Orofacial Pain
Classification & Diagnosis

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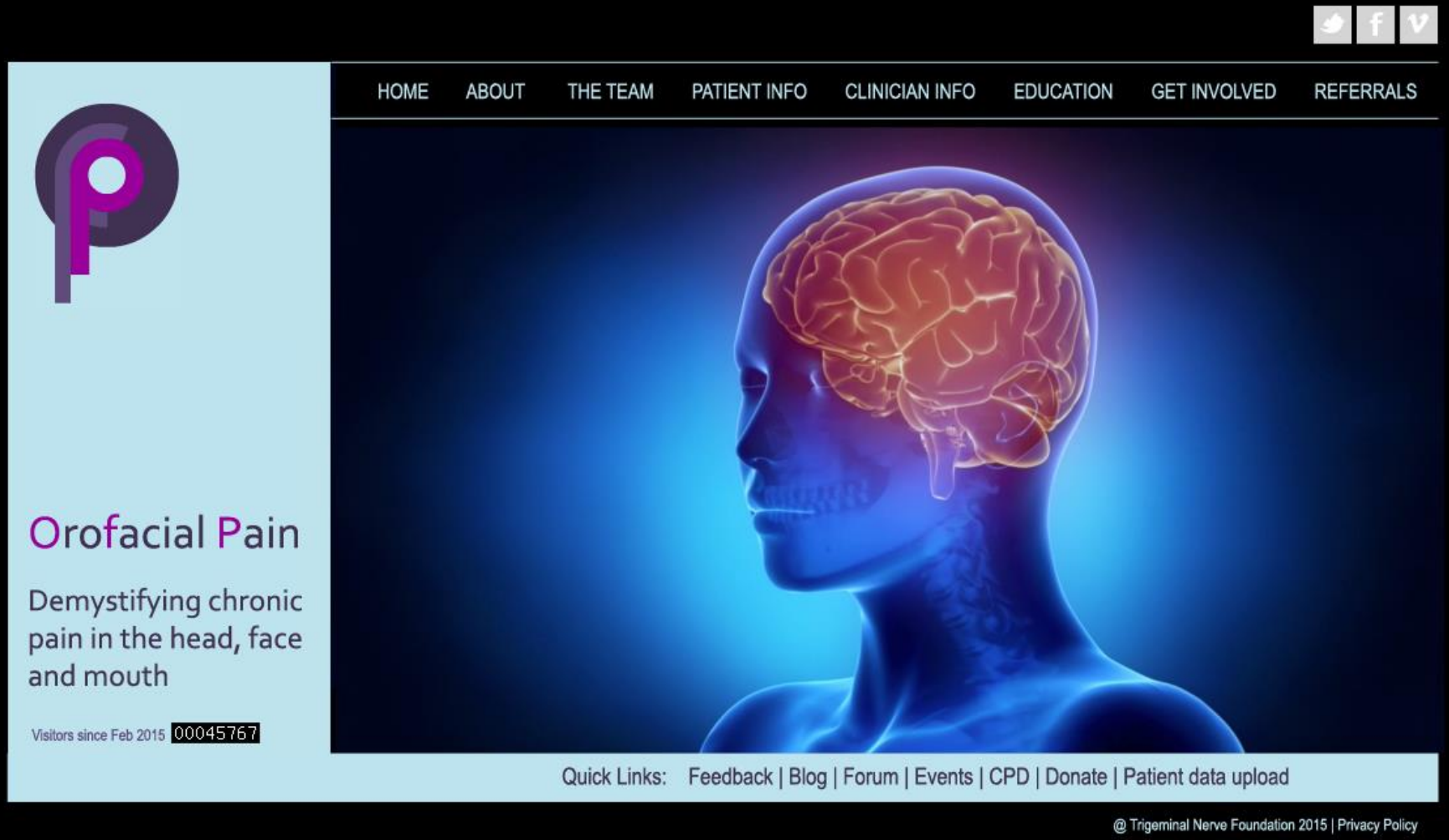


Orofacialpain.org.uk

The diagnosis and medical management of facial pain

Outline

- **An update on pain**
- The trigeminal system
- Chronic pain
- Classification of OFP
- Assessment
- Diagnosis

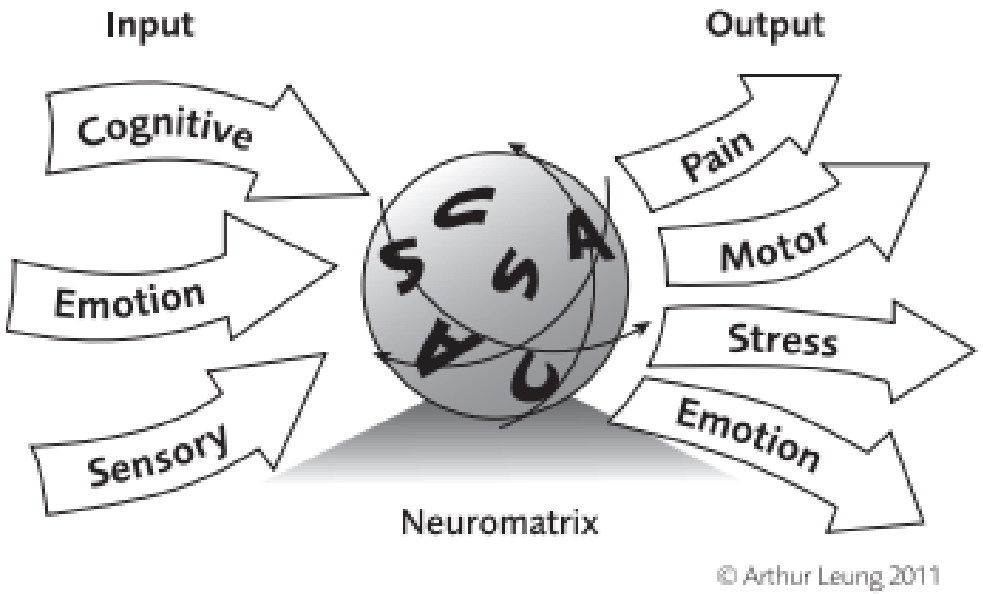
IASP definition of pain

An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

Pain: When poorly managed, is the most common cause for complaints and litigation

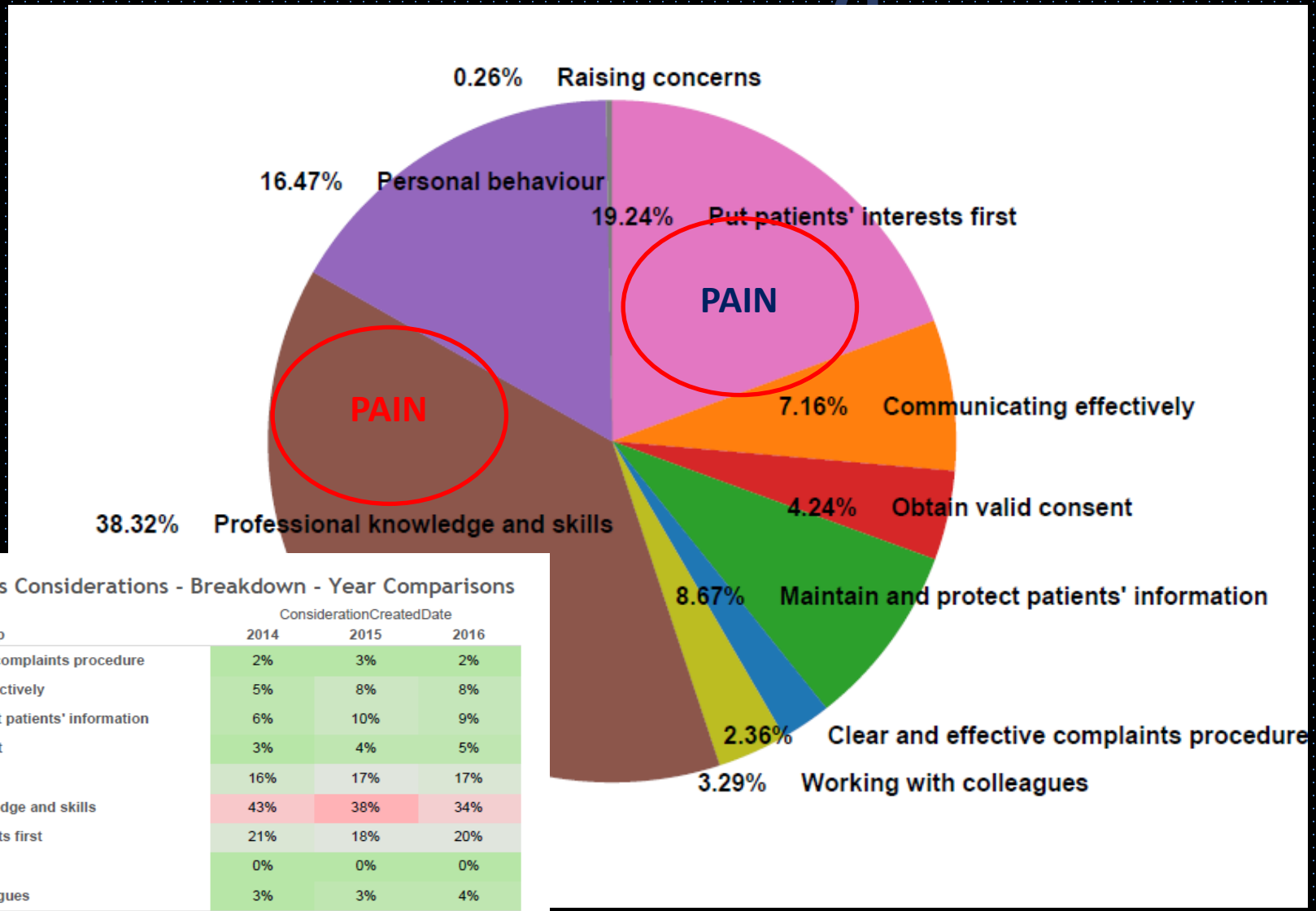
Pain is complex

Figure 2. The concept of the neuromatrix theory for pain



Itself visualised as an entity (like an incessant spinning sphere) comprising the somatosensory (S), cognitive (C) and affective (A) domains, it receives inputs from areas of the brain governing sensation, emotions and cognitions and, in return, churns out a neurosignature (output) which activates various programmes for pain recognition, motor response, emotional and stress reactions. (Adapted from Melzack, Evolution of the neuromatrix theory of pain. The Prithvi Raj Lecture: presented at the third World Congress of World Institute of Pain, Barcelona 2004. Pain Pract. 2005 Jun;5(2):85–94.)

2016 FtP case types



New Standards Considerations - Breakdown - Year Comparisons

	ConsiderationCreatedDate		
Consideration Group	2014	2015	2016
Clear and effective complaints procedure	2%	3%	2%
Communicating effectively	5%	8%	8%
Maintain and protect patients' information	6%	10%	9%
Obtain valid consent	3%	4%	5%
Personal behaviour	16%	17%	17%
Professional knowledge and skills	43%	38%	34%
Put patients' interests first	21%	18%	20%
Raising concerns	0%	0%	0%
Working with colleagues	3%	3%	4%

Dentist’s main responsibility ‘in pain’

- Identifying cancer caused pain and referring appropriately
- Diagnosing and treating dental pain correctly
- Not misdiagnosing non-odontogenic pain as dental pain and then continuing in providing inappropriate dental care
- Preventing nerve injuries and related chronic pain

What is pain?

- Subjective sensation
  - with physical and psychological effects
- Individual response
  - dependant on
  - age / gender / experience / personality / anxiety
  - settings / trust in clinician / fatigue
- Organic and or psychological cause
- Invisible to others

Types of pain .....

Review series introduction

What is this thing called pain?

Clifford J. Woolf

Program in Neurobiology and Department of Neurology, Children's Hospital Boston, and Department of Neurobiology, Harvard Medical School, Boston, Massachusetts, USA.

To paraphrase Cole Porter's famous 1926 song, "What is this thing called pain? This funny thing called pain, just who can solve its mystery?" Pain, like love, is all consuming: when you have it, not much else matters, and there is nothing you can do about it. Unlike love, however, we are actually beginning to tease apart the mystery of pain. The substantial progress made over the last decade in revealing the genes, molecules, cells, and circuits that determine the sensation of pain offers new opportunities to manage it, as revealed in this Review series by some of the foremost experts in the field.

Classifying pain

What exactly, from a neurobiological perspective, is pain? Pain is actually three quite different things, although we and many of our physicians commonly fail to make the distinction. First, there is the pain that is an early-warning physiological protective system, essential to detect and minimize contact with damaging or noxious stimuli. This is the pain we feel when touching something too hot, cold, or sharp. Because this pain is concerned with the sensing of noxious stimuli, it is called *nociceptive* pain (Figure 1A), a high-threshold pain only activated in the presence of intense stimuli (1). The neurobiological apparatus that generates nociceptive pain evolved from the capacity of even the most primitive of nervous systems to signal impending or actual tissue damage from envi-

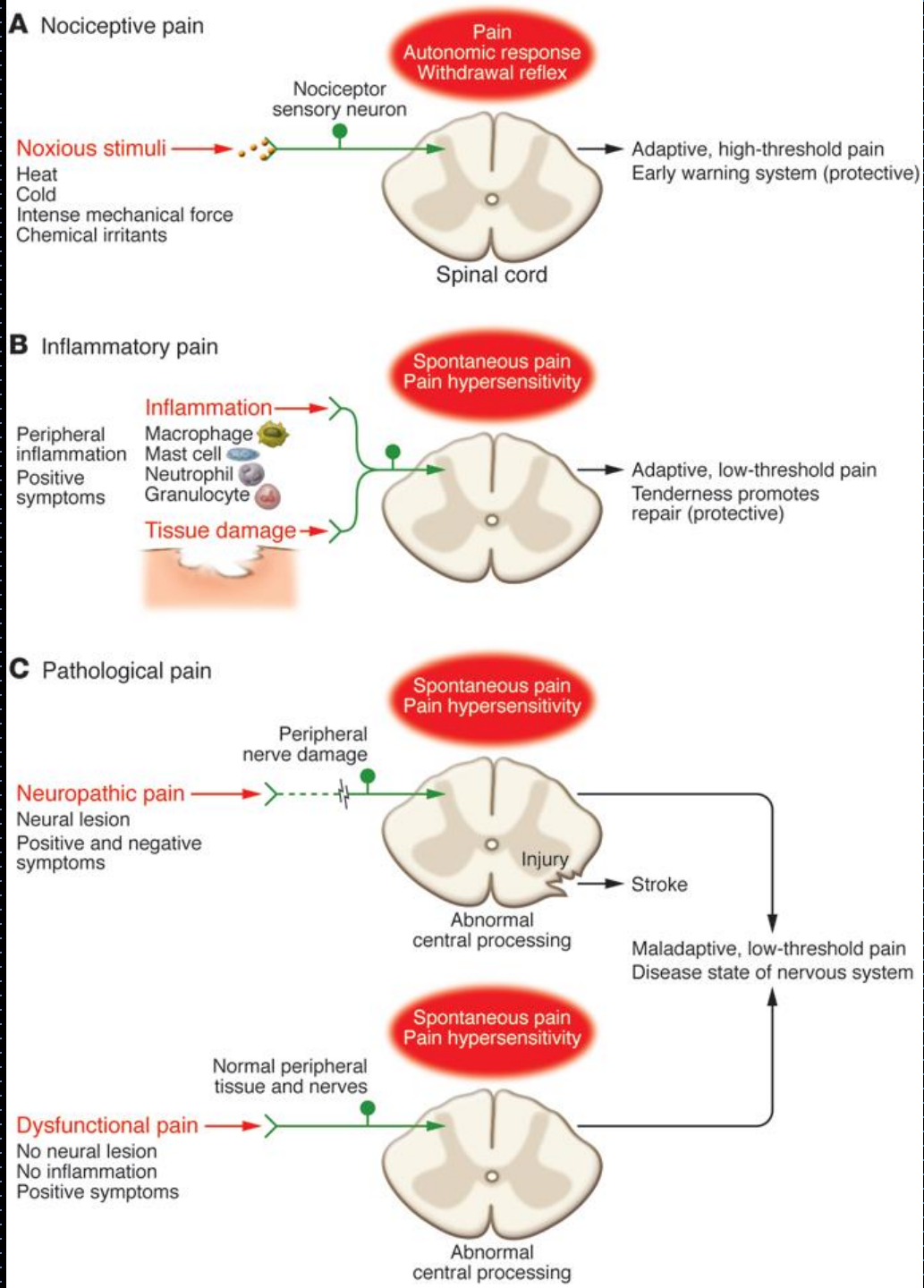
and other syndromes in which there exists substantial pain but no noxious stimulus and no, or minimal, peripheral inflammatory pathology. The clinical pain syndrome with the greatest unmet need, pathological pain is largely the consequence of amplified sensory signals in the central nervous system and is a low-threshold pain. By analogy, if pain were a fire alarm, the nociceptive type would be activated appropriately only by the presence of intense heat, inflammatory pain would be activated by warm temperatures, and pathological pain would be a false alarm caused by malfunction of the system itself. The net effect in all three cases is the sensation we call pain. However, because the processes that drive each are quite different, treatments must be targeted at the distinct mechanisms responsible.

Nociceptive healthy feeling pain ‘pain’

Inflammatory pain health short lived after insult

Neuropathic pains

Dysfunctional pain



Healthy nociceptive pain

Dentine sensitivity

Healthy inflammatory pain/infection/t
Trauma

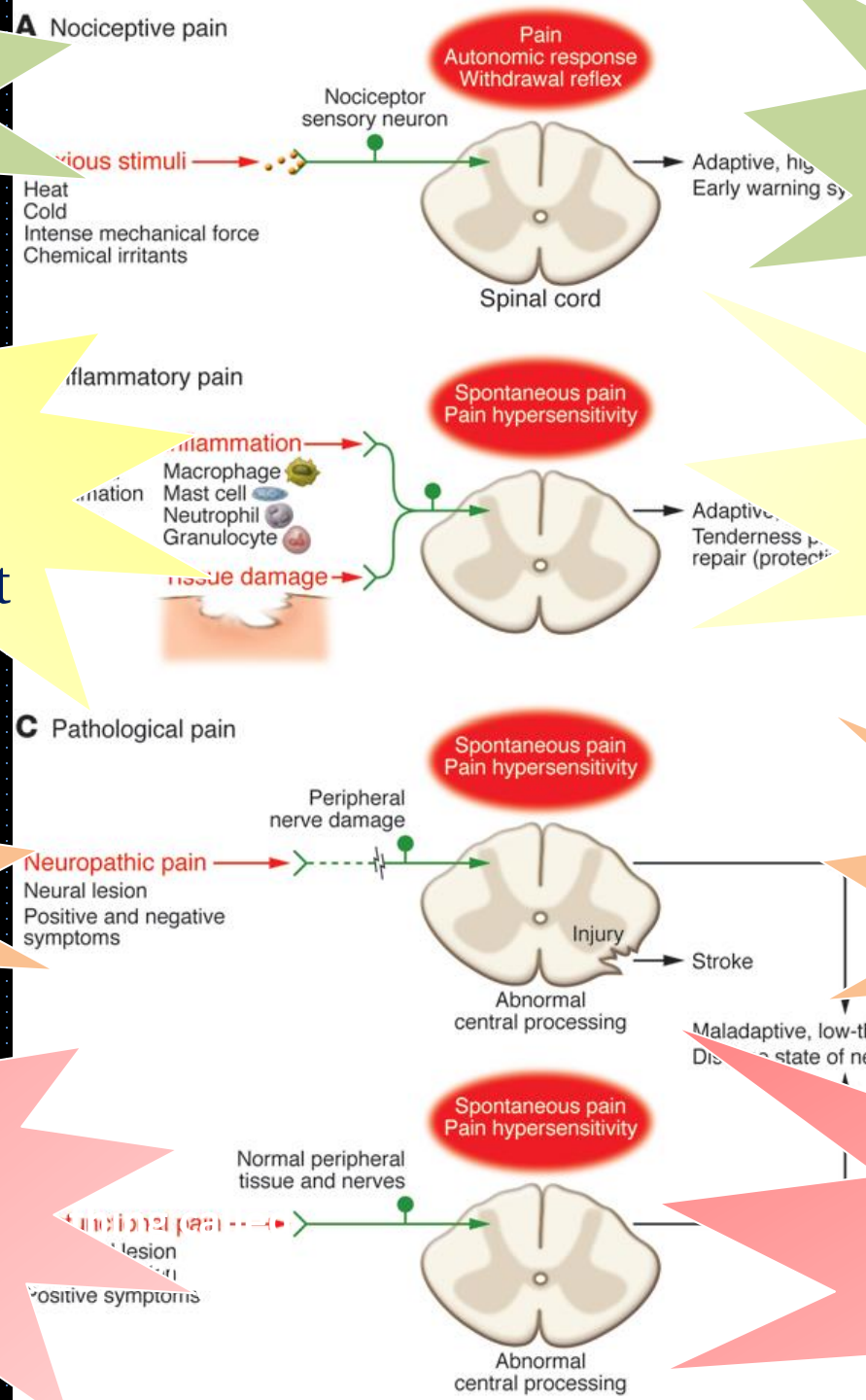
Pulpitis reversible +irreversible
Periapical periodontitis

Chronic neuropathic pain

Posttraumatic neuropathy
PDAP/ PHN

Dysfunctional pain

Fibromyalgia
PIFP
TMD arthromyalgia?



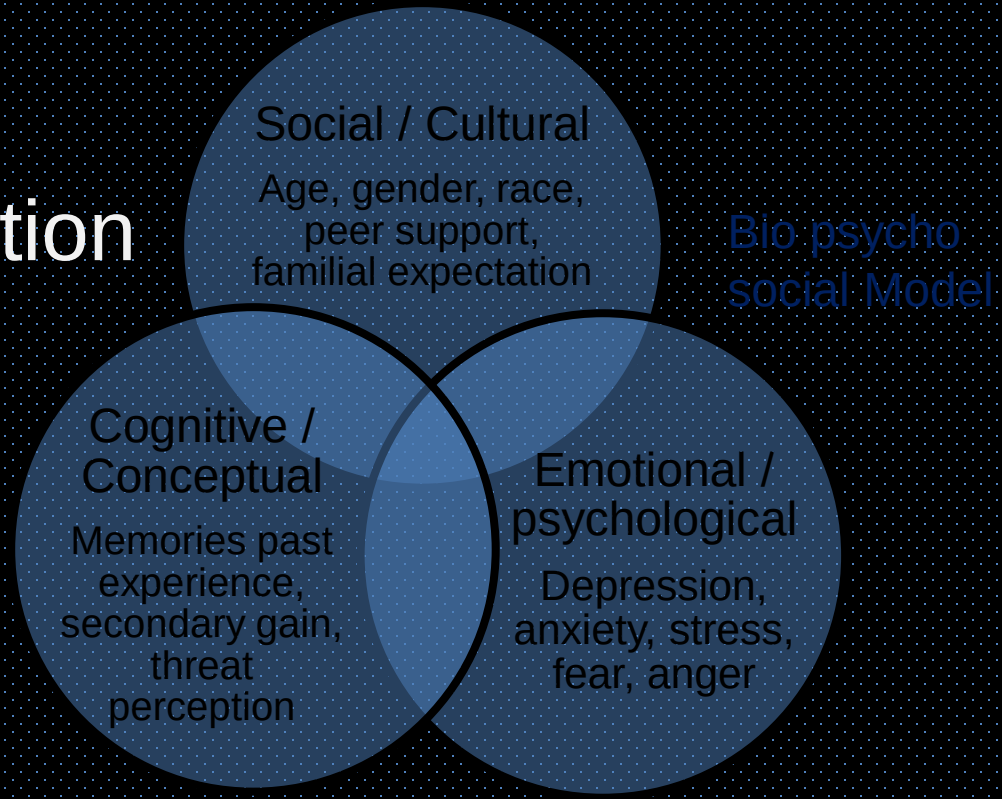
Mechanistic characterisation of pain

Any combination mat be present simultaneously

Peripheral nociceptive	Peripheral Inflammatory	Peripheral neuropathic	Neuropathic autonomic/ vascular	Centralised or dysfunctional pain
• Thermal, chemical, mechanical damage in tissues • NSAID opioid responsive **Examples** • Needle stick • Injection	• Inflammation related to damage of tissues • NSAID opioid responsive • Responds to procedures / antibiotics if infection related **Examples** Acute pain due to injury / Surgery Osteo/Rheumatoid arthritis Cancer pain	• Damage or lesions of peripheral nerves • Responds to NA channel blockers, central (TCAs neuroactive compound Ca channel blockers) pharmacological therapy **Examples** Diabetic neuropathy Neuralgia Chronic post	• **Neuropathic pain** with **autonomic input** • TAC- Facial flushing, ptosis, conjunctival irritation, nasal congestion • Migrainous-Vertigo, dizziness, phono/photo phobia **Examples-** Headaches, TACs	• Characterised by central disturbance in pain processing • Responds to neuroactive compounds altering levels of neurotransmitters involved in pain transmission **Examples** Fibromyalgia Irritable bowel syndrome Myalgic TMD Migraine, Tension headache

Pain Process

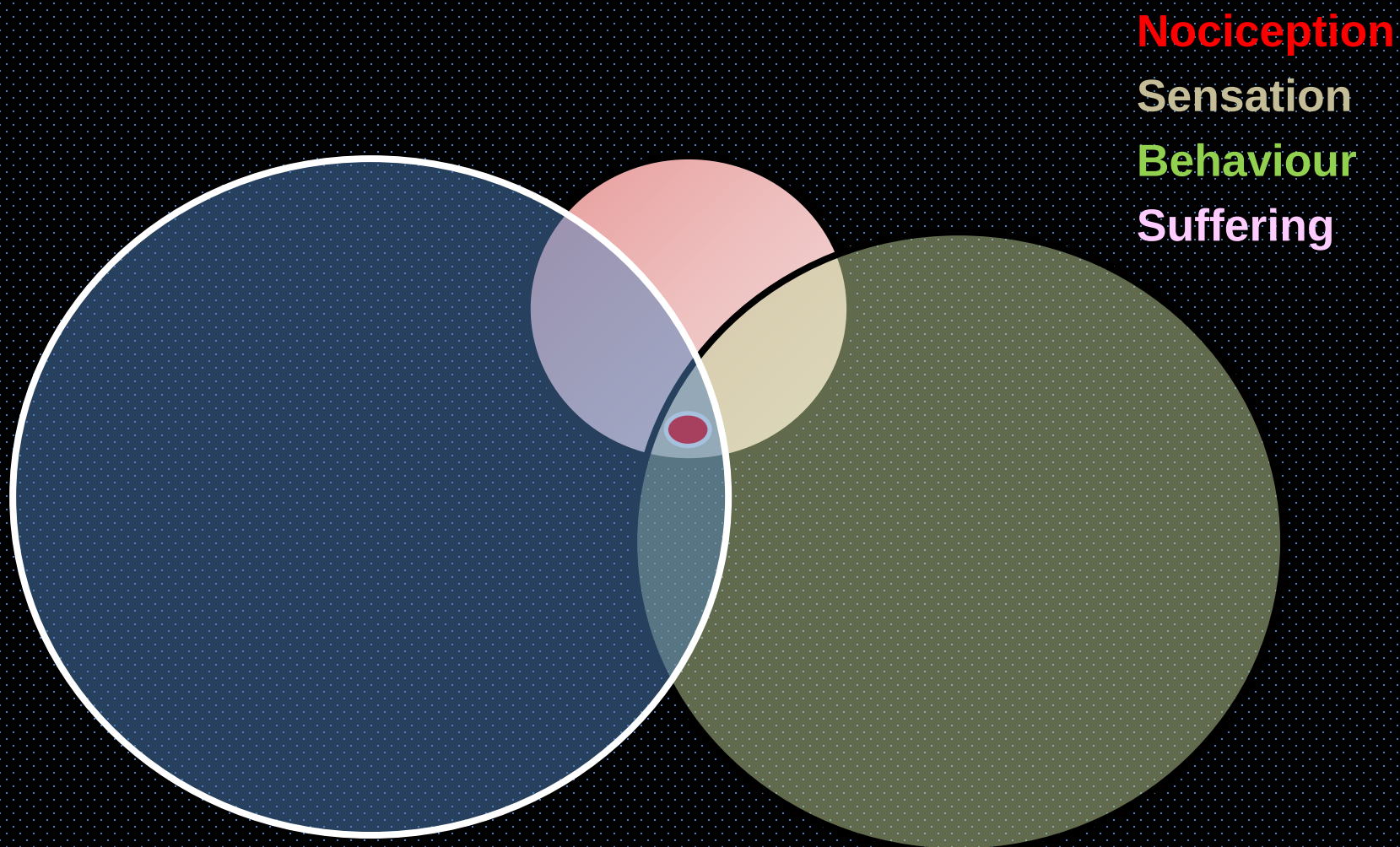
- Nociception
- Sensation/perception
- Behaviour
- Suffering



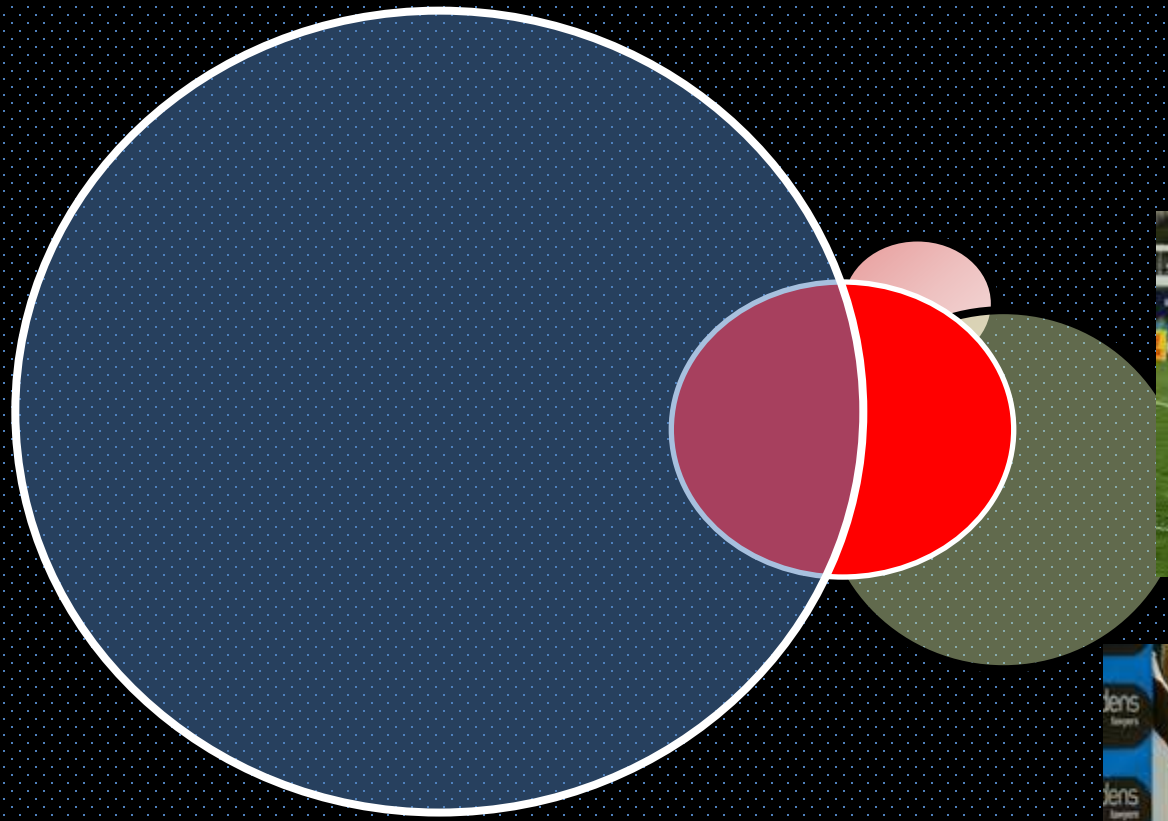
Do genetics influence all of these factors?

