

# Neuralgia in the trigeminal system

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[Trigeminalnerve.org.uk](http://Trigeminalnerve.org.uk)

Neuralgia ,njʊə'raldʒə/  
*noun*



- intense, typically intermittent pain along the course of a nerve

Love conquers all things except  
poverty and toothache.

**Mae West**

# Outline

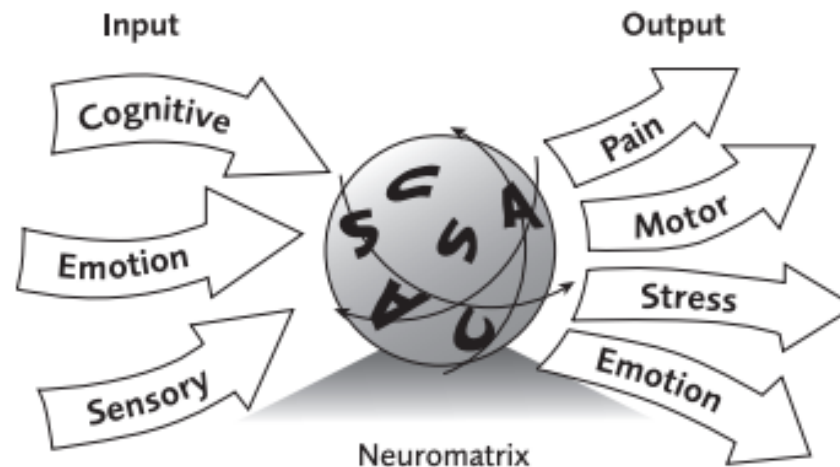
- Overview pain
- Orofacial pain
- An update on trigeminal pain
- An update on classification of pain
  - Excluding;
    - Headaches
    - Trigeminal Autonomic cephalgias
- Causes of neuralgia in the trigeminal system

# 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 is complex

Figure 2. The concept of the neuromatrix theory for pain



© Arthur Leung 2011

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.)

# Chronic pain is common

The report, "Relieving Pain in America: A Blueprint for Transforming Prevention, Care, Education, and Research," says the nation's health care system has largely failed Americans in pain and calls for a "cultural transformation" of the way in which the United States approaches and manages patients with pain.

**"A third of the nation experiences chronic pain. ... Costing us more than we pay as a nation on cardiovascular disease and cancer,"**

**Chronic pain costs the US up to \$635 billion each year** in medical treatment and lost productivity. *The 2010 Patient Protection and Affordable Care Act* required the Department of Health and Human Services (HHS) to enlist the IOM in examining pain as a public health problem.

*(Institute of medicine USA 2011 report on pain)*

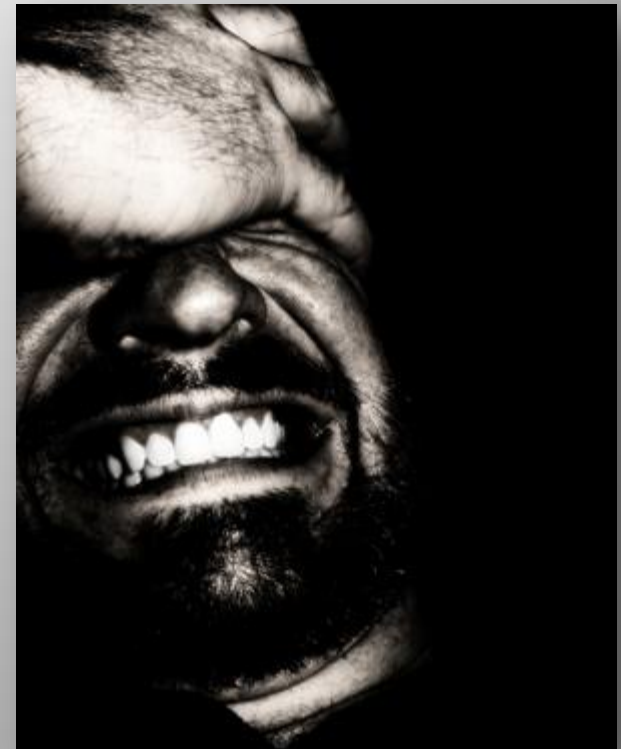
# Chronic pain: Consequences UK

33% of UK population suffer

13% work force is compromised

Diabetic and HIV neuropathy

Accounts for £80 billion year UK





# 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



## Definition of pain

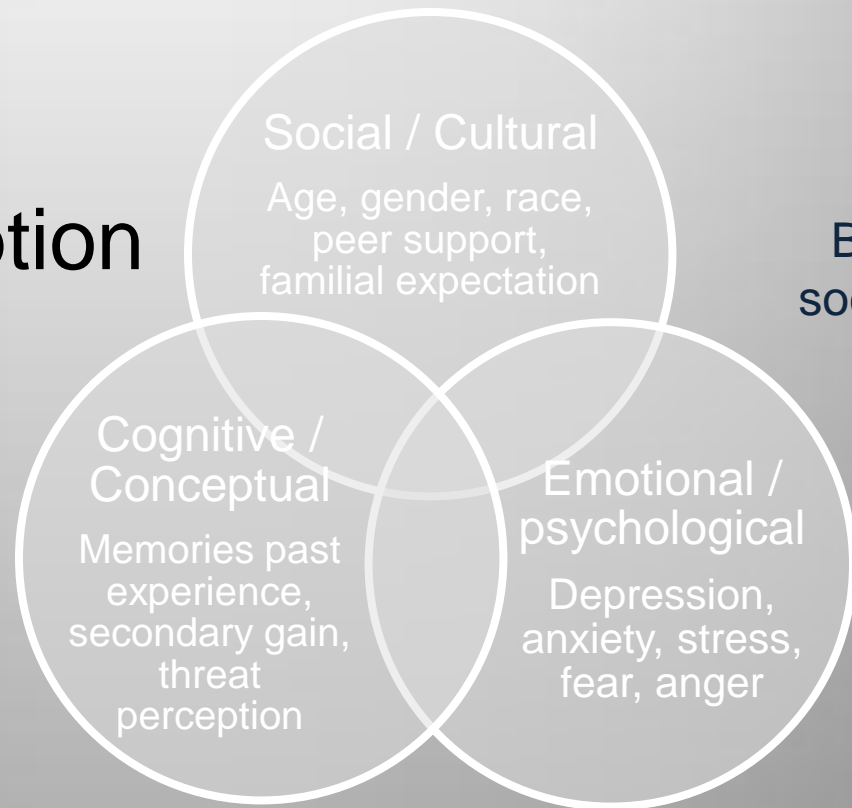
*"An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage"*

**(IASP, 1979)**

# Pain Process

TNI

- Nociception
- Sensation/perception
- Behaviour
- Suffering

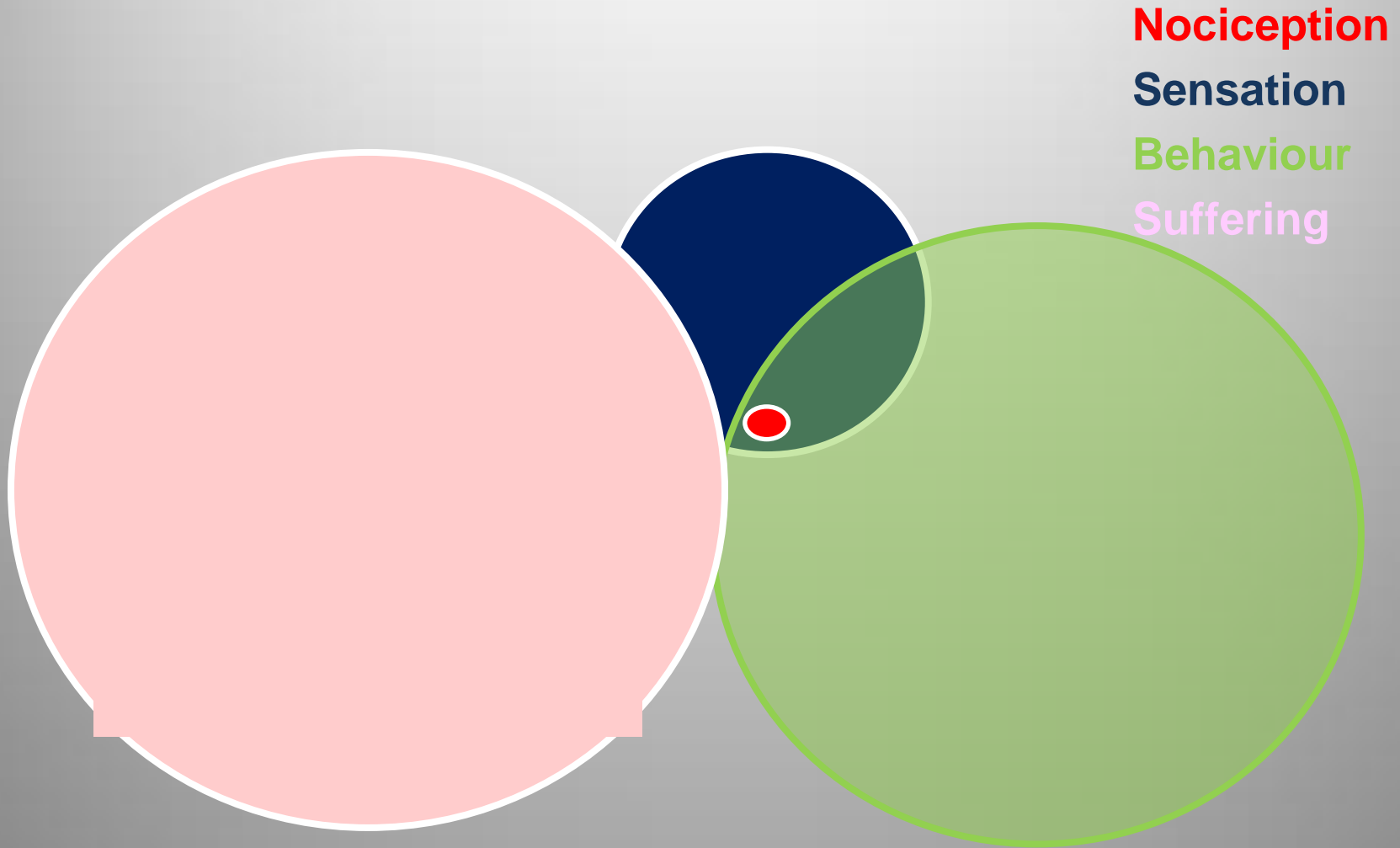


Bio psycho  
social Model

Do genetics influence all of these factors?

# Pain process

TNI



# Rugby player

TNI

**Nociception**

**Sensation**

**Behaviour**

**Suffering**



# Football player

TNI



**Nociception**

**Sensation**

**Behaviour**

**Suffering**



# CNS and PNS

## Receptor

## Primary sensory nerve

- A Delta and C fibres

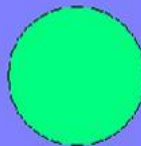
## Secondary sensory nerve

- Lamina I DRG

## Tertiary sensory nerve

- Specific areas of the brain
  - Thalamus
  - Anterior cingulate cortex
  - S1 / S2
  - Insula
  - Brainstem

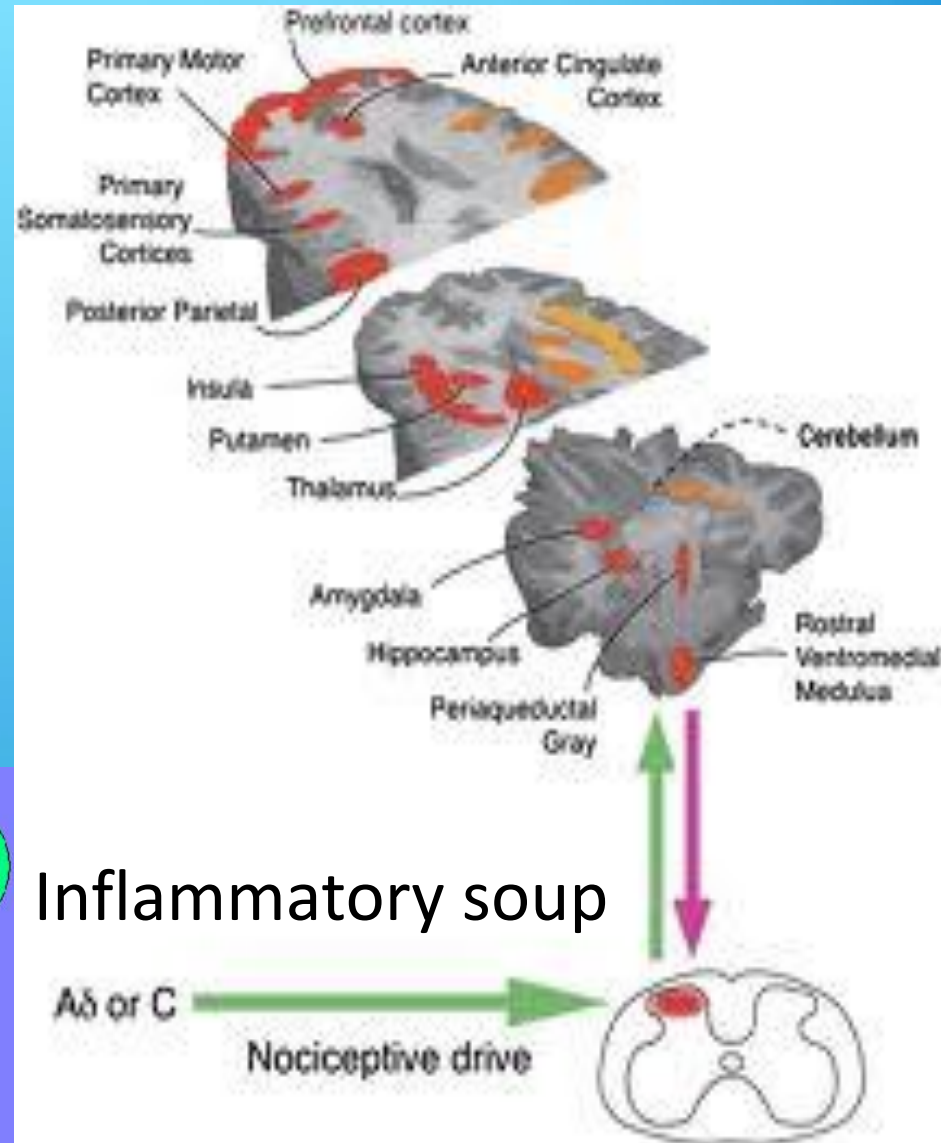
A beta



A delta



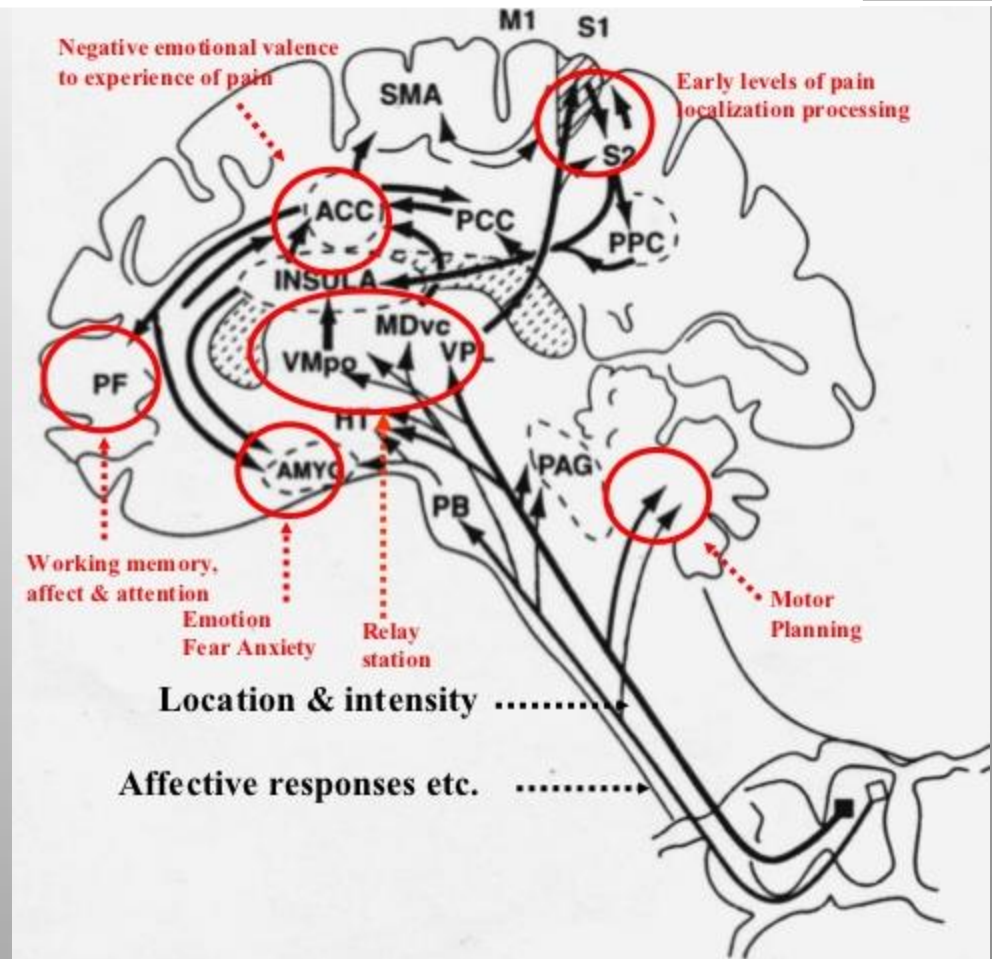
C





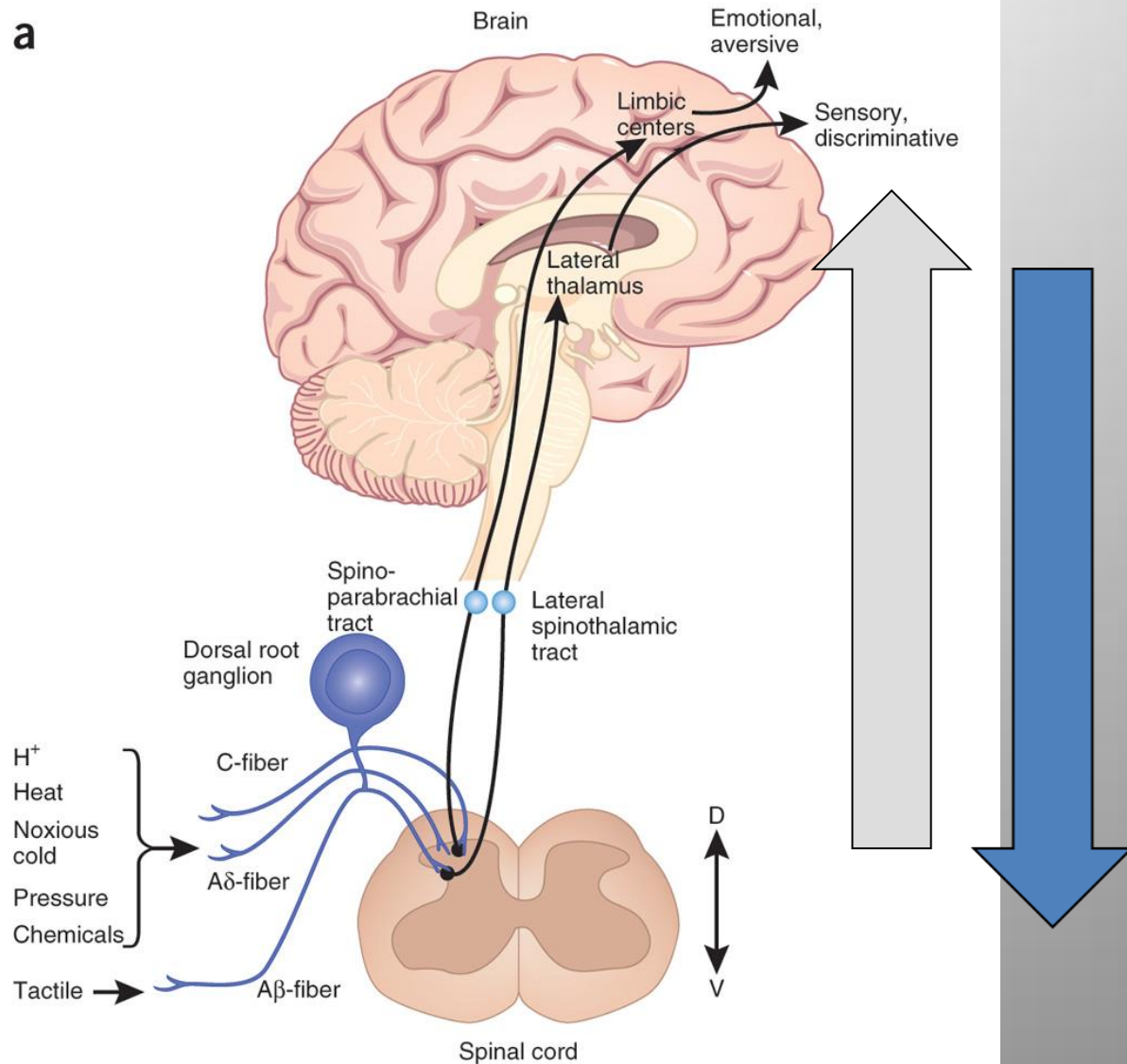
# Sensation -Pain Matrix

The 'Pain Matrix'  
brain areas reactive  
to pain  
26 areas of the brain  
affected so far!