Pain Process

weighted for chronic pain relief potential



Pain Patient dependent



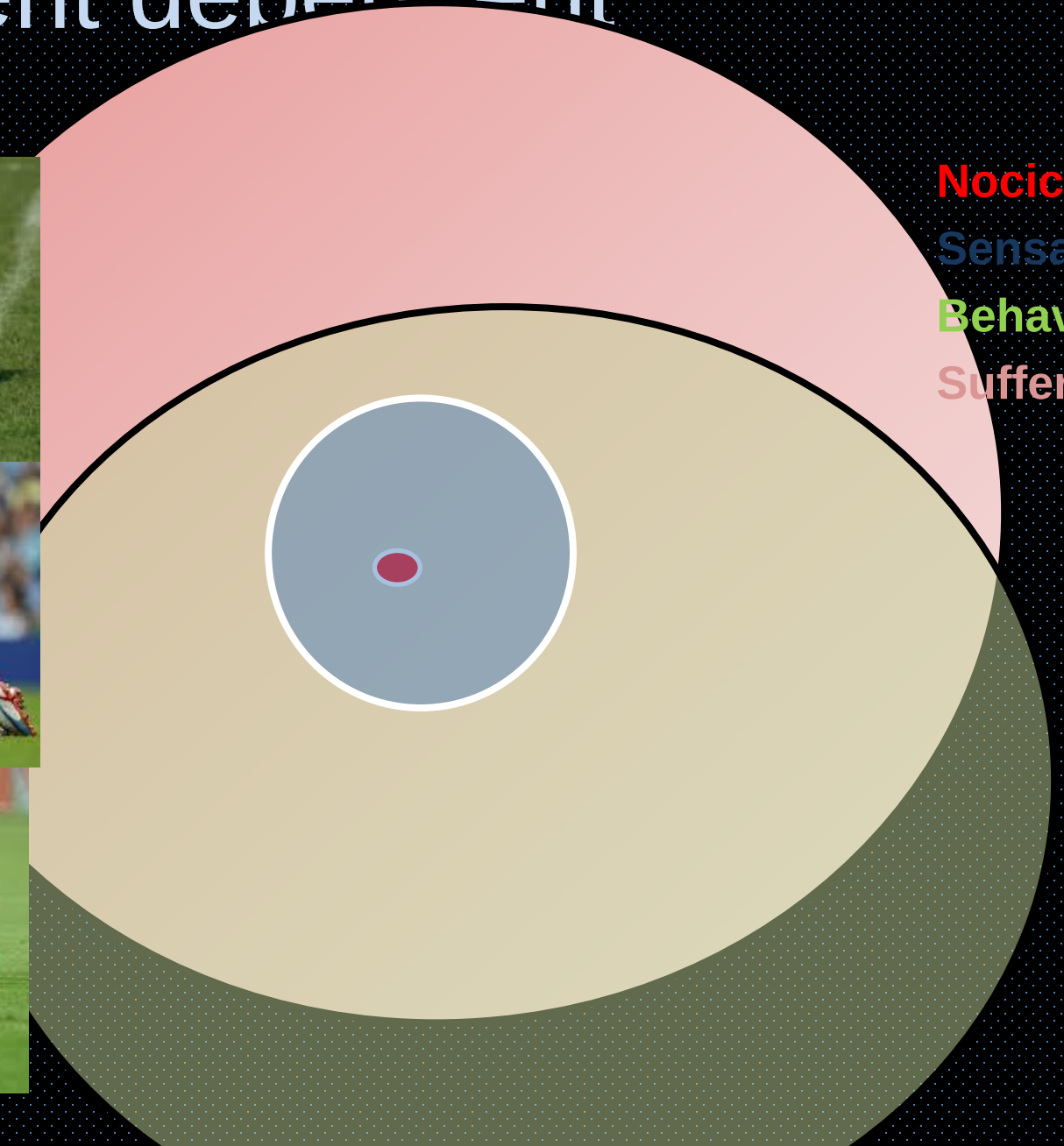
Nociception

Sensation

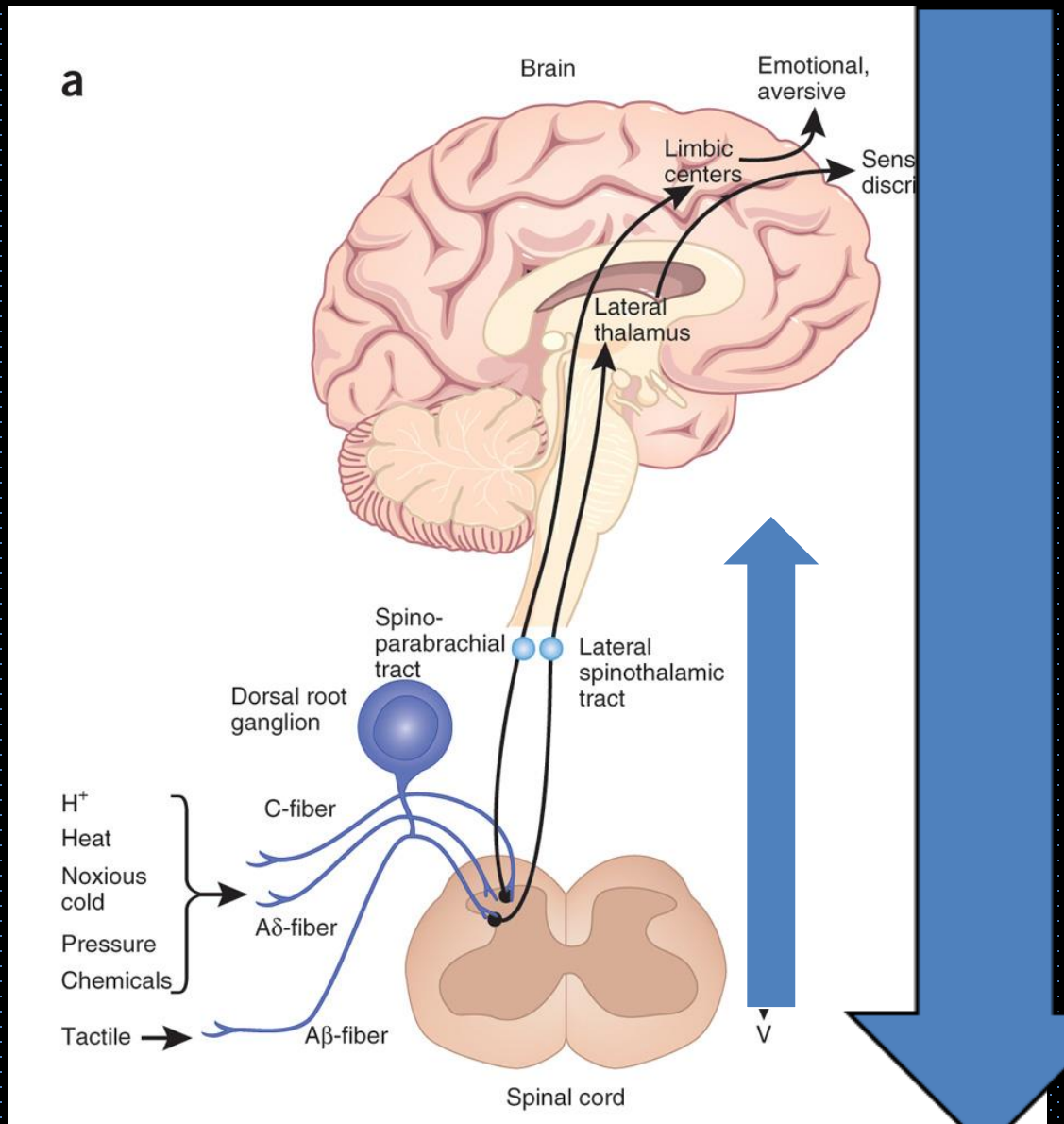
Behaviour

Suffering

Pain Patient dependent



Innate GREAT pain Modulation

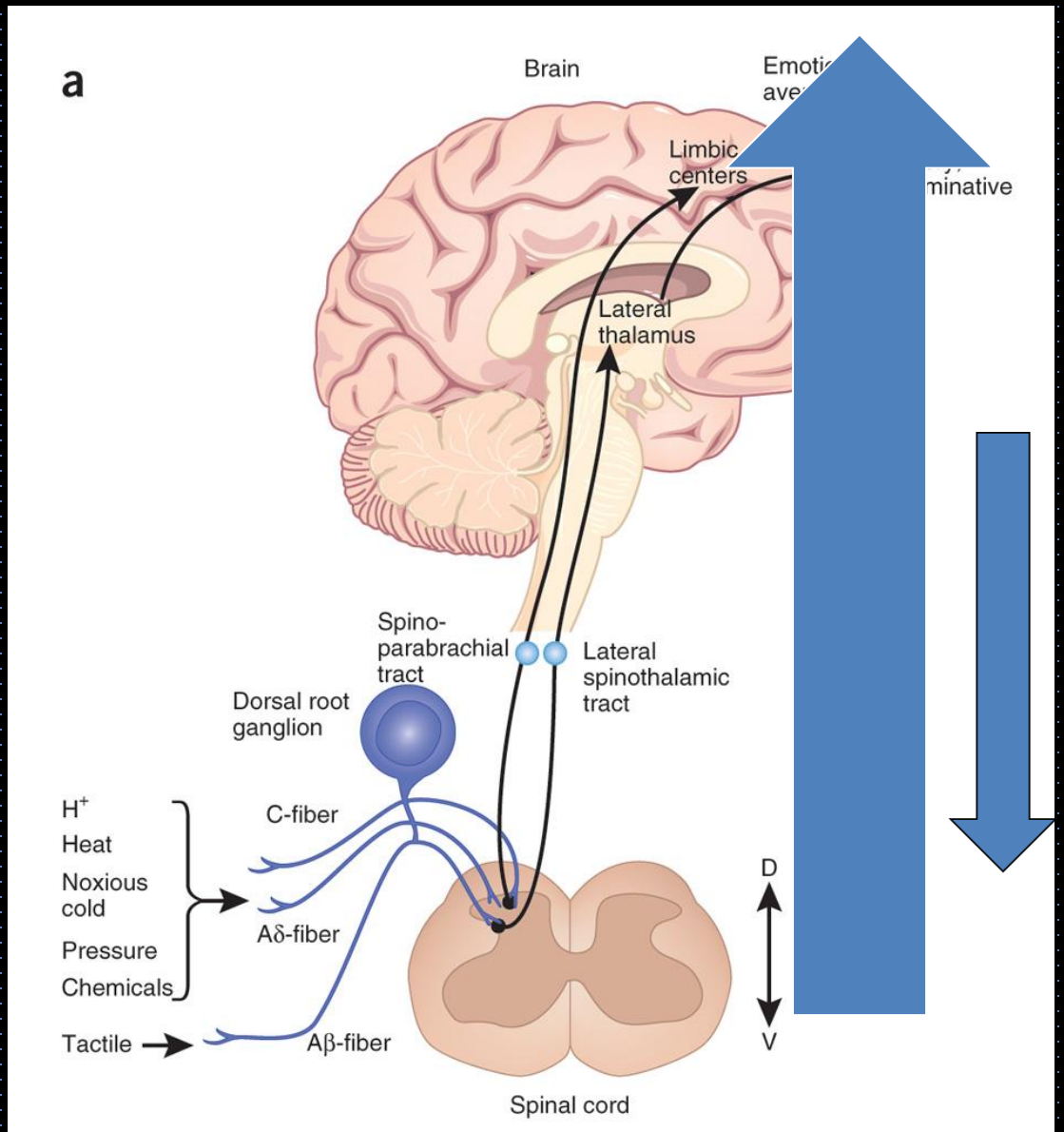


RUGBY PLAYER

Reduced upward facilitation

Increased downward inhibition

Innate POOR pain Modulation

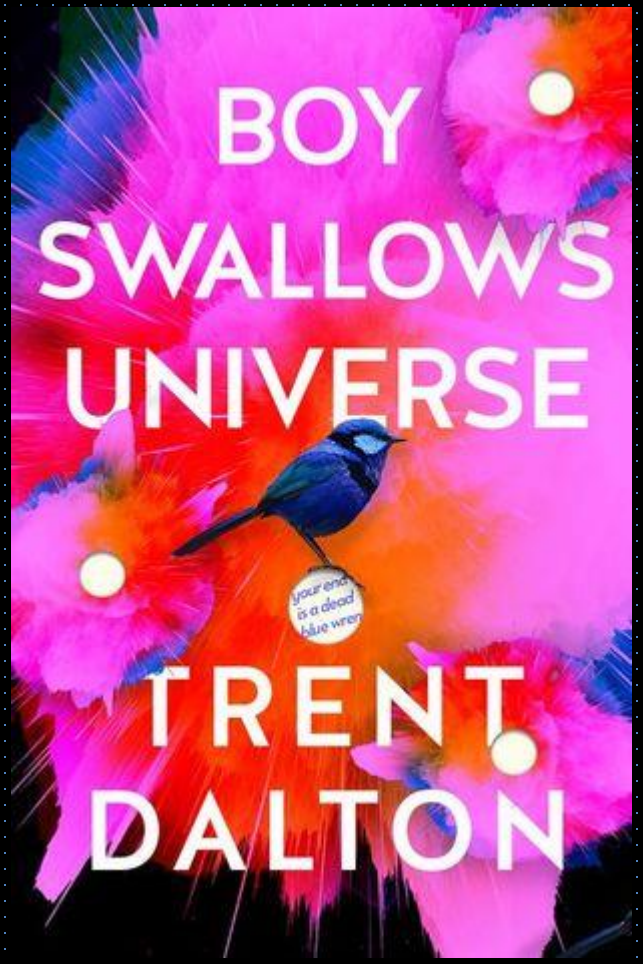


footballer PLAYER

increased upward facilitation

decreased downward inhibition

Past life events……

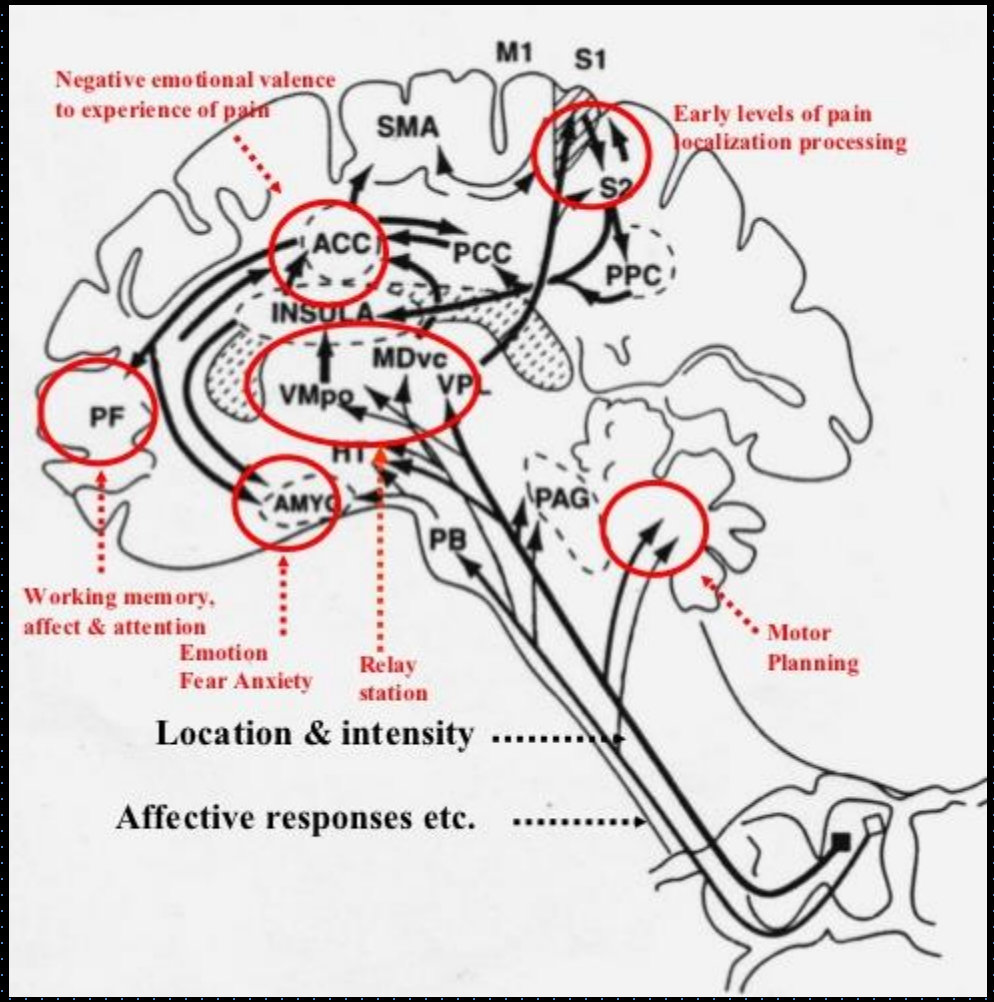


Nociception -Pain Matrix

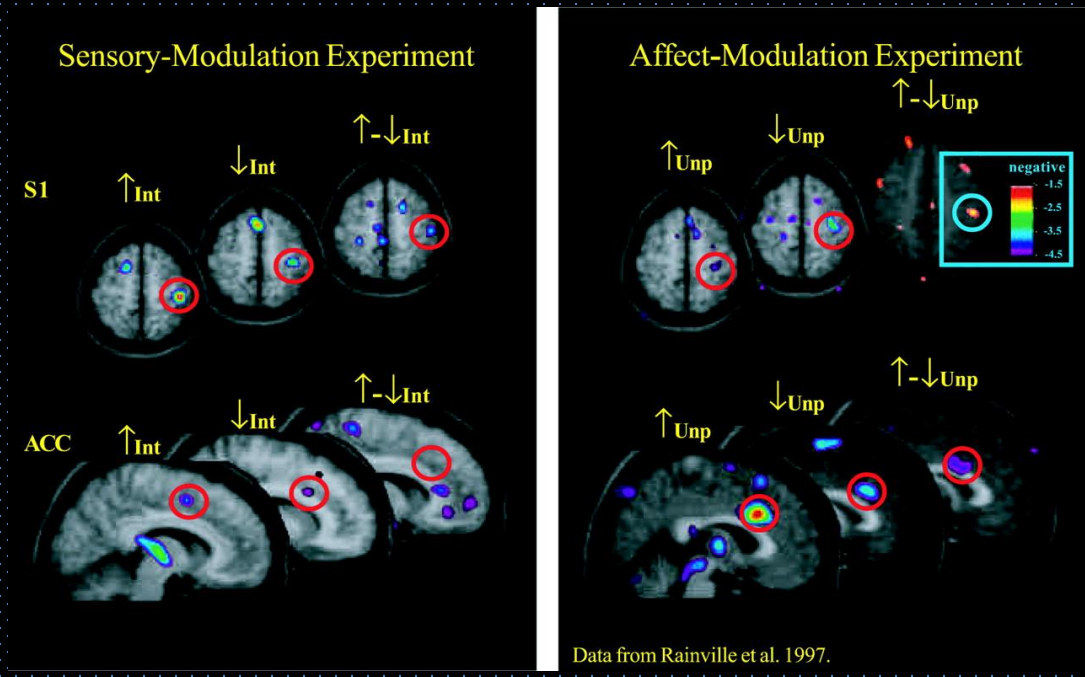
The ‘Pain Matrix’ brain areas reactive to pain

26 areas of the brain affected so far!

Mostly affective= emotional parts of brain resulting in pain **suffering and behaviour**



Sensation
Brain activity to pain

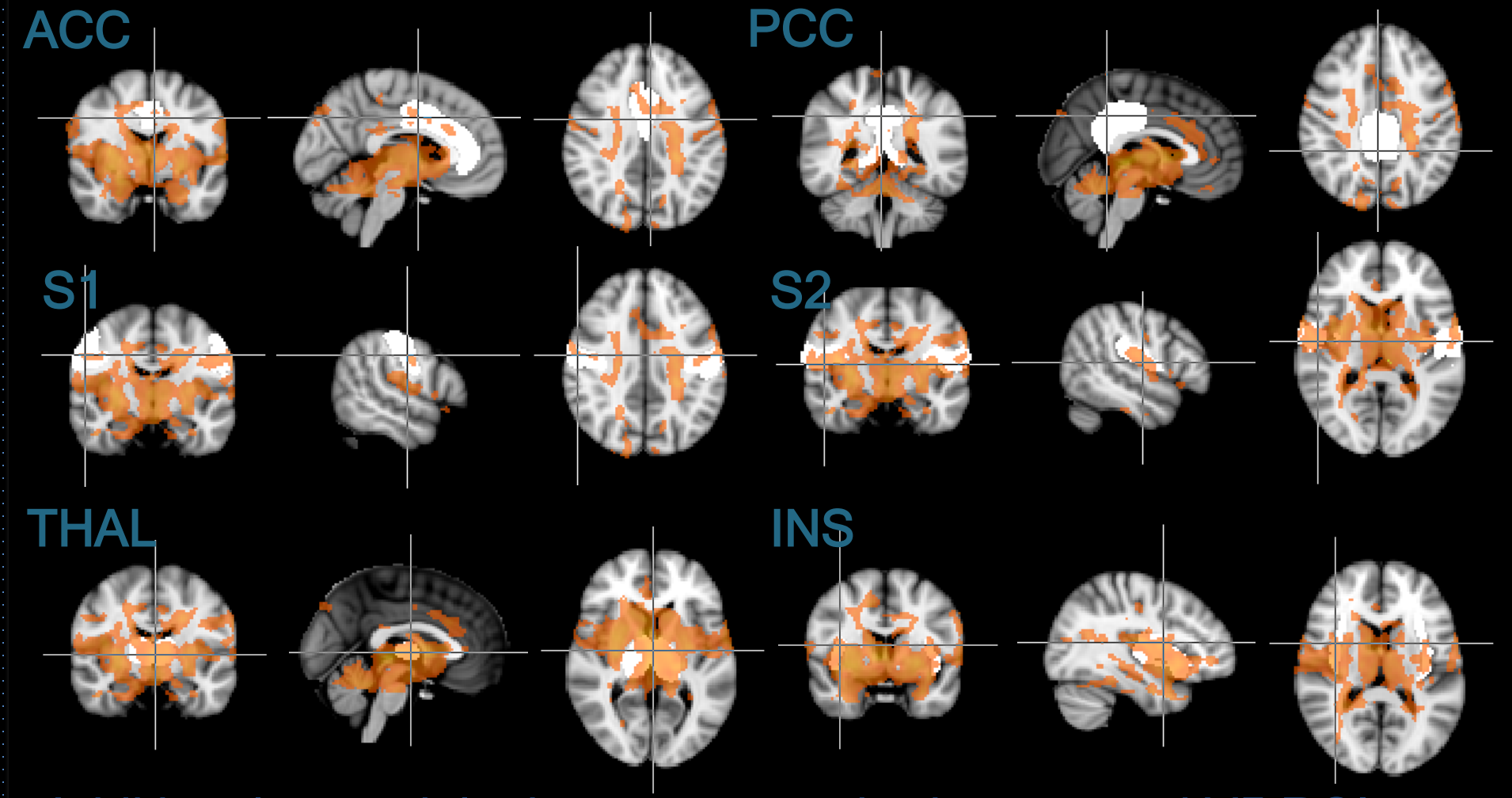


>90% affective
<10% sensory

Cortical Representation of the Sensory Dimension of Pain AJP - JN Physiol **July 1, 2001** vol. 86 no. 1 **402-411**

Ethan Kross [et] al., Proceedings National Academy of Science USA. Social rejection shares somatosensory representations with physical pain

Anatomy of pain



- Additional amygdala, hippocampus, brainstem, and V5 ROIs

Behaviour

- History
- Stress
- Anxiety
- Culture
- Ethnicity
- Beliefs
- Age
- Environment
- Context

Suffering

History
Stress
Anxiety
Culture
Ethnicity
Beliefs
Age
Environment
Context

Personality
Religion
Placebo
Anger
Catastrophising
Fear

How does this chronic pain happen due to surgery?

- Peripheral sensitization of nociceptors
- Central sensitization
- Altered bulbospinal modulation.
  - Both descending facilitatory and inhibitory influences run from brainstem to spinal cord. In chronic pain, levels of descending inhibition can be reduced while facilitation is enhanced. There are established techniques for evaluating the degree of descending pain modulation in operation in volunteers or patients.
- Altered cortical circuitry and connectivity (Plasticity)
  - to be predictive of the emergence of chronic pain.

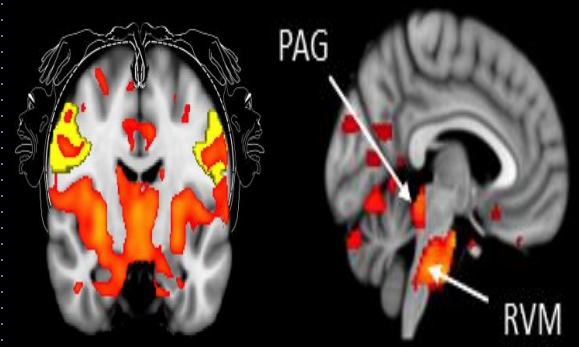
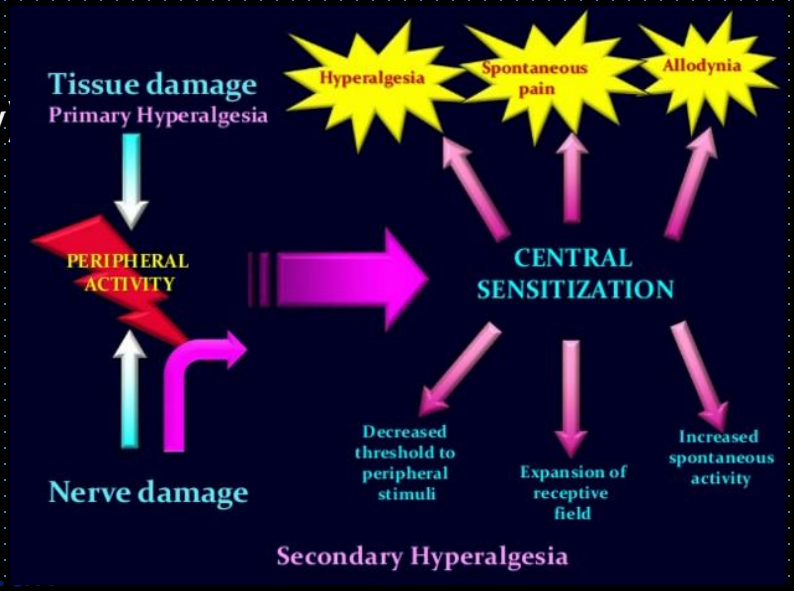


Figure 1: (Left) rCBF increases representing the post-surgical pain experience in primary somatosensory cortex (outlined in yellow). (Right) CBF increases in descending modulatory structures during analgesic response to ibuprofen.



Centralised pain states

- Most common and costly illness in humans
- Used to be termed idiopathic or somatisation
- Characterised by
  - Chronic overlapping conditions-multisystem illness typically begins in childhood or young adult hood
    - Chronic pain or discomfort in several body regions
    - TMD, IBS, Migraine, back pain, Tension headaches, interstitial cystitis, dry eye disease (NIH PA 14-244)
    - Multiple other somatic disorders of CNS origin
      - Fatigue, sleep disorder, mood, memory
  - By stressful trigger
    - Post deployment Gulf war syndrome
    - Post infection (Lyme disease chronic EBV)
    - Post emotional trauma Death of spouse

Essentially…….

- No brain

- No pain!!!!!

Sensation

Follow nociception if physical damage has occurred when the impulse reach the **somatosensory cortex pain is registered = sensation** See animation

If there is no physical damage purely anticipated or emotional

CNS

Prefrontal cortex
Basal Ganglia
(Limbic system)
Insular cortex

Thalamus
Hypothalamus
Brainstem
PAG
Locus coeruleus
Raphe nucleus
Rostroventral medulla

OUCH!

= sensation

CNS

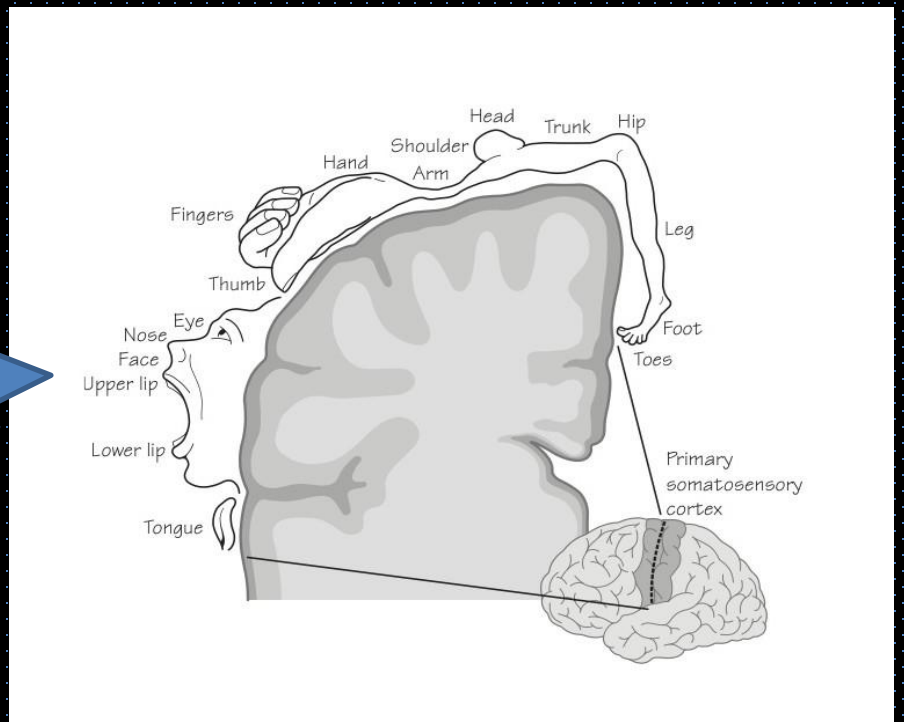
Spinal cord

Dorsal column
or Mesencephalic
nucleus for
Trigeminal nerve

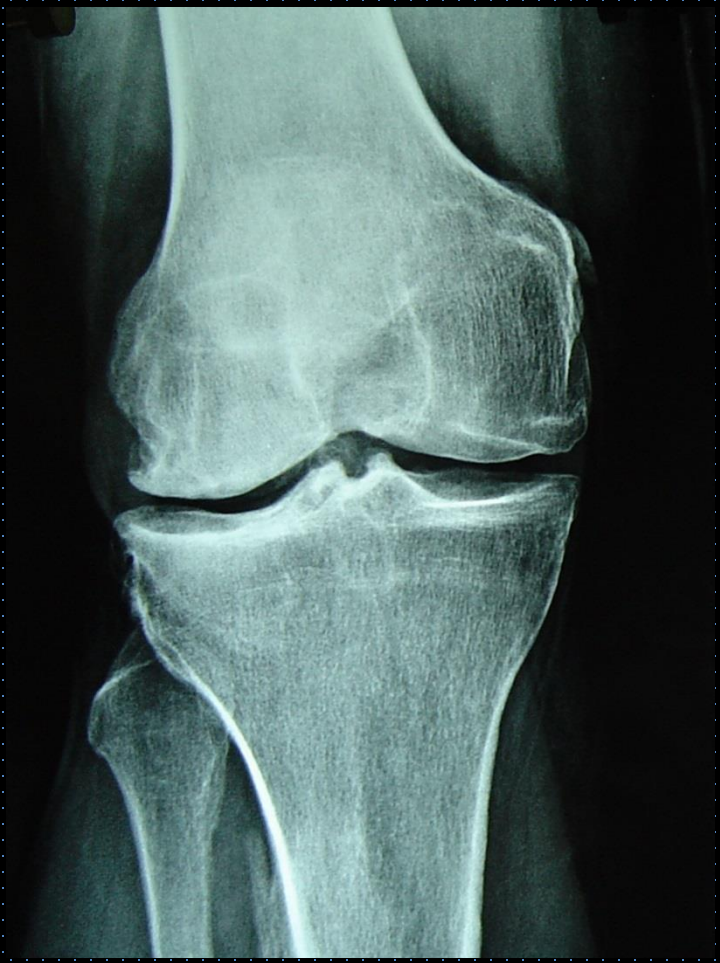
PNS

Primary sensory afferent
C fibre warm + pain
A delta cold +pain
A beta Mechano sensory

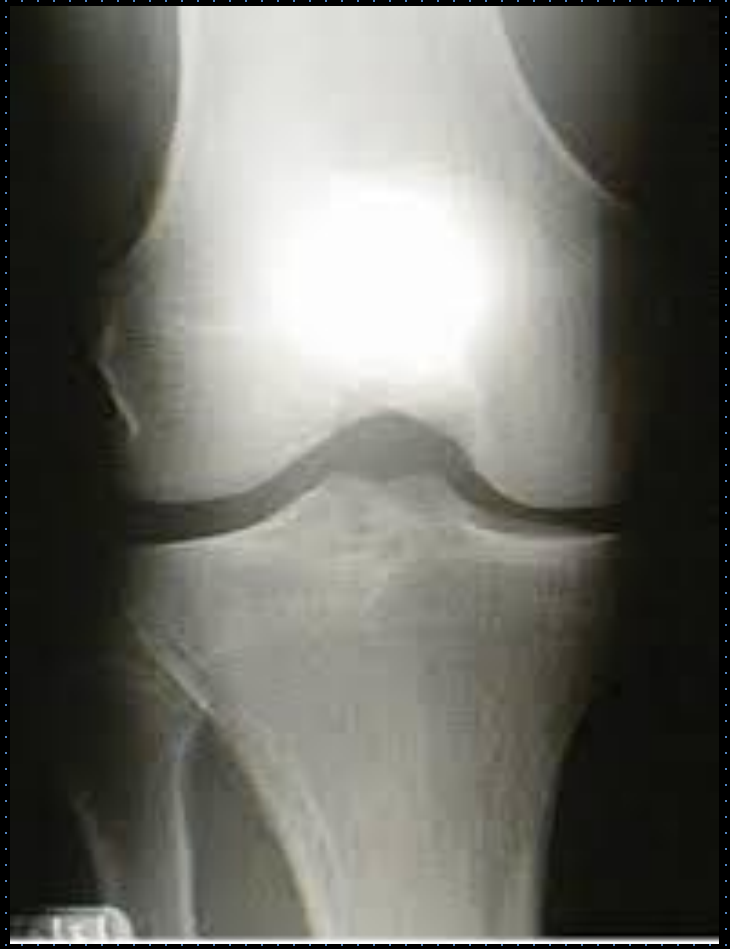
Distal root ganglion (DRG)
equivalent to gasserian
ganglion



Which person has chronic pain?