# Modulation

TNI





# Tapping into natural resources

- Maximising downward inhibition of pain
- Sleep
- Hypnotism
- Meditation
- Education...managing expectations.....



# 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.

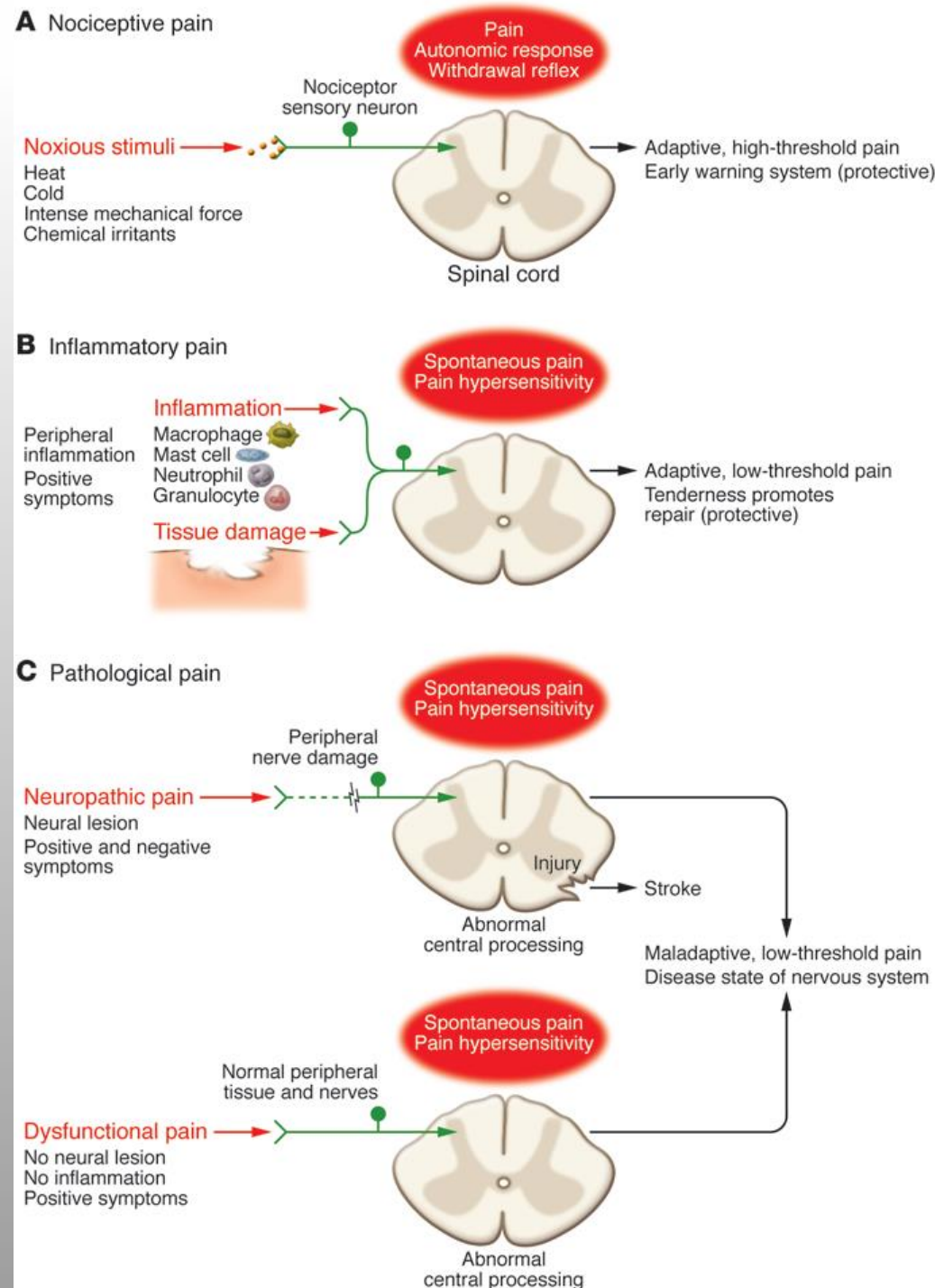
#### Classifying pain

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.

# 4 types of pain

- Nociceptive healthy feeling pain 'pain'
- Inflammatory pain health short lived after insult
- Neuropathic pains
- Dysfunctional pain  
Clifford J. Woolf. **What is this thing called pain?** J Clin Invest. Nov 1, 2010; 120(11): 3742–3744.

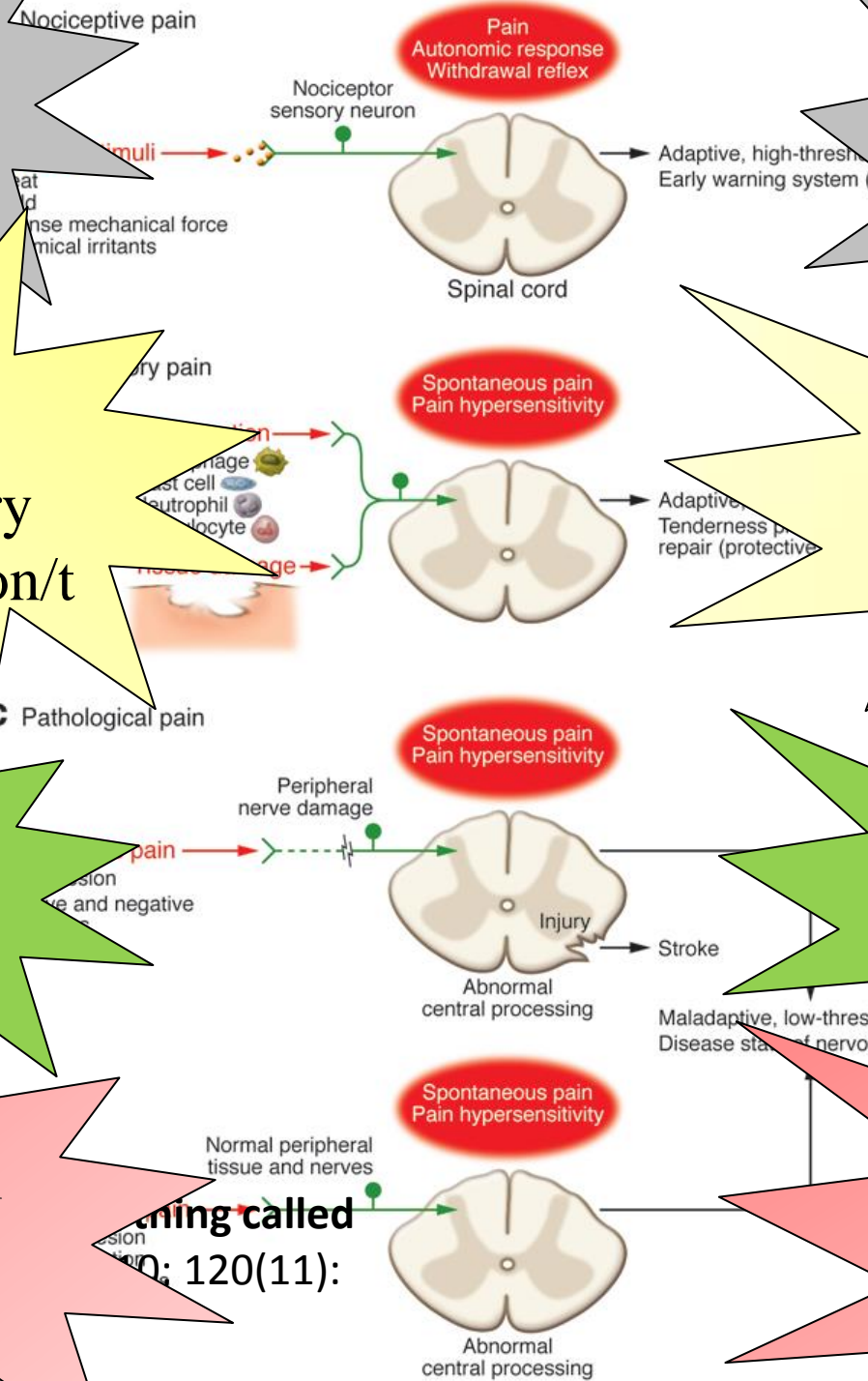


# Healthy nociceptive pain

Healthy  
inflammatory  
pain/infection/t  
Trauma

# Chronic neuropathic pain

# Dysfunctional pain



# Dentine sensitivity

Pulpitis reversible  
+irreversible  
Periapical  
periodontitis

# Posttraumatic neuropathy PDAP/ PHN

# Fibromyalgia

## PIFP

# TMD arthromyalgia

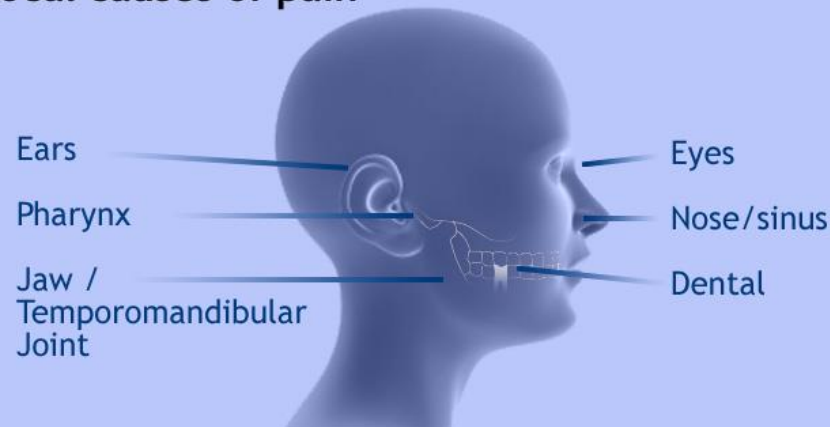
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# Orofacial pain

## Local causes of pain



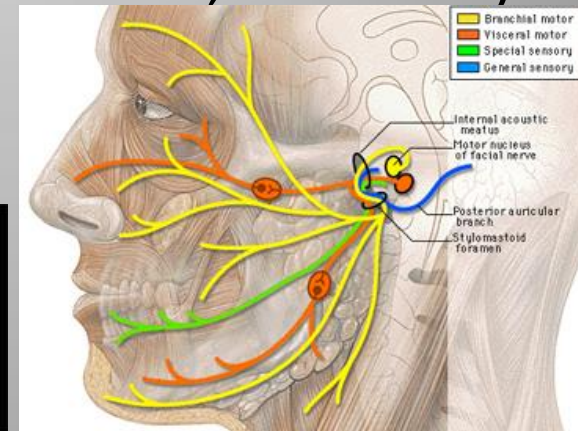
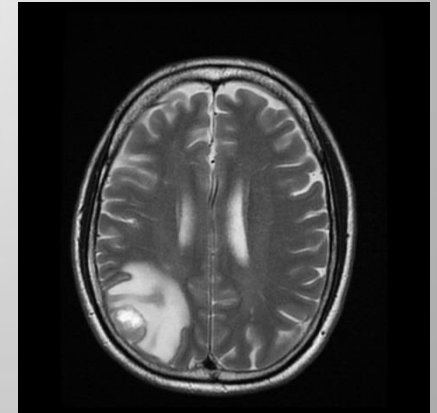
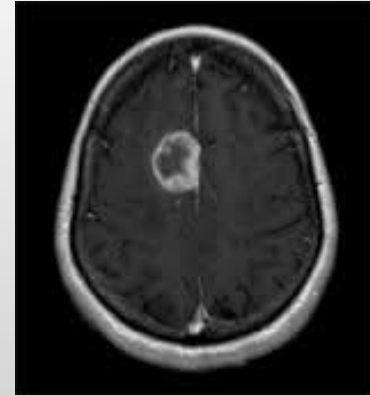
## Other causes



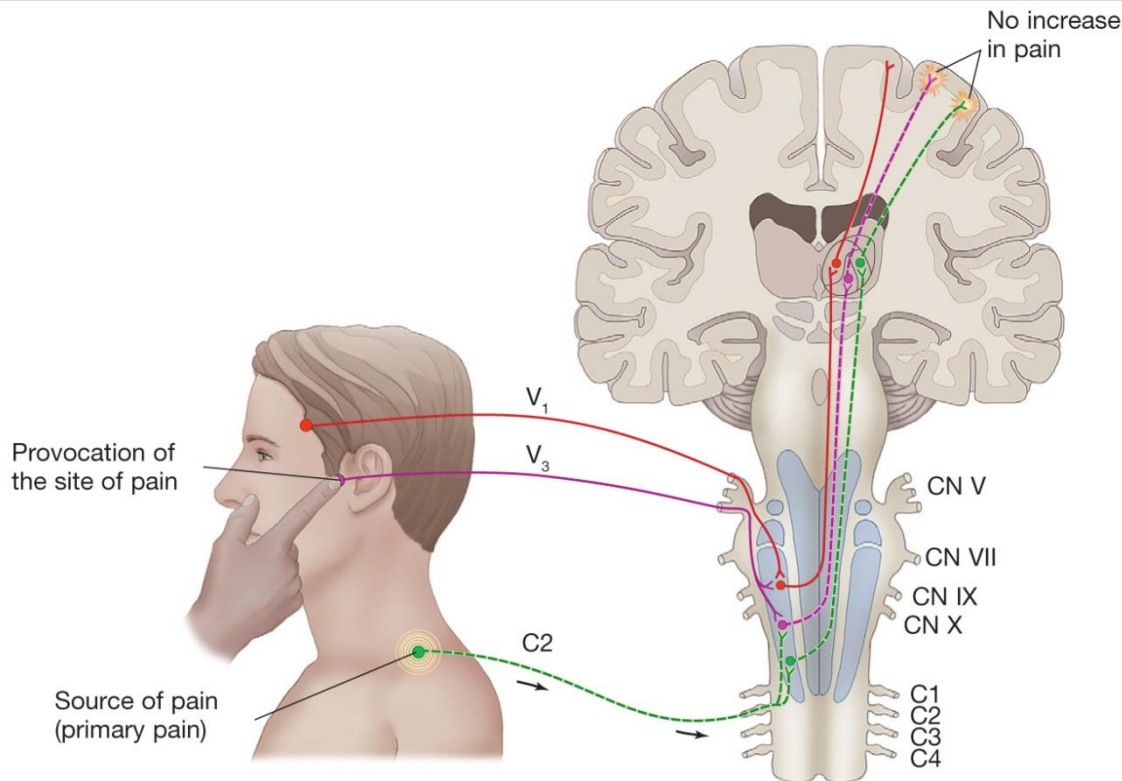
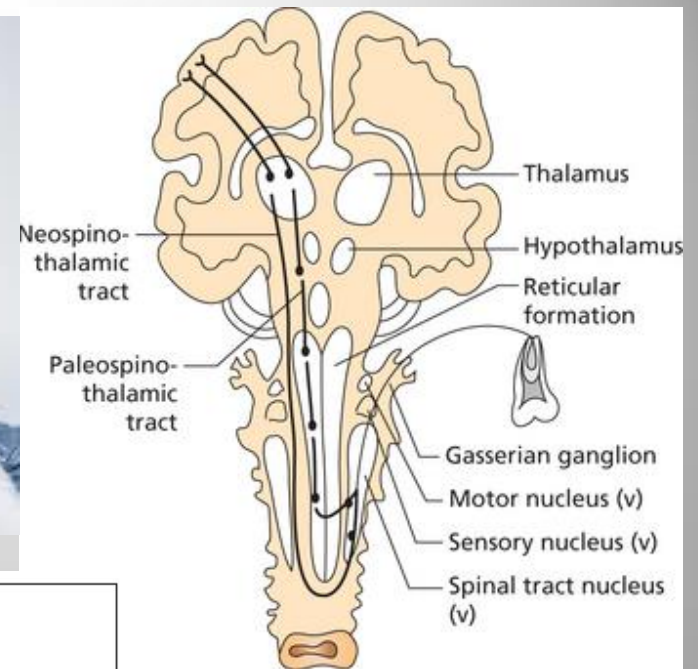
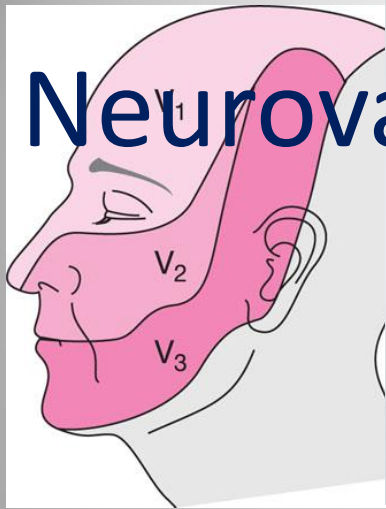
GLOBAL YEAR AGAINST  
**OROFACIAL PAIN**  
OCTOBER 2013 – OCTOBER 2014  
International Association for the Study of Pain

# Neurological causes of OFP

- Primary neuropathies
  - Trigeminal neuralgia
- Secondary neuropathies
  - Trauma (Burns, surgery, trauma, laser, chemical, radiation)
  - Diabetes
  - Demyelination (Multiple sclerosis, GB)
  - Thyroid
  - Addiction (Alcoholism)
  - Cancer
  - Deficiencies Folate, Vit B complex, Ferritin FE Zinc
    - Mal absorption
    - Malnutrition



# Neurovascular





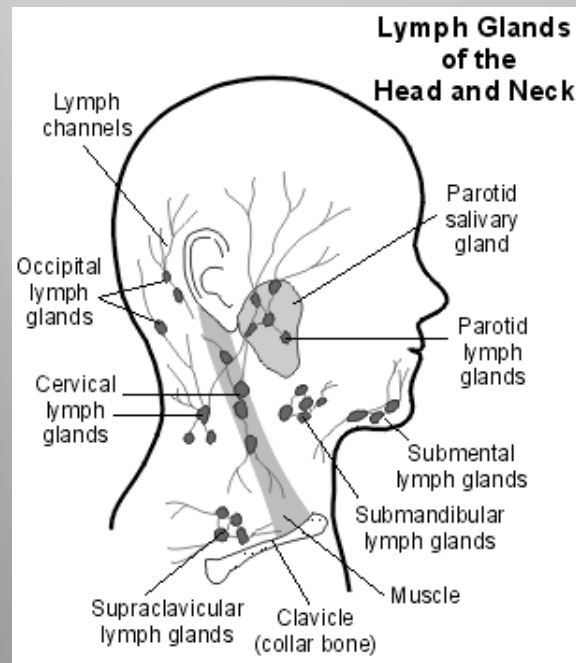
# Temporomandibular joint Disorders

- Arthritides
  - Osteo arthritis
  - Rheumatoid arthritis
  - Stills
  - Reactive
- Dysfunction
  - Jaw locking
  - Clicking
- Arthromyalgia
  - Muscle pain Chewing gum, clenching or brux habits, regular dental treatment