30-40% abnormal X-Ray with **No pain**



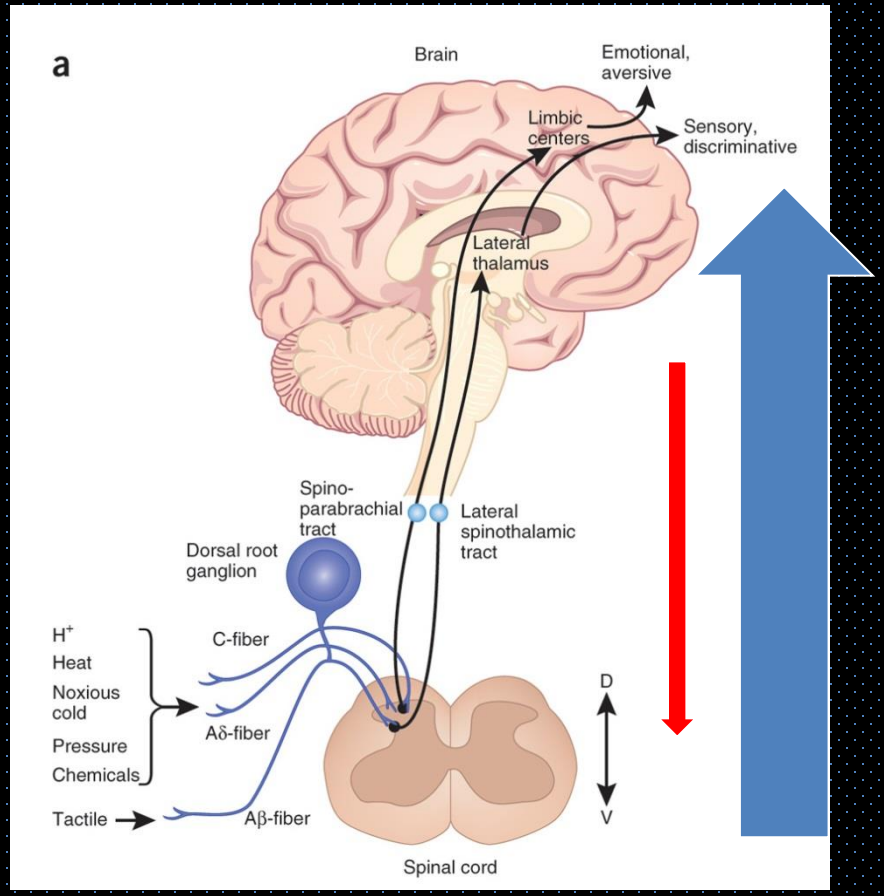
15-20% normal X-Ray with **pain**

Centralisation continuum

Peripheral Centralised

Acute pain Osteoarthritis LB pain Fibromyalgia

Brain continues overlaying pan when ‘irritation’ is gone

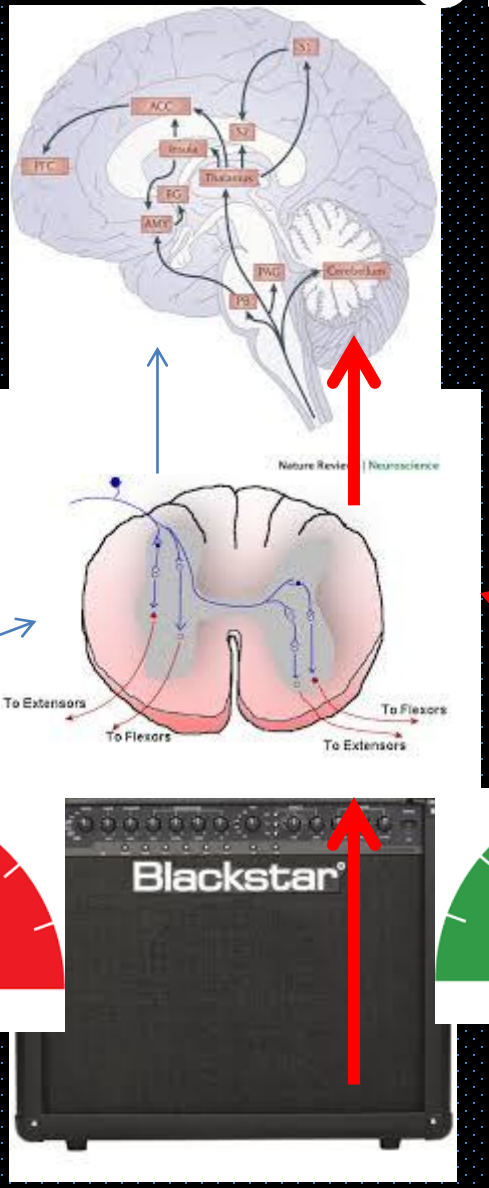


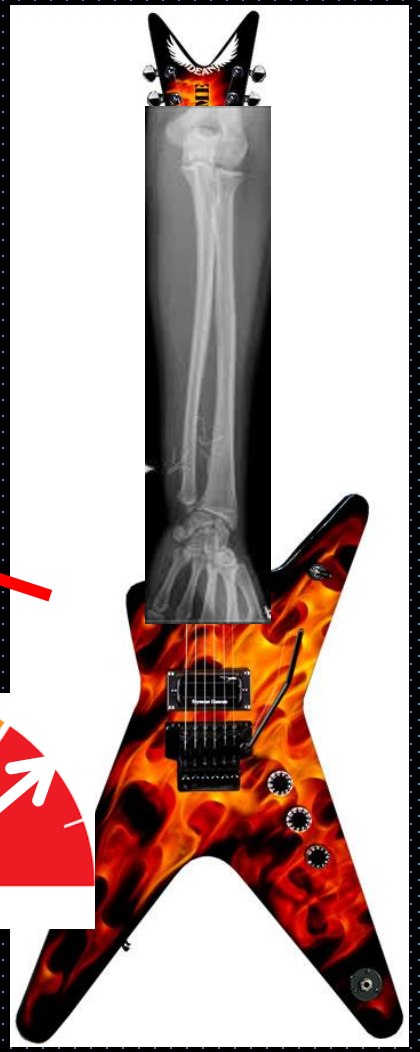
Too much upward facilitation

Not enough downward inhibition

Healthy pain

Unhealthy pain

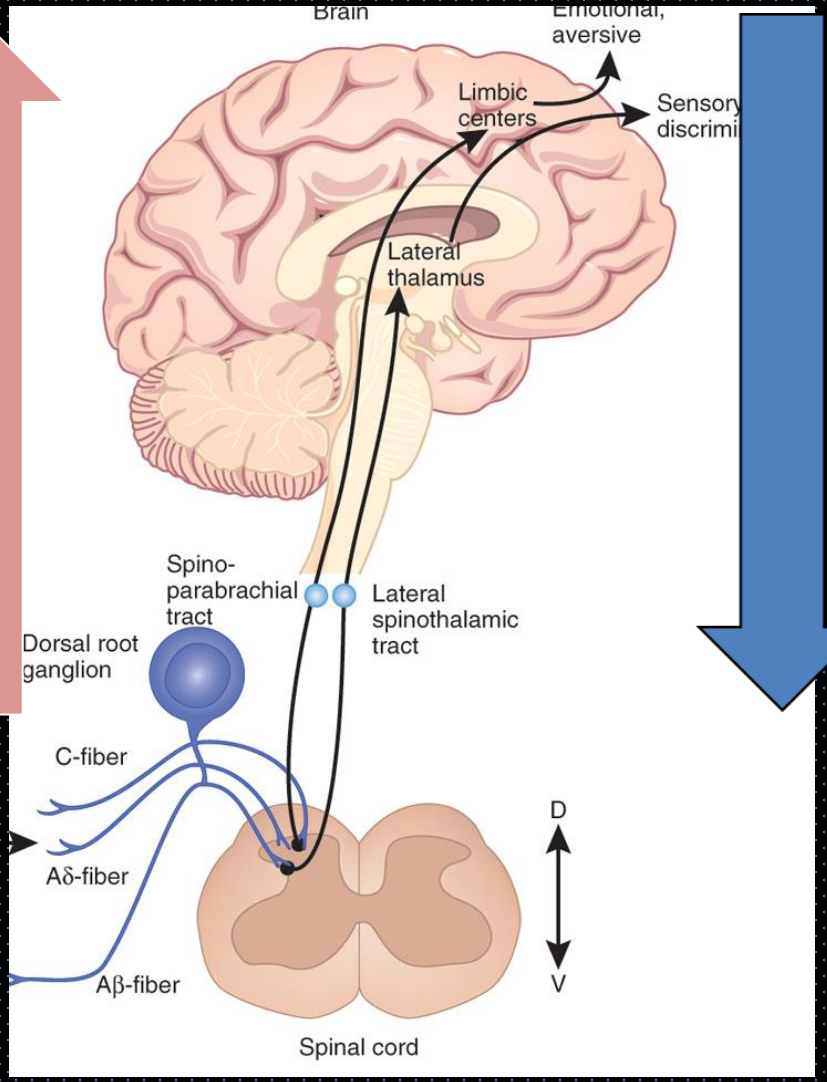




Neural influences on pain and sensory processing

Facilitation

- Substance P
- Glutamate
- EAA
- Serotonin (5HT 2a, 3a)
- Nerve growth factor (NGF)

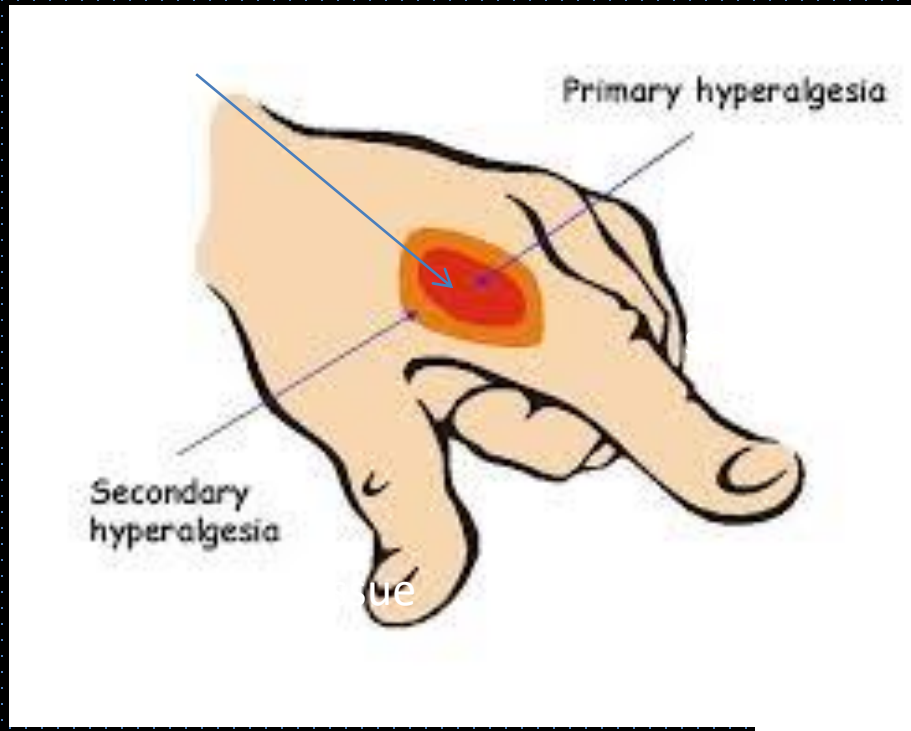


Inhibition

- Norepinephrine
- serotonin (5HT 1a and 1b)
- Dopamine
- GABA
- Cannabanoids

Opioids

Schmidt-Wicke T DJ Clauw Rheumatology 2011 7(9) 518-27



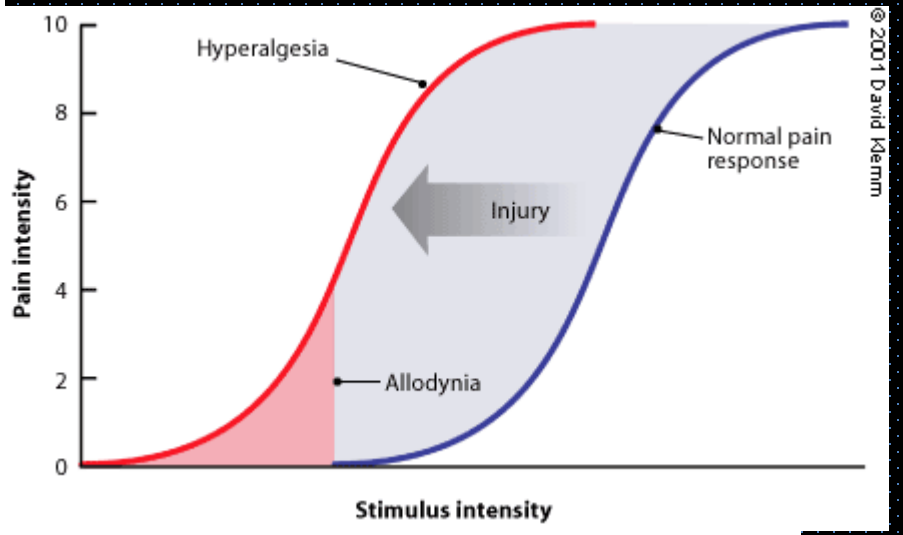


Figure 2. Hyperalgesia. Magnitude of pain is plotted as a function of stimulus intensity. In normal skin, pain increases with stimulus intensity above the pain threshold. For hyperalgesic skin, the stimulus-response function is shifted to the left; there is a lower threshold for producing pain and an increased response to suprathreshold stimuli.

Tertiary hyperalgesia = pain hypersensitivity on contralateral side of your body!

	Primary	Secondary
Mechanical hyperalgesia	Yes	Yes
Heat hyperalgesia	Yes	No

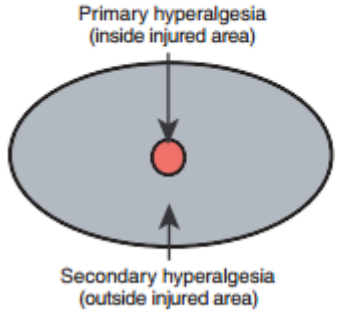


Figure 3. Primary and secondary hyperalgesia differ. After a tissue injury, primary hyperalgesia develops at the site of injury and secondary hyperalgesia develops in uninjured

What kind of patient?

Patient characteristics

- Fragility
- Susceptibility
- Vulnerability
- Genetics
  - *CACNG2* significantly affects susceptibility to chronic pain following nerve injury. *CACNG2* encodes for stargazin, a protein intimately involved in the trafficking of glutamatergic AMPA receptors. The protein might also be a Ca^{2+} channel subunit. And *CACNG2* polymorphisms are associated with chronic pain in a cohort of cancer patients who underwent breast surgery. **Susceptibility to chronic pain following nerve injury is genetically affected by *CACNG2*** Nissenbaum J Genome Res. 2010 Sep; 20(9): 1180–1190.
  - *COMT*, which codes for catechol-*O*-methyltransferase, an enzyme that degrades catecholamine neurotransmitters, including dopamine, epinephrine and norepinephrine. The variant allele V158M results in reduced enzymatic activity due to its effect on thermostability[26] and has been associated with reduced μ-opioid activity in response to pain stimuli resulting in increased pain sensitivity.[27] The low (MM) and intermediate (VM) activity genotypes were significantly increased in frequency in FM patients in two case-control studies,[28,29] however, a much larger Norwegian cohort study (the HUNT study) found no association between the polymorphism and CWP.[30]
  - Paediatrics ABCB1 and OPRM genotypes are associated with clinically meaningful pain variability, whereas NTRK1 and COMT are linked to subclinical effects. First Evidence of a Polygenic Susceptibility to Pain in a Pediatric Cohort Chantal Mamie et al Anesth Analg 2013;116:170–7)

Anxiety, stress and pain

Psychological factors driving pain

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

Lajnert V, et al Depression, somatization and **anxiety** in female patients with temporomandibular disorders (TMD). Coll Antropol. 2010 Dec;34(4):1415-9

Alternative and holistic management of pain

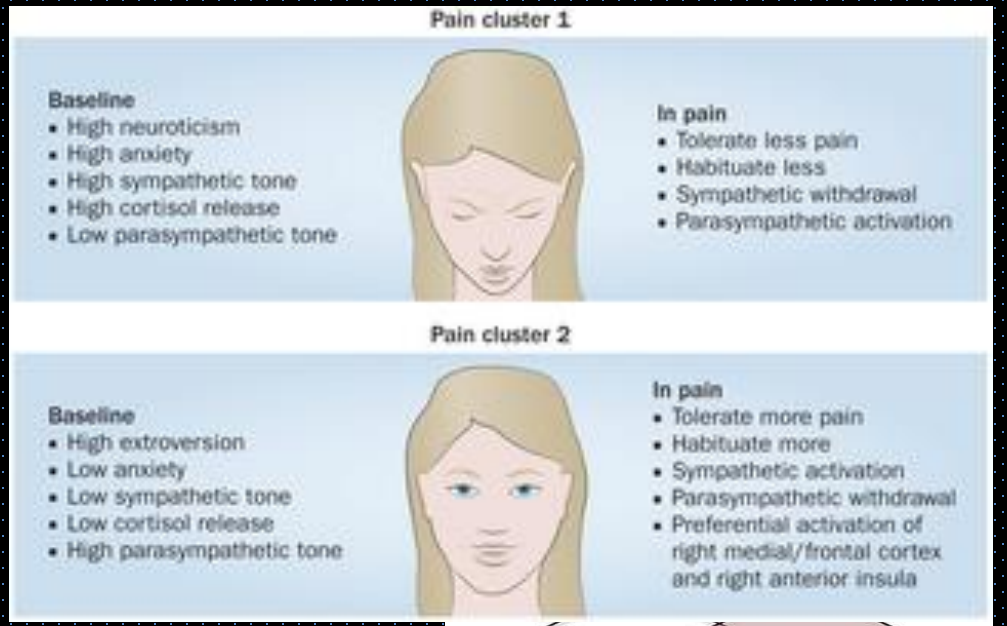
Bauer B et al. Effect of the combination of music and nature sounds on **pain** and **anxiety** in cardiac surgical patients: a randomized study. Altern Ther Health Med. 2011 Jul-Aug;17(4):16-23.

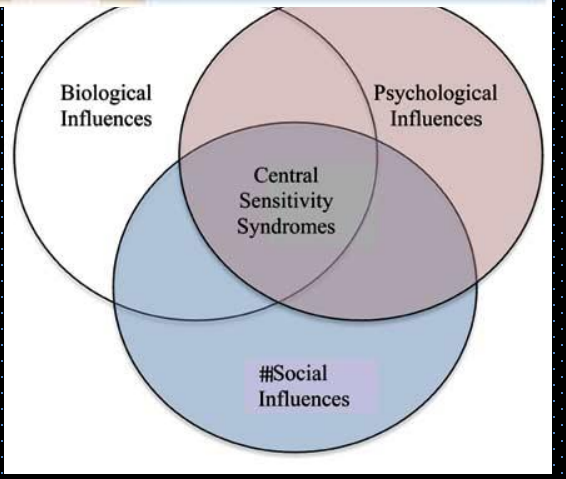
Louw A, et al.The effect of neuroscience education on **pain**, disability, **anxiety**, and **stress** in chronic musculoskeletal **pain**. Arch Phys Med Rehabil. 2011

Psychosocial factors predictive of Chronic pain after surgery

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

Review Katz & Seltzer





Catastrophising

Patients with catastrophizing disorder more likely to develop chronic pain after surgery

Date: October 13, 2014

Source: American Society of Anesthesiologists (ASA)

Summary: Patients with a psychological cognitive disorder known as catastrophizing are more likely to develop persistent, chronic pain after surgery, according to new research. Pain catastrophizing occurs when a patient has an irrational and illogical focus on pain, perceiving that it is worse than it actually is.

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Related Topics

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- Fibromyalgia
- Neuropathy

Mind & Brain
- Caregiving
- PTSD
- Anxiety

Patients with a psychological cognitive disorder known as catastrophizing are more likely to develop persistent, chronic pain after surgery, according to new research presented at the ANESTHESIOLOGY™ 2014 annual meeting. Pain catastrophizing occurs when a patient has an irrational and illogical focus on pain, perceiving that it is worse than it actually is.

"There has been considerable evidence that psychological factors can lead to the development of persistent post-surgical pain," said Asokumar Buvanendran, M.D., lead author and professor, department of anesthesiology, Rush University Medical Center, Chicago. "Pre-surgical evaluation of these factors, including catastrophizing, could help to identify patients having elective surgery who might benefit from treatment of this cognitive disorder to prevent the development of chronic pain."

To evaluate the relationship between catastrophizing

Related Articles

- Chronic pain
- Nociceptor
- Anesthesia
- Pain
- Back pain
- Gate control theory of pain

Sullivan M et al. Perceived Injustice is Associated with Heightened Pain Behavior and Disability in Individuals with Whiplash Injuries. Psychol. Inj. and Law DOI 10.1007/s12207-009-9055-2

Risk Factors
Patient susceptibility -The F-word

- Evolution of thinking regarding Fibromyalgia
- 2-8% population M:F 1:2
- American college of rheumatologists
- Fibromyalgia-ness
  - Symptoms a continuum
  - 2009 modified diagnostic criteria
- Discrete illness
  - Focal areas of tenderness
  - Psychological and behavioural factors present

Wolfe et al Arthritis Rheum Jun 15 2009 61 (6) 715-716
Wolfe et al J Rheumatol Feb 1 2011

Risk factors
Aetiology centralised pain

- Chronic overlapping pain conditions affects up to 20% of population
- Family history
- 2X more common in women
- **Triggered or exacerbated by stressors**
  - Children born in 1958 who had experienced a motor traffic accident or were institutionalised were 1.5-2X more likely to have chronic widespread pain 42 years later (Jones et al 2007 ACR meeting)
  - Peripheral pain syndromes (RA OA SLE) (Clauw D et al JCR 1997)
  - Physical trauma (McBeth 2006 ACR meeting)
  - Post deployment (Clauw D et al J Occup environ Med 2003 Oct 45(10) 1040-8)
  - Infections (Abin et al Sem Arthritis Rheum 2009)
  - Psychological distress
- Genetics

Clauw JAMA 2014 Clauw et al Neuromodulation 1997; 4:134-153; McClean SA et al Med Hypotheses 2004; 63;653-658

Risk Factors
Red heads

- An increasing number of studies show that redheads are differently constituted in terms of pain perception and body reactions. Research reveals that redheads:
  - are more sensitive to cold
  - are less responsive to subcutaneously administered anaesthetics [under the skin]
  - suffer more from toothaches and are more frightened of dentists
  - are at greater risk of developing sclerosis and endometriosis
- Some studies indicate that redheads are more prone to illness because they prefer to keep out of the sun and so lack vitamin D, or because their ability to absorb the vitamin is less efficient.

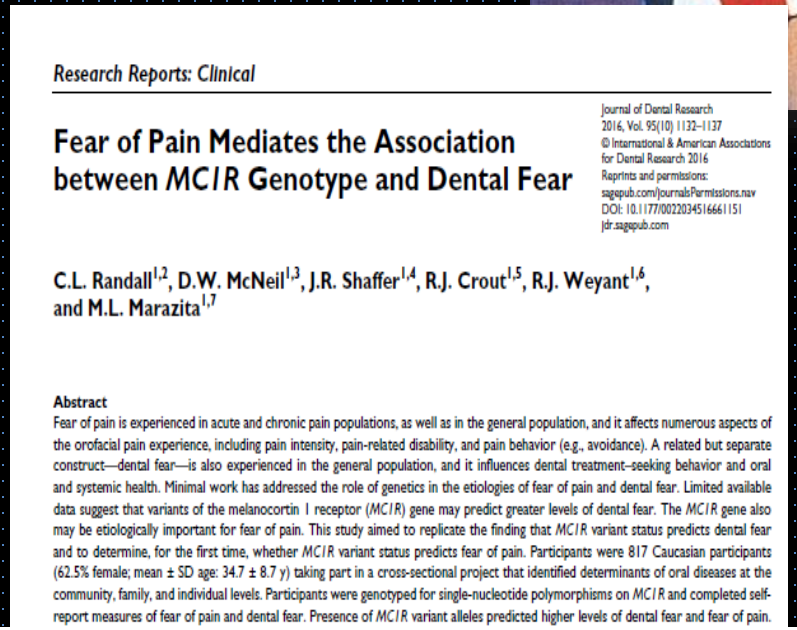
- The focus of the animal studies was **the receptor gene MC1R**, which is responsible for producing the brown skin pigment melanin; redheads have a variant of this gene which does not produce melanin.

Risk Factors
Genetics of pain

- Melanocortin 1 receptor def –Mu opoid receptor def
- Need 20% 20% more anaesthetic
- Melanocortin-1 Gene for Red Hair

2002 “It does appear that redheads have a significantly different pain threshold and require more anaesthetic to block out certain pains,”

2010 Danish study suggests red headed people feel the cold more but could handle eating hot food



Research Reports: Clinical

Fear of Pain Mediates the Association between *MC1R* Genotype and Dental Fear

Journal of Dental Research
2016, Vol. 95(10) 1132–1137

DOI: 10.1177/0022034516661151
jdr.sagepub.com

C.L. Randall[1,2], D.W. McNeil[1,3], J.R. Shaffer[1,4], R.J. Crout[1,5], R.J. Weyant[1,6], and M.L. Marazita[1,7]

Abstract

Fear of pain is experienced in acute and chronic pain populations, as well as in the general population, and it affects numerous aspects of the orofacial pain experience, including pain intensity, pain-related disability, and pain behavior (e.g., avoidance). A related but separate construct—dental fear—is also experienced in the general population, and it influences dental treatment–seeking behavior and oral and systemic health. Minimal work has addressed the role of genetics in the etiologies of fear of pain and dental fear. Limited available data suggest that variants of the melanocortin 1 receptor (*MC1R*) gene may predict greater levels of dental fear. The *MC1R* gene also may be etiologically important for fear of pain. This study aimed to replicate the finding that *MC1R* variant status predicts dental fear and to determine, for the first time, whether *MC1R* variant status predicts fear of pain. Participants were 817 Caucasian participants (62.5% female; mean ± SD age: 34.7 ± 8.7 y) taking part in a cross-sectional project that identified determinants of oral diseases at the community, family, and individual levels. Participants were genotyped for single-nucleotide polymorphisms on *MC1R* and completed self-report measures of fear of pain and dental fear. Presence of *MC1R* variant alleles predicted higher levels of dental fear and fear of pain. Importantly, fear of pain mediated the relation between *MC1R* variant status and dental fear (B = 1.60, 95% confidence interval: 0.281 to 3.056). *MC1R* variants may influence orofacial pain perception and, in turn, predispose individuals to develop fears about pain. Such

Candidate genes so far

- **COMT** (Seeman et al., 2005; Diatchenko et al., 2004)
- **DRD4** (Benjamin et al., 1996, Ebstein et al., 1996)
- **GCH1** (Tegeder et al., 2006)
- **CYP2 D6** (DeLeon et al., 2003; Ammon-Treiber et al., 2003)
- **DAT1** (Mill et al., 2006)
- **OPRM** (Fillingim et al., 2005, Kim et al. 2004)
- **TRPV1** (Kim et al. 2006)
- **IL1** (Solovieva et al., 2004)
- **IL6** (Noponen-Hielta et al., 2005)
- **SCN9A** (Cox et al., 2006)

Patient education

- Patients get the pain they expect!
  - pilot fMRI study, we compared the neural basis of the analgesic and anxiolytic effect of two types of threat modulation: a "behavioural control" paradigm, which involves the ability to terminate a noxious stimulus, and a "safety signalling" paradigm, which involves visual cues that signal the threat (or absence of threat) that a subsequent noxious stimulus might be of unusually high intensity.
  - Our pilot data therefore suggest that analgesic and anxiolytic effects are instantiated in distinguishable neural mechanisms and differ between distinct stress- and pain-modulatory approaches, supporting the recent notion of multiple pathways subserving top-down modulation of the pain experience

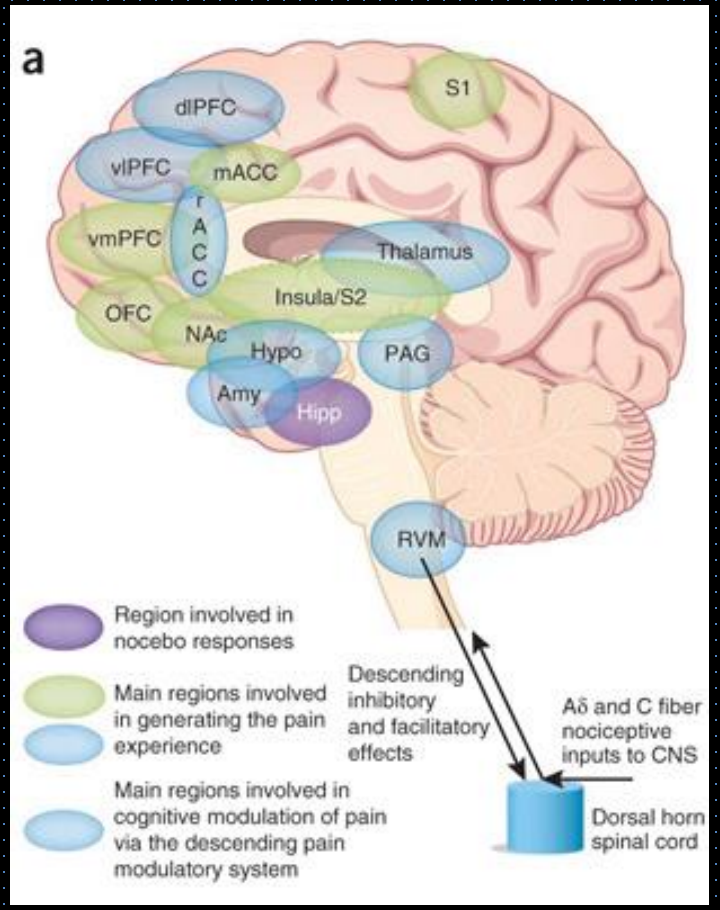
Segerdahl AR Mezue M Okell TW, Farrar JT Tracey I **Corrigendum: The dorsal posterior insula subserves a fundamental role in human pain.** Nat Neurosci. 2015 Nov 25;18(12):1861. doi: 10.1038/nn1215-1861d.
Segerdahl AR, Mezue M, Okell TW, Farrar JT, **Tracey I**. The dorsal posterior insula subserves a fundamental role in human pain. Nat Neurosci. 2015 Apr;18(4):499-500. doi: 10.1038/nn.3969. Epub 2015 Mar 9.
Wiech K Edwards R, Moseley GL , Berna C , Ploner M , Tracey I. **Dissociable neural mechanisms underlying the modulation of pain and anxiety? An FMRI pilot study** PLoS One. 2014 Dec 15;9(12):e110654. doi: 10.1371/journal.pone.0110654. eCollection 2014.

Post surgical pain
Patients get the pain they expect

Brain regions involved in modulation of acute pain

- **Para aqueductal Gray (PAG)**
- **Dorso lateral Insula cortex**
- **Locus coeruleus**
- **In spinal cord**

Distal root ganglion



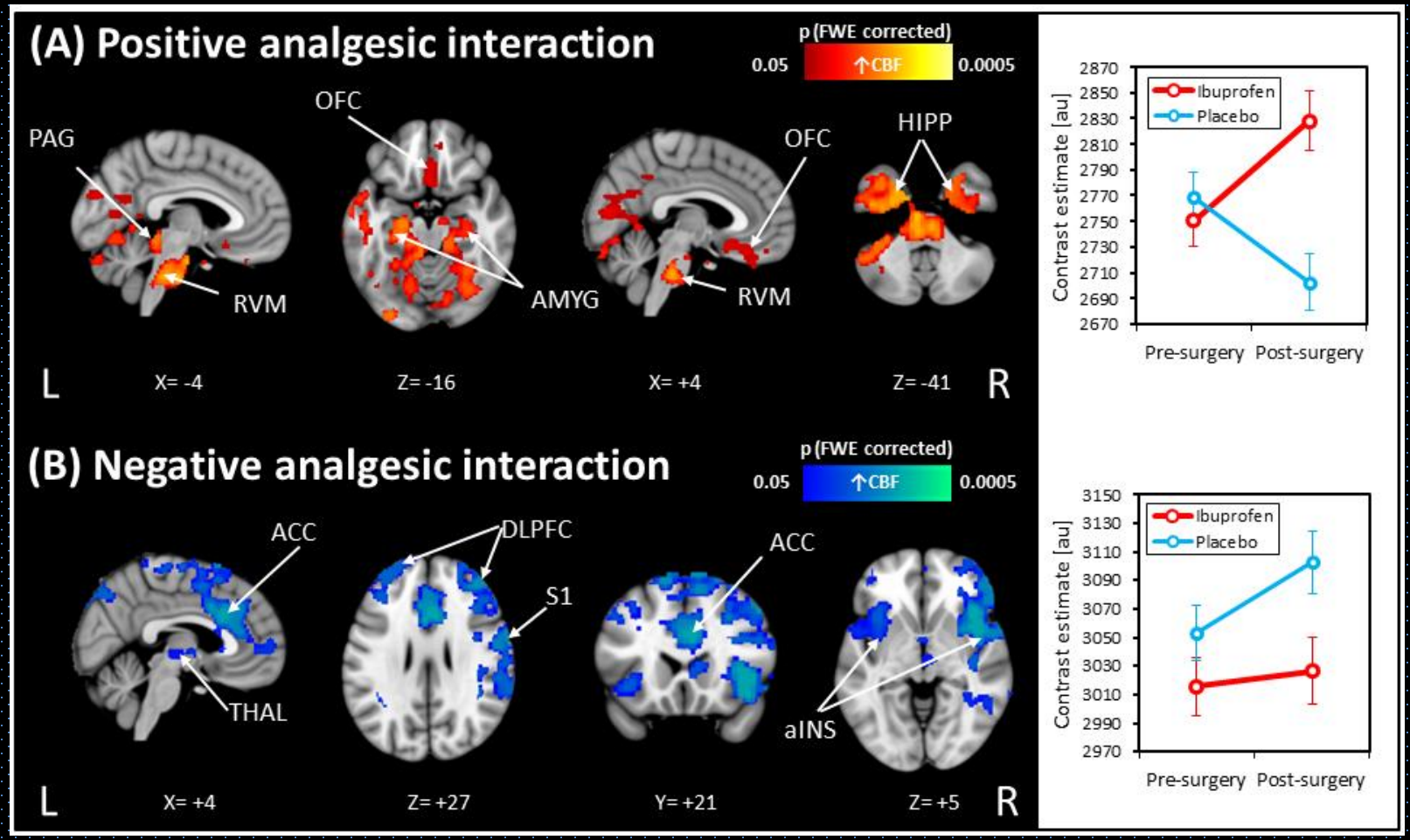
Dorso-lateral Insula cortex is the main area of brain involved in downward modulation of chronic pain

Irene Tracey Getting the pain you expect: mechanisms of placebo, nocebo and reappraisal effects in humans Nature Medicine 16, 1277–1283 (2010)

Managing patients expectations of surgical related pain is effective in pain relief !

Relationship between preoperative expectations, satisfaction, and functional outcomes in patients undergoing lumbar and cervical spine surgery: a multicenter study. Soroceanu A, Ching A, Abdu W, McGuire K. Spine (Phila Pa 1976). 2012 Jan 15;37(2):E103-8

Minimise surgical pain = minimise chronic pain
How do routine analgesic drugs work?



Non psychological contributors to lowered pain thresholds

- Opioids and OTC chronic use increase central pain sensitivity
- Nicotine makes pain worse
- Alcohol GABA agonist 1-2 drinks a day

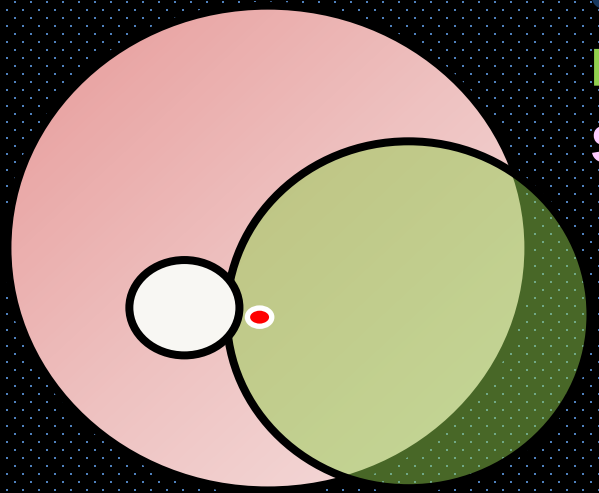
Tapping into natural resources

- Maximising downward inhibition of pain
- Sleep
- Hypnotism
- Meditation

- Education…managing expectations……

How do we minimise the pain?

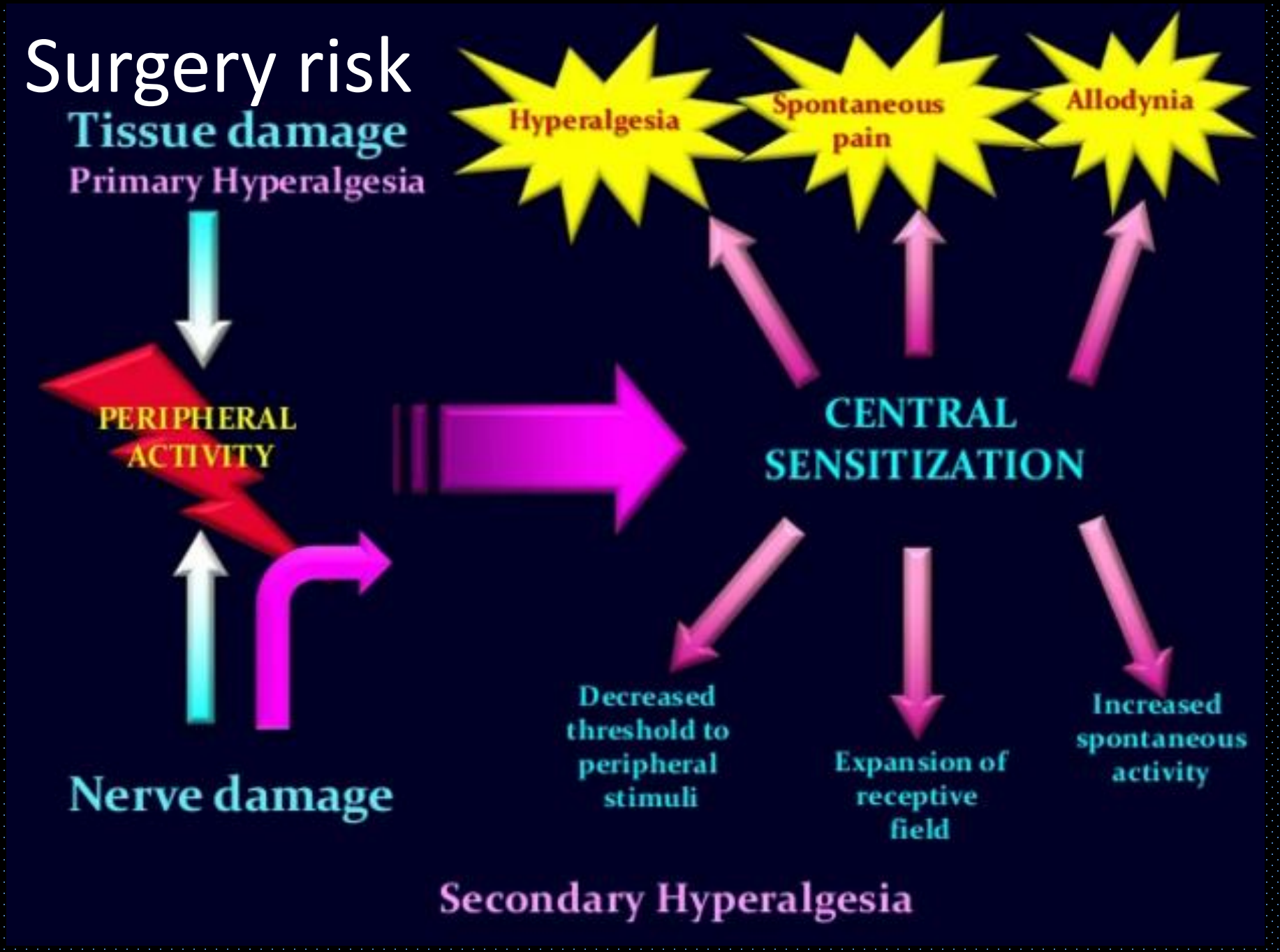
- Clinician
  - -Patient relationship
- Informed consent
  - Patient control
  - Patient expectations
- Anxiolysis
- Surgical technique
- Analgesics



Petrie KJ et al Effect of providing **information** about normal test results on **patients**' reassurance: randomised controlled trial. BMJ. 2007 Feb 17;334(7589):352.