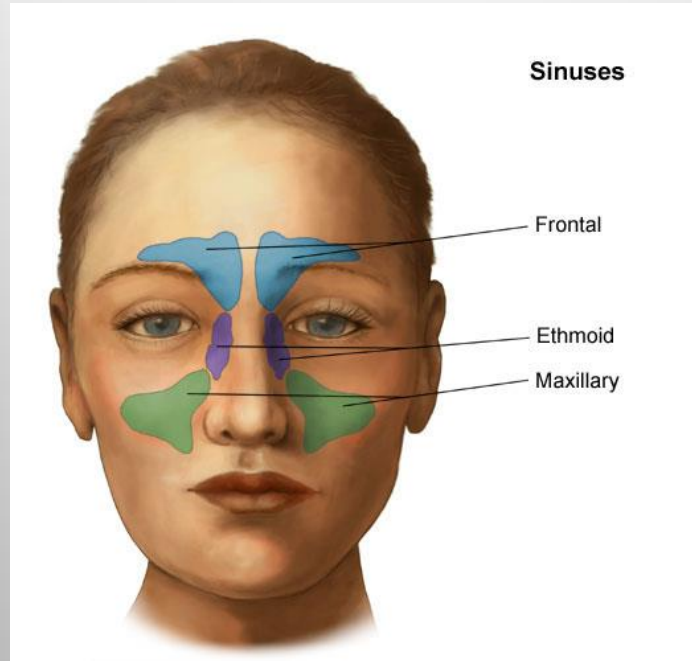
# Skin/ Lymph nodes

- Infection
  - Local dental or skin infection
  - TB
  - Viral Epstein Barr, CMV, Flu, HIV
- Lymphoma
- Leukaemia
- Cancer

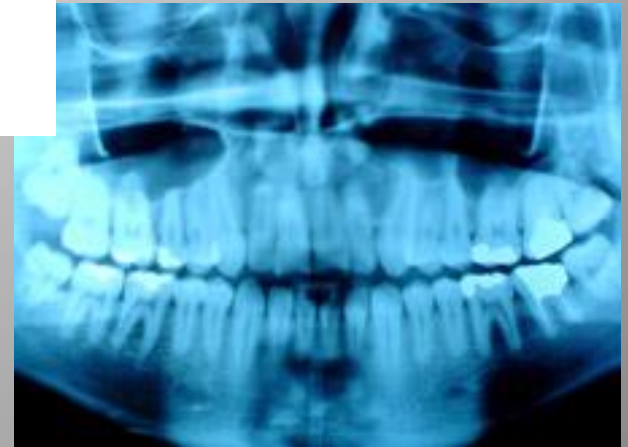
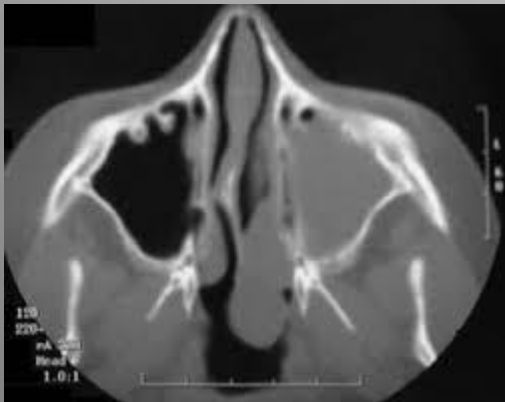


# Sinuses

- Maxillary
- Frontal
- Ethmoidal
- Sphenoidal
- Nasal cavity

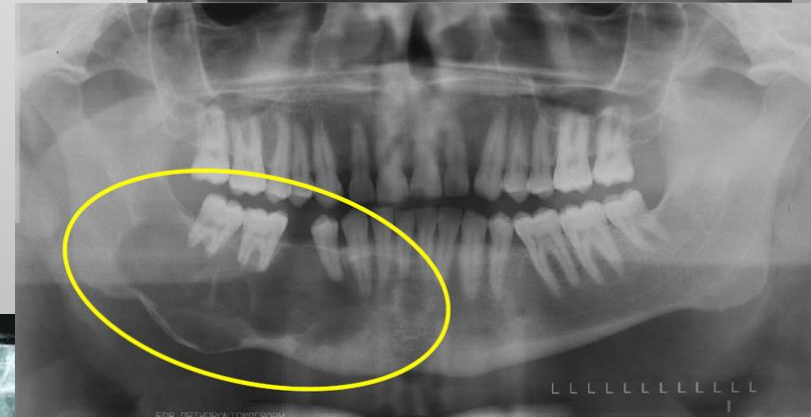
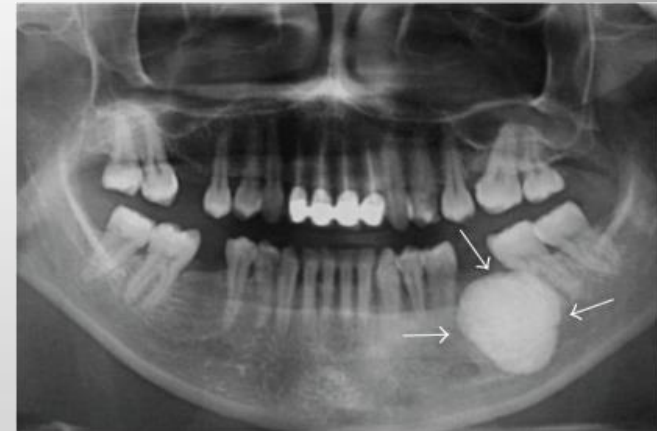


- Sinusitis
- Cysts
- Foreign objects
- Cancer



# Bone Jaws

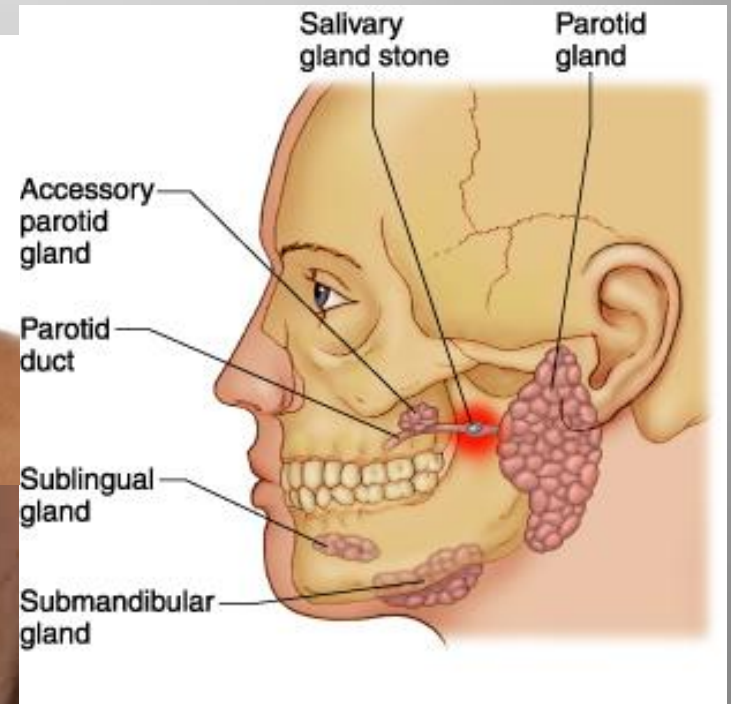
- Infection
  - Spreading dental abscess
  - Osteomyelitis
  - Osteonecrosis
  - Osteoradionecrosis
- Trauma
  - Dental
  - Fracture
- Benign or malignant cancer





# Salivary glands

- Autoimmune connective tissue disease
- Obstructive disease (calculi = stones)
- Tumours



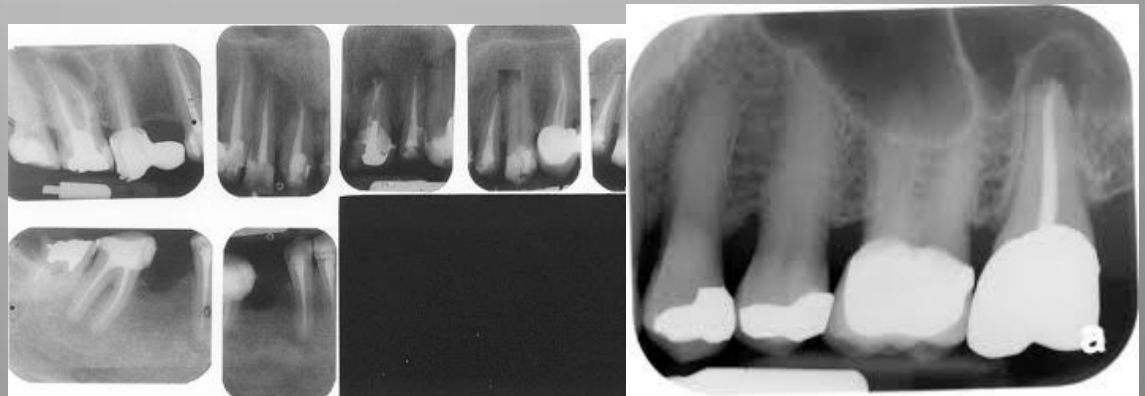
# Mucosa

- Ulcers
- Trauma
- Cancer
- Autoimmune Connective tissue disease



# Teeth

- Toothache
  - Pulpal infection
  - Abscess
  - Trauma
- Cracked tooth syndrome
- Phantom dental pain –persistent dento alveolar pain



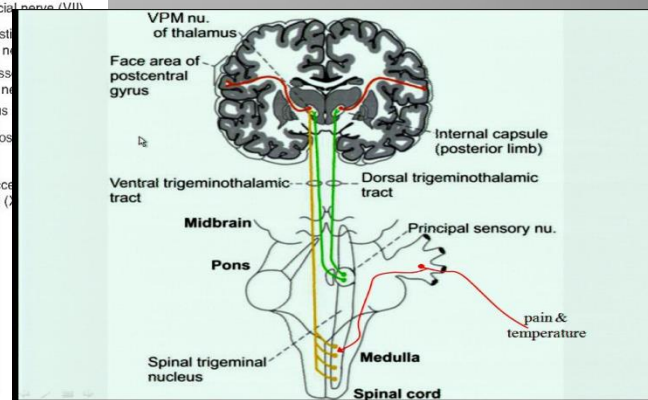
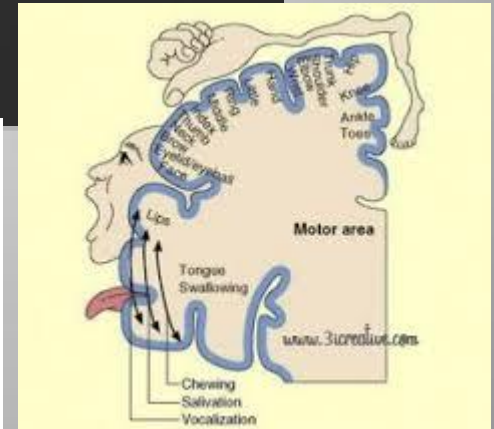
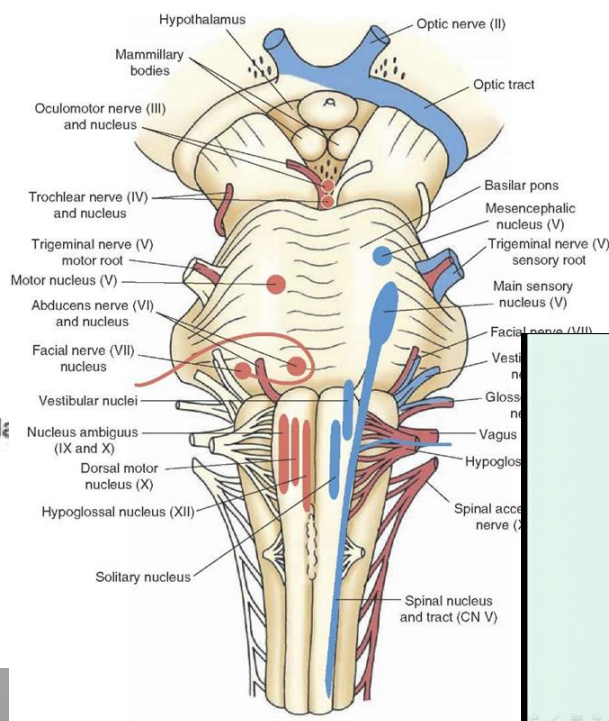
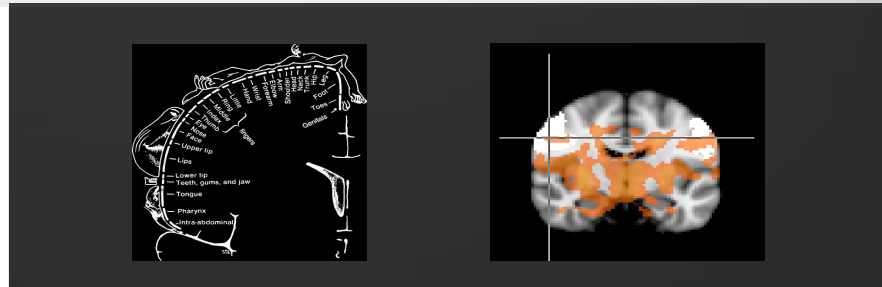
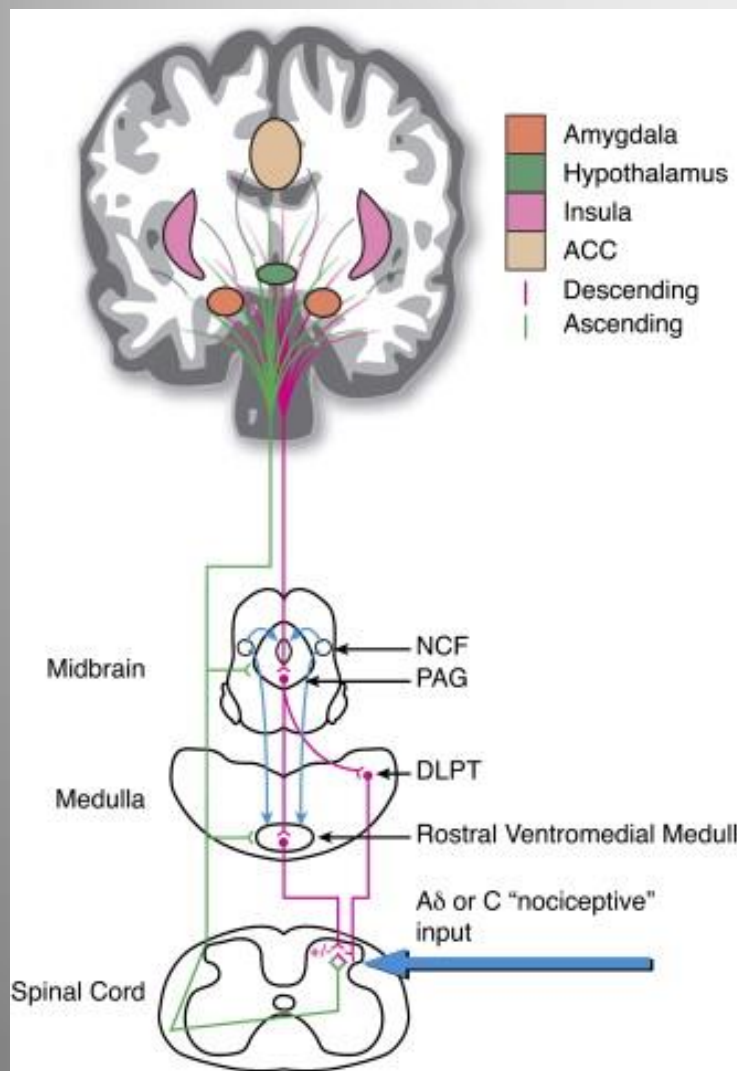
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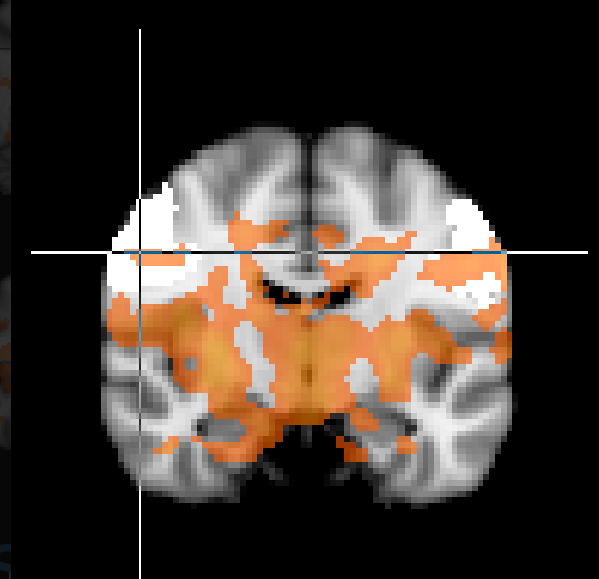
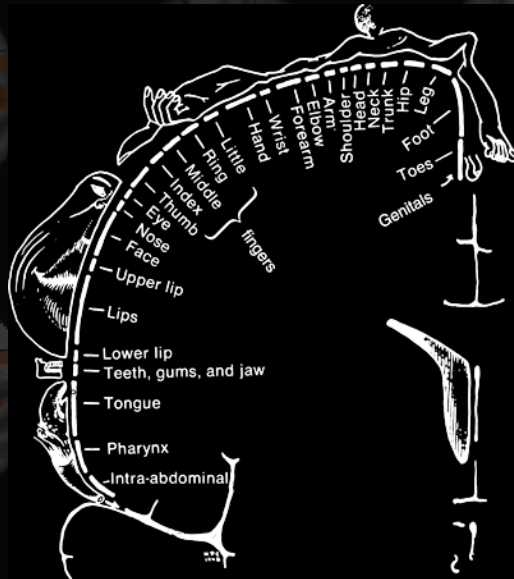


# The trigeminal system

TNI

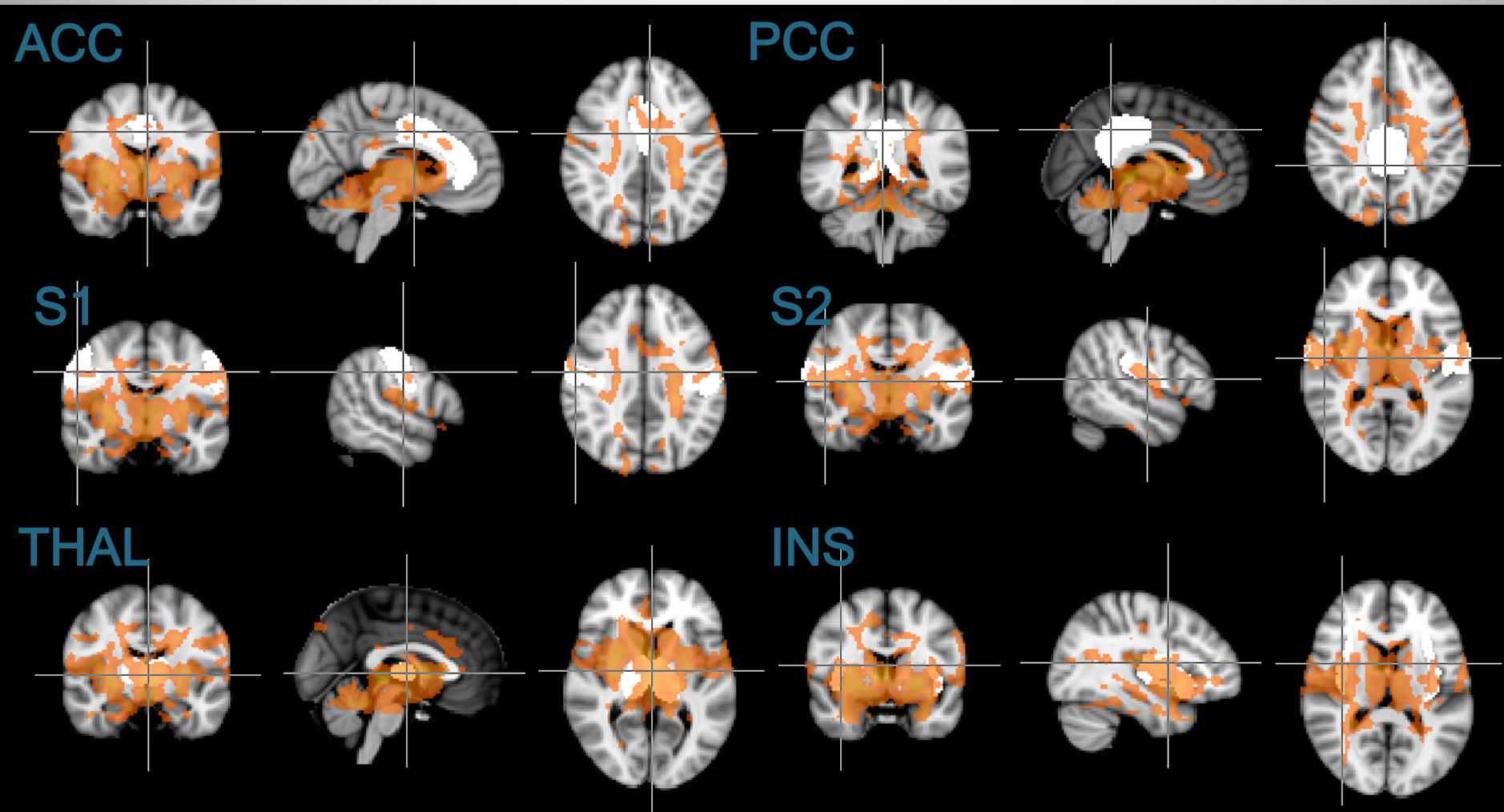


# Anatomy revisited



•Additional amygdala, hippocampus, brainstem, and V5 ROIs

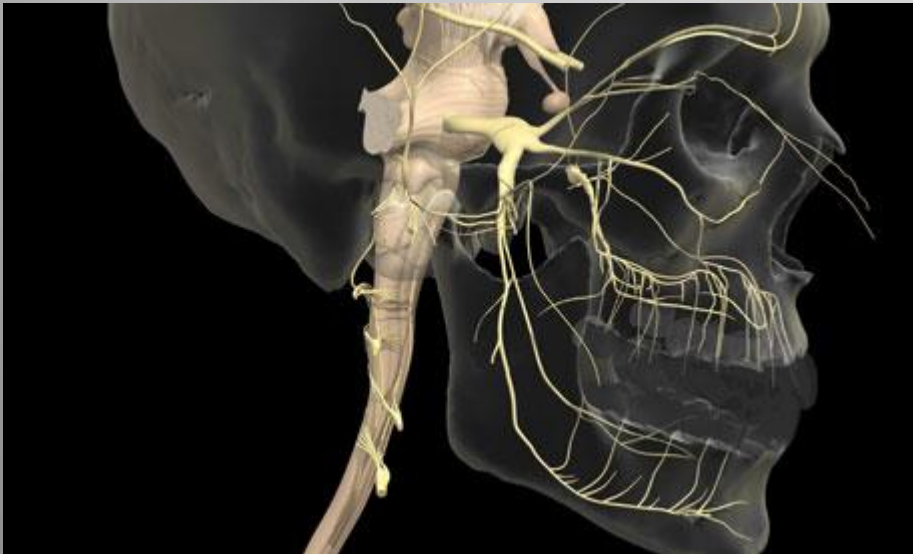
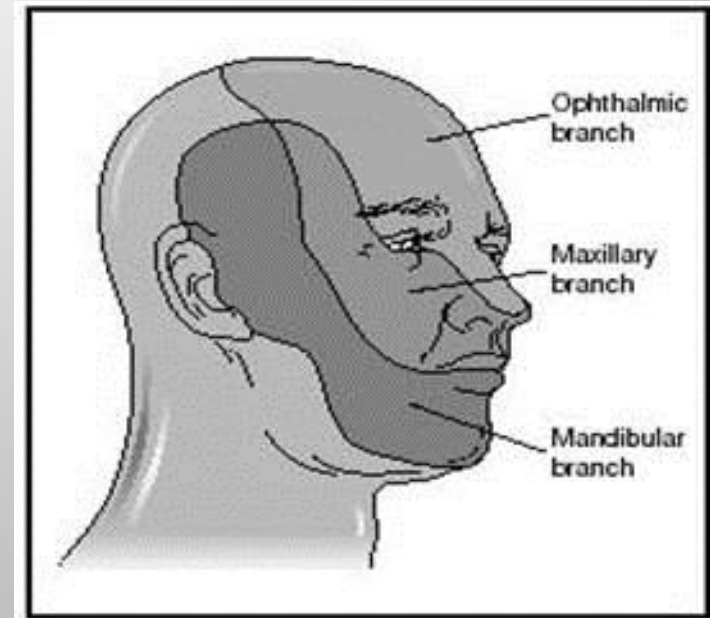
# Anatomy revisited



• Additional amygdala, hippocampus, brainstem, and V5 ROIs

# Types of acute V pain

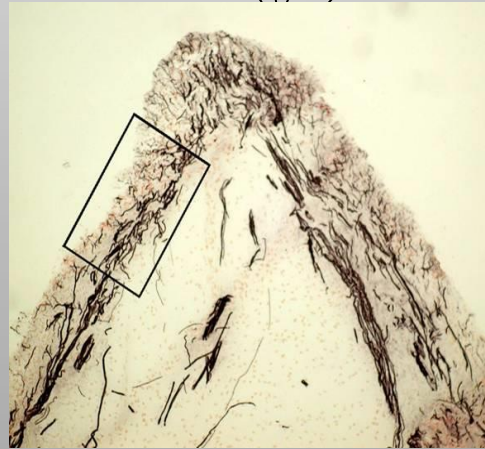
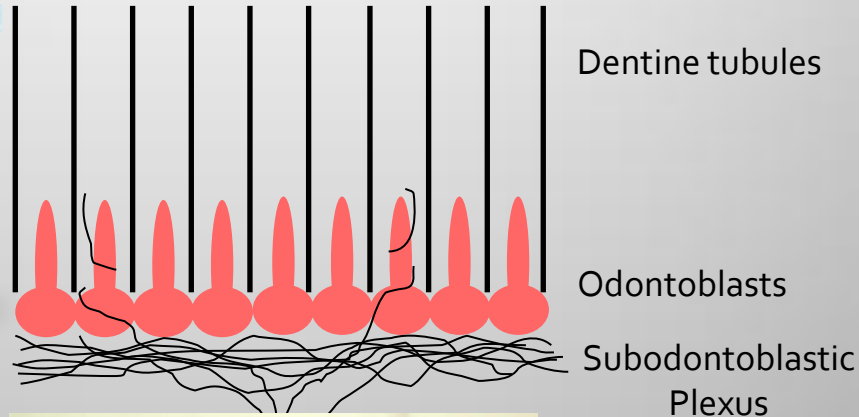
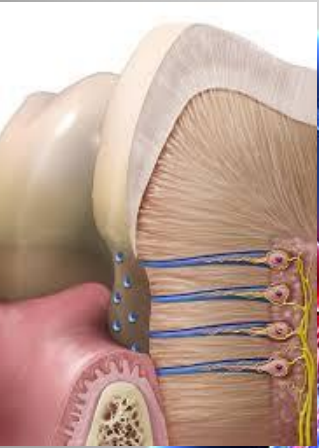
- Nociceptive
- Inflammatory





# Unique features?

Thermal or mechanical stimulations of dentine /tooth pulp **result in a painful sensation** unlike that in other tissues in the body (Cook *et al.*, 1997)



**Roughly 57% of adult population affected by dentine hypersensitivity . Peak age: 30 - 39 years** — Cummins D, J Clin Dent 2009a; 20(Spec Iss): 1–9. **Still under-reported at present** – Gillian D et al, Dental Update 2013 Sep; 40(7): 514-6, 518-20, 523-4

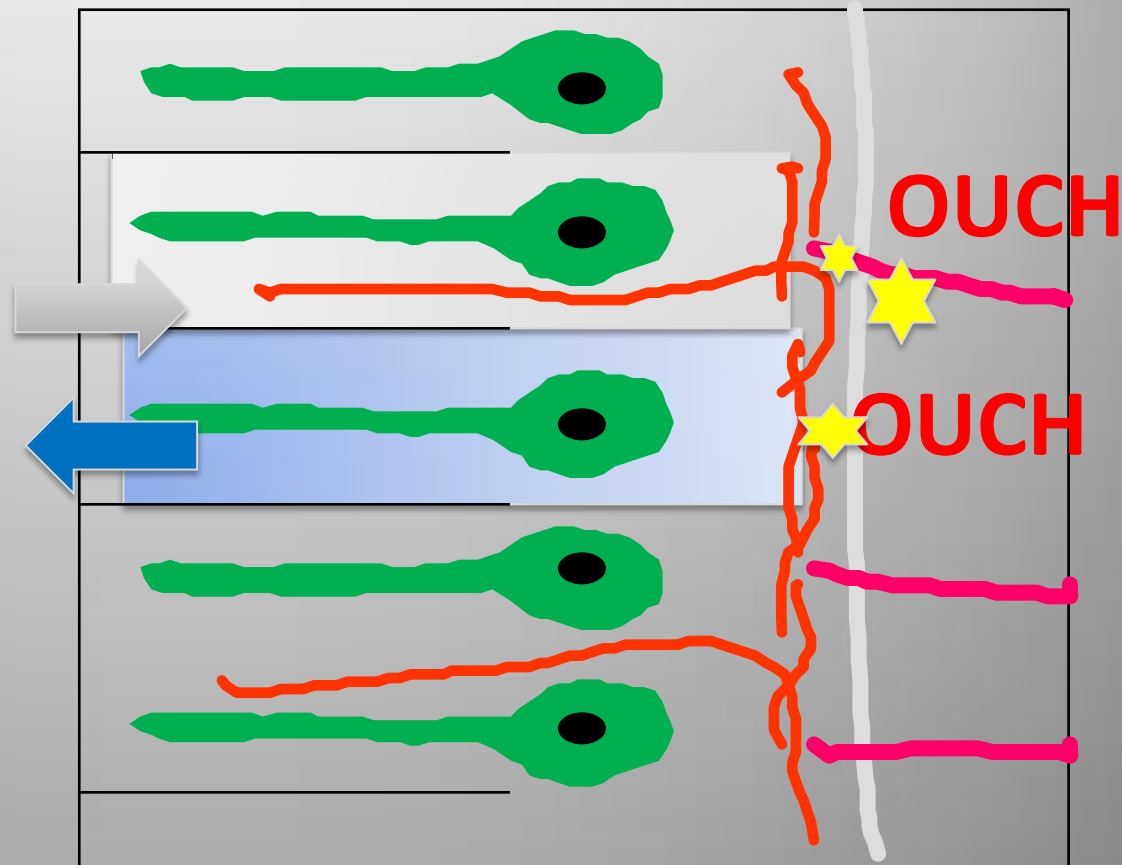
# Theories of dental nociception

TNI

**Activation** -Direct neural stimulation neural theory, whereby nerve endings that penetrate dentinal tubules directly respond to external stimuli

**Transduction**- Odontoblast acting as transducer

**Hydrodynamic theory** fluid movements within the dentinal tubules are detected by nerve endings near the dentin



In all theories the activation of dental primary afferents eventually delivers dental nociception to the central nervous system.

# In health

TNI

In relation to dental innovation.....

Allodynia is NORMAL!

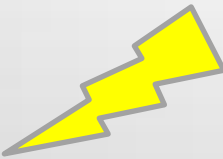




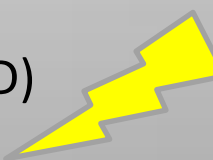
Attrition

Abrasion

Erosion



# Classification of V pain

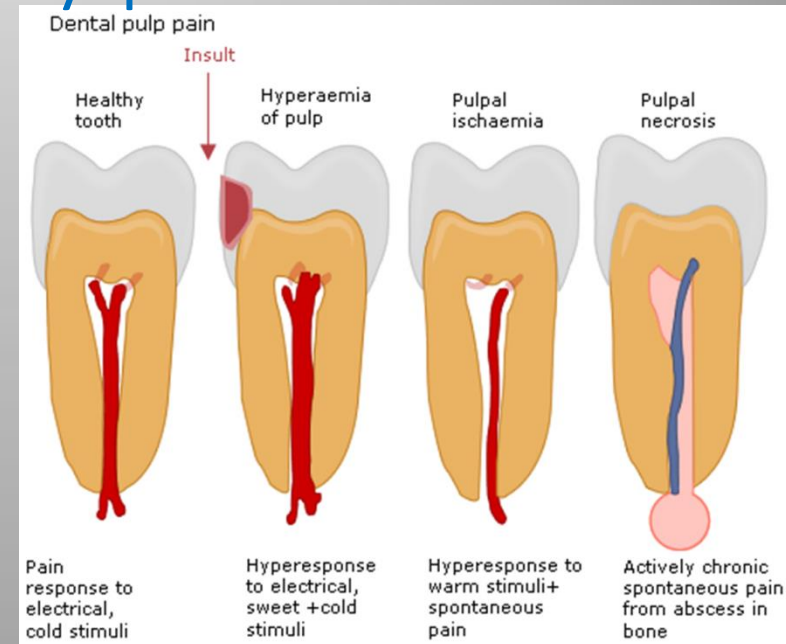
- Nociceptive
  - Dentine sensitivity 
- Inflammatory
  - Odontogenic and non odontogenic 
- Neurovascular
  - Headaches, TACs 
- Neuropathic
  - BMS, TN  Secondary neuropathy 
- Dysfunctional
  - FM,
  - Temporomandibular Disorder (TMD)
  - Myalgic, arthritides, dysfunctional 
  - Persistent idiopathic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



# Acute V dental pain

- **Odontogenic**
  - **Healthy**
    - Dentine sensitivity
  - **Inflammatory pain**
    - Dental impaction pain model post extraction/ surgical pain
    - Peri dental mucosal inflammation
      - Pericoronitis
    - Toothache –
      - Dental pulpitis
      - Irreversible pulpitis
      - Periapical periodontitis
  - Post Surgical pain
  - **Chronic dental pain-**
    - Neuropathic dental pain

## Inflammatory pain

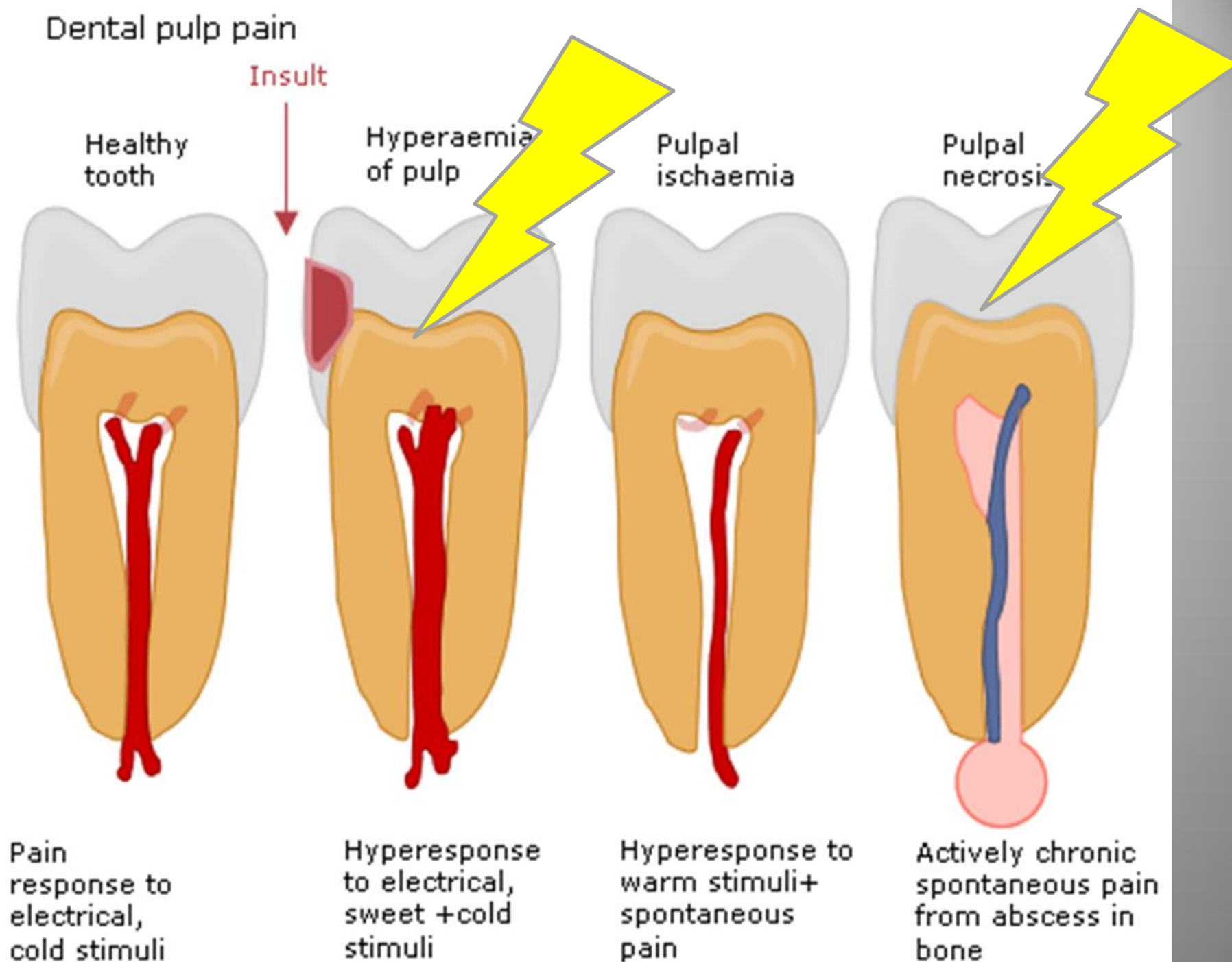


# Dental caries (tooth rot)

TNI



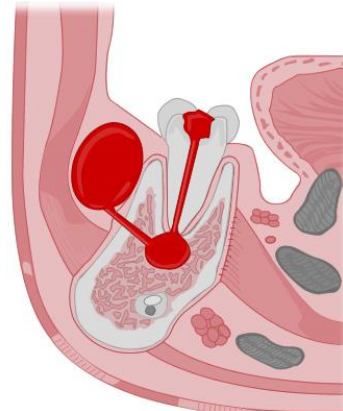
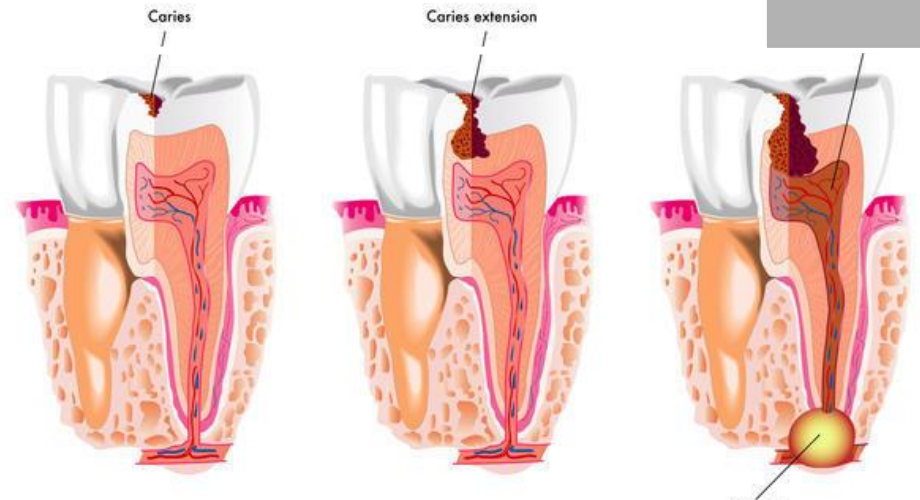
## Dental pulp pain



# Dental abscess

TNI

TOOTH DECAY





# Cracked tooth

TNI



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EXPERT  
REVIEWS

# The classification and differential diagnosis of orofacial pain

*Expert Rev. Neurother.* 12(5), 1–xxx (2012)