Arnold J et al. **Information** sheets for **patients** with acute chest **pain**: randomised controlled trial.BMJ. 2009 Feb 26;338:

Surgery risk



Risk factor
Does poor operative pain control leads to CPSP

- **Prevent CPSP by Modifying surgery and pain management**
  - multi modal analgesic management of severe acute post-surgical pain
  - **minimal access surgery**
  - **intraoperative use of LA**

  International guidelines for prevention and management of post-operative chronic pain following inguinal hernia surgery. Alfieri S, Amid PK, Campanelli G, Izard G, Kehlet H, Wijsmuller AR, Di Miceli D, Doglietto GB.Hernia. 2011 Jun;15(3):239-49

Risk factor
Does poor operative pain control leads to CPSP

- **Prevent CPSP by Modifyin**
  management
  - multi modal analges
    surgical pain
  - **minimal access su**
  - **intraoperative us**

  International guidelin
  operative chronic pain
  Amid PK, Campanelli G,
  Miceli D, Doglietto GB.Herni

Dentists use local anaesthetic for all surgical procedures…….

Thus minimise central sensitisation during surgery!!!

But what about post surgical pain control

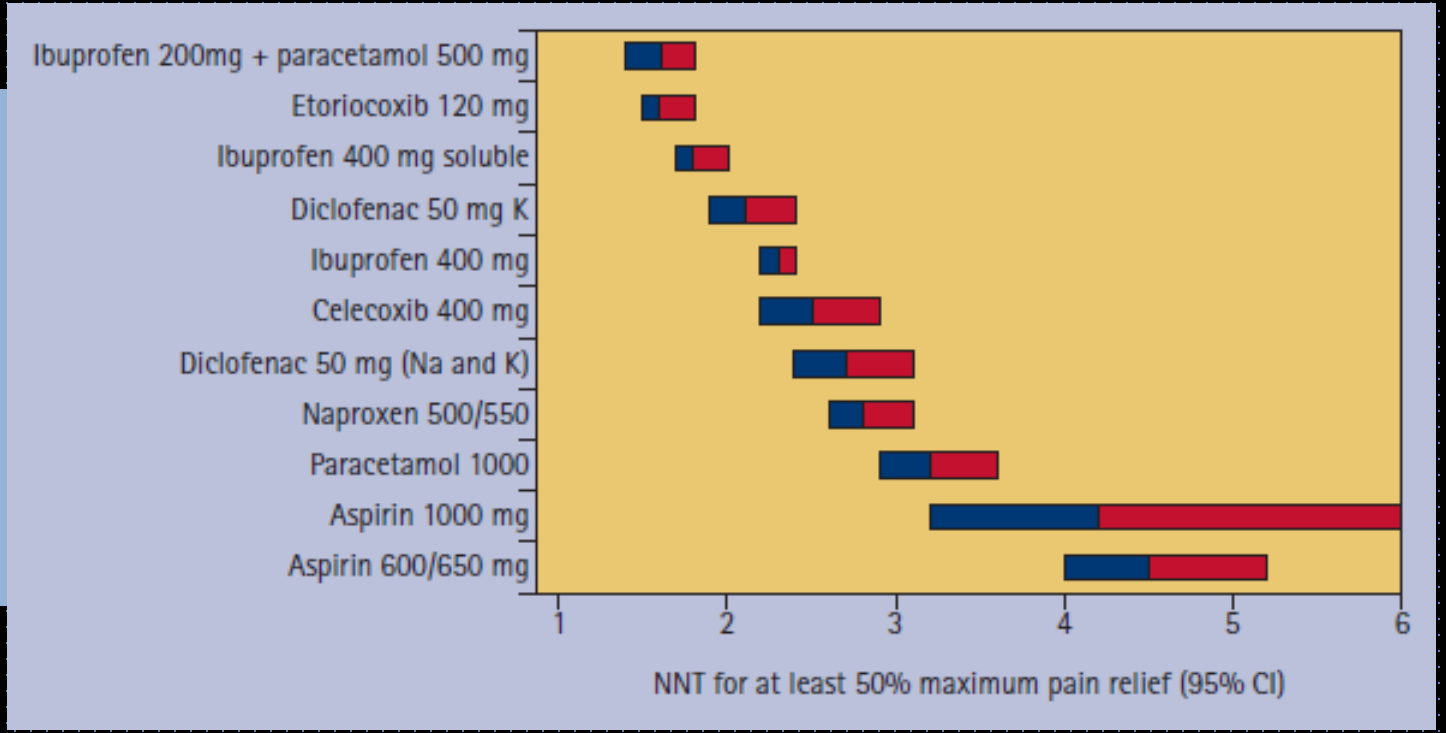
Successful Management of Acute Dental Pain

Ken M. Hargreaves, DDS, PhD

Ibuprofen (600mg or 400-800mg) + Paracetamol (500-1000mg) QDS PO = SYNERGISM
NO OPIOIDS!!!

Risk Factors
Time surgery
Depth impaction
Surgeons experience

Minimise
Min access
Information
LA
?Aggressive early post surgical analgesia

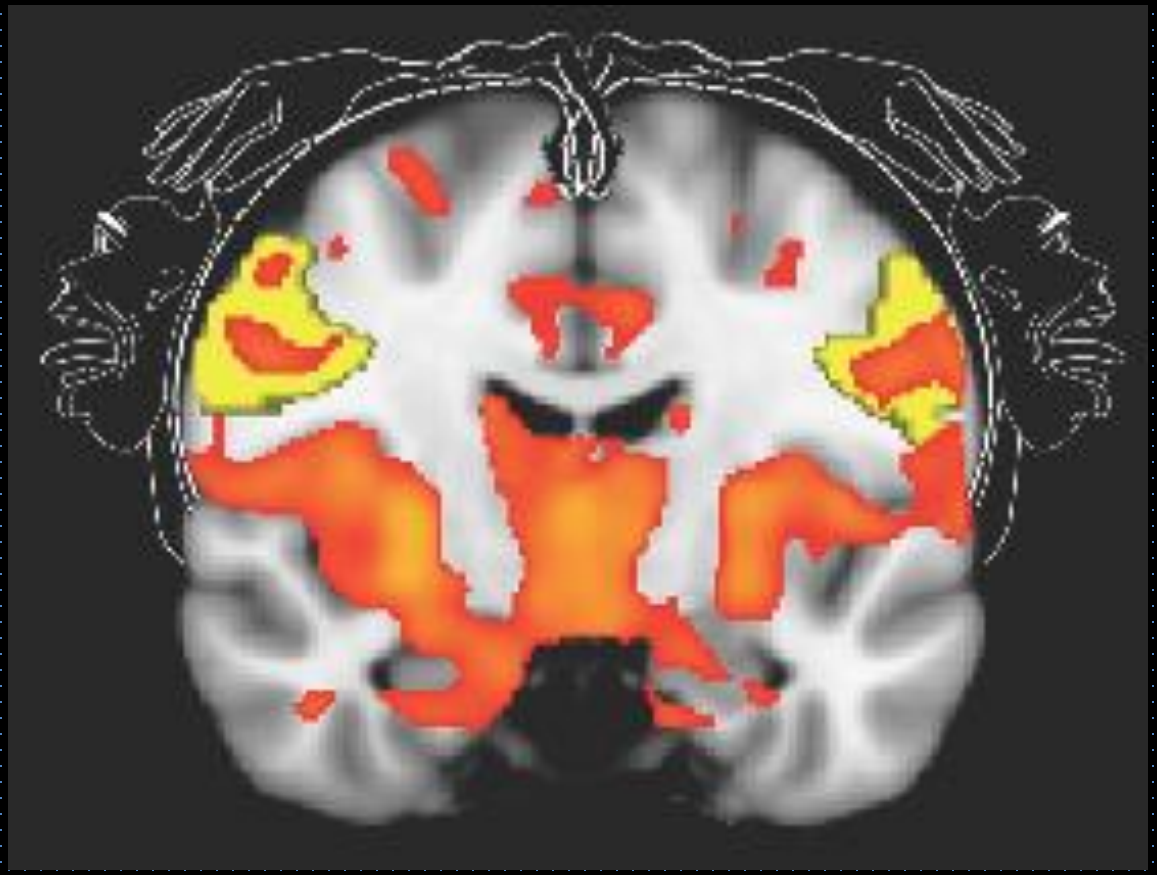


The inflammation induced by surgical trauma results in pain, of which the patient must be forewarned. This will be worst in the first 24 hours post-operatively and should be resolved within three to four days.

Derry S, Wiffen PJ, Moore RA. **Relative efficacy of oral analgesics after third molar extraction--a 2011 update.** Br Dent J. 2011 Nov 11;211(9):419-20. doi: 10.1038/sj.bdj.2011.905.

The diagnosis and medical management of facial pain Outline

- An update on pain
- **The trigeminal system**
- Chronic pain
- Classification of OFP
- Assessment
- Diagnosis



Kings College London-Tara Renton

Trigeminal nerve

- **The great protector…..**

Sensory feedback for all cranial functions

Brains- Consciousness + neural regulation

Breathing

Sight

Smell

Taste

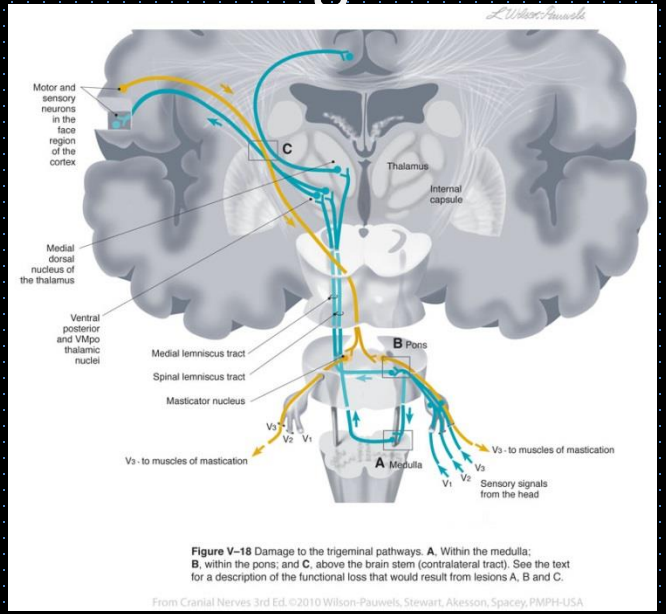


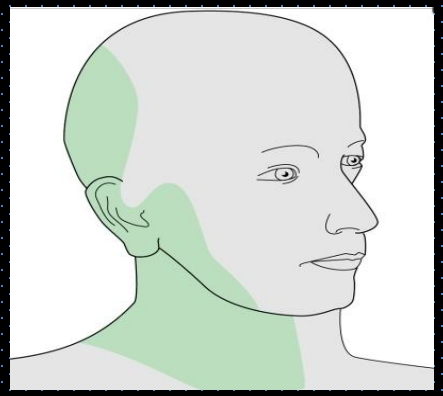
Figure V–18 Damage to the trigeminal pathways. **A**, Within the medulla; **B**, within the pons; and **C**, above the brain stem (contralateral tract). See the text for a description of the functional loss that would result from lesions A, B and C.

From Cranial Nerves 3rd Ed ©2010 Wilson-Pauwels, Stewart, Akesson, Spacey PMPH-USA

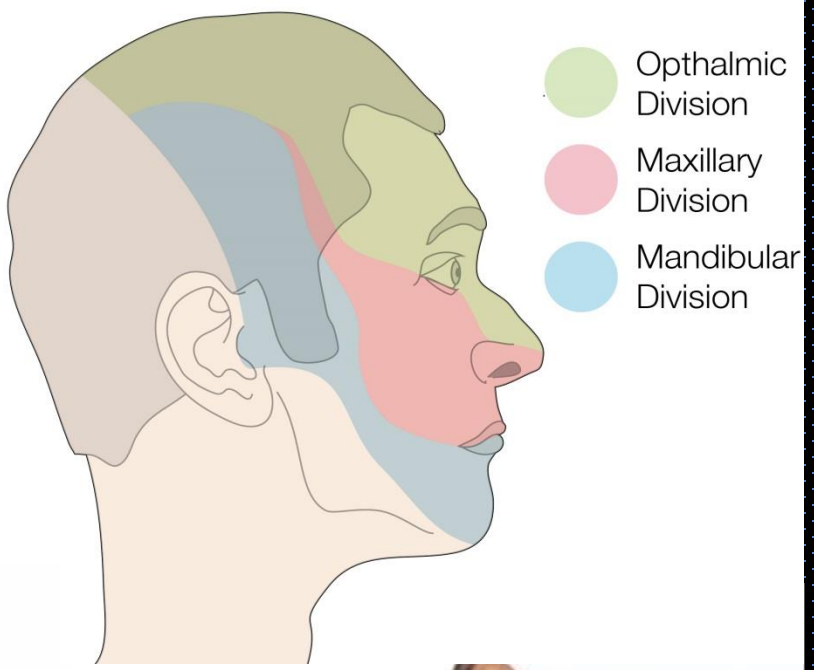
The face…the organ that underpins communication

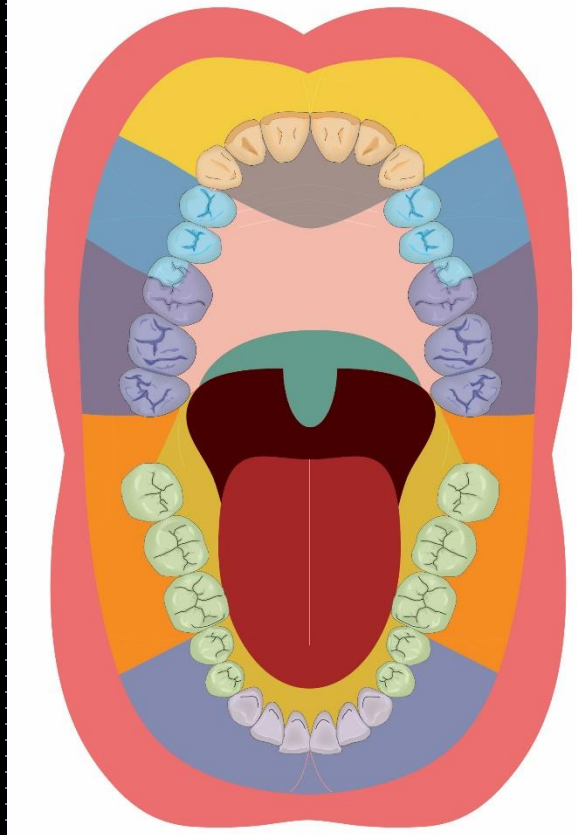
Trigeminal nerve

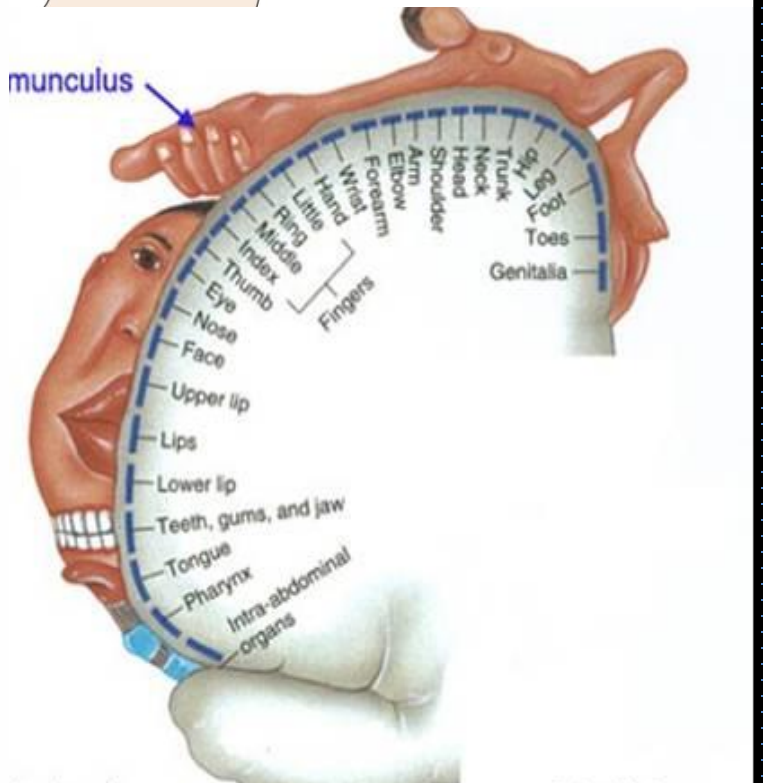
Largest sensory nerve in the body
Constant neurosensory feedback



The dermatomal distribution of C2 and C3 (Adapted from Foester O. The dermatomes in man [Schorstein Lecture, London, 1932]. Brain 1933;56:I-39.)







Trigeminal nerve

Complex region

Consequences

- Social function
- Eating
- Drinking
- Speaking
- Kissing
- Make up / shaving
- Sleeping

IDENTITY?

The diagnosis and medical management of facial pain Outline

- An update on pain
- The trigeminal system
- **Classification of OFP**
- Assessment
- Diagnosis
- Medical management of chronic V pain
- What we see at KCH OFP service

Classifications of OFP

- Woda et al 2005
- International headache society (IHS)
- American Association of Orofacial Pain (AAOP)
- Research Diagnostic Criteria for TMD
- AXES I (physical) and II (psychological)

- Types of classification
- Site / ontological / assumed mechanism/ response to treatment

Classifications of OFP

- Woda et al 2005
- **IASP** Int Assoc Study of Pain
- **ICHD**-3 Beta - The International Classification of Headache Disorders
- American Association of Orofacial Pain (**AAOP**)
- ~~Research~~ Diagnostic Criteria for TMD
- AXES I and II

Pain. 2005 Aug;116(3):396-406.

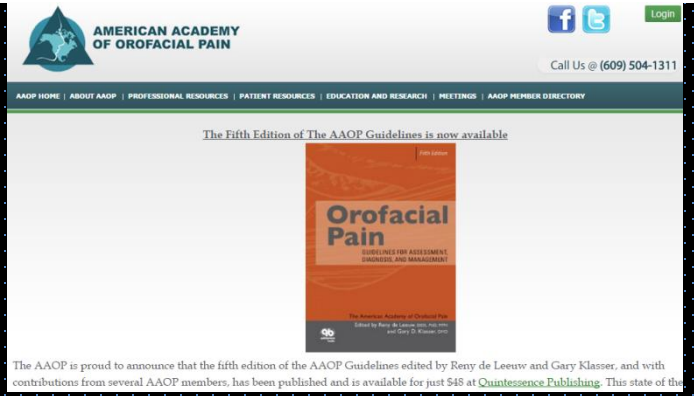
Towards a new taxonomy of idiopathic orofacial pain.

Woda A[1], Tubert-Jeannin S, Bouhassira D, Attal N, Fleiter B, Goulet JP, Gremeau-Richard C, Navez ML, Picard P, Pionchon P, Albuisson E.

Author information

Abstract

There is no current consensus on the taxonomy of the different forms of idiopathic orofacial pain (stomatodynia, atypical odontalgia, atypical facial pain, facial arthromyalgia), which are sometimes considered as separate entities and sometimes grouped together. In the present prospective multicentric study, we used a systematic approach to help to place these different painful syndromes in the general classification of chronic facial pain. This multicenter study was carried out on 245 consecutive patients presenting with chronic facial pain (>4 months duration). Each patient was seen by two experts who proposed a diagnosis, administered a 111-item questionnaire and filled out a standardized 68-item examination form. Statistical processing included univariate analysis and several forms of multidimensional analysis. Migraines (n=37), tension-type headache (n=26), post-traumatic neuralgia (n=20) and trigeminal neuralgia (n=13) tended to cluster independently. When signs and symptoms describing topographic features were not included in the list of variables, the idiopathic orofacial pain patients tended to cluster in a single group. Inside this large cluster, only stomatodynia (n=42) emerged as a distinct homogenous subgroup. In contrast, facial arthromyalgia (n=46) and an entity formed with atypical facial pain (n=25) and atypical odontalgia (n=13) could only be individualised by variables reflecting topographical characteristics. These data provide grounds for an evidence-based classification of idiopathic facial pain entities and indicate that the current sub-classification of these syndromes relies primarily on the topography of the symptoms.



AMERICAN ACADEMY OF OROFACIAL PAIN

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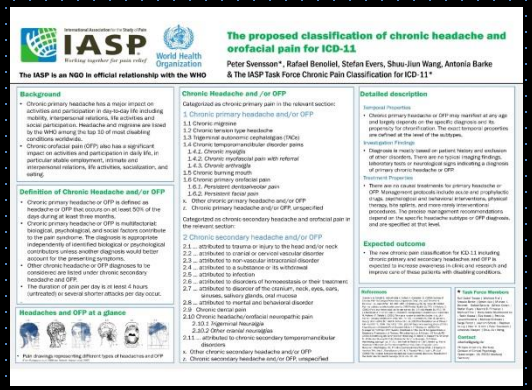
The Fifth Edition of The AAOP Guidelines is now available

Orofacial Pain

GUIDELINES FOR ASSESSMENT, DIAGNOSIS, AND MANAGEMENT

The AAOP is proud to announce that the fifth edition of the AAOP Guidelines edited by Reny de Leeuw and Gary Klasser, and with contributions from several AAOP members, has been published and is available for just $48 at Quintessence Publishing. This state of the

http://www.aaop.org/content.aspx?page_id=2



IASP

World Health Organization

The IASP is an NGO in official relationship with the WHO

The proposed classification of chronic headache and orofacial pain for ICD-11

Peter Svensson*, Rafael Benoliel, Stefan Evers, Shuu-Jiun Wang, Antonia Barke & The IASP Task Force Chronic Pain Classification for ICD-11*

Background

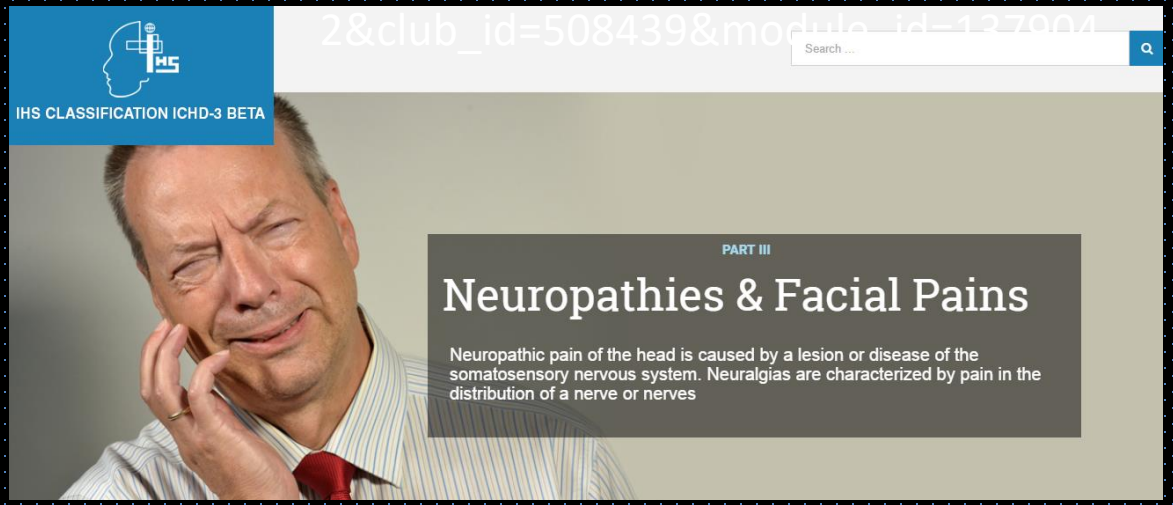
Definition of Chronic Headache and/or OFP

Headaches and OFP at a glance

Chronic Headache and /or OFP

Detailed description

Expected outcome



IHS CLASSIFICATION ICHD-3 BETA

Search ...

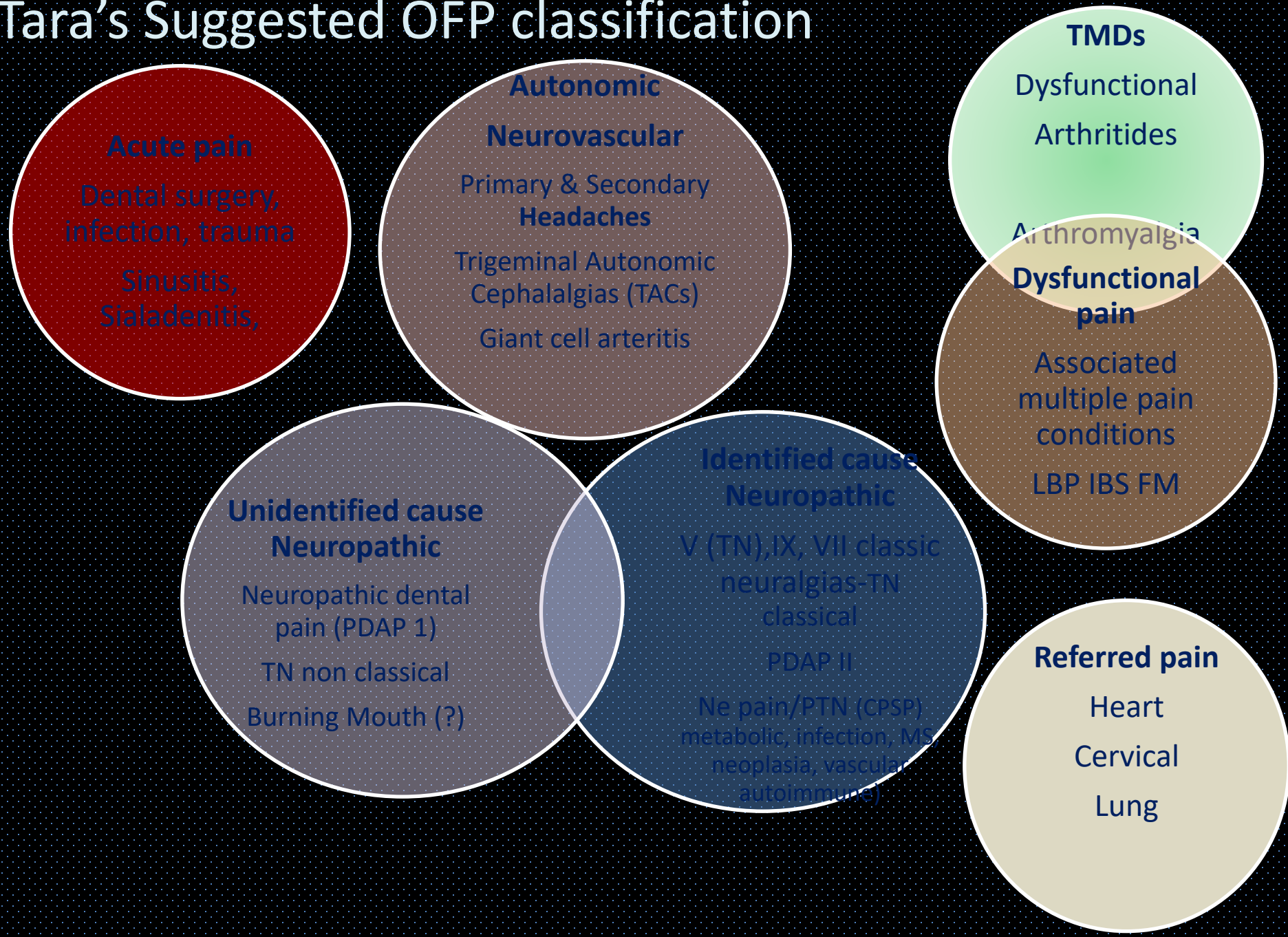
PART III

Neuropathies & Facial Pains

Neuropathic pain of the head is caused by a lesion or disease of the somatosensory nervous system. Neuralgias are characterized by pain in the distribution of a nerve or nerves

https://www.ichd-3.org/

Tara's Suggested OFP classification



Fresh out International classification of orofacial pain

Cephalalgia
An International Journal of Headache
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

Redundant terms?

- Persistent idiopathic facial pain
- Atypical facial pain
- Non-odontogenic tooth pain
- Atypical odontalgia
- Phantom tooth pain

The diagnosis and medical management of facial pain Outline

- An update on pain
- The trigeminal system
- Classification of OFP
- **Assessment**
- Diagnosis

How do we assess pain?

Consider an assessment of the biopsychosocial aspects of pain

This includes:

- Pain intensity
- Pain affect
- Pain-related disability

Sydney University Gargoyle

- From a good history you can diagnose most OFP conditions
- In order to confirm OFP diagnosis, you must exclude;
  - Inflammatory pain
  - Referred pain
  - Cancer related symptoms

Listen to the patient they will tell you the diagnosis

William Osler

Assessment overview

- Age, gender, BMI, Blood pressure
- MH DH SH
- Psychological Axis II
- Pain History SOCRATES
- Clinical assessment
  - V123 C2C3
  - TMJs, Lymph, Skin, Asymmetry
  - Intraoral examination
- Investigations
  - Haematological
  - Radiological
  - Pain Dairy
  - Drug trial e.g. Indomethacin test

Axis 2 Assessment of preceding and injury related psychological problems

- All patients:
  - **EQ-5D**
  - **GAD7 generalised anxiety disorder**
  - **PHQ9 Patient Health Questionnaire**
  - **PHQ 15 MULTIDIMENSIONAL SCALE OF PERCEIVED SOCIAL SUPPORT**
  - **GCPS**
  - SF-MPQ-2 Short-form McGill Pain Questionnaire-2
  - PAIN DETECT PAIN QUESTIONNAIRE Ne pain
  - BPI Facial pain
  - CPSI (sleep)
  - ES-R (abuse)

- Dash board with red flags suicidal thoughts/ depression, anxiety and somatic disorders

Severe Anxiety
Probable Major depression
Somatic disorder
PTSD
Likely NP

Pain history taking is often taught as using SOCRATES

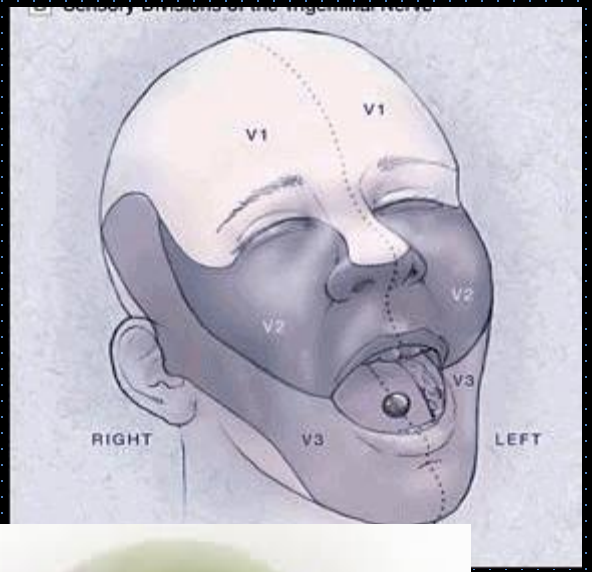
- **S**ite - Where is the pain? Or the maximal site of the pain.
- **O**nset - When did the pain start, and was it sudden or gradual? Include also whether if it is progressive or regressive.
- **C**haracter - What is the pain like? An ache? Stabbing?
- **R**adiation - Does the pain radiate anywhere? (See also Radiation.)
- **A**ssociations - Any other signs or symptoms associated with the pain?
- **T**ime course - Does the pain follow any pattern?
- **E**xacerbating/Relieving factors - Does anything change the pain?
- **S**everity - How bad is the pain?

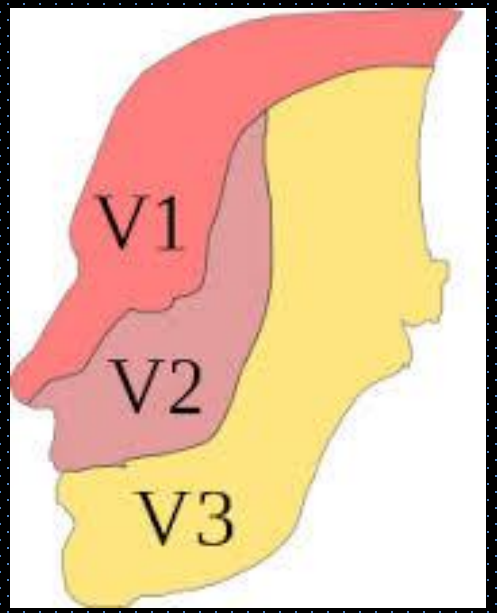
William Osler

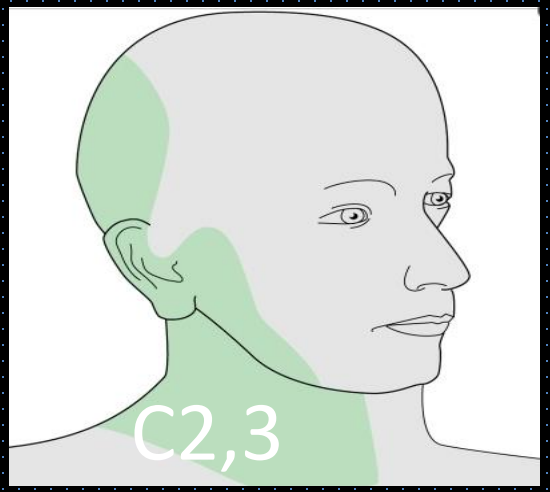
Listen to the patient and they will tell you their diagnosis

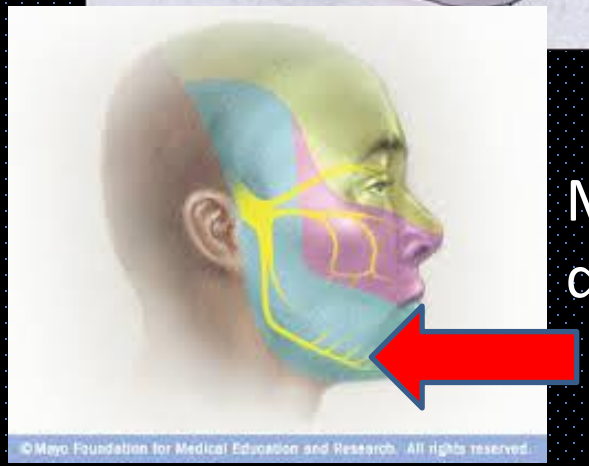
KING'S College LONDON

Get the anatomy correct! (V1 V2 V3 C2 and C3)









Most anatomical diagrams are wrong

The dermatomal distribution of C2 and C3 (Adapted from Foester O. The dermatomes in man [Schorstein Lecture, London, 1932]. Brain 1933;56:I-39.)

Pain Descriptors

- Sharp /shooting /stabbing /severe /life stopping
- Aching, burning, throbbing
- Pins needles, tickling, itchy
- Pulling, stretching, waterfall

Next questions around;

1. Intensity
2. Timing
   - Constant or intermittent
   - Constant with fluctuation (causes?)
   - Intermittent ? Triggers? Elicited or spontaneous?
3. Alleviating and aggravating factors
4. Associated signs and symptoms
   - neuropathy altered sensation, numbness, hypereasthetic
   - Autonomic signs?
     - Flushing face, meiosis, ptosis, nasal congestion, conjunctival irritation

Descriptors

- Sharp /shooting /stabbing /severe /life stopping
- Aching, burning, throbbing
- Pins needles, tickling, itchy
- Pulling, stretching, waterfall

ALLODYNIA?

Neuralgic pain on touch or normal function

= **NEURALGIA**

Next questions around;

1. Intensity
2. Timing
   - Constant or intermittent
   - Constant with fluctuation (causes?)
   - Intermittent ? Triggers? Elicited or spontaneous?
3. Alleviating and aggravating factors
4. Associated signs and symptoms
   - neuropathy altered sensation, numbness, hypereasthetic
   - Autonomic signs?
     - Flushing face, meiosis, ptosis, nasal congestion, conjunctival irritation

OFP pain descriptors

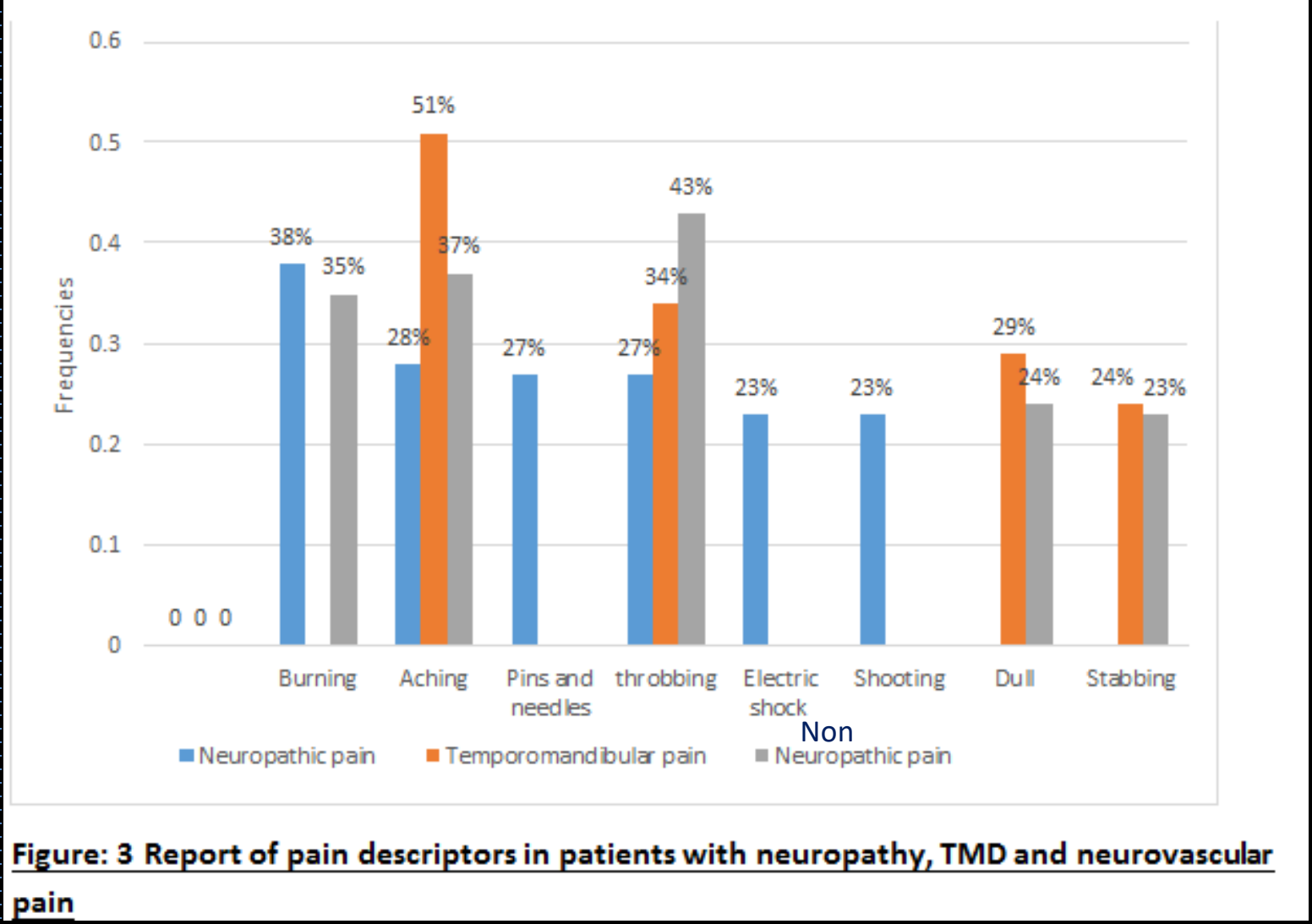


Figure: 3 Report of pain descriptors in patients with neuropathy, TMD and neurovascular pain

OFP pain descriptors

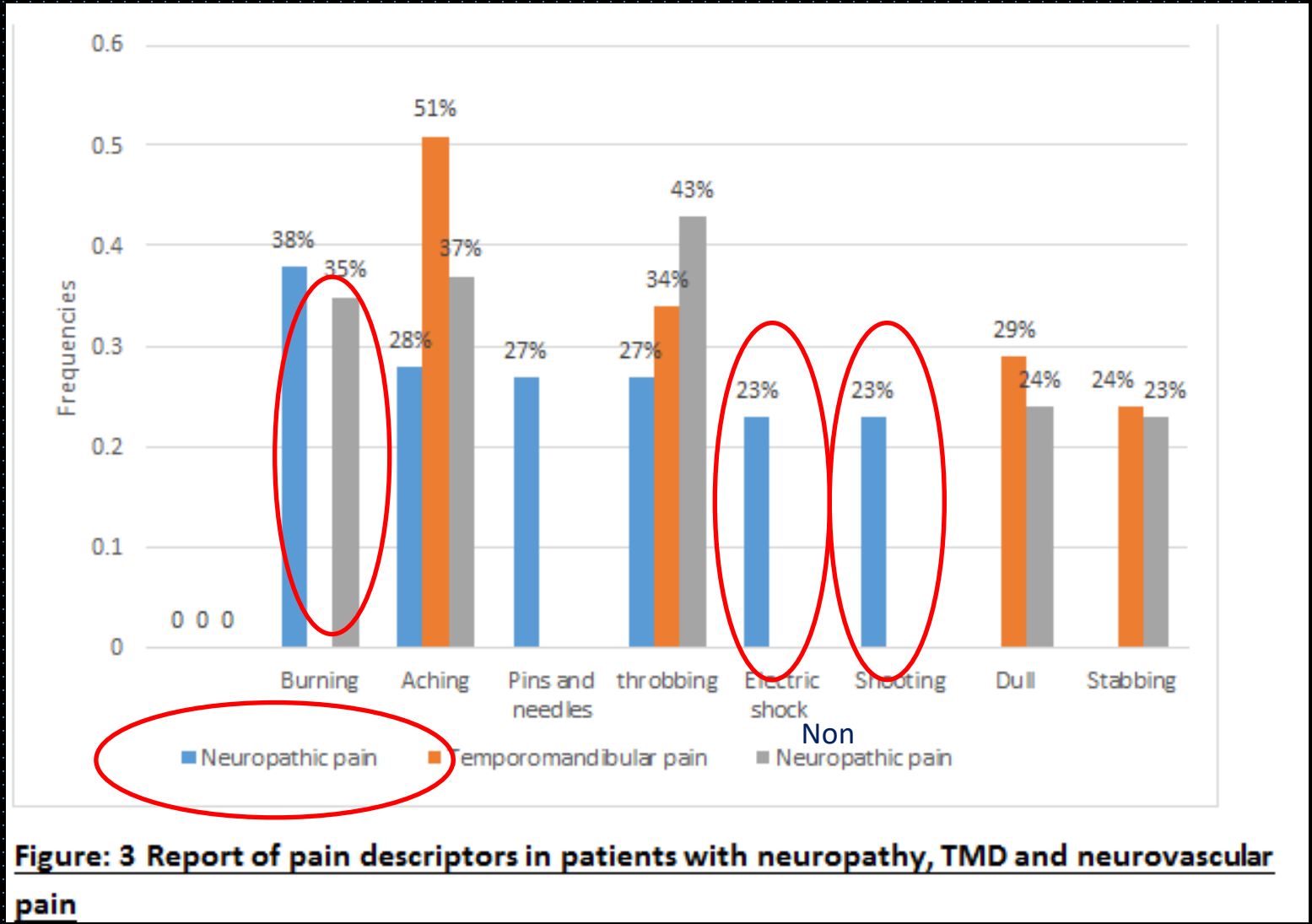


Figure: 3 Report of pain descriptors in patients with neuropathy, TMD and neurovascular pain

Axis 2 Assessment of preceding and injury related psychological problems

- All patients:
  - GAD7 generalised anxiety disorder
  - PHQ9 Patient Health Questionnaire
  - MULTIDIMENSIONAL SCALE OF PERCEIVED SOCIAL SUPPORT
  - OHIP-14 Oral Health Impact Profile
  - PCL Posttraumatic Stress Disorder Checklist
- Patients with pain only (in addition to the previous):
  - CPAQ-8 Chronic Pain Acceptance Questionnaire
  - SF-MPQ-2 Short-form McGill Pain Questionnaire-2
  - PAIN DETECT PAIN QUESTIONNAIRE Ne pain
  - PCS catastrophizing scale
  - PSEQ Pain Self-Efficacy Questionnaire
  - **SLEEP assessment**
  - **Abuse**

Exclude systemic and local pathology

Bloods

- FBC, haematinic (folate, B12, ferritin)
- Thyroid function tests
- HBA1c
- Zinc magnesium Vit D levels
- ENAs and ANAs
- Us+Es required for contrast Gadolinium MRI scan

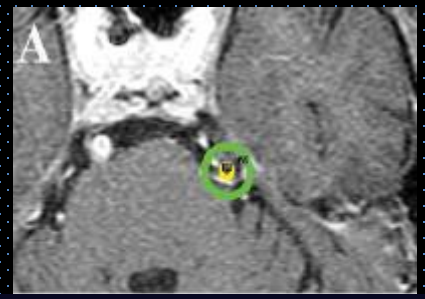
Excluding secondary neuropathy-‘lesional’ neuropathy For BMS diagnosis you must exclude BMD causes:

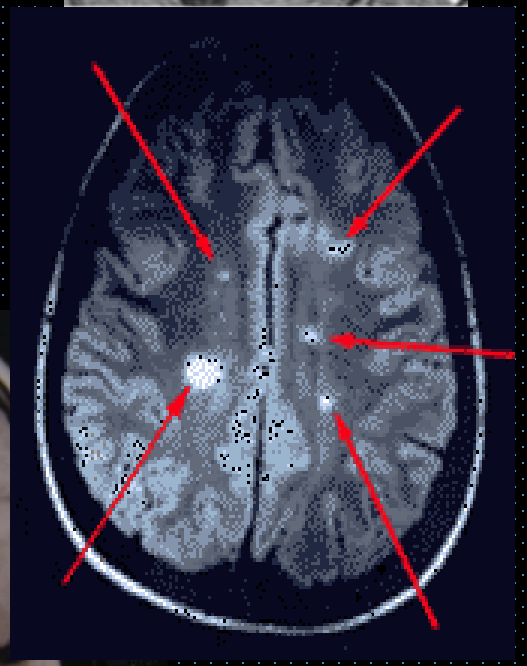
- Endocrine
  - Menopausal
  - Hypo thyroid
  - Diabetes
- Immunological
  - Autoimune
  - Sjögren Syndrome
  - Scleroderma
- Vitamin deficiency (B1, B2, B6, B12, folate, iron)
- Medication reaction (eg, ACE inhibitors, ARBs, antiretrovirals, psychotropic, anticholinergic,clonazepam, [37] chemotherapeutic agents) Ciguatera neurotoxin exposure [38]
- Psychometric
- Allergy
- Supertasters
- Gastric reflux GERD
- Neuropathy ??
- Anemia
- Multiple sclerosis
- Anxiety
- Dehydration
- Mouth breathing/nasal obstruction
- Alcohol-based mouthwash
- Radiation-induced stomatitis
- Vesiculous bullou conditions
  - Aphthous stomatitis
  - Contact stomatitis
  - Erosive lichen planus
  - Pemphigoid
  - Pemphigus
  - Geographic tongue
- Leukoplakia
- Neoplasia
- Chewing tobacco use
- Areca nut extract exposure [39]
- INFECTION
  - Bacterial infection [40]
  - Candidiasis [28]
  - VIRAL

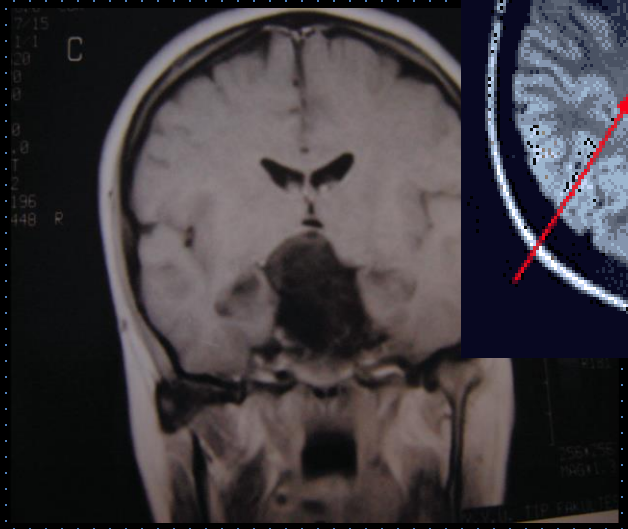
Exclude central pathology

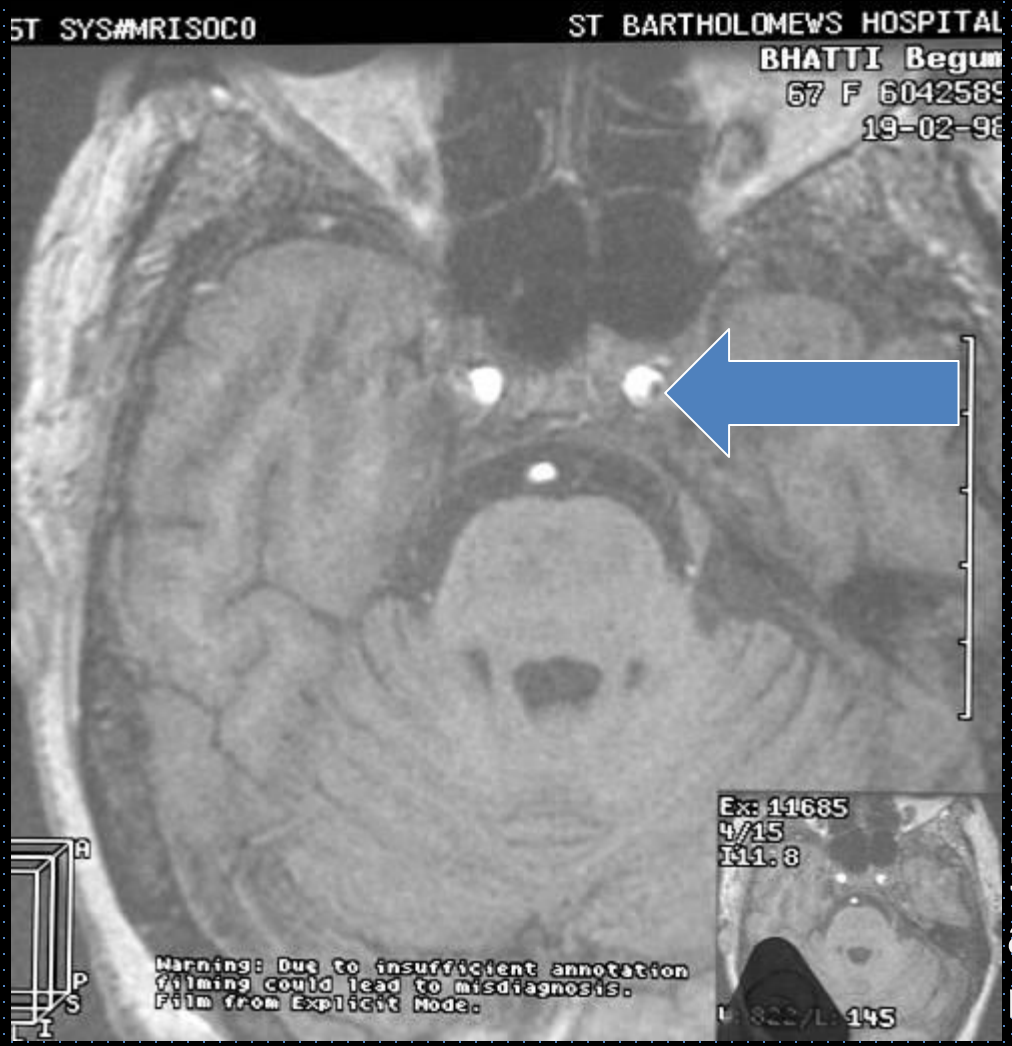
- Classical TN
  - vascular compression
- Multiple sclerosis
  - MRI plaques
- Stroke
- Vasculitis
- Post herpetic neuralgia
- Tumours
  - Cervical pathology

RED FLAGS?









MRI scan

Diagnosis and differential diagnosis of trigeminal neuralgia

Zakrzewska JM.
Clin.J.Pain 2002;18:14-21

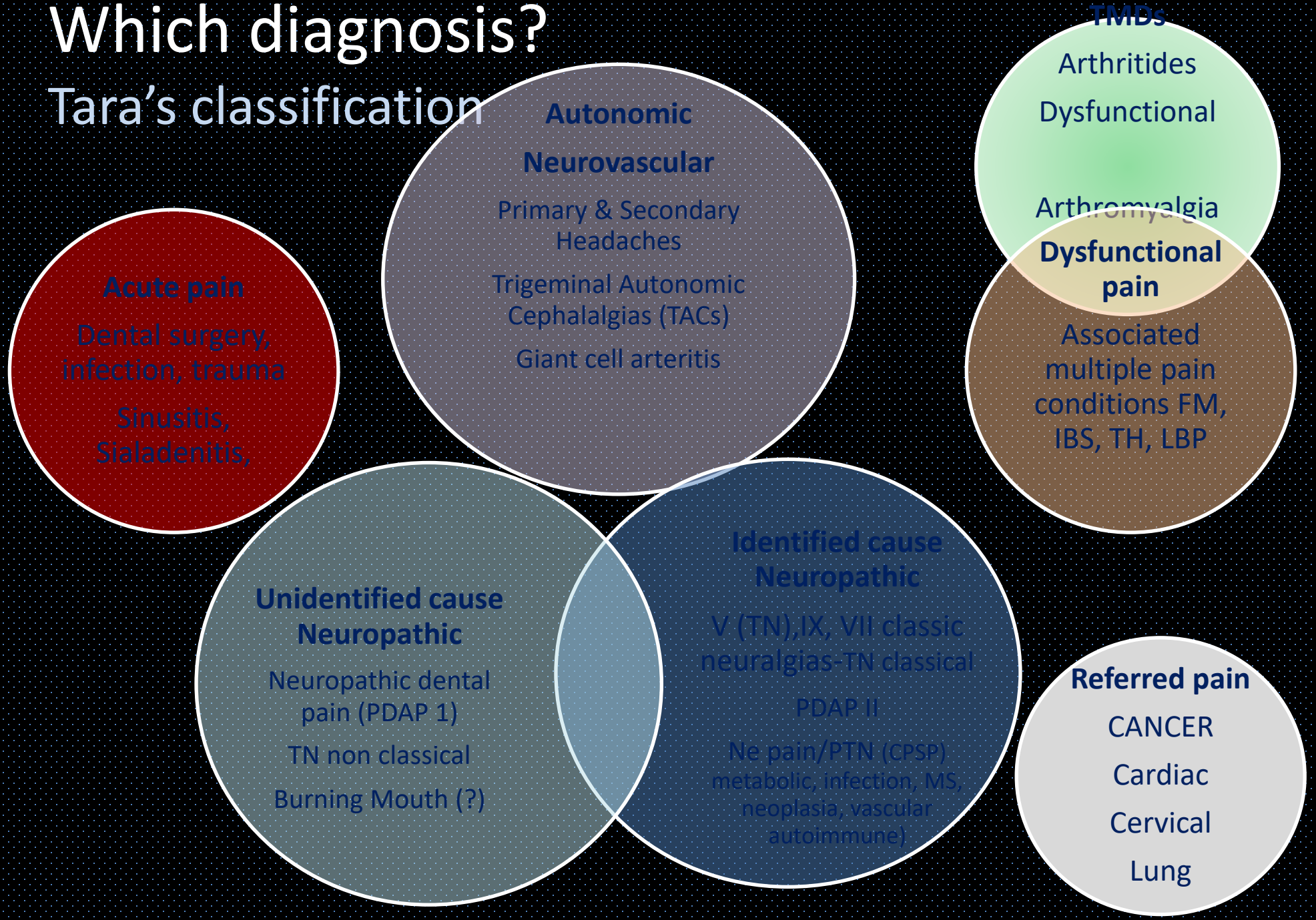
15-88% MRI+ superior cerebellar artery vascular compromise+ve results
25-49% people with NO TN have MRI +ve signs!!!!! (Kakizawa et al 2008,Adamczyk et al 2007)

The diagnosis and medical management of facial pain Outline

- An update on pain
- The trigeminal system
- Classification of OFP
- Assessment
- **Diagnosis**

Which diagnosis?

Tara's classification



FIRST

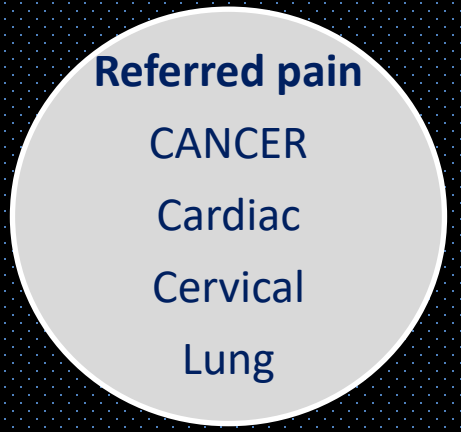
Exclude persistent inflammatory pain

inflammatory pain

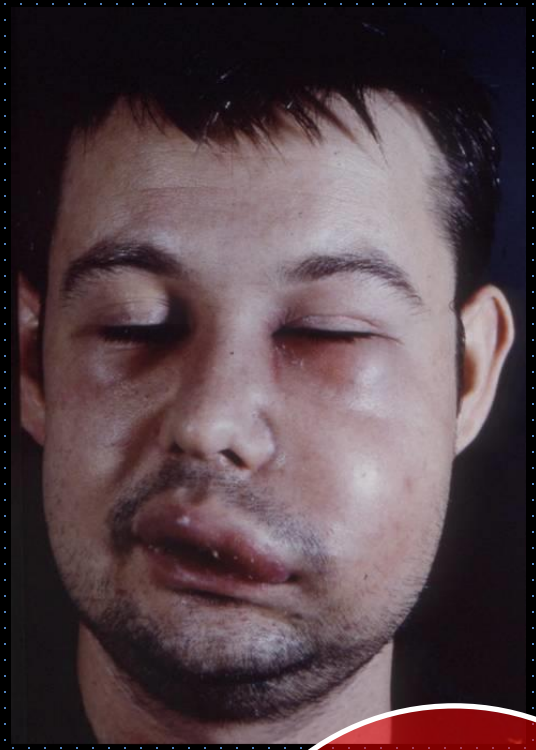
Dental, TMD, Sinus, Skin, Salivary gland, bone, mucosa

and….

CANCER!!!!!



Get the diagnosis right!



Identify cause

When possible **remove cause**:

- Extraction
- Extirpation, and
- Additional drainage pus
- Analgesics
- Rehabilitate patient

Reassess: Is the infection resolved?

If not, what additional treatment is required?

Recheck diagnosis, identify cause and remove.

Is some pus remaining and undrained?

Is incise and drainage required (I&D)?

Are antibiotics (AB) indicated?

If the infection persists with AB therapy……

Is a culture and sensitivity test required to evaluate most effective AB therapy?

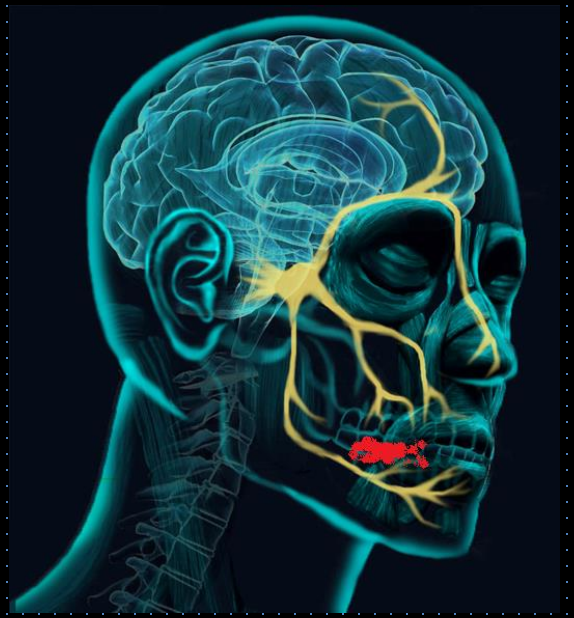
Acute pain

Dental surgery, infection, trauma

Sinusitis, Sialadenitis,

Intraoral pain

- Toothache
- Trauma
- Ulceration or other oral lesions



- Primary or secondary Post traumatic or post lesional neuropathic pain
  - Trigeminal neuralgia
  - Burning mouth Disorder or Syndrome

Acute pain

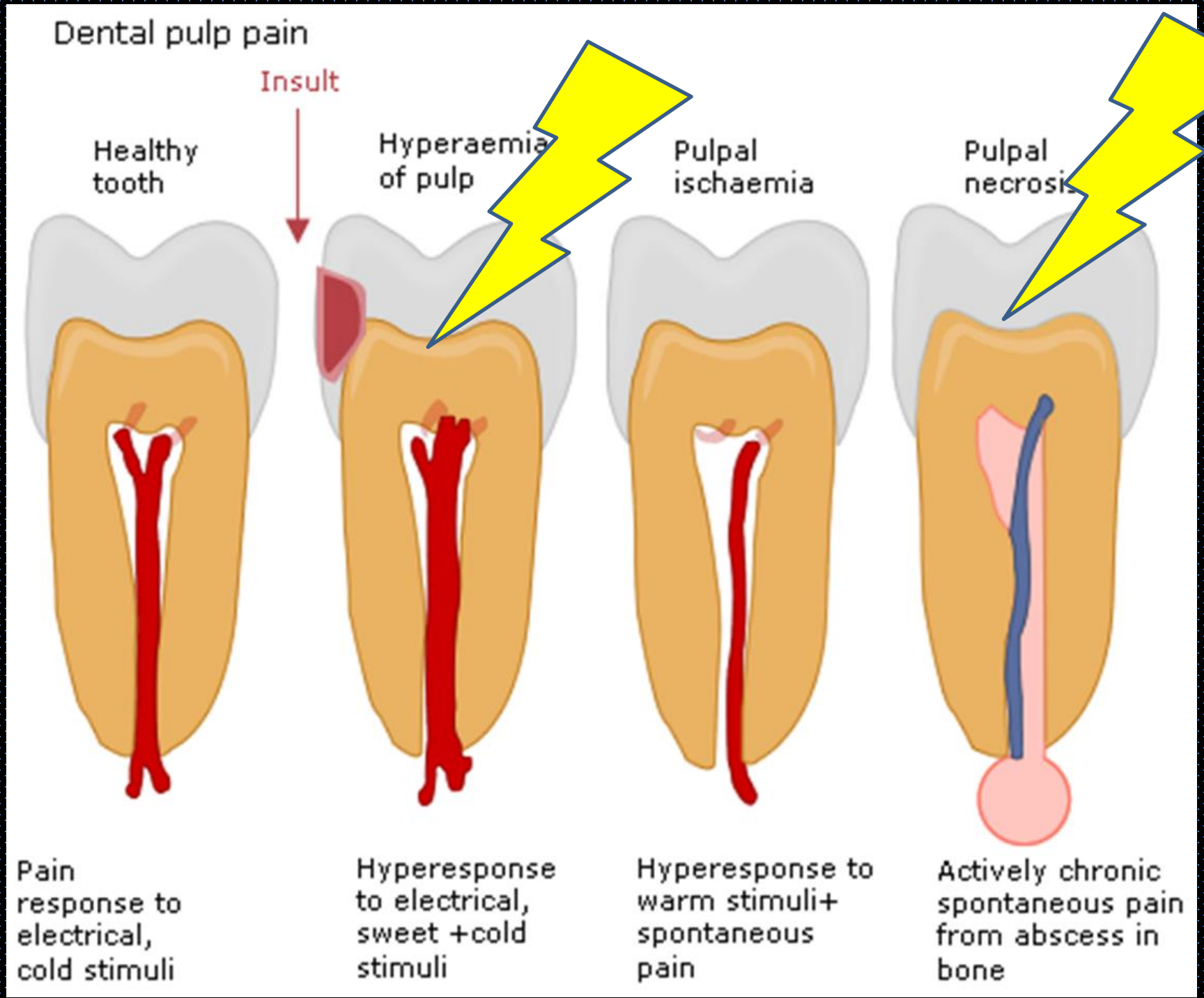
Dental surgery, infection, trauma

Sinusitis, Sialadenitis,

Why do dentists get confused with chronic pain signs?

- Teeth are the ONLY bodily organ, in health, that respond to normal stimulation with pain = ALLODYNIA!

- Toothache mimics……
  - Throbbing pain of headaches
  - Dull ache of sinusitis, salivary gland obstruction
  - Neuralgic pain of stabbing headaches, joint dysfunction, trigeminal autonomic cephalagias, TN
  - To name just a few!!!!!!


Dental pulp pain
Insult
Healthy tooth
Hyperaemia of pulp
Pulpal ischaemia
Pulpal necrosis
Pain response to electrical, cold stimuli
Hyperesponse to electrical, sweet +cold stimuli
Hyperesponse to warm stimuli+ spontaneous pain
Actively chronic spontaneous pain from abscess in bone

In health

Acute pain

Dental surgery, infection, trauma

Sinusitis, Sialadenitis,

In relation to dental innovation……..

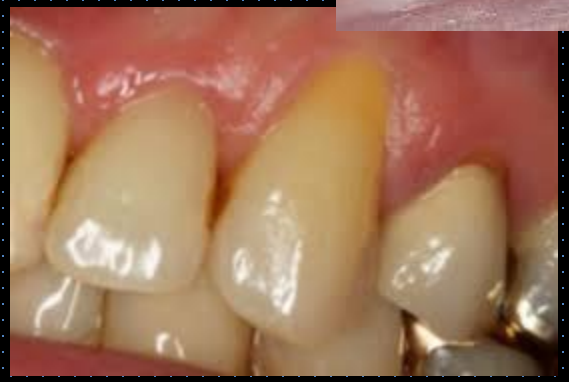
CRACKED TOOTH!!!!

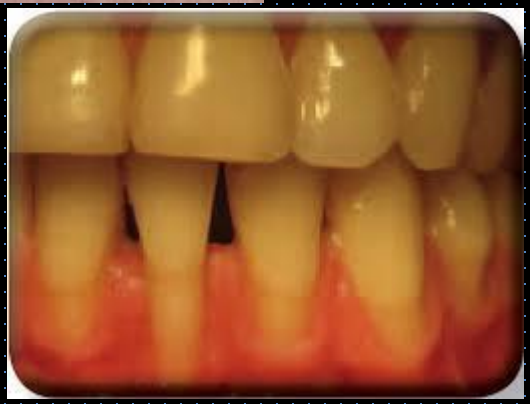
Allodynia is NORMAL!

Attrition

Abrasion

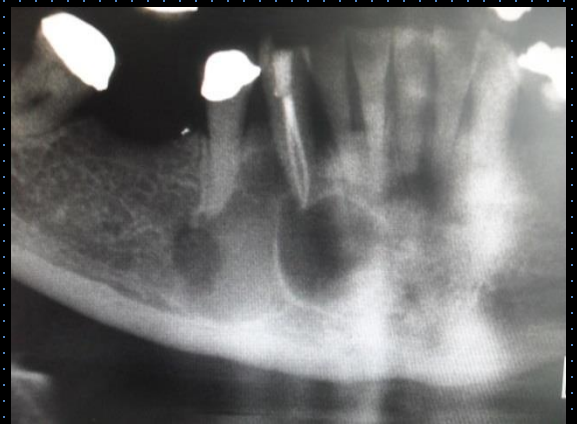
Erosion

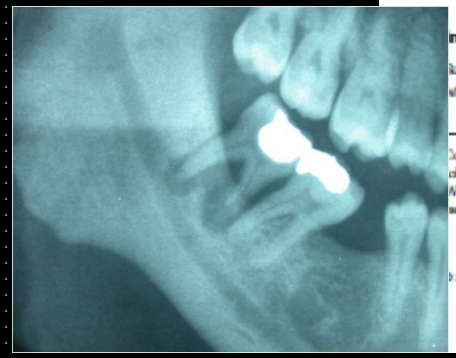




Periapical lesions

In one retrospective study, the incidence of mental paresthesia resulting from periapical infection or pathology was 0.96%. In another 0.24% of cases in the same study, mental paresthesia was a complication of root canal treatment (caused by severe overfill in one case and iatrogenic perforation of mechanical instrumentation through the root and into the mental nerve in the second case).





INTERNATIONAL ENDODONTIC JOURNAL

doi:10.1111/iej.12352

The detection of periapical pathoses in root filled teeth using single and parallax periapical radiographs versus cone beam computed tomography – a clinical study

A. Davies[1], F. Mannocci[1], P. Mitchell[1], M. Andiappan[2] & S. Patel[1]

[1]Department of Endodontology, Kings College Dental Institute, Guys Hospital, London Bridge, London; and [2]Biomedical Statistics, Kings College Dental Institute, Denmark Hill, London, UK

Abstract

Davies A, Mannocci F, Mitchell P, Andiappan M, Patel S. The detection of periapical pathoses in root filled teeth using single and parallax periapical radiographs versus cone beam computed tomography – a clinical study. *International Endodontic Journal*

Aim To clinically assess the diagnostic yield from single parallel periapical, two parallax radiographs and cone beam computed tomography (CBCT) by comparing the prevalence of periapical lesions associated with individual roots, and the total number of root canals in root-treated teeth in patients referred for root canal retreatment.

Methodology Single and parallax periapical radiographs, and CBCT scans, were taken of 100 teeth in 78 patients who had been referred for root canal retreatment. The presence of a periapical lesion associated with each specific root and the number of identifiable root canals were assessed using each imaging modality by a consensus panel of two examiners. The number of root canals was confirmed using the dental-operating microscope during treatment. In addition, the panel was asked to decide whether they felt they had adequate information to manage each case.

Results A total of 209 paired roots were assessed for periapical lesions. Lesions were identified in 41%, 38% and 68% of paired roots when using single radiographs, two parallax radiographs and CBCT respectively. The number of root canals identified were 186, 218, 242 and 239 when using parallel, parallax, CBCT and the dental-operating microscope, respectively.

Conclusions CBCT detected significantly more periapical lesions and root canals than both single and parallax periapical radiographs. There was no significant difference between CBCT and the clinical microscope in the identification of root canals. Whilst two parallax radiographs detect significantly more root canals than a single radiograph, they did not increase detection of periapical lesions when compared to a single periapical radiograph.

Keywords: cone beam computed tomography, intra-oral radiographs, parallax radiographs, periapical lesions.