**Tara Renton<sup>\*1</sup>,  
Justin Durham<sup>2</sup> and  
Vishal R Aggarwal<sup>3</sup>**

<sup>1</sup>Department Oral Surgery, Kings  
College London Dental Institute,  
Denmark Hill Campus, Bessemer Road,

There are currently four main pain classification systems relevant to orofacial pain (OFP): the International Association for the Study of Pain (IASP), International Classification of Headache Disorders (ICHD-II), the American Academy of Orofacial Pain (AAOP) and the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD). Of the four, the RDC/TMD is the most biopsychosocial system with the remaining three focusing more on the biomedical. Unsurprisingly clinical scientists and clinicians have both reported perceived deficiencies in the published systems

# Classification of **Chronic** orofacial pain

## Neurovascular

Headaches

MOH

Chronic daily

Tension HA

Migraine

Trigeminal autonomic  
cephalgias

Cluster HA

SUNCT

SUNA

Hemifacial Cont

Praxysmal Hemicrania

Giant cell arteritis

## Neuropathic

**Trigeminal N**

Classic/ symptomatic

**PHN**

**Glosspharyngeal N**

**Burning Mouth Syndrome**

**Secondary Sensory  
Neuropathy**

DM, MS, GB, Chemo, Thyroid  
D, Vit Deficiency

**Post Traumatic**

## Idiopathic

Persistent idiopathic  
(ATFP / ATO)

PDAP

Temporomandibular  
Disorders

Dysfunctional

Athritides

Arthromyalgia

# Neurovascular



- Exclude sinister headaches 1%
  - » >50 yrs Tumour 1%
  - » Subarachnoid haemorrhage - recent trauma LoC
- Migraine 10-17%

Five or more lifetime headache attacks lasting 4-72 hours each and symptom-free between attacks  
moderate to severe pain, unilateral +/- aura visual signs
- Cluster headaches 5% - SUNCT

Male:female ratio 4:1 to 20:1 / 30yrs +  
Severe episodic pain lasting 15-180 minutes  
Unilateral Orbital, supraorbital or temporal  
8x a day to every other day for a period of 2 -12 weeks
- Tensions type headaches

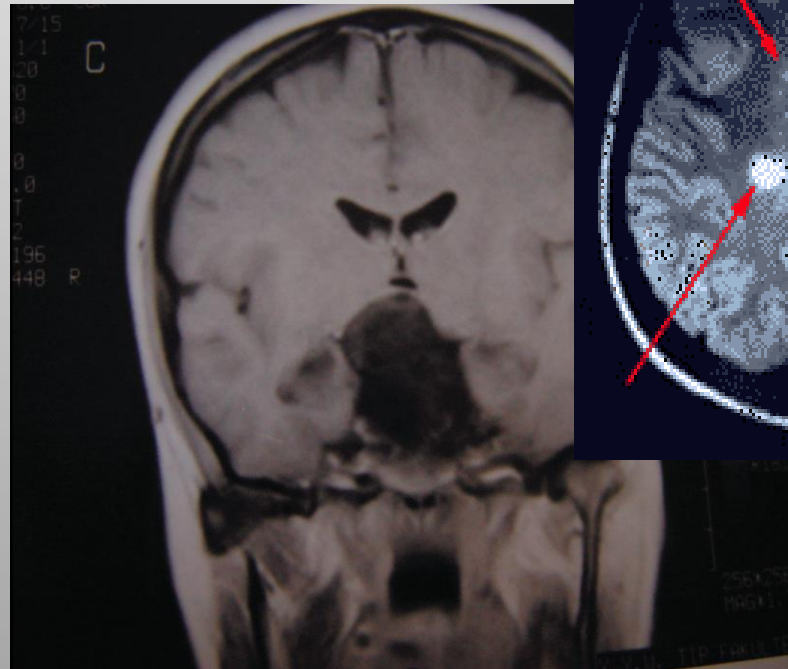
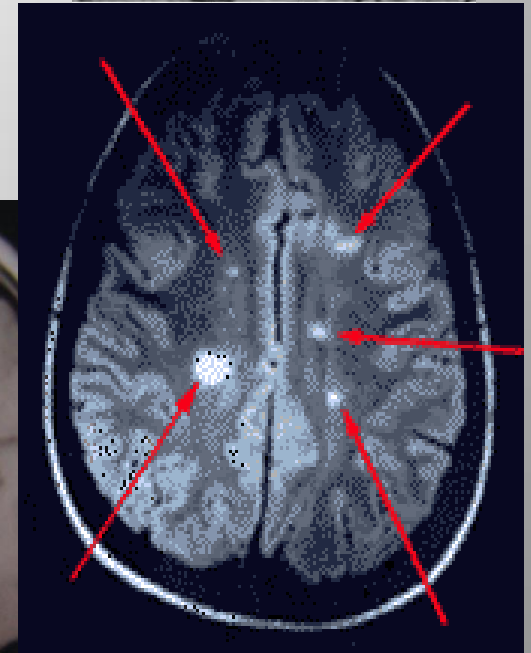
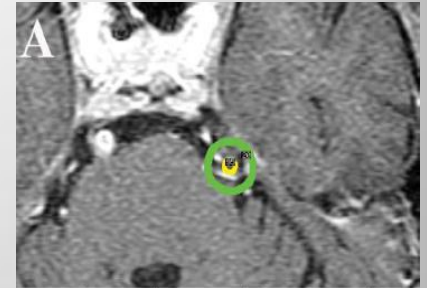
*30-78% population -Highest socioeconomic impact*  
At least 10 episodes occurring <1 day a month on average  
Infrequent episodes lasting from 30 minutes to 7 days  
Typically bilateral
- Medication over use headaches *30-78%*

# Neuropathic with 'neuralgia'

- Trigeminal neuralgia (TN)
  - » Typical
  - » Atypical
- Post herpetic neuralgia (PHN)
  - » > 50 yrs 60% likely to develop pain post shingles
  - » Ramsay Hunt syndrome
- Glossopharyngeal neuralgia
  - Acute pain pharynx, tongue base, mastoid regions
- Secondary sensory Neuropathy
  - Post traumatic V neuralgia
    - Lingual nerve injuries
    - Inferior alveolar nerve
  - Diabetes
  - HIV
  - Chemotherapy
  - MS

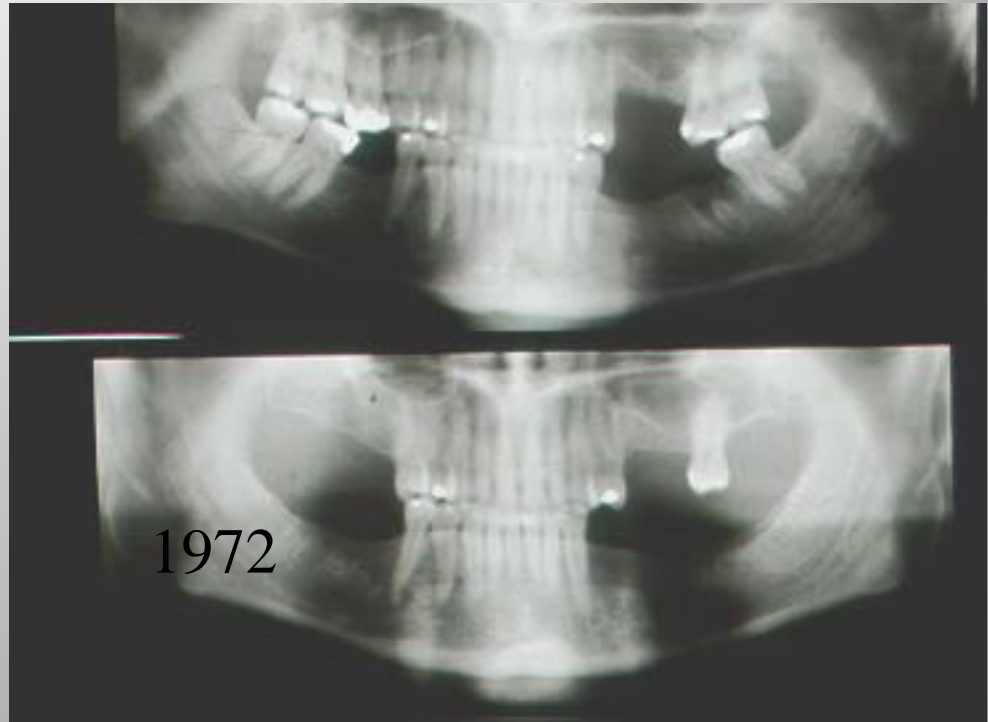
# Exclude central pathology

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



# Idiopathic chronic OFP

- TMD
  - » Functional - chewing gum
  - » Arthritides
  - » Derangement
- Persistent idiopathic
  - » Extraoral / facial
  - » Intraoral / odontalgia



1984



**Facial arthromyalgia**

**Tension headache**

**Atypical facial pain**

**Atypical odontalgia**

**BMS**

**Cervical pain**

**Fibromyalgia**

**Back pain**

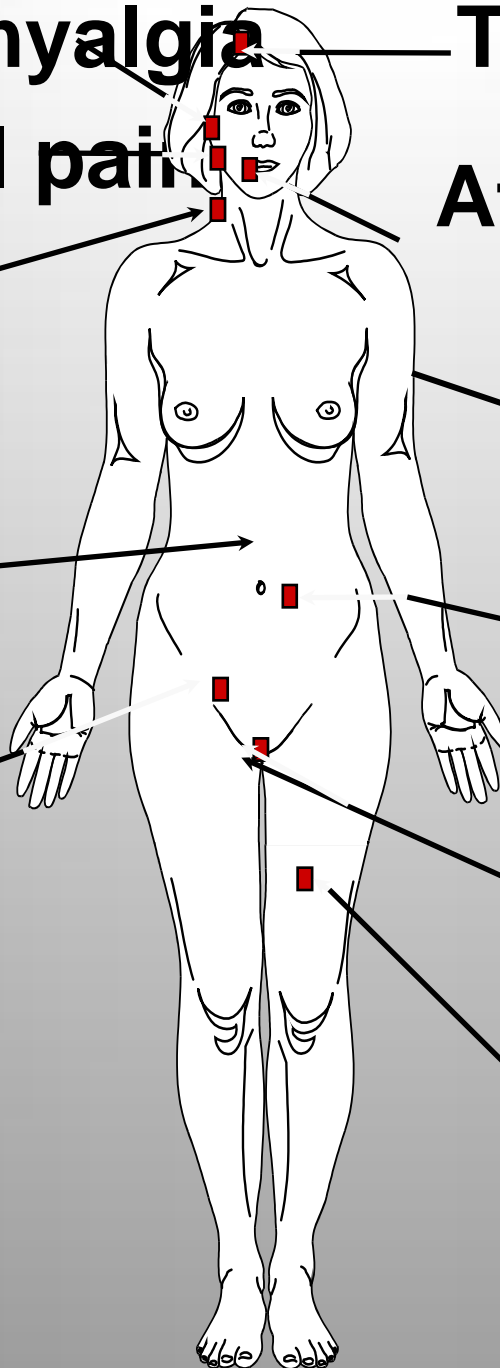
**Irritable  
bowel**

**Pelvic pain**

**Menorrhagia**

**Vulvodynia**

**Pruritus**



# Outline

- Overview pain
- An update on trigeminal pain
- An update on classification of pain
  - Excluding;
    - Headaches
    - Trigeminal Autonomic cephalgias
- Causes of neuralgia in the trigeminal system

# Causes of 'neuralgia' in the trigeminal system

## Most common

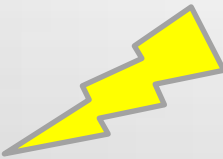

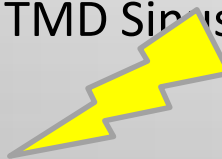

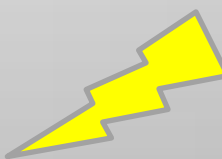
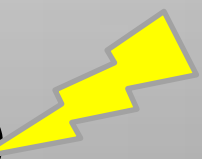
- Toothache
- TMD
- Post traumatic neuropathy
- Non dental pathology-cysts, SOLs, sinus, ear, salivary

## ○ Least common

- Secondary peripheral painful neuropathies
  - PHN getting rarer
- Trigeminal neuralgia
- SUNCT, SUNA
- IX neuralgia
- Nervous intermedius neuralgia



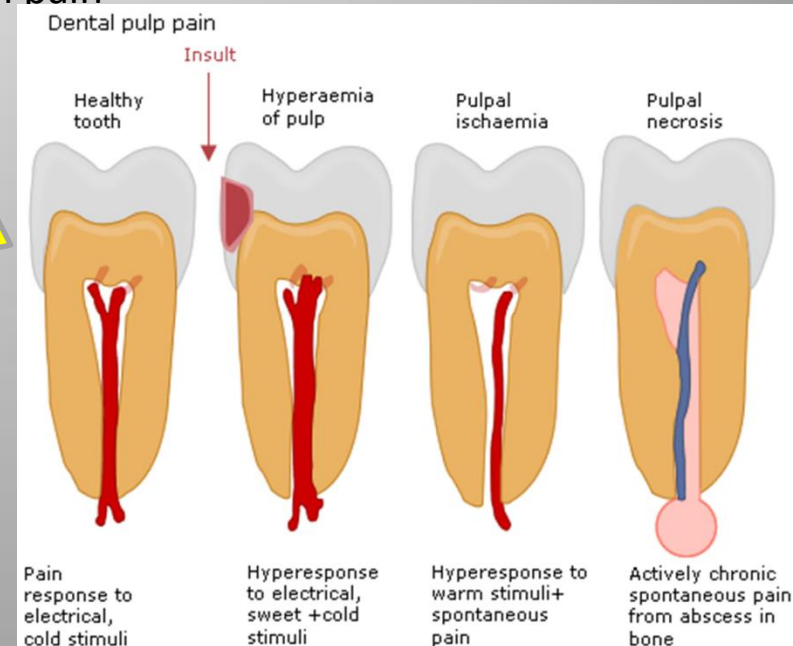
# Neuralgic V pain

- Nociceptive
  - Dentine sensitivity 
- Inflammatory
  - Odontogenic pulpitis, periapical abscess 
  - non odontogenic TMD Sinusitis Ear ache salivary gland
- Neurovascular
  - Headaches, TAC 
- Neuropathic
  - BMS, TN,  Secondary neuropathy 
- Dysfunctional
  - FM, 
  - Temporomandibular Disorder (TMD),
  - Myalgic, arthritides, dysfunctional
  - Persistent idiopathic facial pain (intra oral and extraoral)

# Acute V dental pain

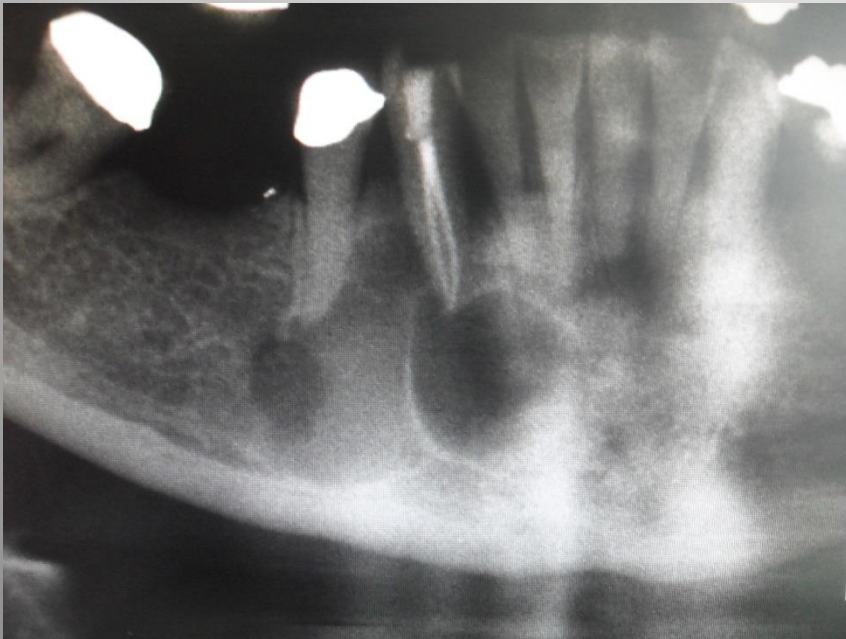
- **Odontogenic**
- **Healthy**
  - Dentine sensitivity
- **Inflammatory pain**
  - Dental impaction pain model post extraction/ surgical pain
  - Peri dental mucosal inflammation
    - Pericoronitis
    - Dental pulpitis
    - Irreversible pulpitis
    - Periapical periodontitis
  - Toothache –
- **Post surgical pain**
- **Chronic dental pain-**
  - Neuropathic dental pain

Nociceptive pain



# TN or toothache?

TNI





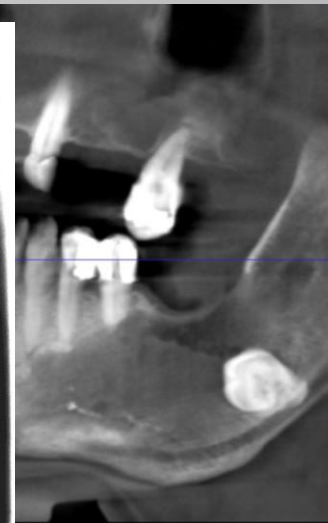
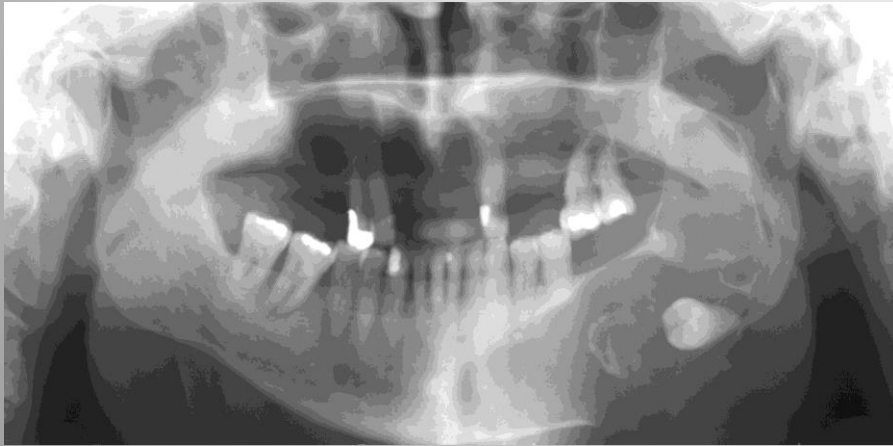
# ? TN or toothache?

TNI



# TN or toothache?

TNI

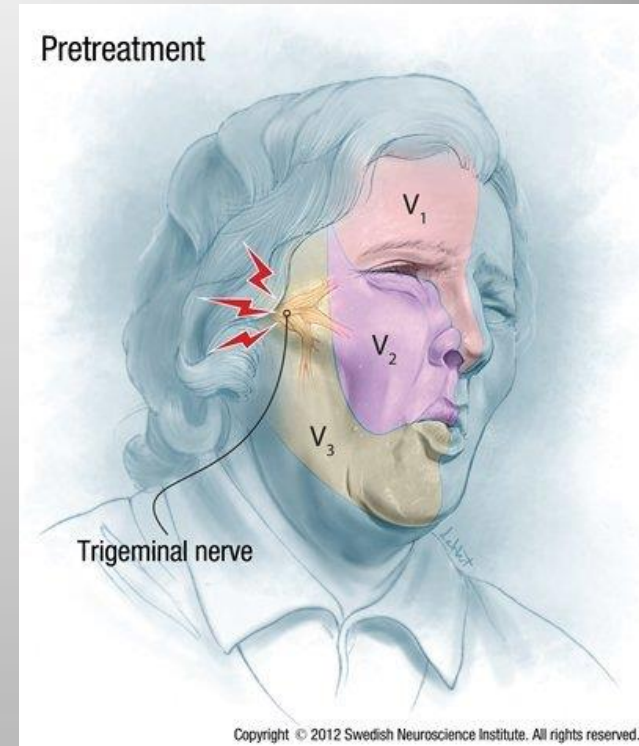


TN or toothache?



# Temporomandibular Joint Pain *Disorder* **TMD**

- Incidence
  - Prevalence
    - 20-50 year
    - No clinical radiographic pathology
    - Anxiety
    - Female:male 15:1
    - Spontaneous onset and cessation
    - DD TMJ trauma to joint / arthritides
- Other structures Otitis, Parotitis, ?headache, masseteric hypertrophy, infratemporal fossa Ca



## Commentary

## A new definition of neuropathic pain

**1. Introduction**

IASP has recently published a new definition of neuropathic pain according to which neuropathic pain is defined as “pain caused by a lesion or disease of the somatosensory system” ([www.iasp-pain.org/resources/painDefinition](http://www.iasp-pain.org/resources/painDefinition)). This definition replaces the 17-year old definition that appeared in the *Classification of Chronic Pain* published by IASP in 1994 [7], which defined neuropathic pain as “pain initiated or caused by a primary lesion, dysfunction, or transitory perturbation of the peripheral or central nervous system”. Even though the definition has not been changed dramatically, there are two important changes in the new version: (1) the word “dysfunction” has been removed and (2) a lesion or disease affecting the nervous system has been specified to be a lesion or disease of the somatosensory system.

**2. Background**

The history behind this change dates back several years with a long, and at times, heated debate about the inclusion of the term

nisms are known, but many are not. Increased understanding of pain mechanisms should put us in a better position to treat patients and design rational treatment strategies. There has indeed been progress since the last update of the neuropathic pain definition 17 years ago. For example, primary erythromelalgia and paroxysmal extreme pain disorder are both rare pain conditions for which we had no explanation 10 years ago, and therefore pain associated with these could not then have been classified as neuropathic. It is now clear that both disorders are due to specific and separable mutations in the SCN9A gene that codes for one of the many subtypes of neuronal voltage-gated sodium channels: the Na<sub>v</sub> 1.7 channel [1]. While these observations have not yet resulted in a specific or preventive treatment for the rare genetic pain states, there is now a clear target that can be addressed. Another pain condition that has seen progress is Fabry’s disease, which can now be treated with enzyme replacement therapy [6]. Biomarkers for an inflammatory component in neuropathic pain are also being discovered, and again, these may lead to new specific treatments. Other examples will certainly be added as our knowledge of diseases and their causes increases.

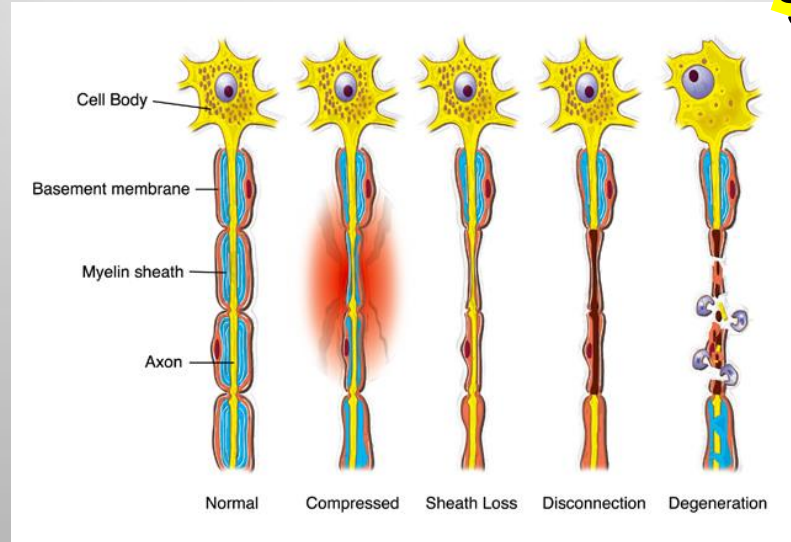


# Painful sensory neuropathy

Viral  
Herpes Zoster  
PHN  
HIV

Chemotherapy

Diabetes



**SURGERY & LA**

Alcoholism

Vitamin deficiency  
B 1,3,6,12, E

Radiation, Burns

Demyelination  
CTD, MS, GB

Post traumatic Peripheral sensory nerve injury  
PTN



# Trigeminal post traumatic neuropathy

- When is the Lingual nerve inferior alveolar nerve
  - Local analgesia
  - Wisdom teeth
  - Fractures
  - Pathology
  - **IAN**
    - Implants
    - Endodontics
    - Orthognathic surgery



# Possible Mechanisms of nerve injury

- **Chemical**

- Agent
- Vasoconstrictor
- Buffer
- Preservative
- Metabolites

**Hydrostatic pressure** from injection, direct mechanical injury to the nerve by the needle, or chemical injury from the local anesthetic solution itself (Haas, 2006)

Extrafascicular administration of clinically used concentrations of local anesthetic solutions may **alter perineurial permeability**, causing **endoneurial edema**, increasing endoneurial fluid pressure, causing **Schwann cell injury** and **axonal dystrophy** with endoneurial fibrotic changes as a late consequence (Myer et al., 1986).

- **Mechanical**

- Epineural, endoneurial, epi fascicular, endo fascicular
- Direct /Indirect

Increased perineurial permeability, resultant edema, and pressures intrafascicles, the normally hypertonic endoneurial fluid becomes hypotonic (Hogan, 2008). **Thus, a local anesthetic solution applied non-traumatically and externally to a peripheral nerve bundle may cause deleterious effects by increasing intraneural hydrostatic pressure.**

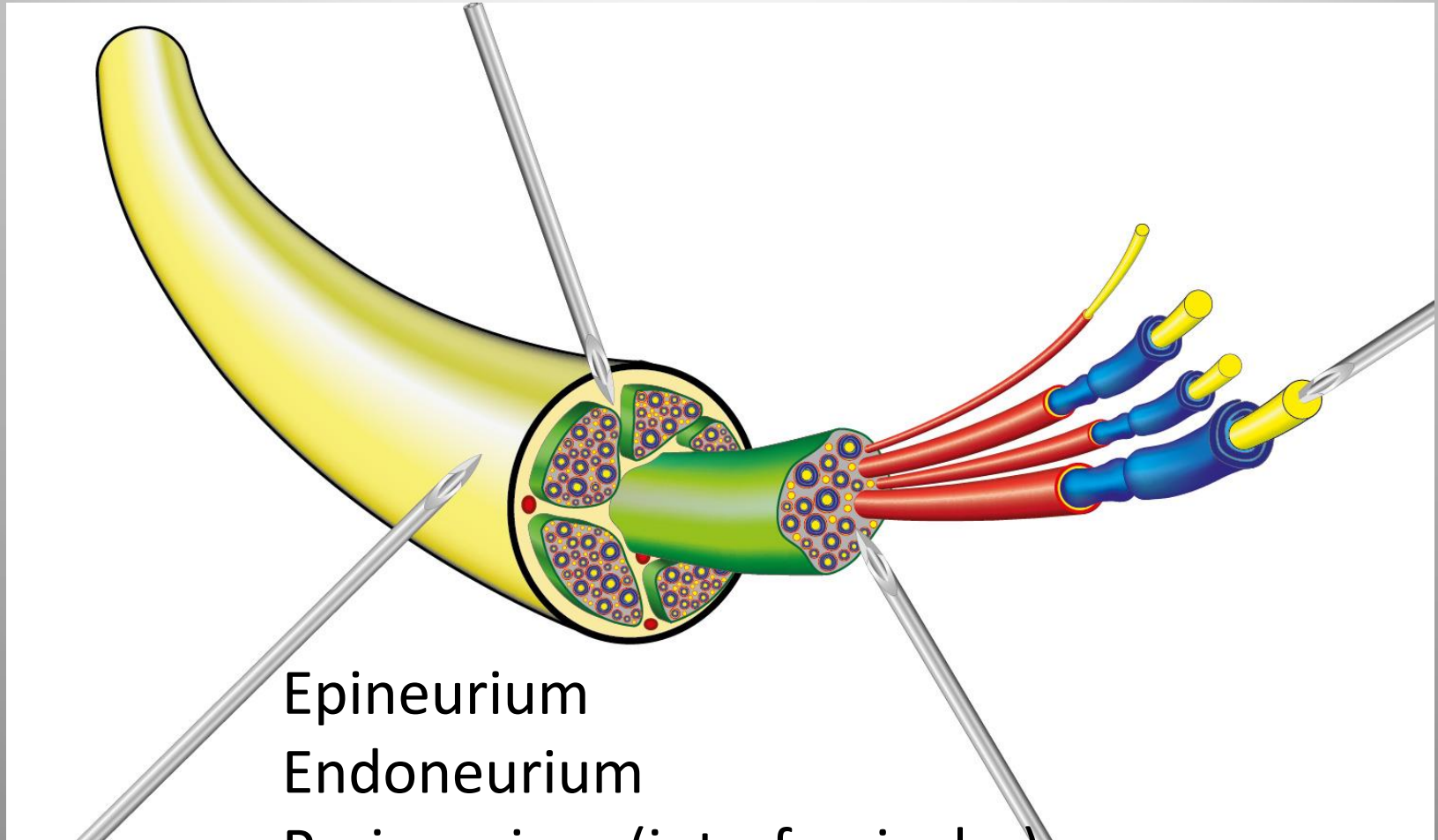
- **Haemorrhage**

- Chemical iron content very irritant to neural tissue
- Primary or secondary haemorrhage/ scarring epi or intra neural

- **Infection**

<http://trigeminalnerve.org.uk/>

# How does neural damage happen?



Epineurium

Endoneurium

Perineurium (intrafascicular)

Intraneural/ axonal A beta / A delta / C fibres

<http://trigeminalnerve.org.uk/>

# Types of tissue damage + possible mechanisms

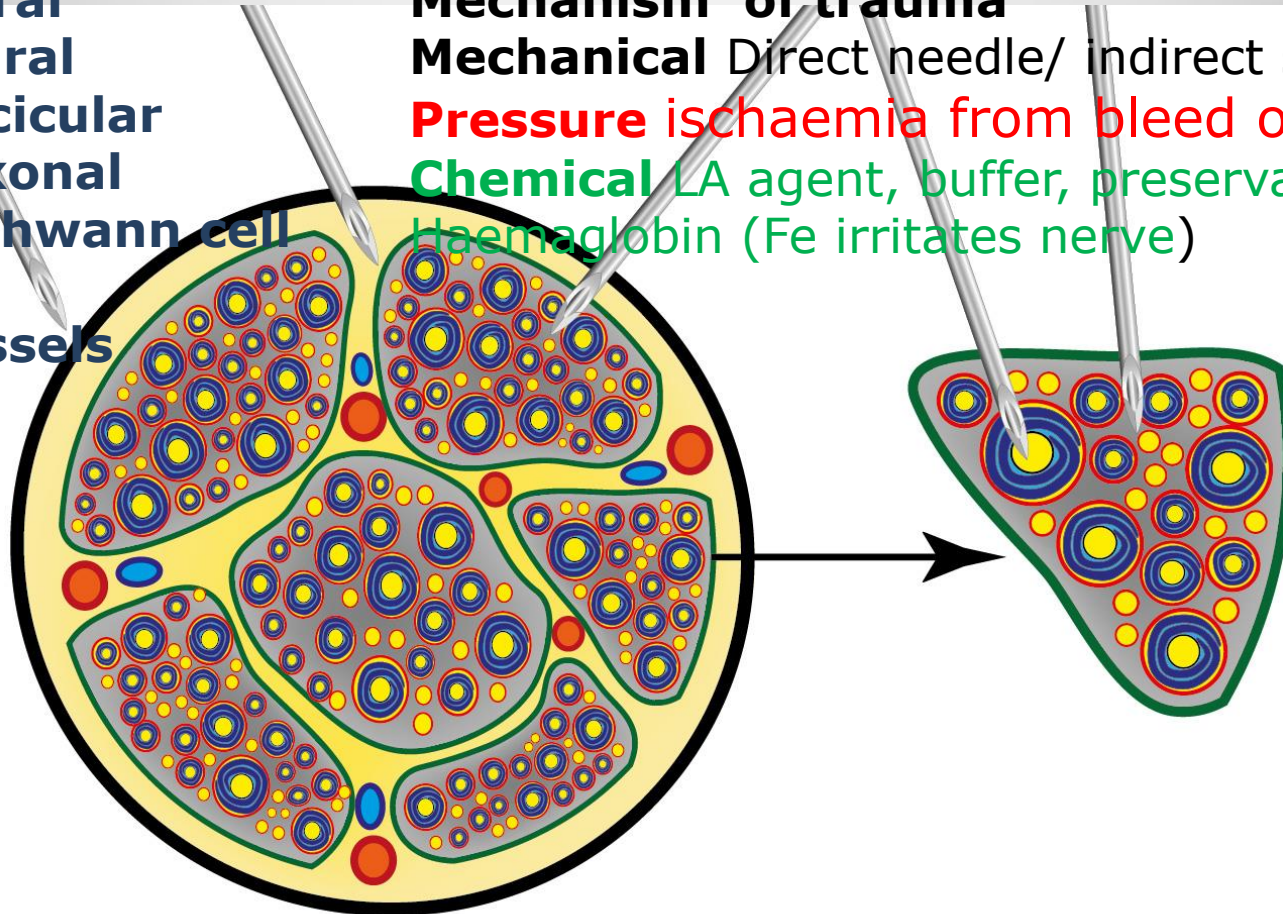
Extraneural  
Intra neural  
Intra fascicular  
Neural axonal  
Neural schwann cell  
(myelin)  
Blood vessels  
Fat

## Mechanism of trauma

**Mechanical** Direct needle/ indirect scarring

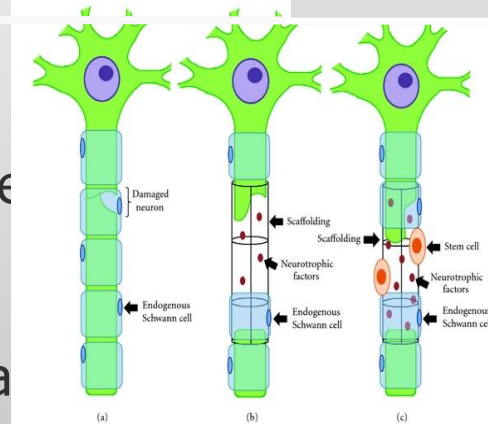
**Pressure** ischaemia from bleed or LA

**Chemical** LA agent, buffer, preservative, carrier  
Haemoglobin (Fe irritates nerve)



# Neural consequences

- Direct damage
- **Type A and B nerve block**
  - Ischaemia Inflammation (neuritis at any site)
  - Oedema
- **Prolonged conduction block**
  - Can recover 10-12 weeks with no myelin damage
  - If prolonged heamatoma or irritation then may not recover
- **Axonal disruption** (advancing Tinell's sign)
  - Axonal damage and degeneration
  - + endoneurium damage sensory motor mixing incomplete recovery
  - +perineurium distal degeneration and neuroma in-continuity
- **Unlikely resolution or recovery**
  - Disruption of epi, peri or endo neurium
  - Direct myelin damage
  - Wallerian degeneration(subsequent degradation of myelin)
- **Neurotmesis- nerve section** Requires Immediate repair





# Risk factors for PTN

- >50years
- Multiple insults

## Features

- Non respondent to anti inflammatory pain killers (NSAIDs Paracetamol)
- Better in mornings
- Does not disturb sleep
- Worsens during day
- Worsens with stress, tiredness and illness
- Either
  - Constant burning
  - Elicited neuralgic
  - Or combination

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



# Features of Neuropathic pain

TNI

- Pain      **hyper aesthesia**

**Allodynia pain** with non noxious stimulus  
pain on touch/cold/hot

**Hyperalgesia** increased pain to painful stimulus



- Altered sensation

Paraesthesia –pins and needles, formication, many descriptions

Dysaesthesia – uncomfortable sensations often burning

- Numbness- **hypo aesthesia**

# Consequences for the patient

620 patients with nerve injuries seen over 4 years at KCH

## **Pain**

**70% of Lingual or Inferior Alveolar Nerve injuries**

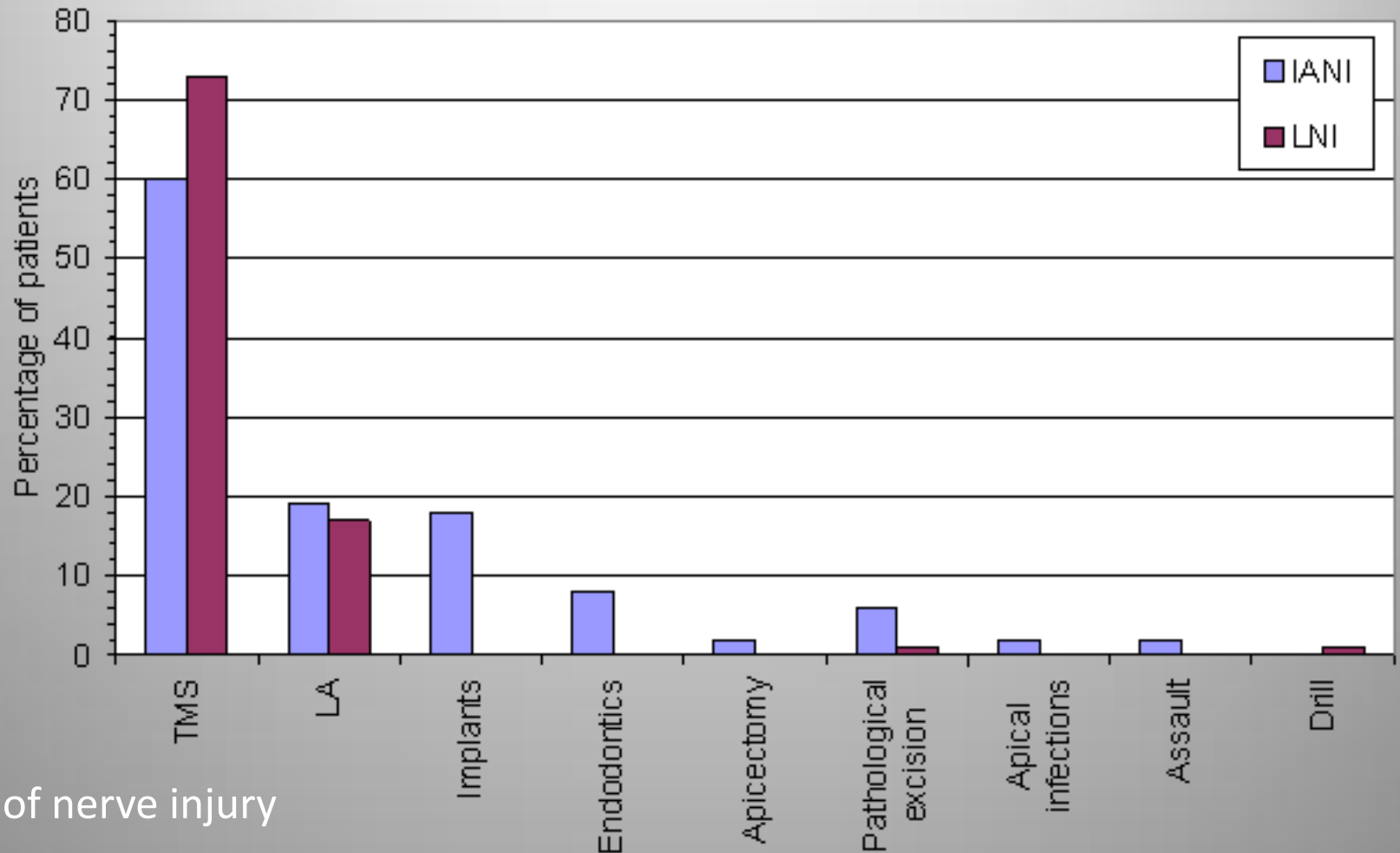
## **Functional**

Eating, speaking, drinking, sleeping, kissing, make-up, shaving, tooth brushing

## **Psychological**

50% chronic pain sufferers are depressed

# What procedures?



Risk of nerve injury

	Wisdom teeth	Local anaesthesia	Implants	Root canal	Orthognathic
Permanent	1 in 500	1in 47K	0.001-3%	???????	BSSO 14-20%
Temporary	2%	75% of 1 in 14K			