Received 27 January 2014; accepted 28 July 2014

Introduction

Success rates of primary root canal treatments vary, with 60–100% of cases demonstrating healing or healed outcomes (Ng *et al.* 2007). However, when outcome is assessed by cone beam computed tomography (CBCT), a lower initial rate of healing is demonstrated (Patel *et al.* 2012b). With many millions of root canal treatments performed annually, the small number that develop post-treatment apical periodontitis translate to a large number of cases that may potentially require further management (Roda & Gettleman 2011). Root canal treatment usually fills

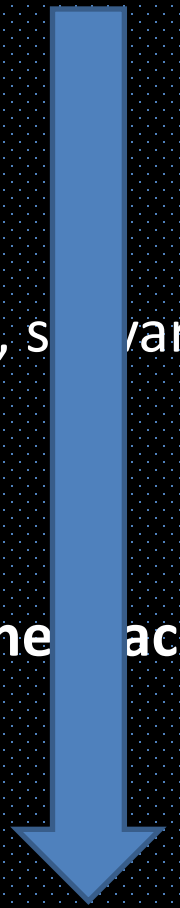
Correspondence: Dr. Sharon Patel, Department of Endodontology, Kings College Dental Institute, Floor 25 – Tower Wing, Guys Hospital, London SE1 9RT, UK (e-mail: sharonpatel@gmail.com).

Neuralgia in the Trigeminal system

- Nociceptive
  - Dentine sensitivity
- Inflammatory
  - Odontogenic and non odontogenic
- Neurovascular
  - Headaches, TACs
- Neuropathic
  - BMS, TN Secondary neuropathy
- Dysfunctional
  - FM,
  - Temporomandibular Disorder (TMD)
  - Myalgic, arthritides, dysfunctional
  - Persistent idopathic facial pain (intra oral and extraoral)

Causes of ‘neuralgia’ in the trigeminal system

- Most common
  - Toothache
  - TMD
  - Post traumatic neuropathy
  - Non dental pathology-cysts, SOLs, sinus, ear, s vary
- Least common
  - Secondary peripheral painful neuropathies
    - PHN getting rarer
  - **Trigeminal autonomic cephalalgia (Cluster he ache)**
    - **SUNCT, SUNA, HC ,PH**
  - Trigeminal neuralgia
  - IX neuralgia
  - Nervous intermedius neuralgia



Inflammatory pain mimicking toothache

TMDs, Sinusitis

- **S -site**
  - **Usually unilateral**
  - **Temporal occipital**
- O -onset
  - Spontanous age 20-40yrs
  - **Male**
- C -character
  - **severe neuralgic**
- R -radiation
  - Unilateral radiation
- **A –** associations
  - **Autonomic signs, parasympathetics. Face sweating, redness, conjunctval irritation, nasal congestion, meiosis, ptosis**
- T -timing
  - Intermittent
  - Night time onset
- E -exacerbating & relieving factors

exacerbating & relieving factors

exercise, alcohol, GTN

Relief oxygen

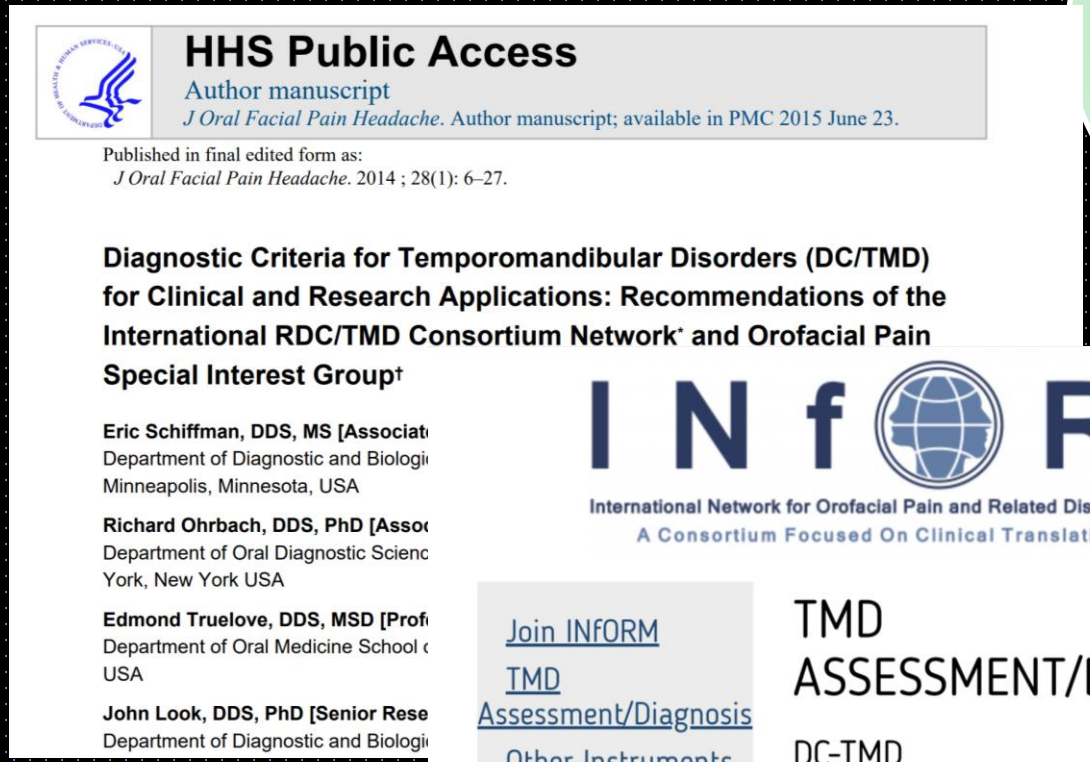
- S –SUICIDAL severe **impactful**

Definition Types of Temporomandibular disorders Diagnostic Criteria

- TMJ ~~RDC~~ now DC
  - Degenerative joint disease
    - Arthritides
    - +/-pain OA, RA, Reactive
  - Myalgia or arthralgia
    - Muscle or joint pain
  - Myofascial
  - Internal derangements
    - Disc displacement with or without reduction (with or without open or closed locking)
    - subluxation
  - Headache attributable to TMD

http://www.iadr.org/INfORM/DC-TMD

Degenerative
Myofacial pain
Arthro/myalgia
DDWOR /DDWR
subluxation
Headache



HHS Public Access

Author manuscript

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Published in final edited form as:

J Oral Facial Pain Headache. 2014 ; 28(1): 6–27.

Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for Clinical and Research Applications: Recommendations of the International RDC/TMD Consortium Network* and Orofacial Pain Special Interest Group†

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Department of Diagnostic and Biologi

INfORM

International Network for Orofacial Pain and Related Disorders Methodology

A Consortium Focused On Clinical Translation Research

Join INfORM
TMD Assessment/Diagnosis
Other Instruments
Meetings
Other Resources
INfORM Officers

Follow IADR INfORM

TMD ASSESSMENT/DIAGNOSIS

DC-TMD

Diagnostic Criteria for Temporomandibular Disorders (2014)

Overview

The Diagnostic Criteria for Temporomandibular Disorders (DC/TMD), by Schiffman et al, are available in the *Journal of Oral & Facial Pain and Headache, 2014*. The DC/TMD is intended for use in both clinical settings and applied research settings. Schiffman et al describe the rationale and methodology underlying the changes from the RDC/TMD to the DC/TMD. The extensive development process can be explored in

TMJ Research Diagnostic Criteria

- TMJ RCD
  - Arthritides
    - +/-pain OA, RA, Reactive
  - Myalgia
    - Muscle pain
  - Dysfunction
    - Internal derangements
    - +/-pain
    - +/- open closed locking
    - +/- crepitus, clicks

COMMENTARY | GUEST EDITORIAL

GUEST EDITORIAL

Temporomandibular disorders
A term past its time?

Continued use of the term "temporomandibular disorders" no longer can be defended in light of the many scientific advances that have been made in this field.

As G.K. Chesterton once wrote, "A man does not know what he is saying until he knows what he is not saying."[1] Use of the term "temporomandibular disorders" (TMD) clearly fits this description. Having been introduced into the literature as a means of solving a complex taxonomic dilemma, the term has been misused to the degree that it now has led to more confusion than clarification. Perhaps it is time to reconsider the value of maintaining this diagnostic classification. In making this decision, it is important to understand how we arrived at the current situation.

"TEMPOROMANDIBULAR DISORDERS": HISTORY OF THE TERM

The earliest descriptions of conditions involving the temporomandibular joint (TMJ) date back to the fifth century and were concerned with the diagnosis and treatment of dislocation. Subsequently, there were references to so-called fixations that were related to TMJ ankylosis and arthritis. Thus, initially, although all of the conditions affecting the TMJ were not fully understood, there was a disease concept of TMJ involvement.

Before the establishment of formal dental education in the late 1800s, physicians generally treated these conditions. However, in 1918, a key article by Prentiss[2] sparked some interest in this subject in the dental community. Prentiss wrote, "When the teeth are extracted the condyle is pulled upward by the powerful musculature and pressure on the meniscus results in atrophy." Subsequently, articles by Summa,[3] Monson,[4] Wright[5] and Goodfriend[6] emphasized the role of missing teeth, lost vertical dimension and the resulting displacement of the mandible as the cause of the symptoms these patients experienced.

However, it was in 1934 that the first of a series of articles by otolaryngologist James Costen[7] not only resulted in greater involvement of dentistry in the management of TMJ disorders and diseases, but also changed the entire diagnostic concept. In this article, Costen described a series of TMJ, ear and "sinus" symptoms that ultimately came to bear his name (Costen syndrome), which he attributed to nerve impingement from overclosure of the bite. Although it subsequently was shown that his anatomical explanations for the causes of the symptoms were incorrect,[8,9] his work still had a profound and long-lasting effect on the diagnosis and treatment of TMJ pain and dysfunction. As a result of his influence, there was a shift

Daniel M. Laskin, DDS, MS

Inflammatory Joint Pain – TMDs

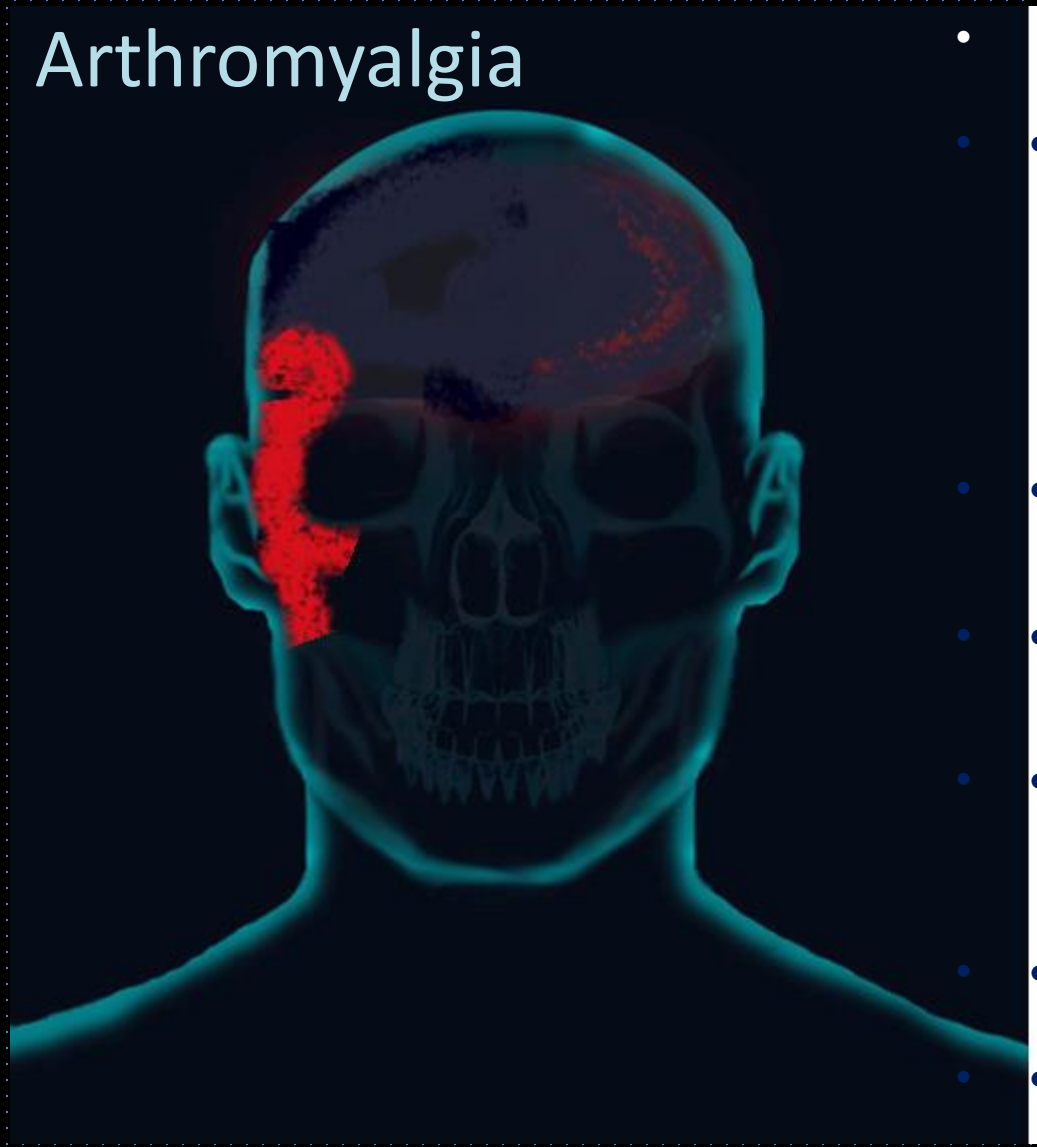
Temporomandibular disorders

Research Diagnostic Criteria

- **Arthomyalgic**
  - **Muscular and joint pain**
- **Dysfunctional**
  - **Clicking**
  - **Crepitus**
  - **locking**
- **Arthritides**
  - **+/- systemic arthritic conditions**

TMDs Temporomandibular disorders

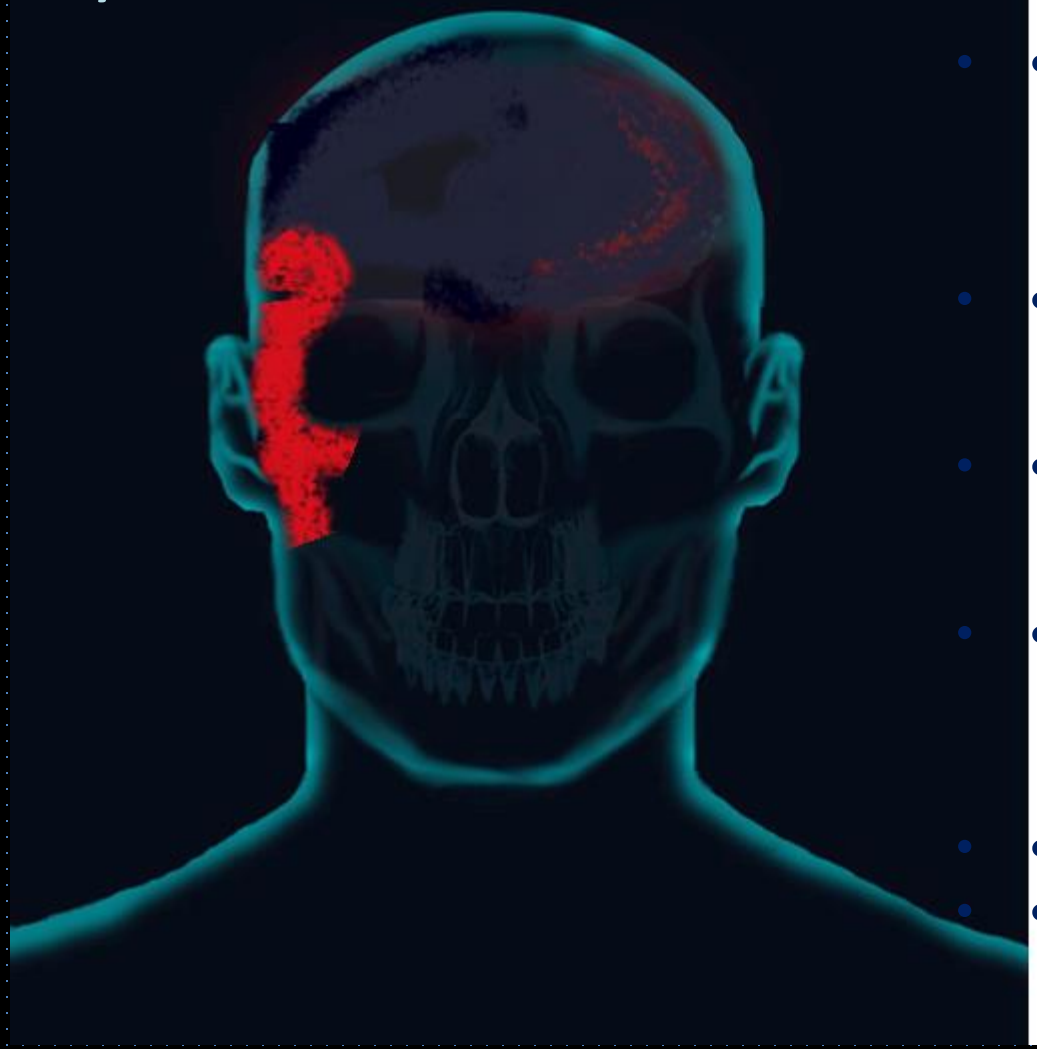
Arthromyalgia



- **S -site**
  - **Usually unilateral can be Bilateral**
- O -onset
  - Related to dental treatment
  - Chewing gum
  - Stress
  - Parafunctioal
  - Sponatnanous - Fibromylagia
  - **Female**
- C -character
  - **Dull throbbing ache**
  - **Worse on opening wide**
- R -radiation
  - Unilateral radiation cheek bones, temporal, auricular
- **A –** associations
  - Worse am with nocturnal bruxist habit
  - Worse end of day clenching or chewing gum habit
- T -timing
  - Intermittent or constant
- E -exacerbating & relieving factors
  - jaw function and opening dental Rx
  - Mporves with rest soft diet, BRA and NSAIDs or paracetamol
- S –severity Mild Moderate

TMDs Temporomandibular disorders

Dysfunctional

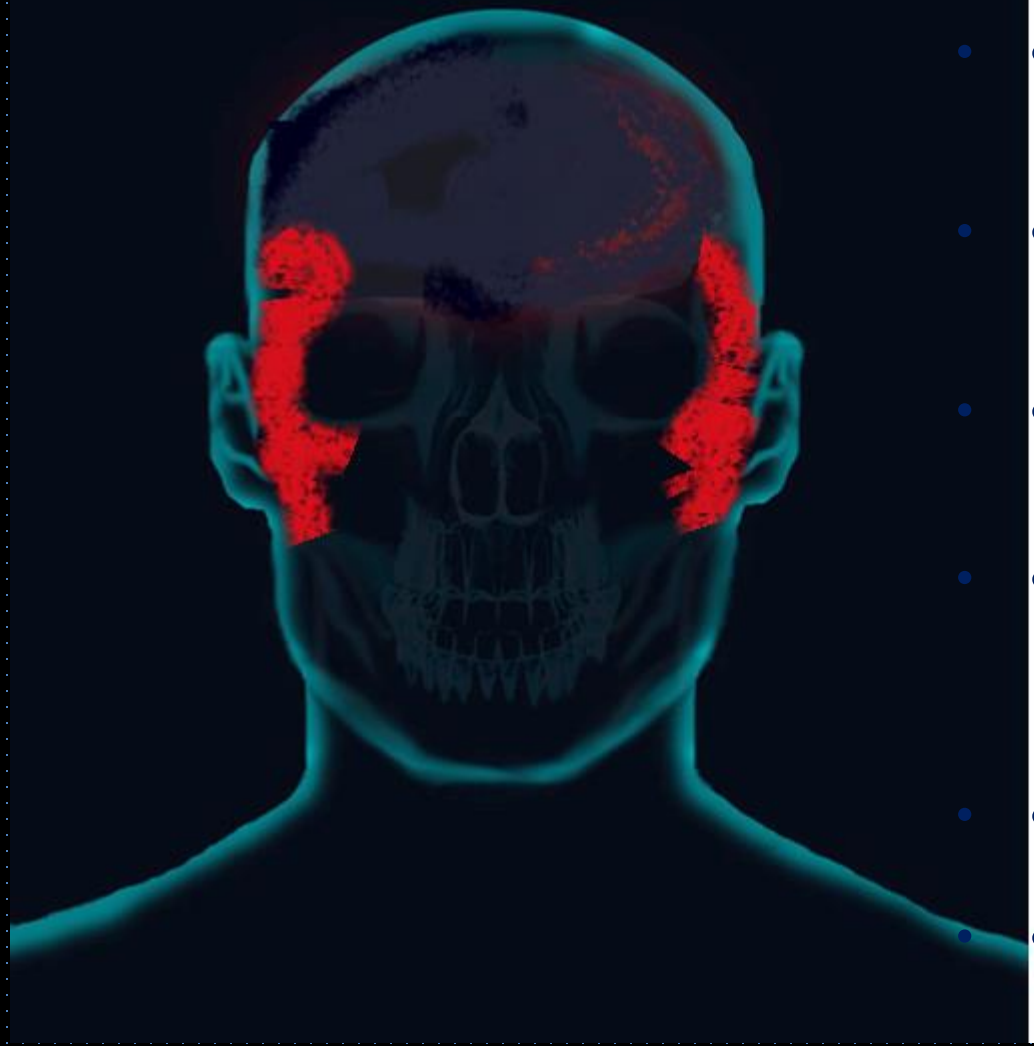


- **S -site**
  - **Usually unilateral can be Bilateral**
- O -onset
  - Opening wide
  - Eating, talking
  - **Female**
- C -character
  - Acute pain (Neuralgia) on locking
  - Open or closed locking
- R -radiation
  - Unilateral radiation cheek bones, temporal, auricular
- **A –** associations
  - Hyperflexia collagen disorders (Ehlos Danlos), Pregnancy, Recent trauma
- T -timing Intermittent
- E -exacerbating & relieving factors
  - jaw function and opening dental Rx
  - Improves with rest soft diet, BRA and NSAIDs or paracetamol
- S –severity Mild Moderate

Recurrent debilitating open or closed locking that requires hospital reduction under sedation or GA is the ONLY indication for TMJ surgery aside from Trauma or neoplasia

TMDs Temporomandibular disorders

Arthritides (RARE)



- **S -site**
  - **Usually Bilateral can be unilateral**
- O -onset
  - Spontaneous -
  - Female
- C -character
  - Dull throbbing ache
  - Worse on opening wide
- R -radiation
  - Unilateral radiation cheek bones, temporal, auricular
- **A –** associations
  - Recent Flu Reactive arthritis
  - Osteo arthritis
  - Rheumatoid/ Stills Rh in kids
- T -timing
  - Intermittent or constant
- E -exacerbating & relieving factors
  - jaw function and opening ental improves with NSAIDs or paracetamol
- S –severity Mild Moderate

TMD mimicking dental pain

- Referral of pain from masseter to maxillary and mandibular molar teeth

TMDs

Dysfunctional

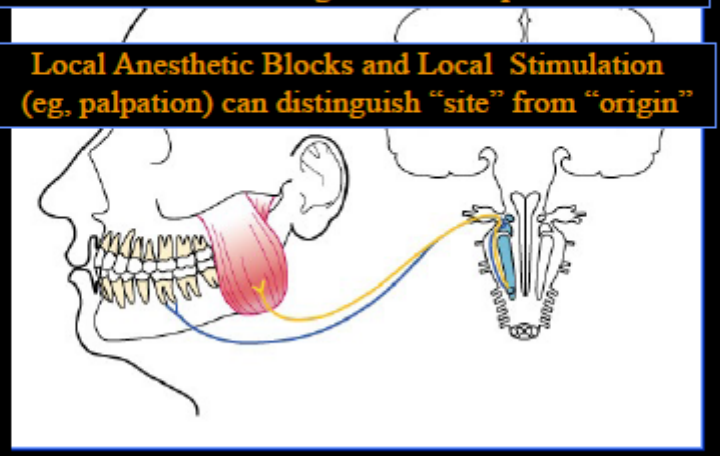
Arthitides

Arthromyalgia

Convergence & Referred Pain

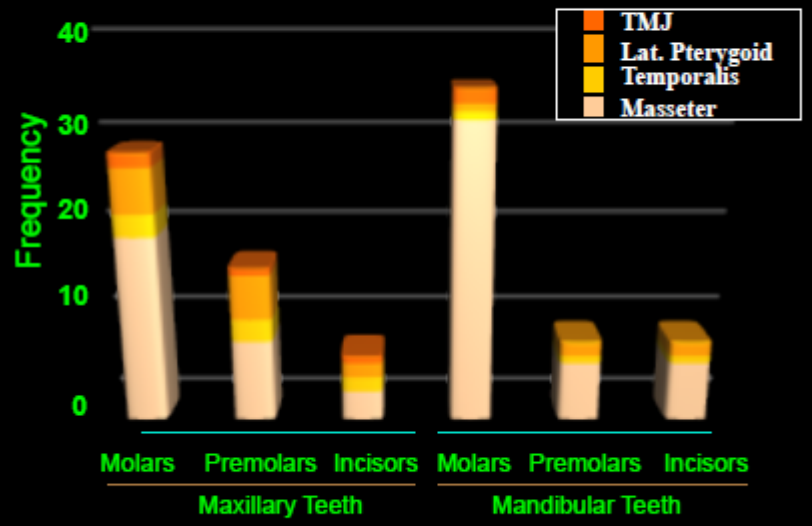
The "Site" of pain perception is Different from the "Origin" of nociceptor activation

Local Anesthetic Blocks and Local Stimulation (eg, palpation) can distinguish "site" from "origin"



Seltzer & Hargreaves, *in:* Seltzer & Bender's Dental Pulp, 2002

Frequency of Pain Referral to Teeth



Wright *JADA* 131:1307, 2000

N = 230

TMD Cochrane reviews

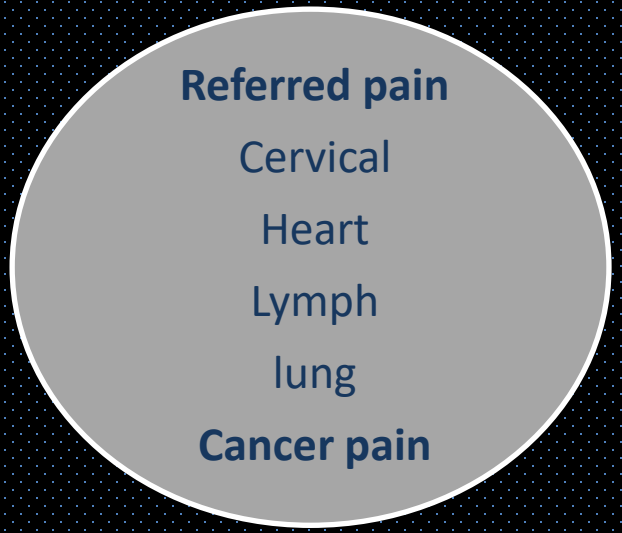
- **NO OCCLUSAL THERAPIES**
- **DENTAL INTERVENTION IS NOT INDICATED**
- Koh H Robinson PG Occlusal adjustment for treating and preventing temporomandibular joint disorders. J Evid Based Dent Pract. 2006.
- Al-Ani MZ Stabilisation splint therapy for temporomandibular pain dysfunction syndrome. Evid Based Dent. 2004;5(3):65-6.
- Koh H, Robinson PG Occlusal adjustment for treating and preventing temporomandibular joint disorders. J Oral Rehabil. 2004 Apr;31(4):287-92
- Bessa-Nogueira RV, Vasconcelos BC, Niederman R The methodological quality of systematic reviews comparing temporomandibular joint disorder surgical and non-surgical treatment. BMC Oral Health. 2008 Sep 26;8:27

NO EVIDENCE !

TMD or Salivary gland pain?

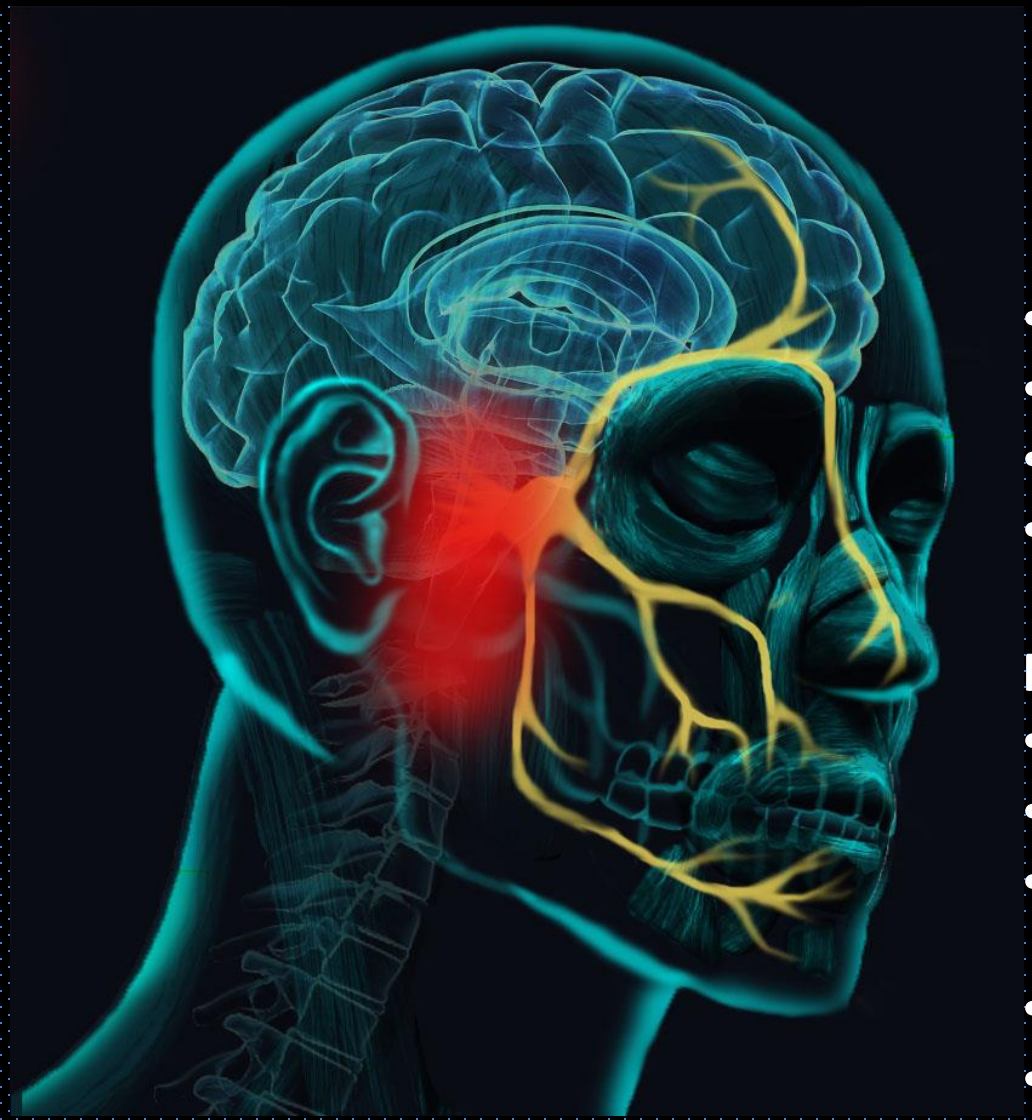
- • **S -site**
  - **Parotid**
  - **Submandibular Rarely sublingual**
  - **Obstructive disease unilateral**
  - **Systemic Mumps, HIV, CMV Bilateral**
- • O -onset
  - During meals for obstructive ‘
  - **Meal Time syndrome’**
- • C -character
  - Dull throbbing ache
  - Acute on eating and slaivation
- • R -radiation
  - Unilateral radiation cheek bones, temporal, auricular
- • **A –** associations
  - Meal time syndrome for obstructive
- • T -timing
  - Intermittent or constant
- • E -exacerbating & relieving factors
  - Eating
  - mproves with NSAIDs or paracetamol
- • S –severity Mild Moderate

Exclude referred pain



- Elephant in the room
- Any spontaneous sudden onset pain =/- neuropathy (motor or sensory)
- Is Neoplasia until otherwise proven

Referred pain?