# Risk nerve injury M3M surgery

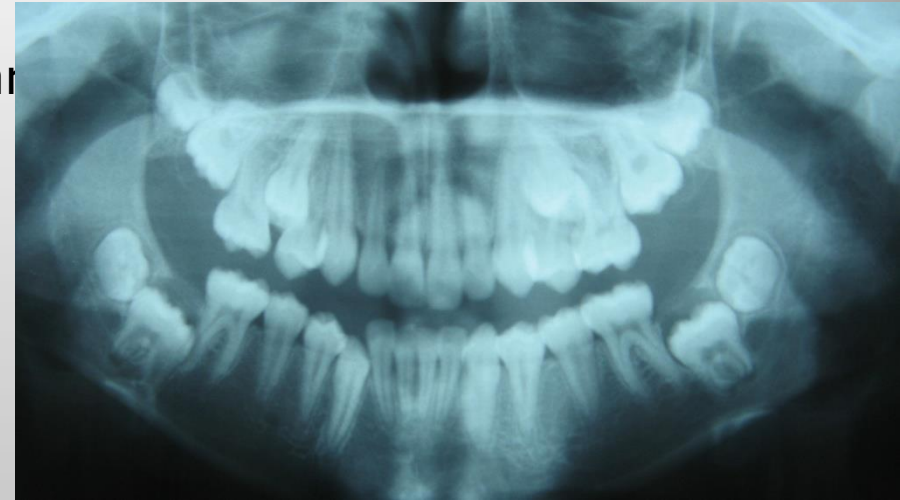
**10 million M3Ms removed** USA per year  
60% elective surgery

Costing **\$US 4.2 billion**

**11000 pts permanent nerve injury!**

**'Silent epidemic' of iatrogenic nerve injury**

No evidence substantiates prophylactic removal. Freidman J Am J Public Health 2007;97:1554



- Mythology of 8s
- Overall 12% associated with pathology
  - same as appendicitis and cholecystitis
  - 8% pericoronitis
  - 3% caries lower 7s
  - 0.048% resorption of adjacent tooth
  - 0.0085% internal resorption
  - 0.0165% cyst formation

# Inferior alveolar nerve injury



# Prevention of IAN injury

TNI

teeth can be high risk when crossing  
Inferior Dental Canal

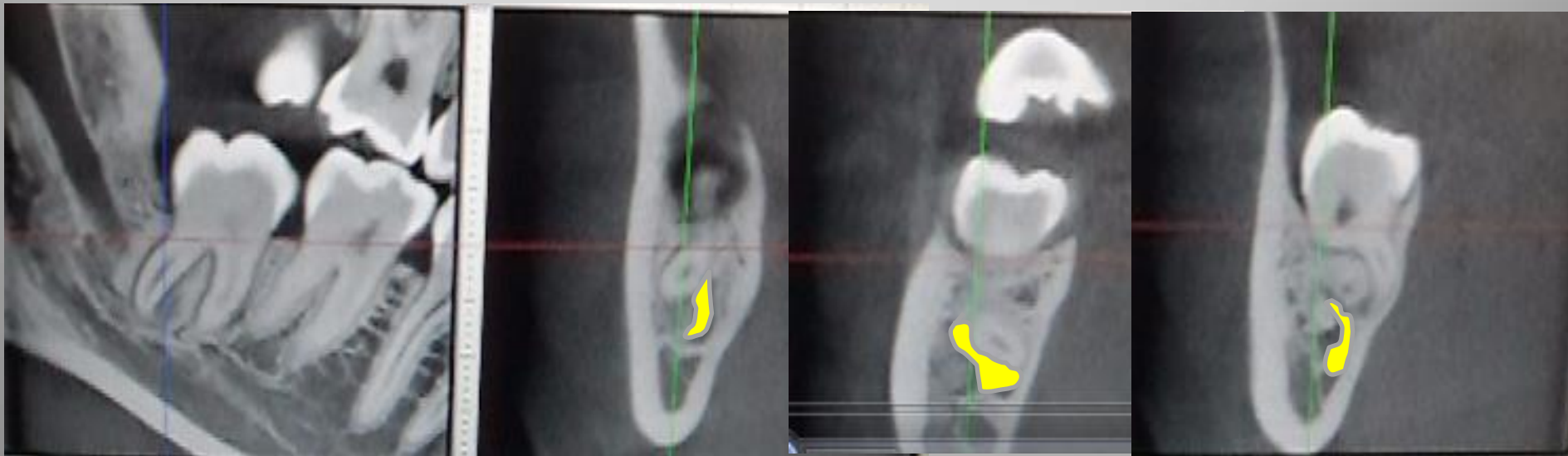




# Prevention of IAN injury

TNI

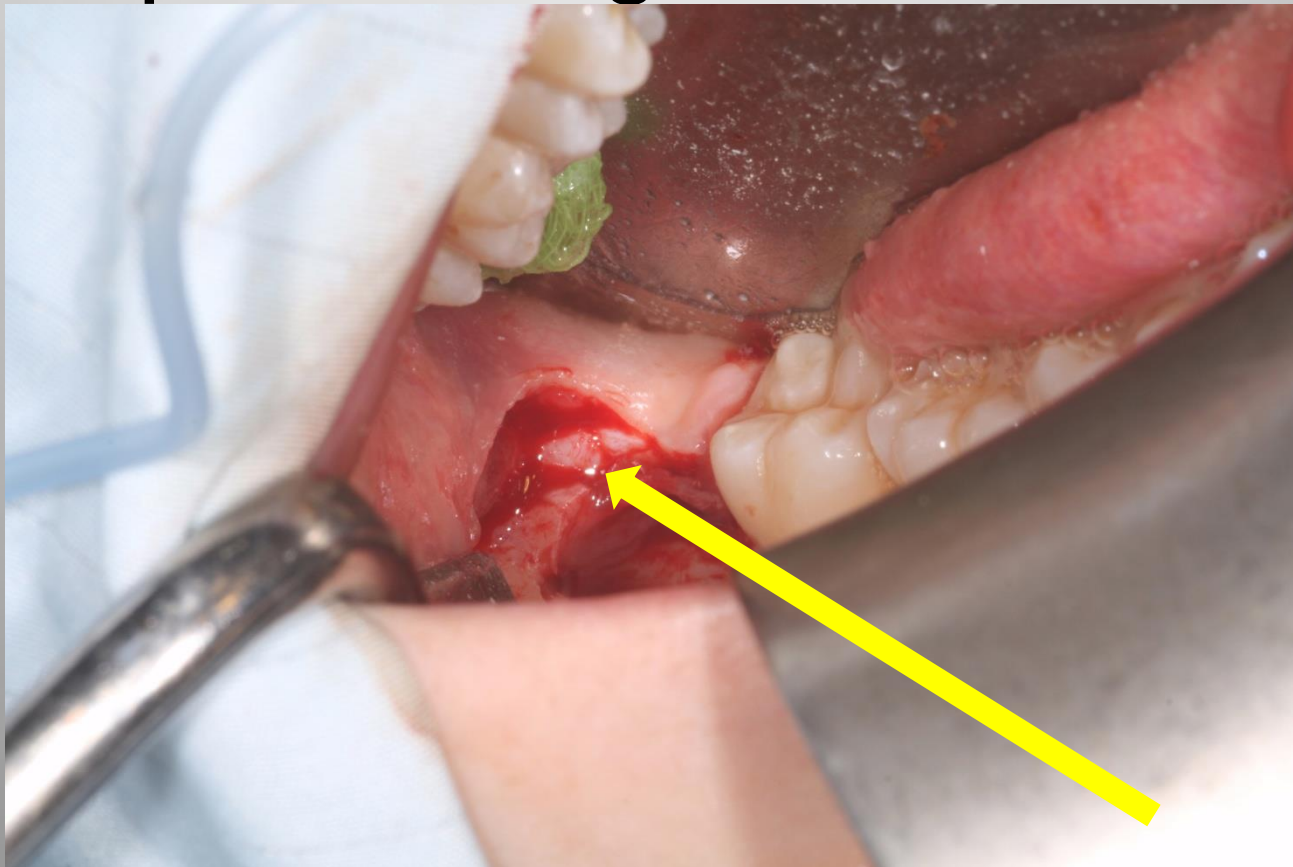
The nerve doesn't have to 'perforate' tooth.....



'Snake' nerves

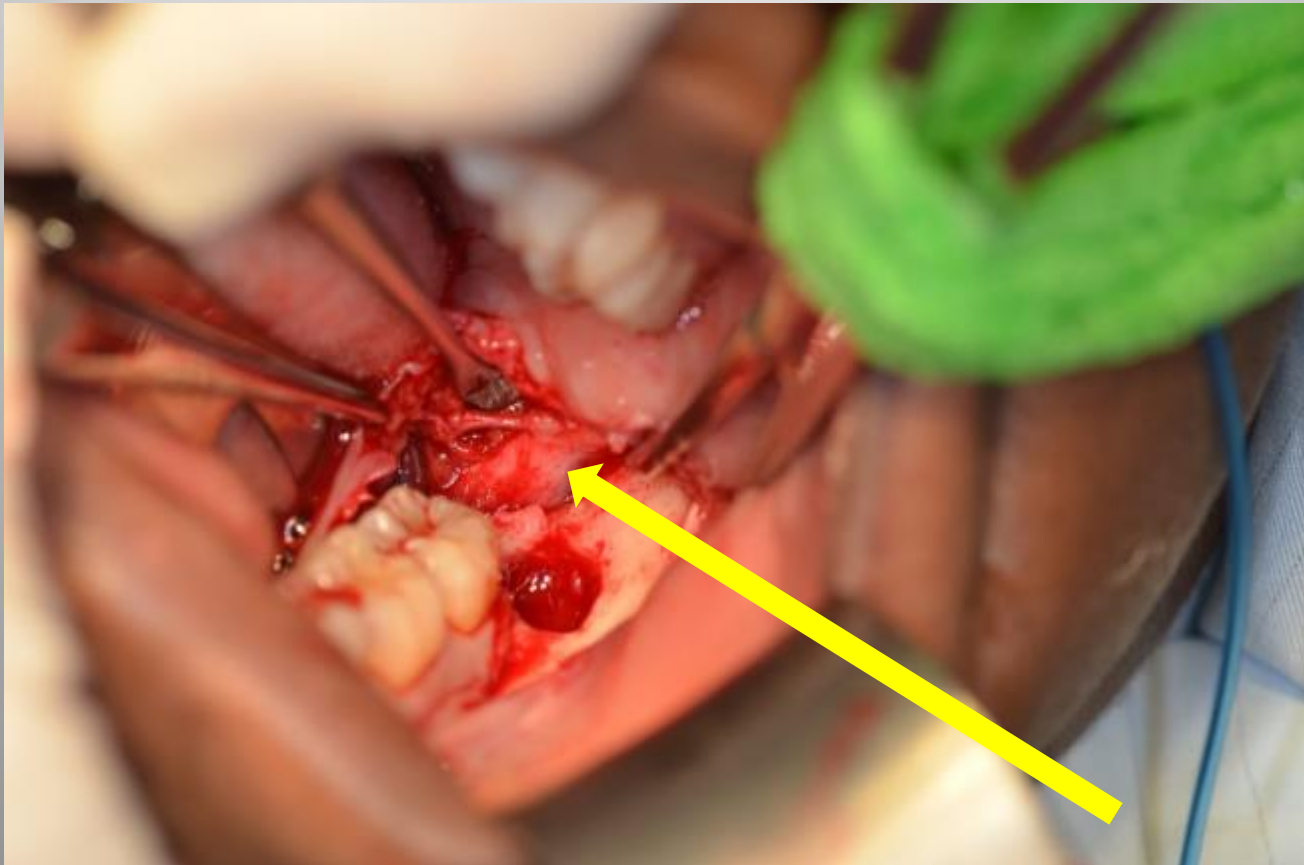
# Prevention of lingual nerve injury in

Spot the lingual nerve!



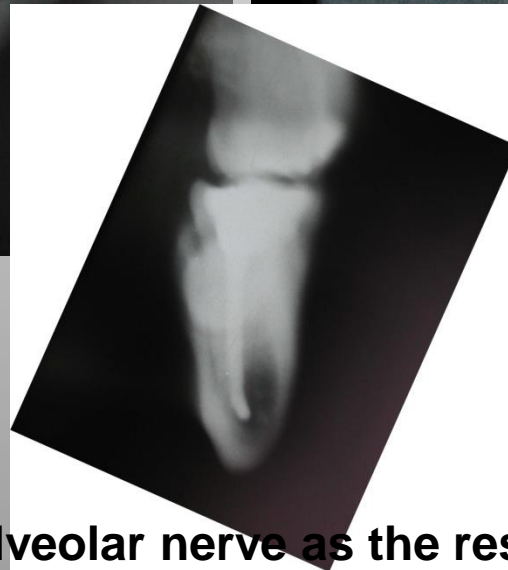
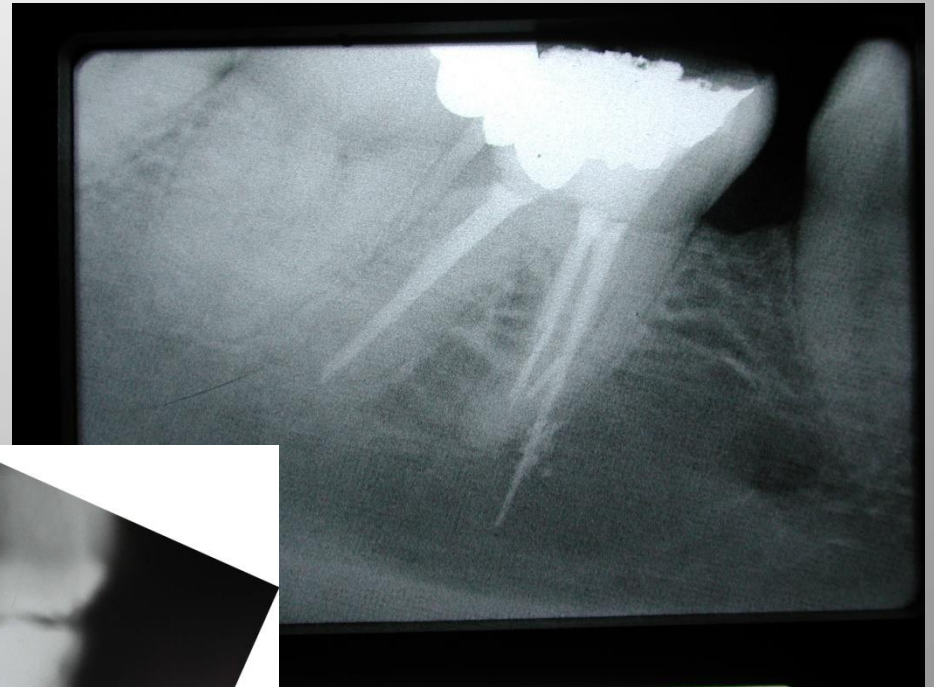
# Prevention of lingual nerve injury in

## During lingual nerve exploration



# Prevention of root canal IAN injury

TNI

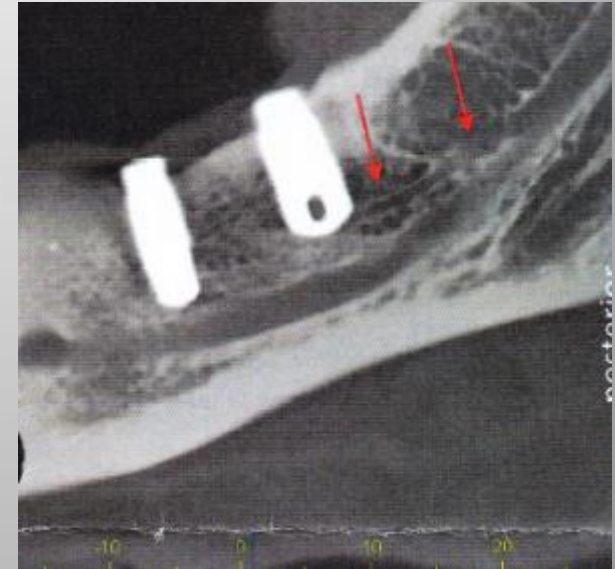
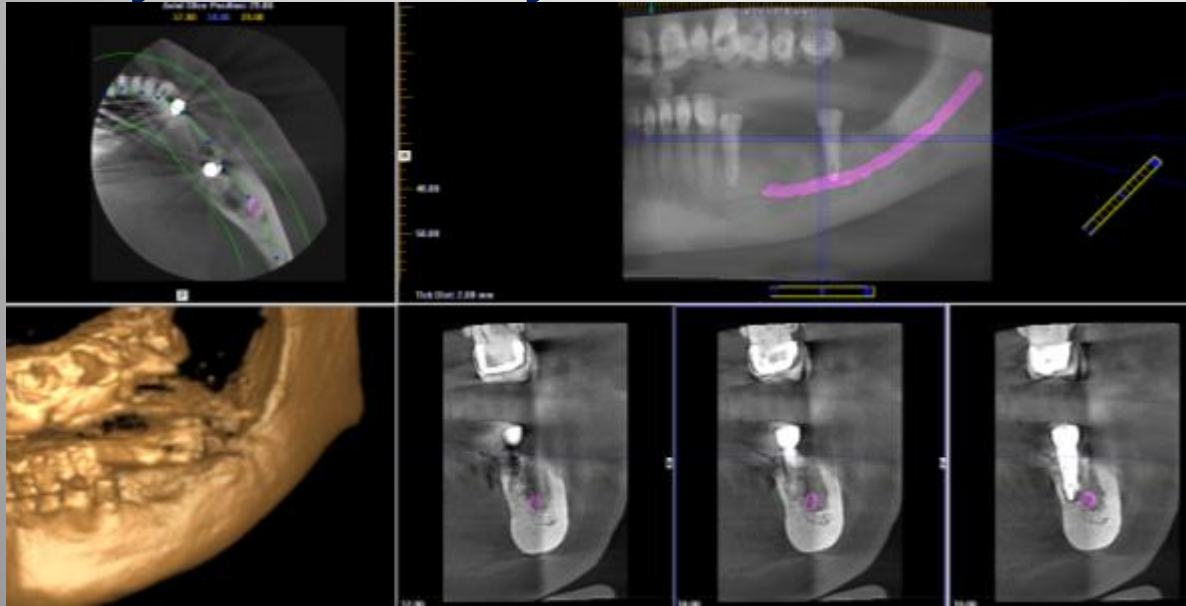


**Damage to the inferior alveolar nerve as the result of root canal therapy.**  
**Pogrel MA. J Am Dent Assoc. 2007 Jan;138(1):65-9**



# Prevention of dental Implant nerve injury

Are you sure you know where the nerve is?