- **Tonsillar CA Pain?**
- **Cardiac Angina?**
- **Cervical pain?**
- **Migraine?**

Remember your red flags

- Spontaneous neuropathy
- Trismus
- Persistent lymph node or swelling or ulcer (painless)
- Patient prior Ca else where
- Patient over 50 years
- Unexplained aneamia

Red flags of malignancy

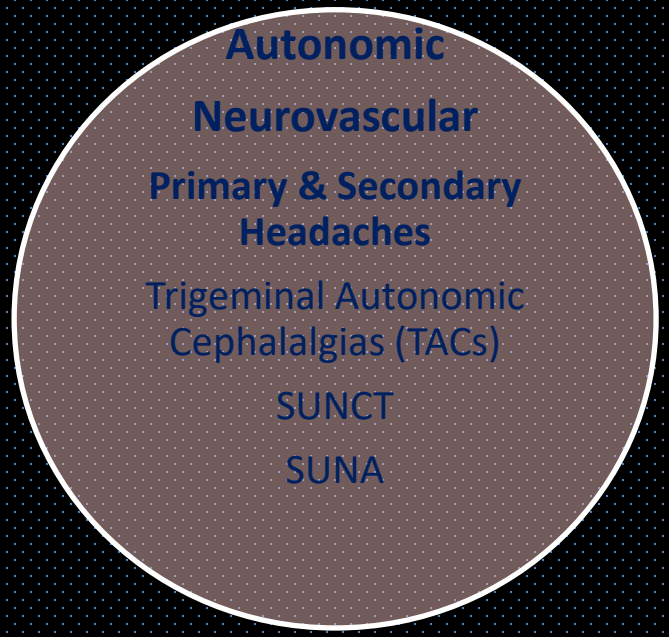
- Over 50 years
- Previous history of Carcinoma
- Smoking /alcohol/ Betel nut/ Pan
- Night fevers
- Weight loss
- Blood loss/ aneamia

- 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

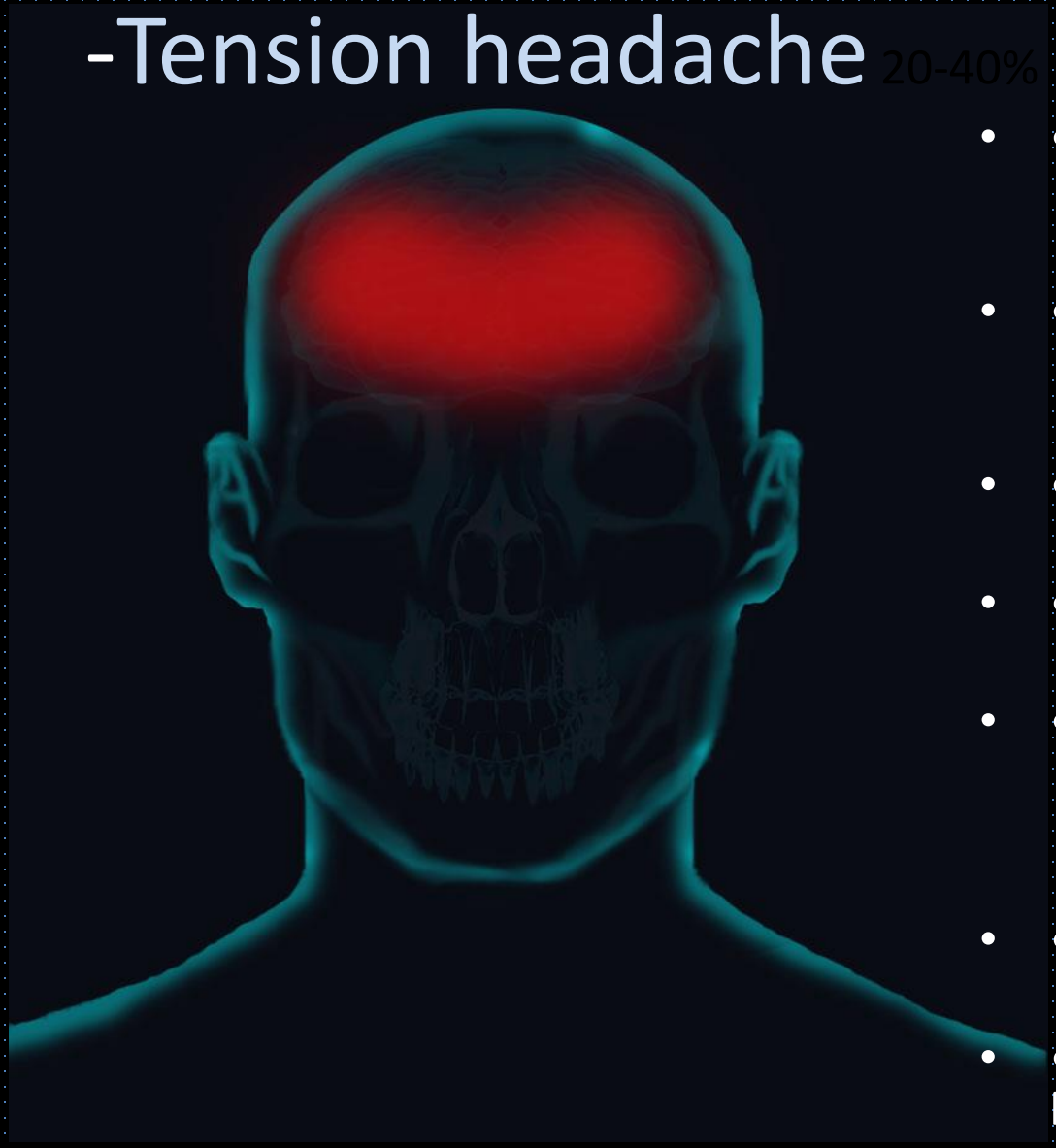
Neuropathic pain + Autonomic signs

- Headaches
  - Primary
  - Secondary
- Trigeminal autonomic cephalagias
- Giant cell arteritis



Neurovascular causes of facial pain

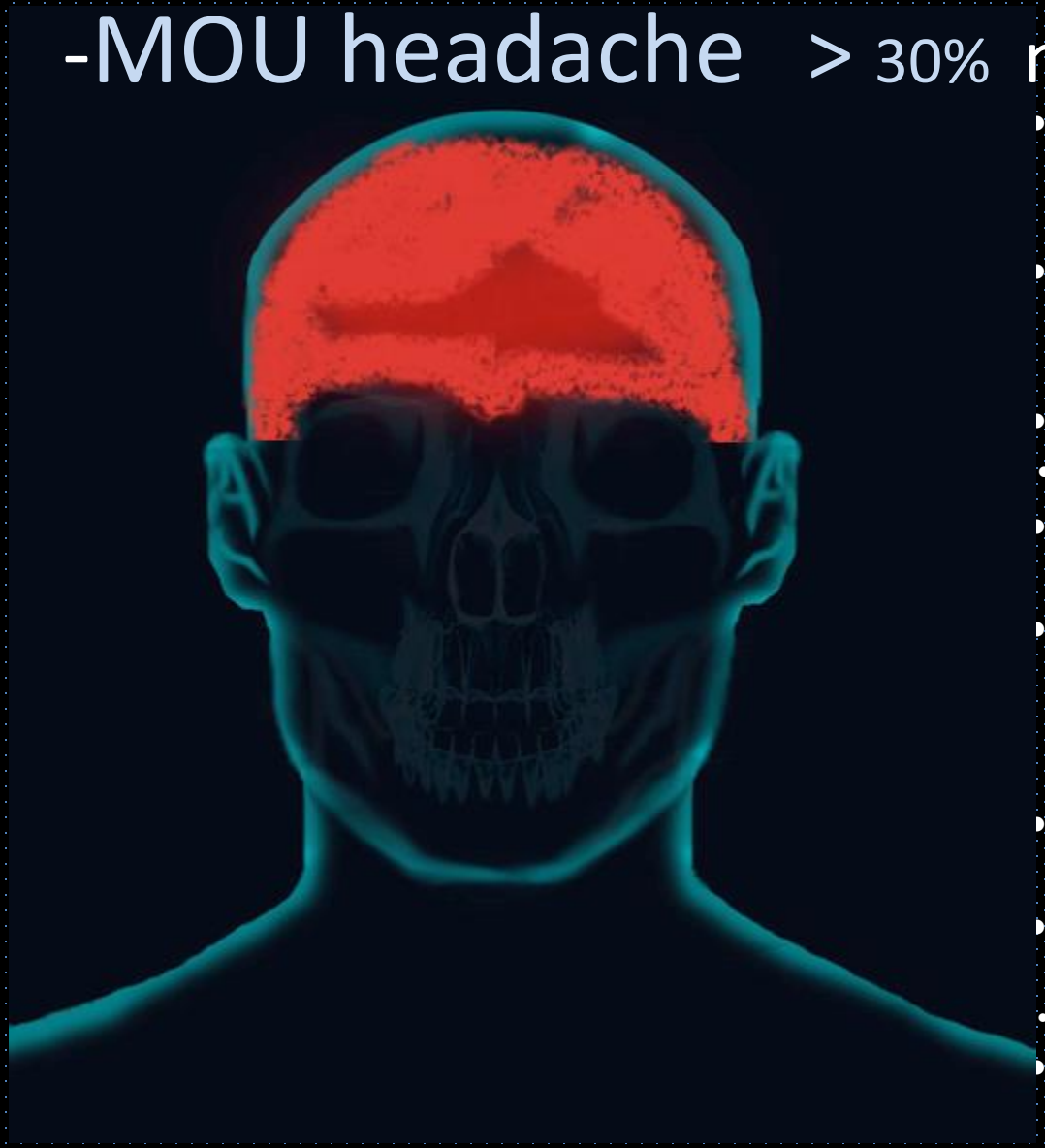
-Tension headache 20-40%



- • **S -site**
  - **Usually bilateral**
  - **Frontal, Temporal**
- • O -onset
  - • stress, tension, tiredness
  - **Femaile**
- • C -character
  - **Mild moderate dull throbbing**
- • R -radiation
  - Orbital sinuses
- • **A –** associations
  - Relieved with NSAIDs paracetamol and rest
  - Doesn’t happen on holiday
- • T -timing
  - Daily worse end of day
- • E -exacerbating & relieving factors NSAIDs paracetamol
- • S –severity Mild Moderate

Neurovascular causes of facial pain

-MOU headache > 30% medication overuse

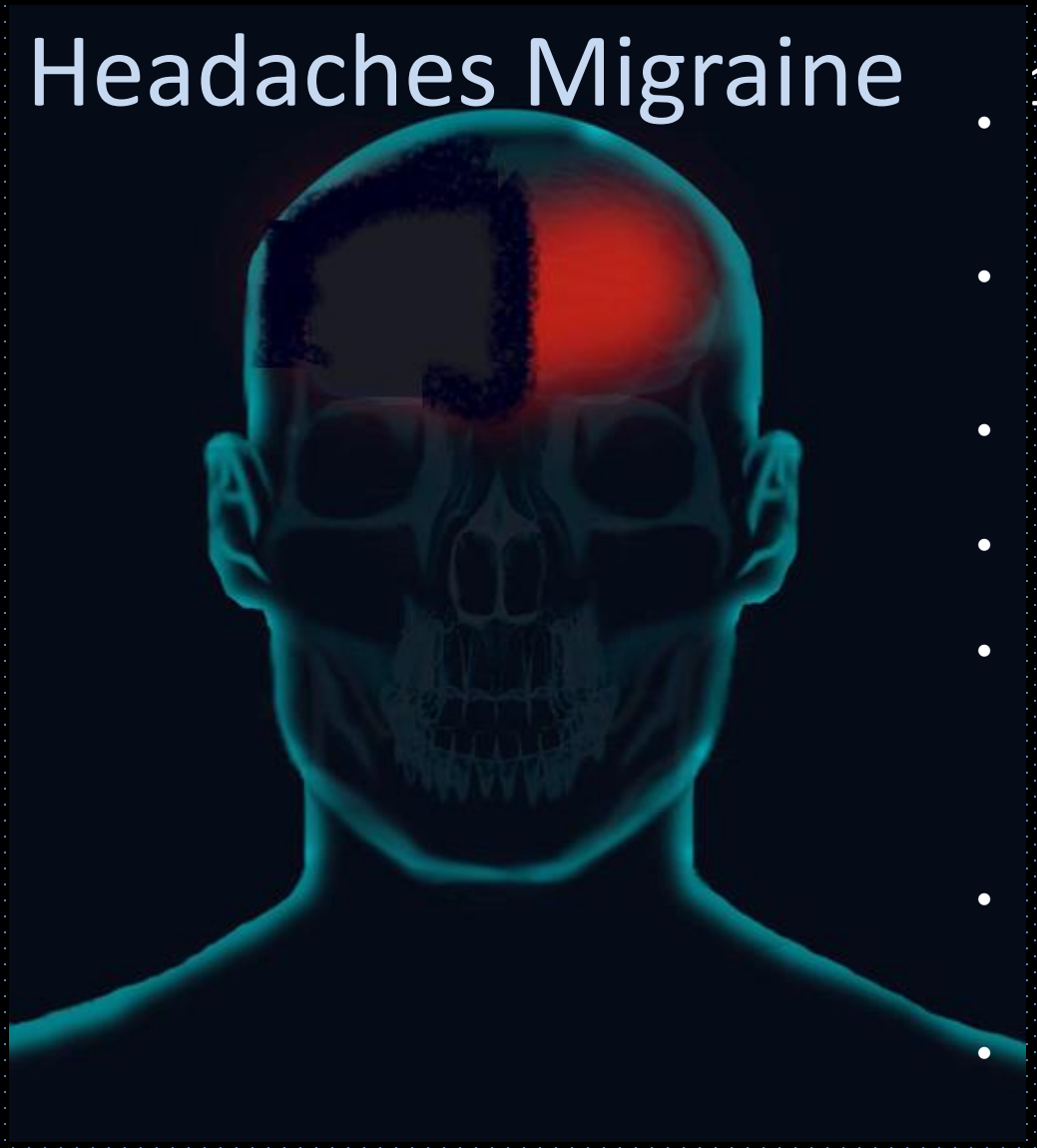


- **S -site**
  - **Usually bilateral**
  - **Frontal, Temporal Occipital, Orbital**
- O -onset
  - Daily constant
  - **Female**
- C -character
  - **Mild moderate dull throbbing**
- R -radiation
  - All over
- **A –** associations
  - Relieved with NSAIDs paracetamol and rest
  - Doesn't happen on holiday
  - **Long history of daily OTC analgesics**
- T -timing
  - Daily worse end of day
- E -exacerbating & relieving factors
  - NSAIDs paracetamol
  - stress, tension, tiredness
- S –severity Mild

- **Previously were Tension or Migraine headaches**

Neurovascular causes of facial pain

Headaches Migraine



Onset related to head injury?

10-20%

- **S -site**
  - **Usually unilateral V1**
  - **CAN be V2 and V3**
- O -onset
  - Spontanous age puberty-40yrs
  - **Female**
- C -character
  - Moderate dull throbbing
- R -radiation
  - Localise V1 temporal occipital
- **A – associations**
  - **+/ - Aura Visual, nausea, photophobia**
  - **Neuropathy –hypersensitivty**
  - **Paralysis rare**
  - **Eostrogen depletion**
- T -timing
  - Monthly
  - Less than 13 days a month
- E -exacerbating & relieving factors dehydrtion stress tiredness
- Relief hydration, rest. MSAIDs, tryptans
- S –moderate –severe **impactful needs dark quiet room often miss work**

Migraine many patients present with facial dental / sinus pain

Migraine mimicking

Onset related to head injury?

Ongoing toothache can exacerbate headaches

- **S -site**
  - **Usually unilateral V1**
  - **CAN be V2 and V3**
- O -onset
  - Spontanous age puberty-40yrs
  - **Female**
- C -character
  - Moderate dull throbbing
- R -radiation
  - Localise V1 temporal occipital
- **A – associations**
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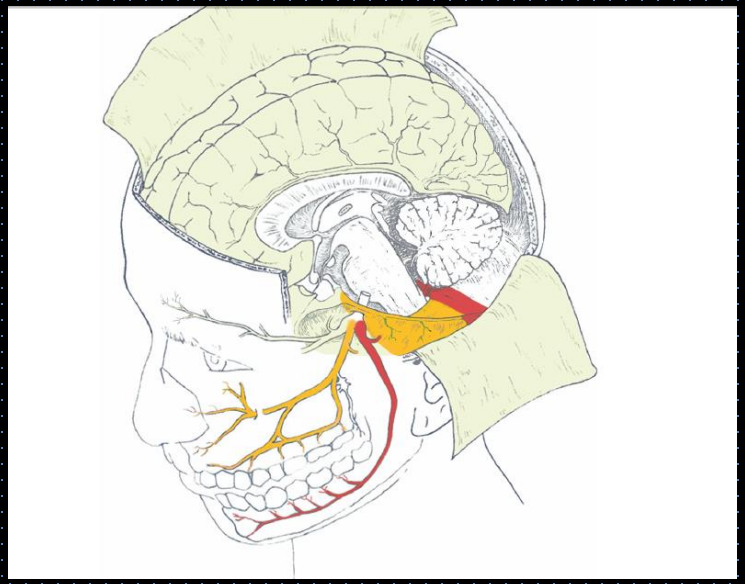
- T -timing
  - Monthly
  - Less than 13 days a month
- E -exacerbating & relieving factors dehydrtion stress tiredness
- Relief hydration, rest. MSAIDs, tryptans
- S –**impactful needs dark quiet room often miss work**

Conditions that can mimic toothache

- Migraine



Autonomic

Neurovascular

Primary & Secondary Headaches

Trigeminal Autonomic Cephalalgias (TACs)

SUNCT

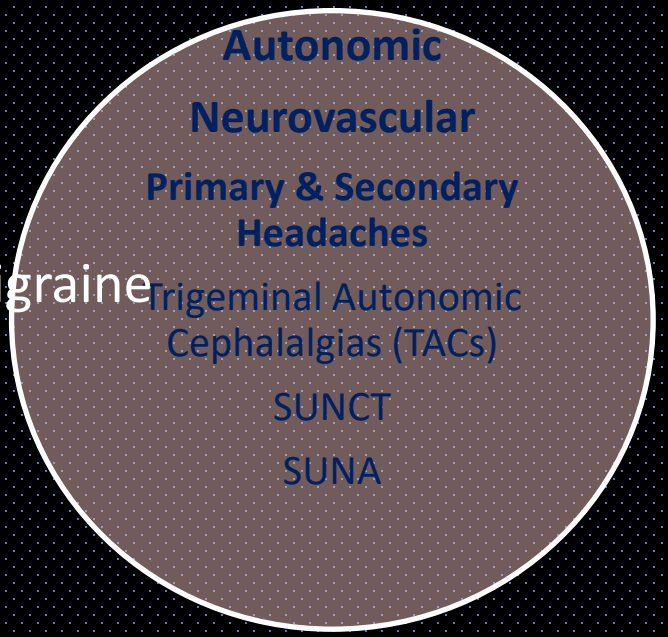
SUNA

Case Series of Four Different Headache Types Presenting as Tooth Pain

Aurelio A. Alonso, DDS and Donald R. Nixdorf, DDS, MS**

Migraine OR toothache

- Migraine headache sites usually temporal, supraorbital, frontal, retrobulbar, parietal, auricular and occipital. However may occur in **malar region and upper and lower teeth** base of nose and median wall of orbit and neck….
  - **Harold G Wolff (1963)**
- Diagnostic difficulty The Sinus Allergy ad Migraine study (SAMS)
  - 1.6% maxillary unilateral
  - 1.6% bilateral maxillary
  - V2 and v3 unilateral 3.2%
  - *Eross E Dodick DO and Eross M Headache 2007*
- Prevalence of facial pain in migraine
  - *Yoon M-S Cephalgia;2009: (30)92-96*
- 88% of people self or physician diagnosed sinusitis had migraine
  - *Lipton et al Headache 2001*
  - *Schreiber et al Arch Int Med 2004*



Trigeminal autonomic cephalalgias
Familiarise yourself with TAC conditions

Autonomic

Neurovascular

Primary & Secondary Headaches

Trigeminal Autonomic Cephalalgias (TACs)

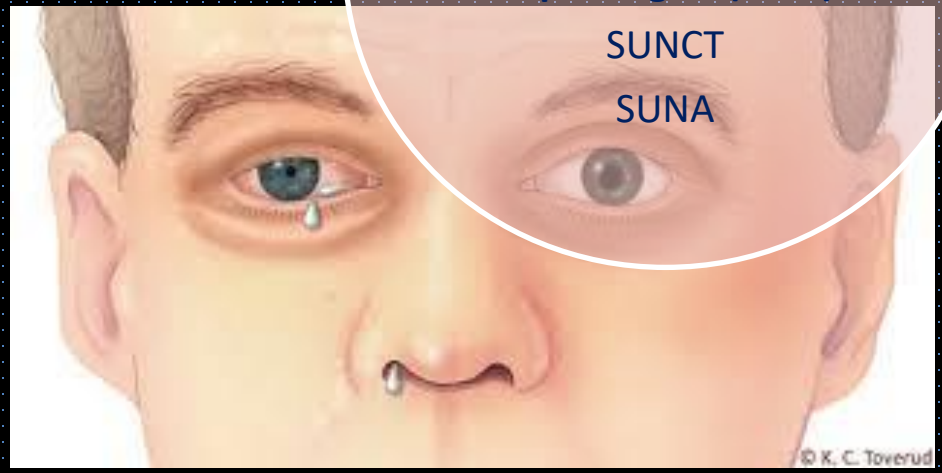
SUNCT

SUNA

Patient seen by our neurology team

The most common diagnosis made in the Clinic was TACs (42 %), followed by migraine (34 %) and post-traumatic trigeminal neuropathy (11%).

TACs are the most common diagnosis made in our Multidisciplinary Orofacial Pain clinic. The data stress the importance of a multidisciplinary team approach to seeing these complex patients.



V2 Migraine and TACs are probably most 'persistent idiopathic facial pains'

Trigeminal autonomic cephalalgias

- Cluster headache
- Sudden Onset unilateral conjunctival irritation (SUNCT)
- Hemicranial continua
- Paroxysmal Hemicrania

Differential Diagnosis TACs CH/ SUNCT/PH

	Cluster headache	Paroxysmal hemicrania	SUNCT
Sex F: M	1:2.5	1:1	1:2
Pain:			
Type	Stabbing, boring	Throbbing, boring, stabbing	Burning, stabbing, sharp
Severity	Excruciating	Excruciating	Excruciating
Site	Orbit, temple	Orbit, temple	Periorbital
Attack frequency	1/alternate day –8/day	1–40/day (>5/day for more than half the time)	3–200/day
Duration of attack	15–180 min	2–30 min	5–240 s
Autonomic features	Yes	Yes	Yes
Migrainous features	Yes	Yes	Very rarely
Alcohol trigger	Yes	Occasional	No
Cutaneous triggers	No	No	Yes
Indometacin effect	-	++	-
Abortive treatment	Sumatriptan injection or nasal spray Oxygen	Nil	Nil
Prophylactic treatment	Verapamil Lithium Topiramate	Indometacin	Lamotrigine Topiramate Gabapentin

Autonomic

Neurovascular

Primary & Secondary Headaches

Trigeminal Autonomic Cephalalgias (TACs)

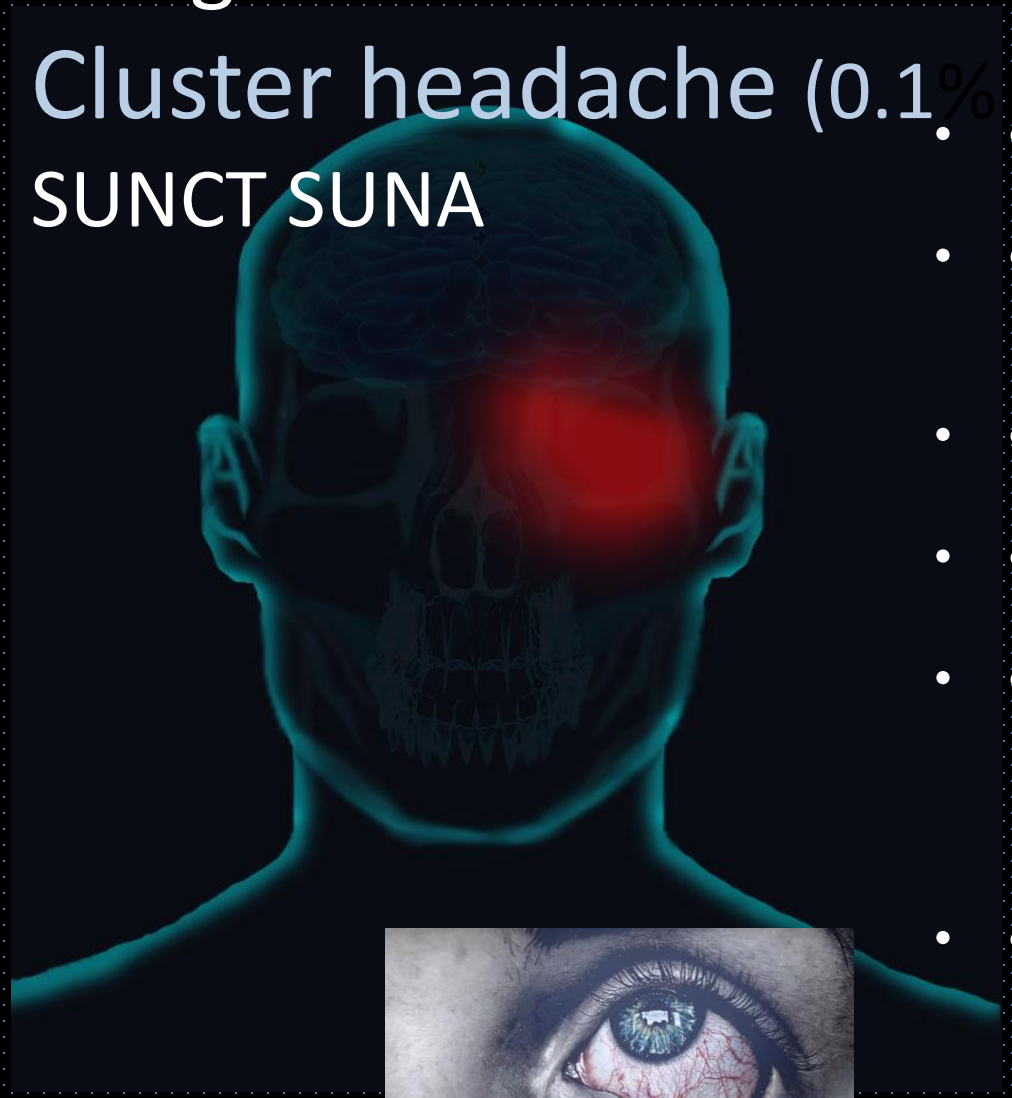
SUNCT

SUNA

Trigeminal Autonomic Cephalalgia – TAC

Cluster headache (0.1% = suicidal headache

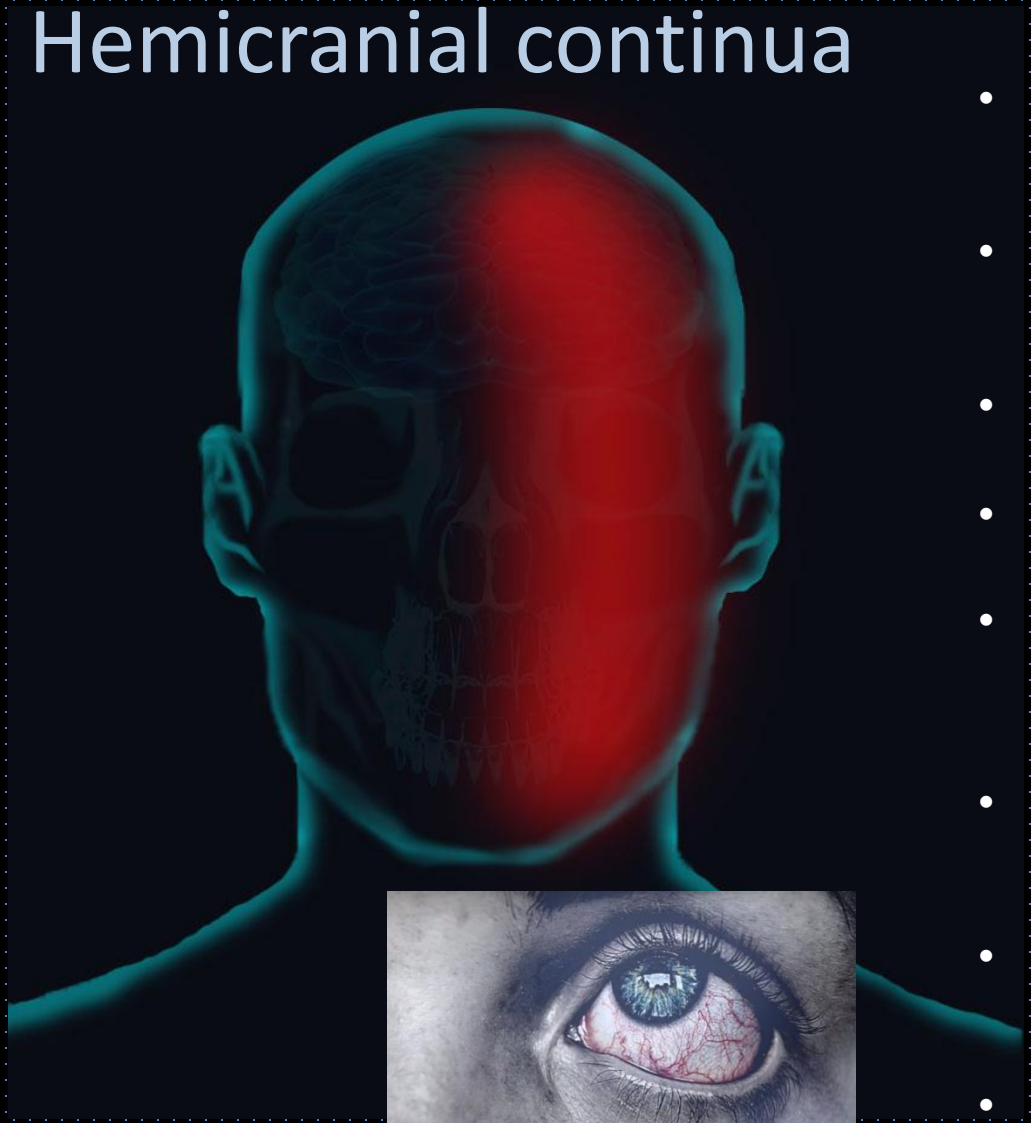
SUNCT SUNA



- **S -site**
  - **Orbit – V1**
- O -onset
  - Spontaneous age 20-40yrs
  - **Male**
- C -character
  - severe neuralgic
- R -radiation
  - Localise orbit and V1
- **A – associations**
  - **<u>Autonomic signs</u>, parasympathetics. Face sweating, redness, conjunctval irritation, nasal congestion, meiosis, ptosis**
- T -timing
  - seasonal 6-8 weeks one attack-18 per day last 3-20 minutes
- E -exacerbating & relieving factors exercise, alcohol, GTN
- Relief oxygen
- S –**severity SUICIDAL**

Trigeminal Autonomic Cephalalgia – TAC

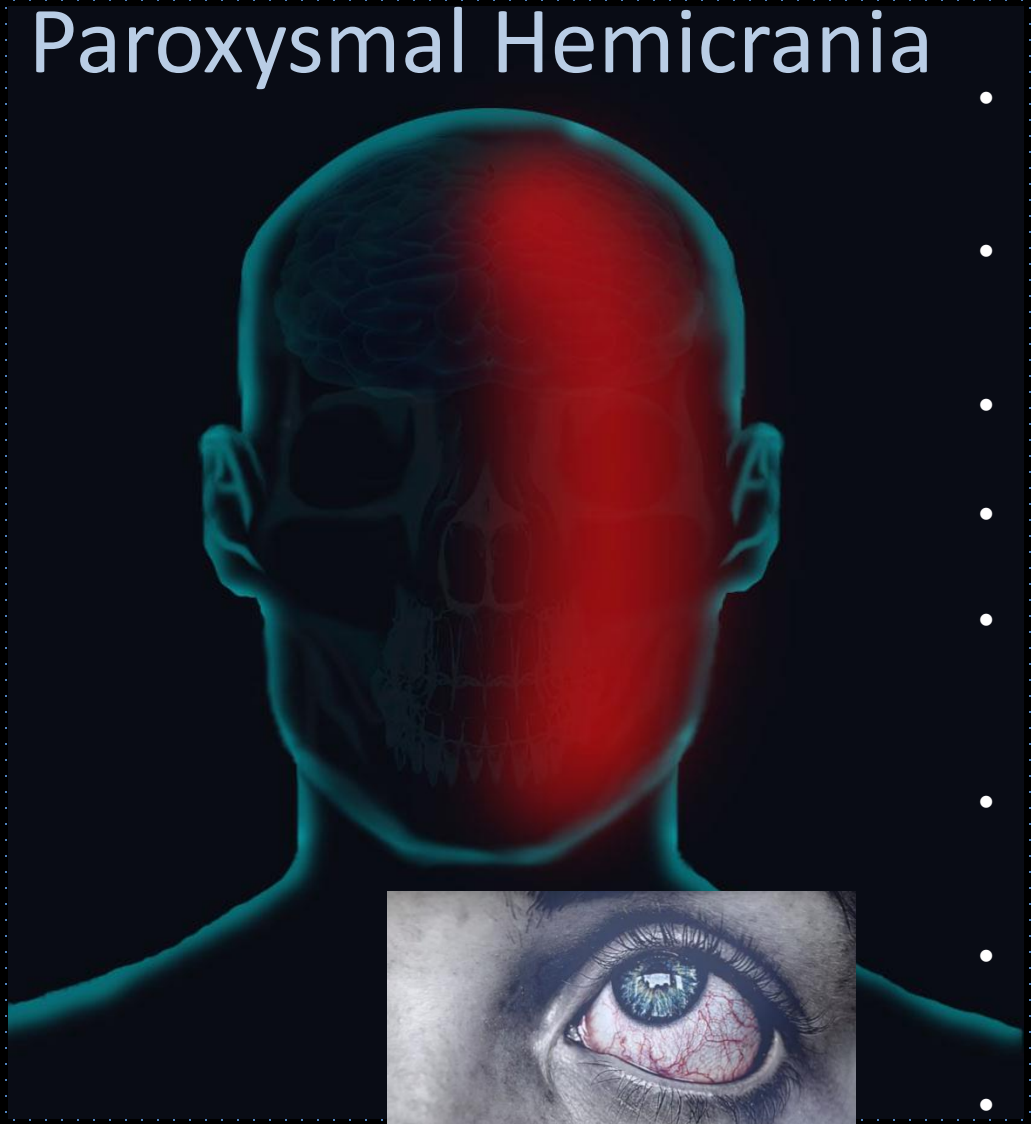
Hemicranial continua



- **S -site**
  - **Usually unilateral**
  - **Temporal occipital**
- O -onset
  - Spontaneous age 20-40yrs
  - **Male**
- C -character
  - **severe neuralgic**
- R -radiation
  - Unilateral radiation
- **A – associations**
  - **Autonomic signs, parasympathetics. Face sweating, redness, conjunctval irritation, nasal congestion, meiosis, ptosis**
- T -timing
  - Constantly present may fluctuate
- E -exacerbating & relieving factors
- exacerbating & relieving factors exercise, alcohol, GTN
- Relief oxygen
- S –SUICIDAL severe **impactful**

Trigeminal Autonomic Cephalalgia – TAC

Paroxysmal Hemicrania



- **S -site**
  - **Usually unilateral**
  - **Temporal occipital**
- O -onset
  - Spontaneous age 20-40yrs
  - **Male**
- C -character
  - **severe neuralgic**
- R -radiation
  - Unilateral radiation
- **A –** associations
  - **Autonomic signs, parasympathetics. Face sweating, redness, conjunctval irritation, nasal congestion, meiosis, ptosis**
- T -timing
  - Intermittent
  - Night time onset
- E -exacerbating & relieving factors

exacerbating & relieving factors
exercise, alcohol, GTN

- Relief oxygen
- S –SUICIDAL severe **impactful**

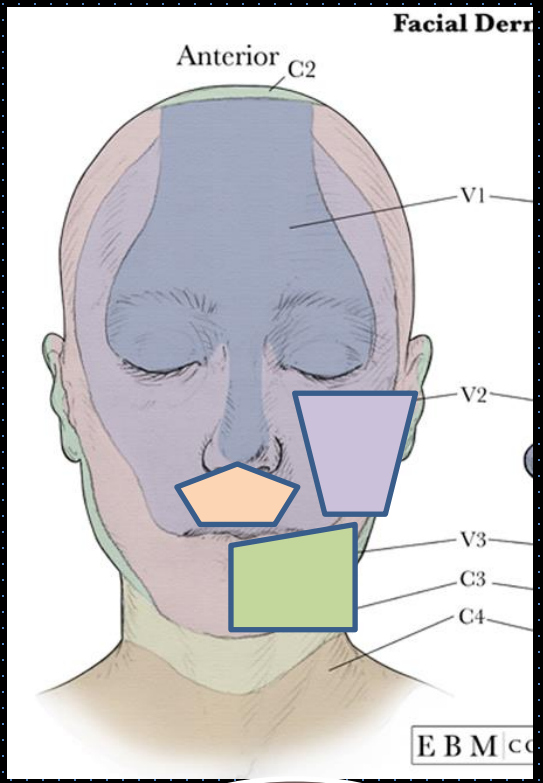
TAC OR toothache?

- Patients with TACs consult;
  - 34-45% dentists
  - 27-33% ENT
  - > 15% of PH patients have pain similar to dental pain
- Average 4.3 physicians consulted
- 4% have sinus surgery
- Most common misdiagnosis
  - Migraine 45%
  - TTH 16%
  - Sinusitis 23%
  - Dental 23%
  - TN 16%
  - Klapper et al 2000; van Viet et al 2003; Bahca and Goadsby 2004; Larner al 2009
  - 2008; vanAlboom etal 2009

- Regions of orofacial region most affected by Neurovascular pain

Premaxillla 30%, V2 17%, V3 31%

- Age of onset 21 years and 51 years
- Duration 9-16 hours
  - Benoliel R et al Cephalalgia 2008



Autonomic

Neurovascular

Primary & Secondary Headaches

Trigeminal Autonomic Cephalalgias (TACs)

SUNCT

SUNA

Strange pain like toothache? Treatment not working?

- Exclude migrainous symptoms
  - Nausea
  - Vertigo
  - Cold and touch sensitivity
  - Photo phobia
  - Phono phobia
  - Aura

Behaviour…retire to dark room and lie down

TREAT Migraine

- Exclude autonomic symptoms
  - Red eye conjunctival irritation
  - Tearing
  - Nasal congestion
  - Facial flushing
  - Drooping eyelid (Ptosis)
  - Enlarged pupil (Meiosis)

Behaviour…aggressive irritated restless

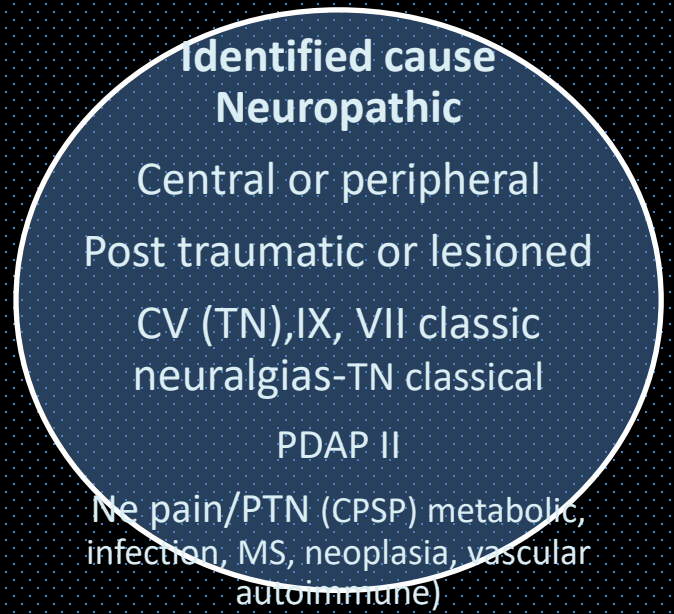
TREAT TAC

Secondary Neuropathic pain

- Central
  - TN classical or non classical
    - vascular compromise, MS. SOL
    - Stroke, IC bleed
- Peripheral
  - Disease / Lesioned

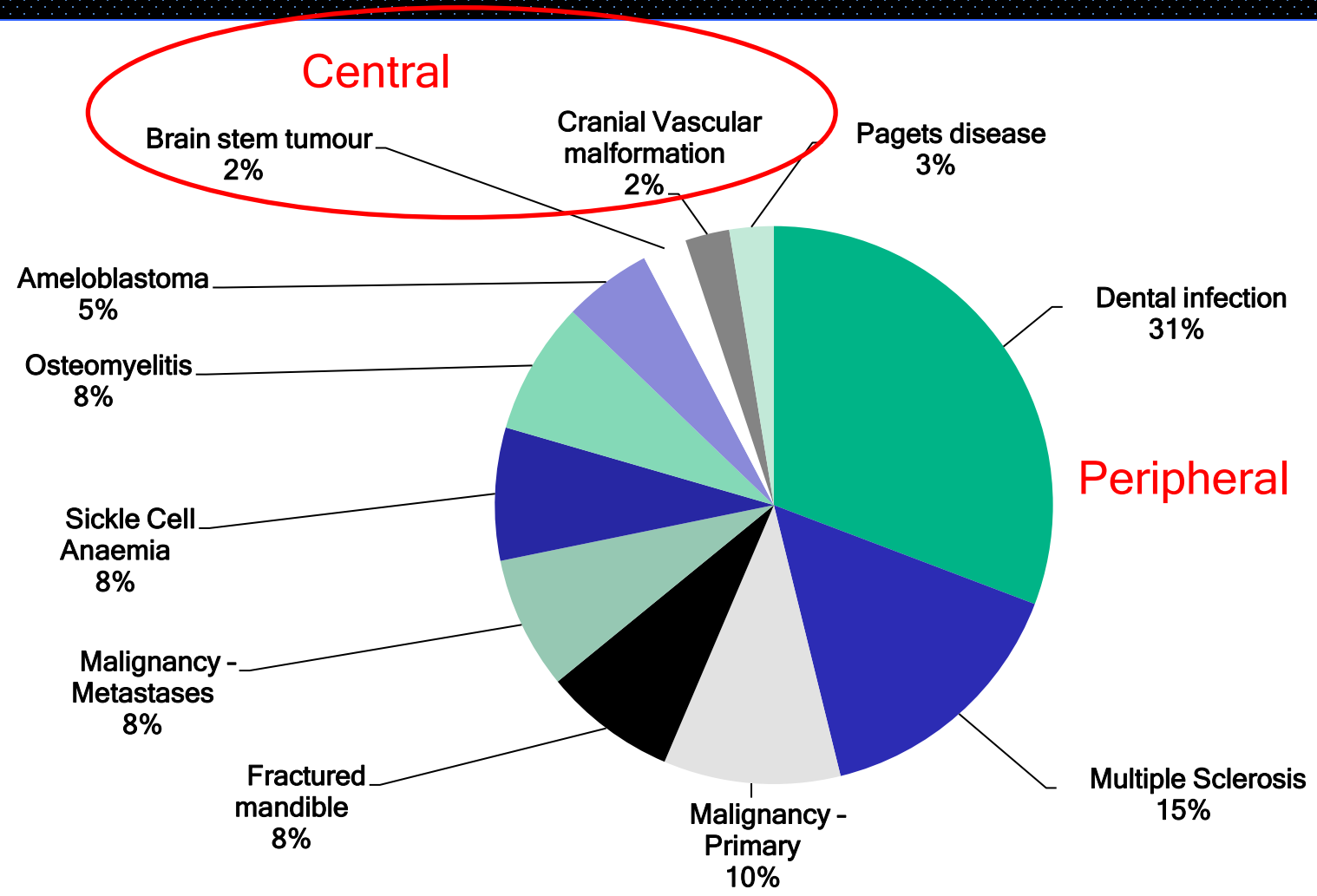
    OR

  - Traumatic
    - *Chemical*
    - *Thermal*
    - *Radiation*
    - *Mechanical trauma*
      - *Chronic post surgical pain, Phantom limb pain, spinal cord injury, trauma and postoperative neuropathic pain,*
      - *Post Traumatic pain Complex regional pain syndromes (CRPSs) (principally type 1)*



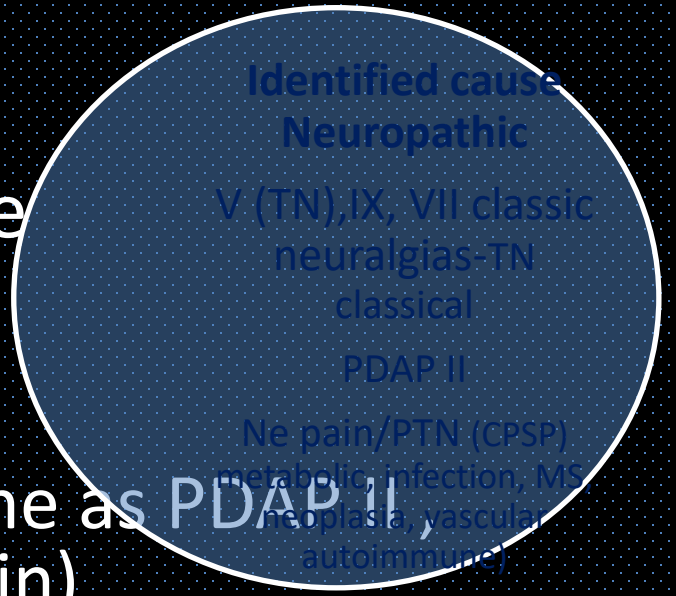
Trigeminal neuropathy

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

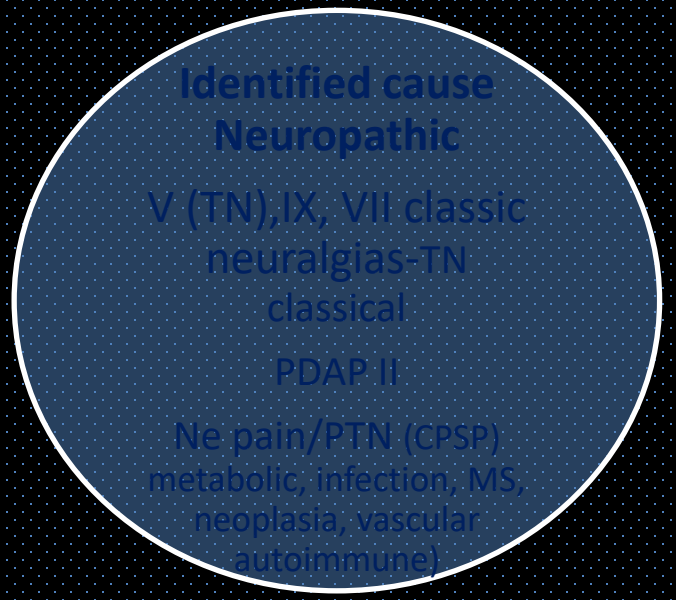
Secondary neuropathic pain
Demonstrable lesion on the nerve



Peripheral

- Post traumatic neuropathy (Same as PDAP II , PPTTN, Chronic post surgical pain)
- V (TN),IX, VII classic neuralgias
  - Mechanical Trauma, surgery
  - Chemo therapy
  - Radiation
  - Thermal
- Non traumatic lesional
  - Burning mouth DISORDER

Secondary neuropathic pain peripheral lesional



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

Chronic post surgical pain?

- Chronic pain after surgery is a well recognised problem and affects upwards of 20-30% of patients undergoing limb amputation, thoracotomy and breast surgery.
- There is confusing nomenclature for surgical induced pain without identifiable neuropathy and nerve damage these include:

Surgically induced neuropathic pain	**SNPP**
Chronic post surgical pain	**CPSP**
Post traumatic neuropathy	**PTN**
Postoperative neuropathic pain	**PPNP**
Phantom limb pain	**PLP**

- Over the last 10 years it has become evident that significant numbers of patients suffer from chronic pain as a result of routine surgery with over 30-40% of patients presenting in chronic pain clinics being diagnosed with CPSP. Macrae (2008) suggested a definition including;

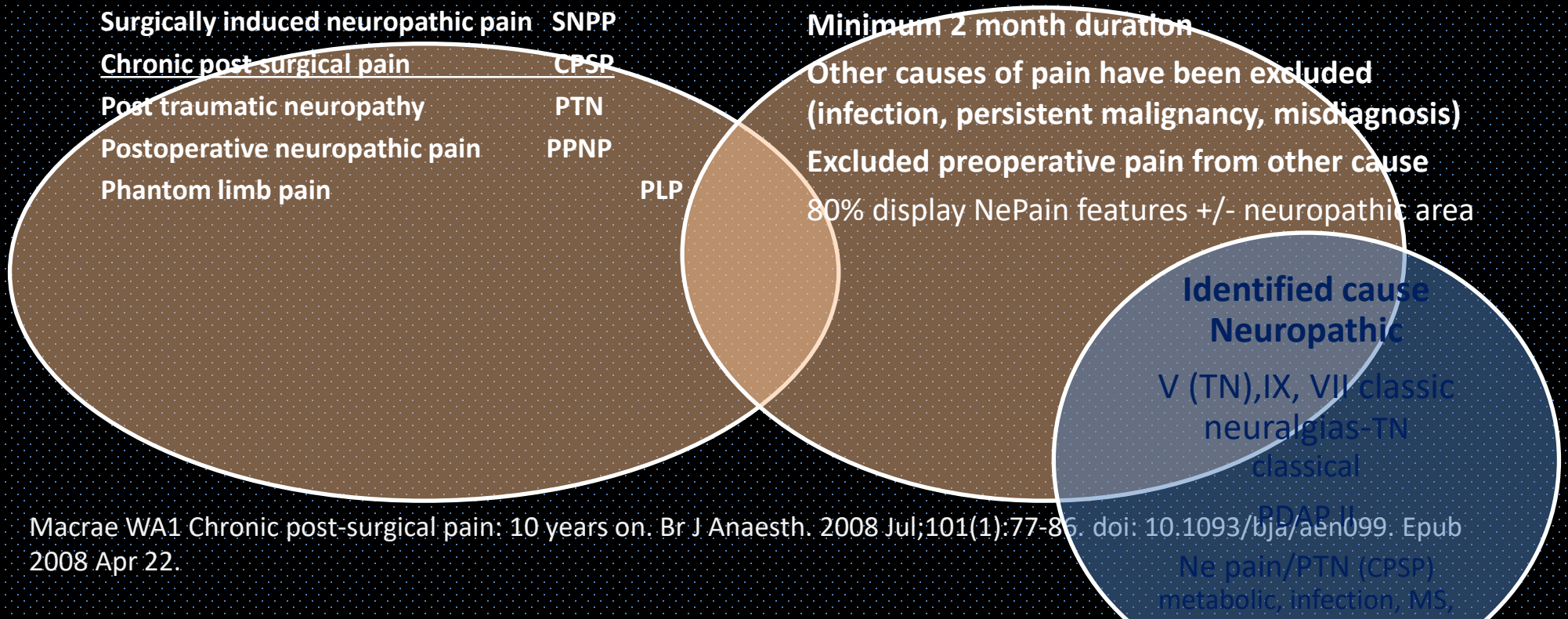
Pain developed after surgery

Minimum 2 month duration

Other causes of pain have been excluded (infection, persistent malignancy, misdiagnosis)

Excluded preoperative pain from other cause

80% display NePain features +/- neuropathic area



Macrae WA1 Chronic post-surgical pain: 10 years on. Br J Anaesth. 2008 Jul;101(1):77-86. doi: 10.1093/bja/aen099. Epub 2008 Apr 22.

CPSP after Dentistry

PAIN UPDATE

Persistent pain after dental implant placement

A case of implant-related nerve injury

Robert Delcanho, BDSc, MS, Cert Orofacial Pain, FFPMANZCA, FICD; Elizabeth Moncada, DDS, MS, Cert Orofacial Pain

CLINICAL PROBLEM

A 48-year-old woman visited one of us (R.D.) for a second opinion regarding severe pain that began six weeks earlier after placement of an implant into her mandibular right first premolar region (Figure). She recalled that profuse bleeding had to be controlled when the implant site was prepared, and she experienced sharp pain as the implant was being inserted. As the local anesthetic wore off, she developed severe deep aching and burning pain at the implant site. She then experienced sharp stabbing pains whenever she touched or brushed the area around the implant. Also, her right lower lip felt unpleasant, which caused difficulty in drinking and affected kissing. The patient reported that she had been pain free before the implant procedure.

The patient returned to the dentist who had placed the implant. A radiograph revealed that the implant was not impinging on the inferior alveolar canal (IAC). Initial treatments included a course of antibiotics, a combination of analgesics and anti-inflammatory drugs. A moderate degree of pain relief was achieved, and the dentist advised her to wait and see if the pain resolved before possibly referring her to a neurologist or pain specialist.

The patient's medical history was significant for depression, anxiety, insomnia, irritable bowel syndrome

lasting. The inflammatory process is a complex biologi response to tissue injury by normally functioning vascu lar and somatosensory nervous systems. It is a protecti response intended to eliminate the initial cause of the injury and to foster healing and repair of the injured pa By contrast, NP is "caused by a lesion or disease of the somatosensory nervous system."[2] Because it is not feasi clinically to determine the degree of nerve contusion o injury or the extent of ongoing inflammation in the su cal area, clinicians should presume that both nociceptiv and neuropathic factors are present. Authors of previou Pain Updates in The Journal of the American Dental Association (JADA) have reviewed NP,[3–6] which is char acterized by its burning, prickling, electrical and sharp nature. NP can be spontaneous or evoked, with distinc associated positive (heightened sensation) signs, negati (sensory deficit) signs or both. There almost always is a area of abnormal sensation (Table 1, page 1270).

The incidence of nerve injury after dental surgical procedures, including third-molar extractions and pla ment of implants, is higher than that commonly be- lieved (possibly up to 40 percent), and, for the latter, t incidence is increasing.[7–13] The term "peripheral painf traumatic trigeminal neuropathy" (PPTTN) has been proposed for NP that occurs within three months of s procedures.[14] In the mandible, NP after dental implan placement may occur without evidence of direct impl intrusion into the IAC. The course of the IAC within t mandible is variable, so furcations and small branches the inferior alveolar nerve (IAN) outside the main can may have been traumatized. To further minimize the

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Persistent Pain after Dental Surgery

Tara Renton, BDS MDSc PhD FDS RCS FRACDS (OMS) ILTM, Professor in Oral Surgery[1]

Journal List › Rev Pain › v.5(1); 2011 Mar › PMC4590080

REVIEWS IN PAIN

Rev Pain. 2011 Mar; 5(1): 8–17. PMCID: PMC4590080
doi: 10.1177/204946371100500103

Persistent Pain after Dental Surgery

Tara Renton, BDS MDSc PhD FDS RCS FRACDS (OMS) ILTM

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Abstract

- This article aims to cover post surgical trigeminal neuropathy and other conditions related to ... trigeminal pain not specifically covered elsewhere in this series.
- Is estimated to occur in 4–5% of patients overall, considerably le ... compared with other site surgeries.
- Due to the high volume surgery undertaken in this region chronic post surgical pain remains common.
- Relatively few clinicians are aware of this condition and as a resu... it is frequently poorly managed.

Introduction

Rev Pain

Identified cause Neuropathic

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

PDAP II

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

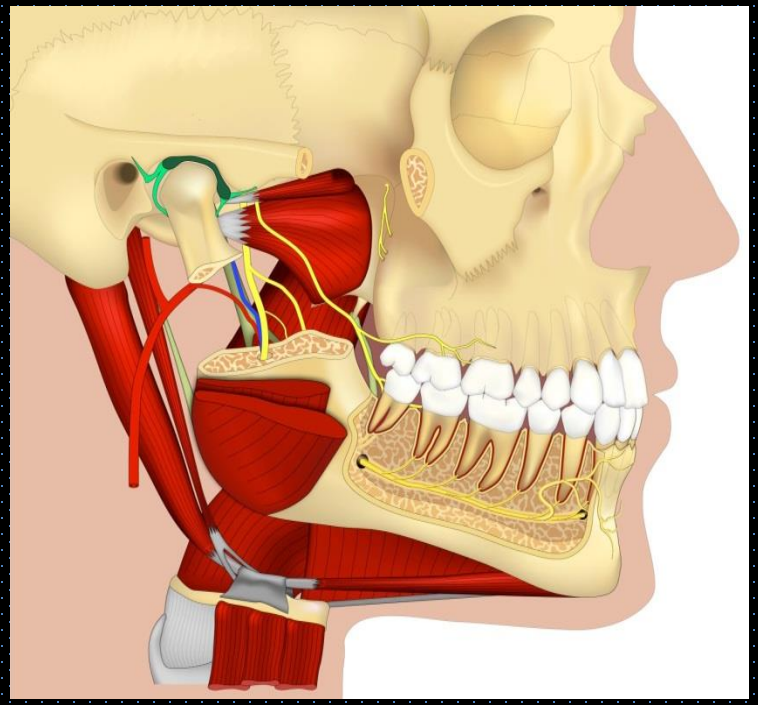
Chronic pain after dental surgery

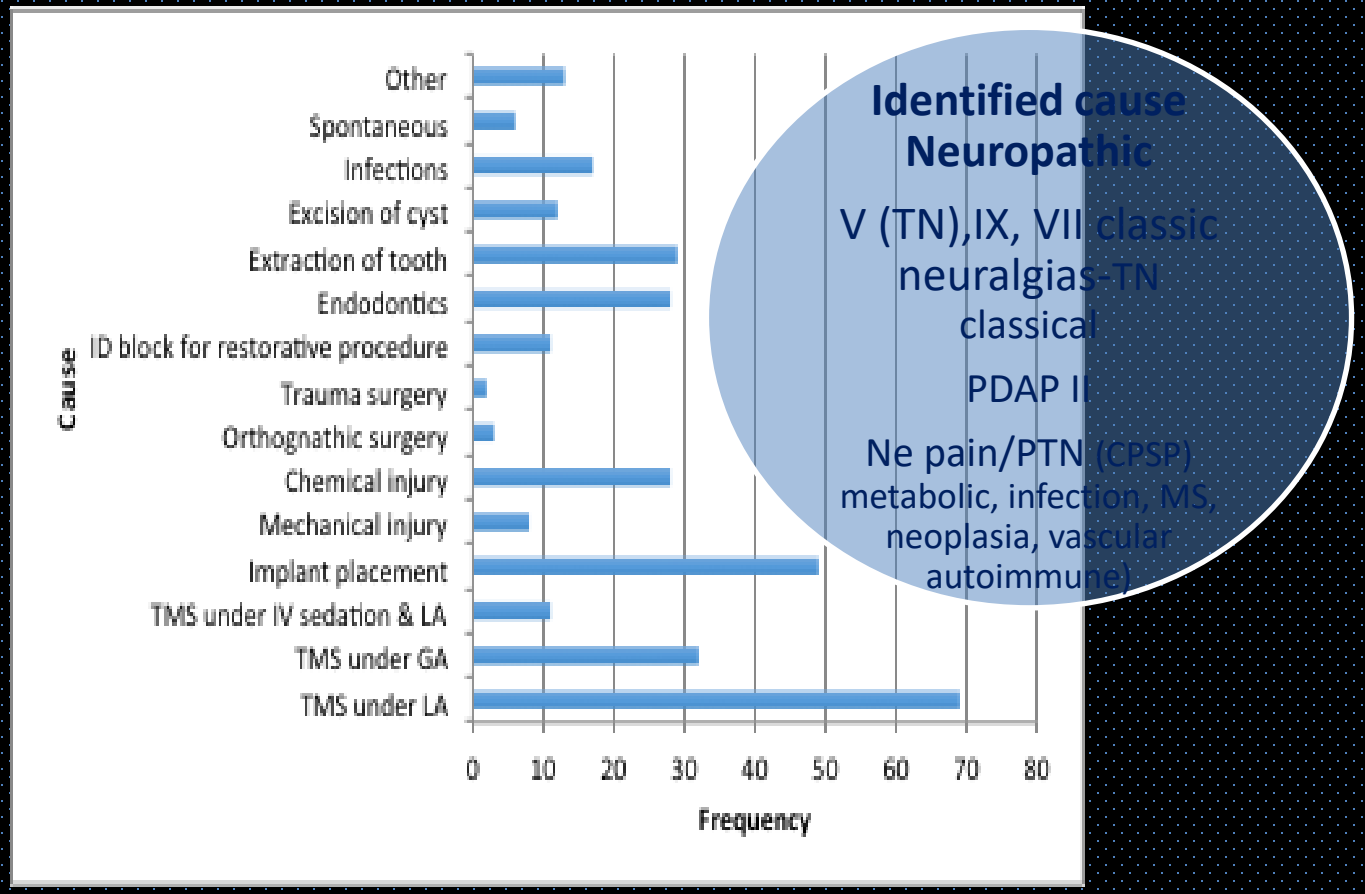
Two distinct chronic pain syndromes have been reported after dental surgery

- Persistent dentoalveolar pain =post-traumatic dysaesthesia
- Phantom tooth pain. The incidence of phantom tooth pain after endodontic therapy has been reported as 3%. For other pain syndromes, the incidence has been reported as varying from 5% to 13%. An interesting finding from the study by Lobb and colleagues was that most patients who suffered chronic pain after dental surgery did not revisit the dental surgeon. This does suggest that many dental

Dental causes of Trigeminal Post Traumatic Neuropathy

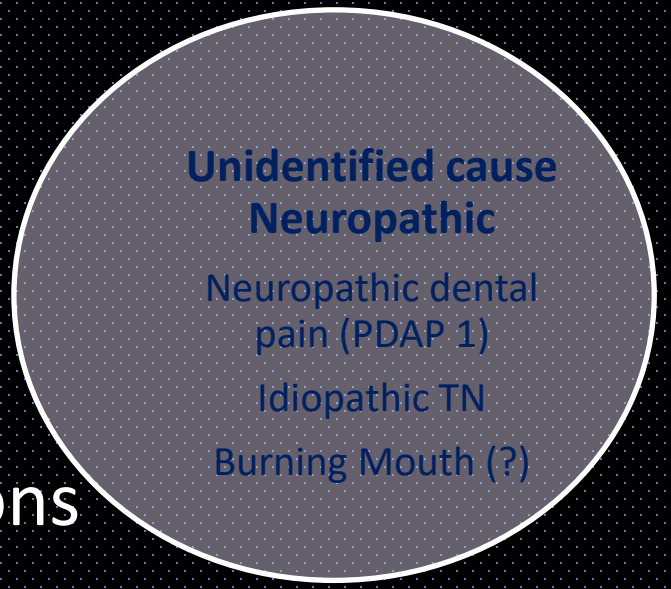
(+/- pain)





Summary of 535 TNIs assessed by TR 2016

Neuropathic pain or toothache?
Idiopathic neuropathic pain



Congenital neuropathic pain conditions

- Burning Mouth Syndrome
- Idiopathic Trigeminal neuralgia
- Primary neuropathic pain in intraoral region

=

- Pre TN
- Pre Tic
- Persistent dentoalveolar pain
  - PDAP 1
  - = Atypical odontalgia

OFP pain descriptors

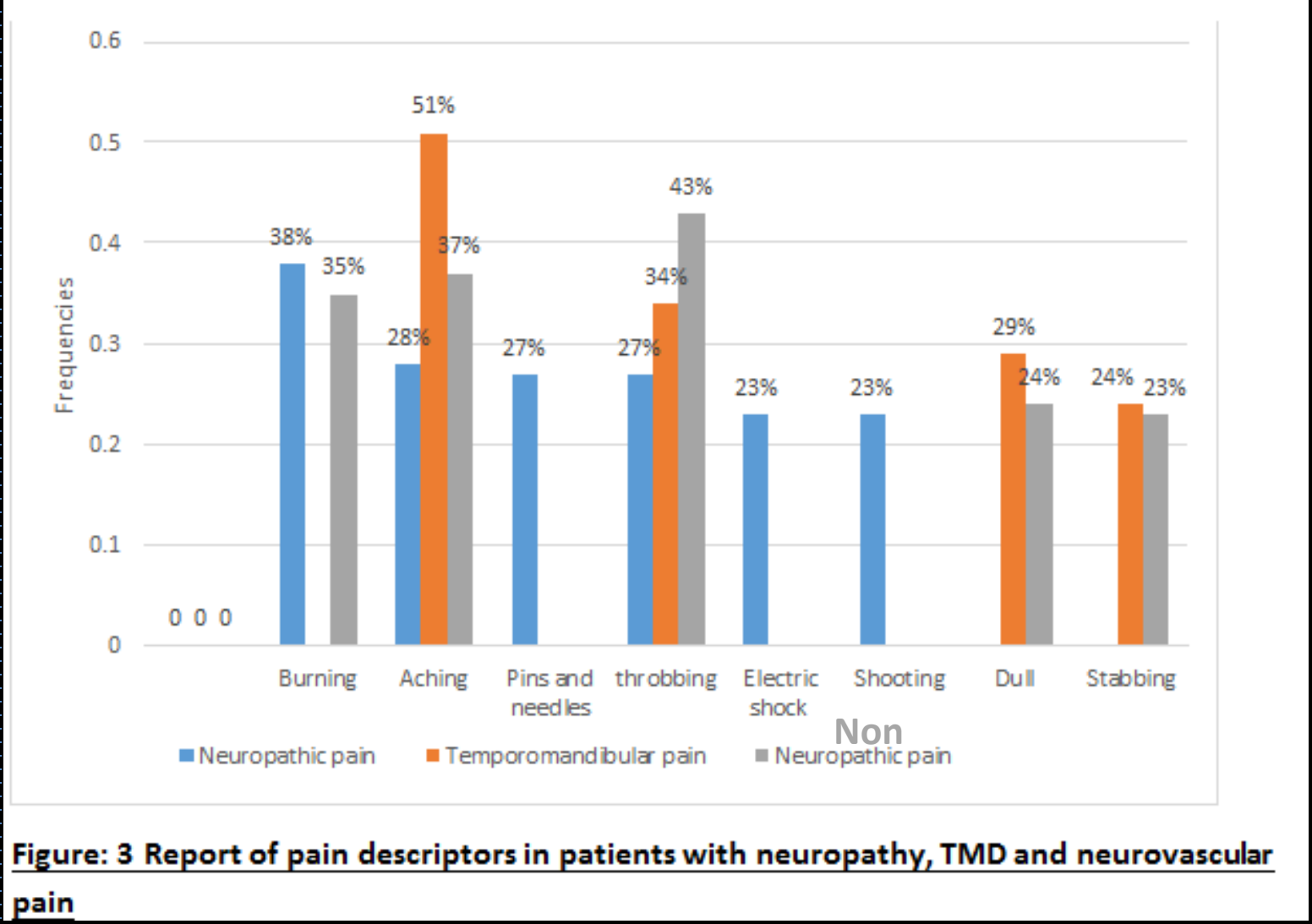


Figure: 3 Report of pain descriptors in patients with neuropathy, TMD and neurovascular pain

Pre screening for neuropathic pain in dentistry

Should it be routine?

Important to

- Identify primary orofacial and dental neuropathic pain to prevent inappropriate treatments
- Chronic neuropathic pain DOES NOT RESPOND to peripheral SURGERY
- **Surgery can make Neuropathic pain worse**
- Pre screening tools
  - DN4, Pain Detect, LANSS questionnaires

Shackleton TA. Failure of root canal treatment misdiagnosed as neuropathic pain: case report. J Can Dent Assoc. 2013;79:d94

Kaufmann R. Failure of root canal treatment misdiagnosed as neuropathic pain. J Can Dent Assoc. 2014;80:e28

- Fibromyalgia
- Irritable bowel syndrome
- Lower back pain
- Multiple pain syndrome

Dentist’s main responsibility ‘in pain’

- Identifying cancer caused pain and referring appropriately
- Diagnosing and treating dental pain correctly
- Not misdiagnosing non-odontogenic pain as dental pain and then continuing in providing inappropriate dental care
- Preventing nerve injuries and related chronic pain

Thank you

www.Trigeminalnerve.org.uk
www.orofacialpain.org.uk

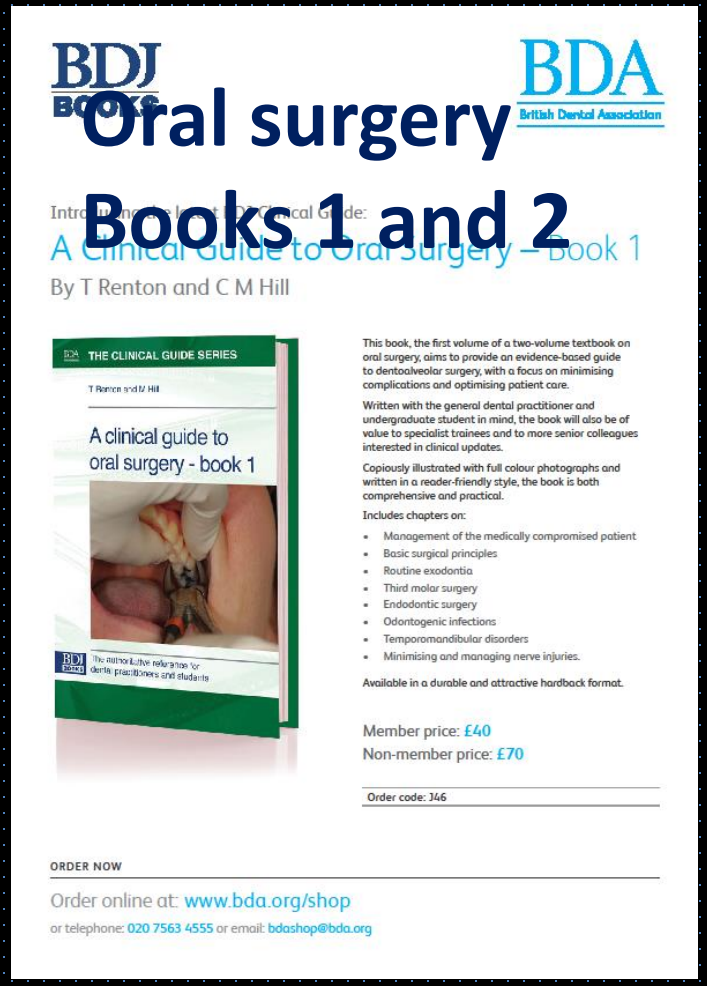
Andrew Mason University Dundee for illustrations
Zehra Yilmaz Post Doc Analysis and research assistance

Orofacial pain

BDJ book on pain management for dental teams coming soon!

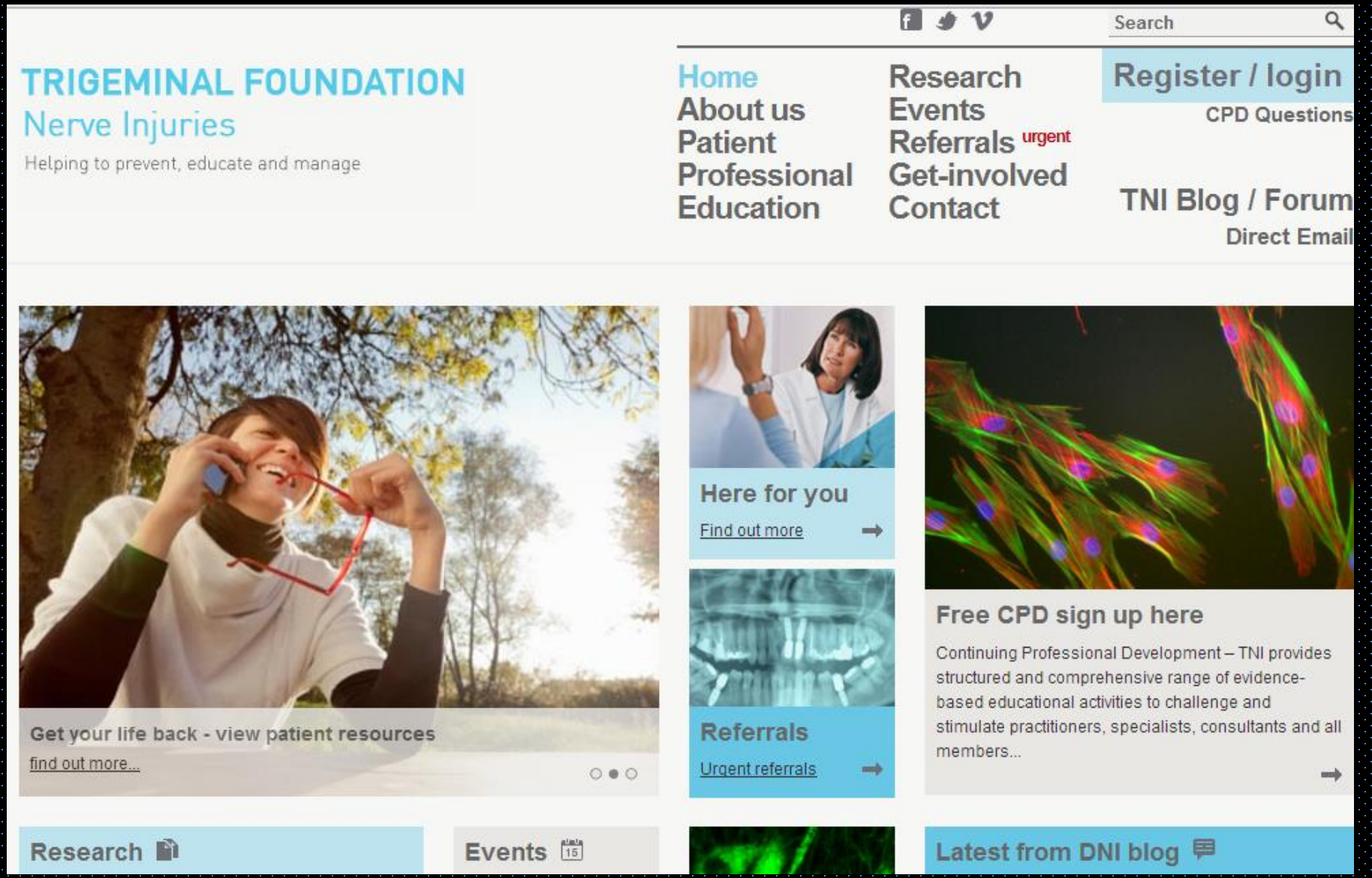
Tara Renton
Editor

Oral surgery Books 1 and 2



New KCL Orofacial pain Masters programme starting October 2017

Trigeminalnerve.org.uk



Thank you