# Novel Management of nerve injuries

Mechanism	Duration	Treatment
Known/suspected nerve section		Immediate exploration
TMS IANI –retained roots	<30 hours	Immediate exploration
Implant	<30 hours	Remove implant
Endodontic	<30 hours	Remove tooth / overfill
Implant / Endodontic	>30 hours	Treat therapeutically
TMS IANI large neuropathic area, pain and disability	<3 months	Consider exploration
TMS LNI – large neuropathic area, pain and disability	<3 months	Consider exploration
TMS IANI –	>6 month	Treat therapeutically
TMS LNI–	>6 month	Treat therapeutically
LA, fracture, orthognathic		Treat t therapeutically



# TRIGEMINAL FOUNDATION

## Nerve Injuries

Helping to prevent, educate and manage



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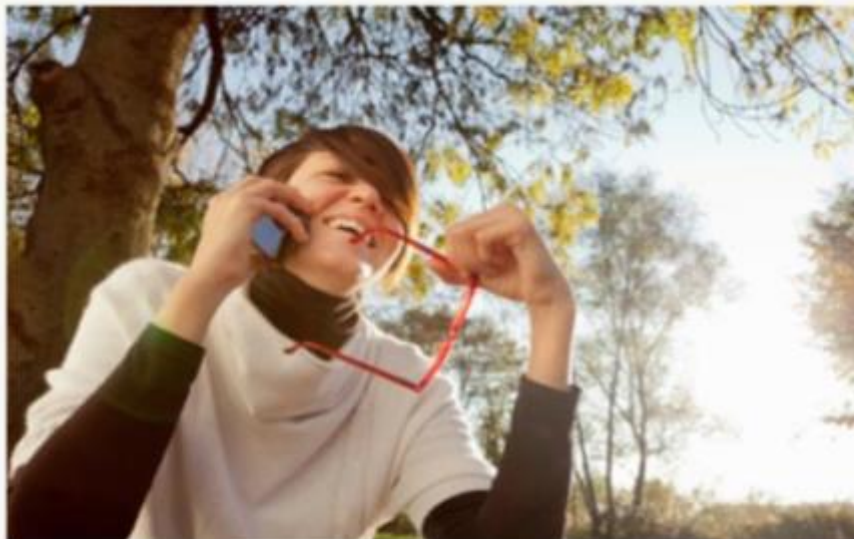
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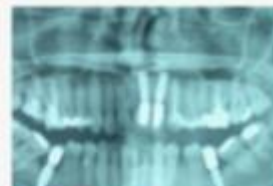
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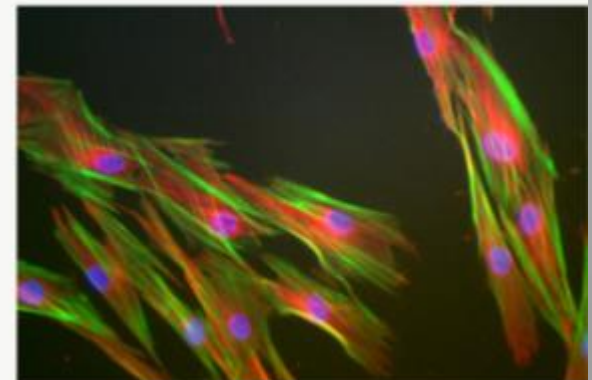
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Latest from DNI blog 

# Trigeminal neuralgia

IASP defines trigeminal neuralgia as

**“ a sudden, usually unilateral, severe, brief, stabbing, recurrent pain in the distribution of one or more branches of the fifth cranial nerve”.**

- Incidence rate of trigeminal neuralgia 4.3 per 100,000 population in Rochester Minnesota, for 1945 through 1984
- The age-adjusted (to total 1980 US population) rate for women (5.9) was significantly higher than that for men (3.4).
- Annual incidence rates increased significantly with age in both women and men. Data based on evidence in the medical records suggest that trigeminal neuralgia is a rather rare and unpredictable disease:
- The number of episodes varies from 1 to 11, and length of episode from 1 day to 4 years.

- Trigeminal Neuralgia

- If patient < 40 years exclude MS any age consider Parkinsons

Ann Neurol. 1990 Jan;27(1):89-95. Incidence and clinical features of trigeminal neuralgia, Rochester, Minnesota, 1945-1984.

Katusic S1, Beard CM, Bergstralh E, Kurland LT.

# Trigeminal Neuralgia

- Character of pain
  - Flashing, shooting, sharp, unbearable
  - elicited
- Severity
  - Moderate to severe
- Site, radiation
  - Distribution of trigeminal nerve
- Duration, periodicity
  - Bouts last for seconds, pain free periods
- 5<sup>th</sup>-6<sup>th</sup> decade
- F:M 3:2
- V2 and V3
- Can be bilateral
- Does not occur at night
- Responds to Tegretol
- Absent autonomic signs
- No neuropathic area

# TN Investigations

- MRI – patients under 40 years  
to exclude multiple sclerosis and to assess if micro  
vascular compression
- CT - tumours of posterior fossa
- Haematological tests
- Biochemical tests
- Neurological – sensory testing and hearing



## MRI scan

Diagnosis and differential  
diagnosis of trigeminal  
neuralgia

Zakrzewska JM.

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

# Other Neuralgias

## – Post Herpetic Neuralgia

- 20% of patients (60% > 50 yrs) progress to neuropathic pain after Shingles caused by a reactivation of the varicella-zoster virus (VZV).
- In the trigeminal system most commonly V1 and V2
- If patient is < 40 years check immuno status (15 times higher in HIV-infected patients )
- If caught early treat with high dose antivirals
  - Acyclovir (Zovirax)† 800 mg orally five times daily for 7 to 10 days  
10 mg per kg IV every 8 hours for 7 to 10 days
  - Prednisone 30 mg orally twice daily on days 1 through 7; then 15 mg twice daily on days 8 through 14; then 7.5 mg twice daily on days 15 through 21
- Ramsay hunt syndrome HZ of geniculate ganglion (facial nerve, CT)

## – Post Traumatic Neuralgia

- Avoid trigeminal nerve injury



# Post ophthalmic herpes zoster – hyperaemia and corneal scarring

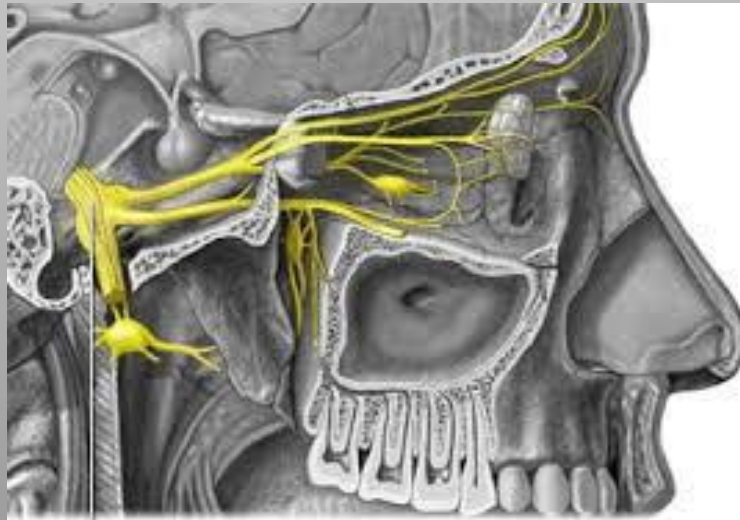


- Always consider immune compromise in pts presenting with HZ
- Aggressive therapy with antivirals, steroids and anticonvulsants has minimised progression to PHN

Thank you

<http://trigeminalnerve.org.uk>

<http://orofacialpain.org.uk> (Dec  
2014)





# Orofacial Pain

Demystifying chronic pain  
in the trigeminal system

info@orofacialpain.co.uk



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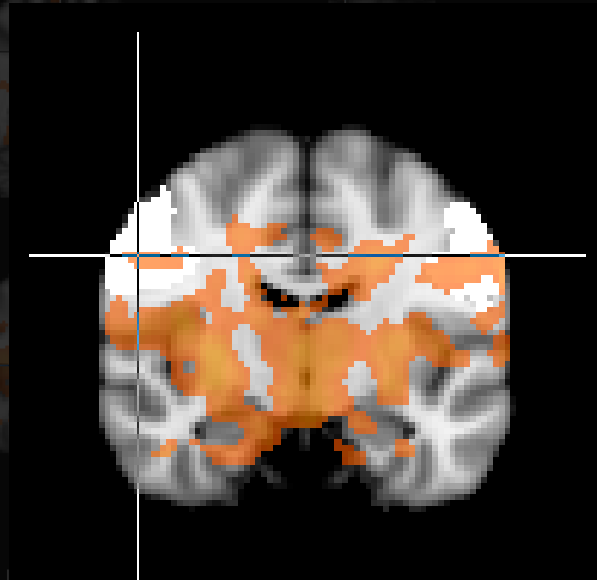
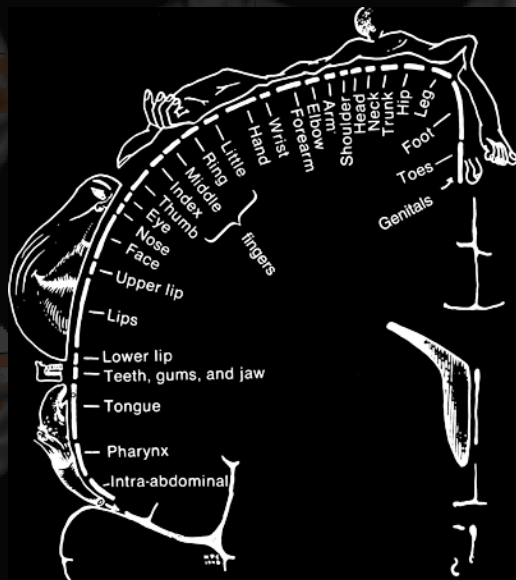
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Launching December 18th 2014

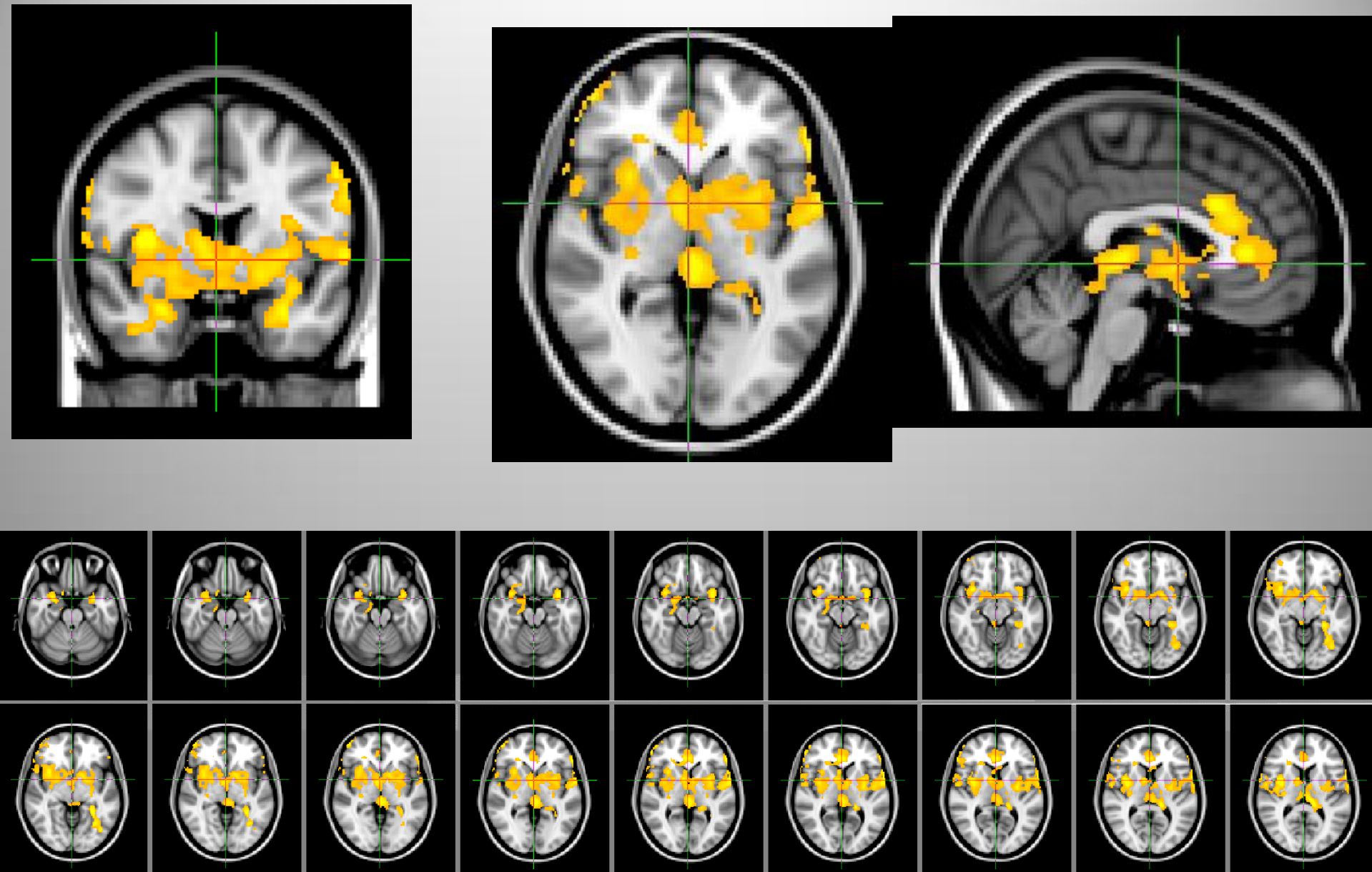
# Anatomy revisited



•Additional amygdala, hippocampus, brainstem, and V5 ROIs



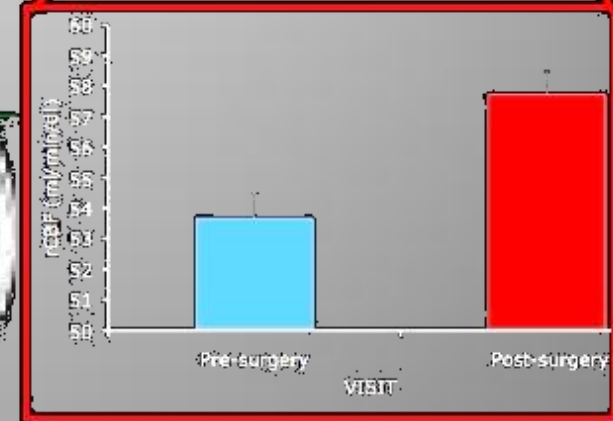
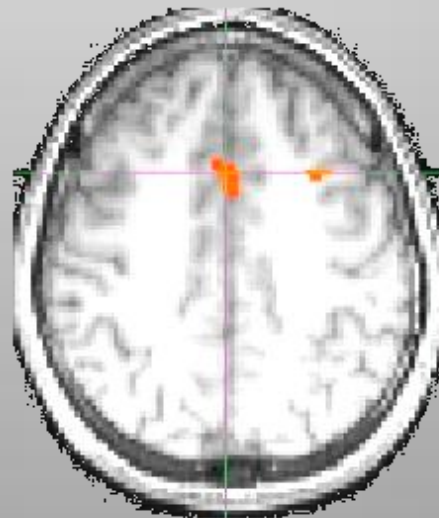
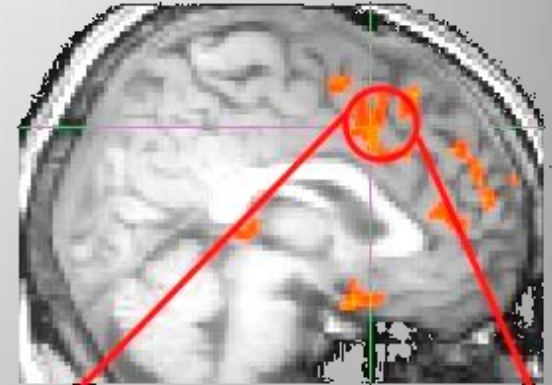
Main effect of TME pain, right tooth, cluster corrected  $\alpha < 0.05$



# Central pain activity

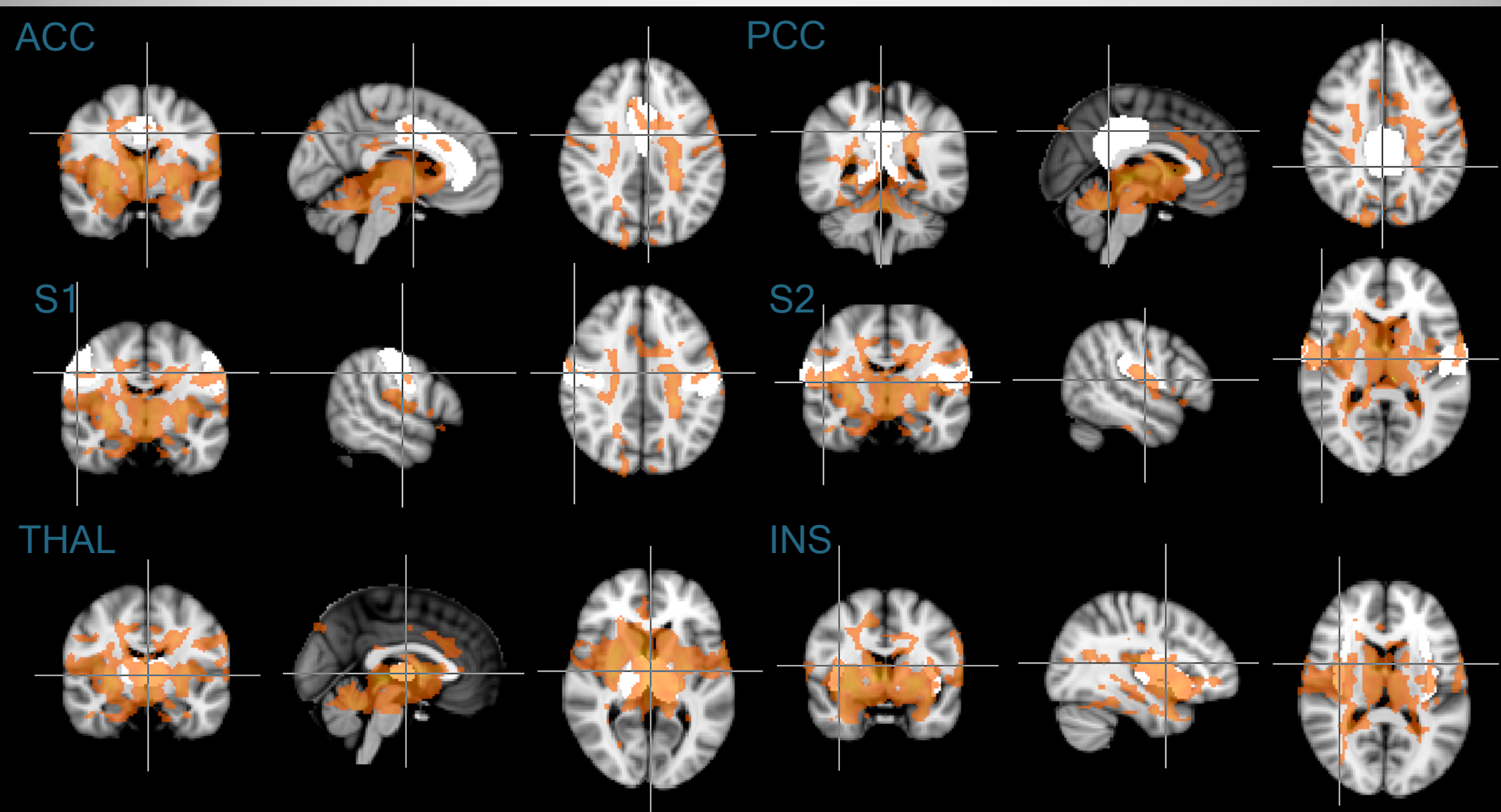
## – Pain related areas

- Spinal cord C1-S5
  - C1-8/T1-12/L1-5/S1-5
  - distal root ganglion
    - » Ventral horn = motor
    - » Dorsal horn = sensory
- Brain stem
  - Cranial nerve
  - Thalamus
  - Hypothalamus
  - Cerebellum
- Forebrain
  - Cortex-sensation
  - Limbic system -memory
  - Basal ganglia-movement





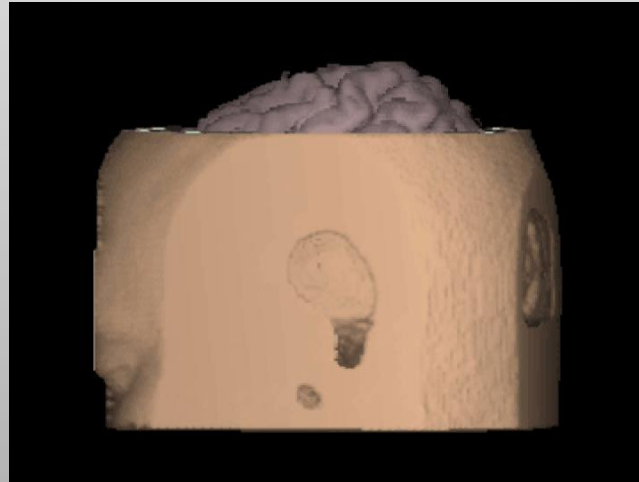
# Anatomy revisited



• Additional amygdala, hippocampus, brainstem, and V5 ROIs

# Pain in the Brain

## fMRI video



# The genetic basis of V pain

