Neuralgia in the trigeminal system

Tara Renton
Professor Oral Surgery KCL

Trigeminalnerve.org.uk
Neuralgia ˌnɪəˈraldʒə/
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

Input
- Cognitive
- Emotion
- Sensory

Neuromatrix

Output
- Pain
- Motor
- Stress
- Emotion

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

- Nociception
- Sensation/perception
- Behaviour
- Suffering

Do genetics influence all of these factors?
Pain process

- Nociception
- Sensation
- Behaviour
- Suffering
Rugby player

Nociception
Sensation
Behaviour
Suffering
Football player

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

Inflammatory soup
The ‘Pain Matrix’ brain areas reactive to pain
26 areas of the brain affected so far!
Modulation
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 neurological 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

What is this thing called pain?

Clifford J. Woolf.

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    • Trigeminal Autonomic cephalgias
• Causes of neuralgia in the trigeminal system
Orofacial pain

Local causes of pain
- Ears
- Pharynx
- Jaw / Temporomandibular Joint
- Eyes
- Nose/sinus
- Dental

Other causes
- Vascular
- Psychogenic
- Neurological
- Other
- Referred
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

• Arthritis
  – 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 dentoalveolar pain
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The trigeminal system
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).

Theories of dental nociception

**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

In relation to dental innovation........

Allodynia is NORMAL!

Attrition
Abrasition
Erosion

TNI
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 idopathic 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

Dental caries (tooth rot)
Dental pulp pain

Healthy tooth → Insult → Hyperaemia of pulp → Pulpal ischaemia → Pulpal necrosis

Pain response to electrical, cold stimuli
Hyperresponse to electrical, sweet + cold stimuli
Hyperresponse to warm stimuli + spontaneous pain
Actively chronic spontaneous pain from abscess in bone
Dental abscess
Cracked tooth
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The classification and differential diagnosis of orofacial pain


Tara Renton¹, Justin Durham² and Vishal R Aggarwal³

¹Department of Oral Surgery, Kings College London Dental Institute, Denmark Hill Campus, Denmark Hill Road

There are currently four main pain classification systems relevant to orofacial pain (OPF): 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

<table>
<thead>
<tr>
<th>Neurovascular</th>
<th>Neuropathic</th>
<th>Idiopathic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headaches</td>
<td>Trigeminal N</td>
<td>Persistent idiopathic (ATFP / ATO)</td>
</tr>
<tr>
<td>MOH</td>
<td>Classic/ symptomatic</td>
<td>PDAP</td>
</tr>
<tr>
<td>Chronic daily</td>
<td>PHN</td>
<td>Temporomandibular Disorders</td>
</tr>
<tr>
<td>Tension HA</td>
<td>Glosspharyngeal N</td>
<td>Dysfunctional Athritides</td>
</tr>
<tr>
<td>Migraine</td>
<td>Burning Mouth Syndrome</td>
<td>Arthromyalgia</td>
</tr>
<tr>
<td>Trigeminal autonomic cephalgias</td>
<td>Secondary Sensory Neuropathy</td>
<td></td>
</tr>
<tr>
<td>Cluster HA</td>
<td>DM, MS, GB, Chemo, Thyroid D, Vit Deficiency</td>
<td></td>
</tr>
<tr>
<td>SUNCT</td>
<td>Post Traumatic</td>
<td></td>
</tr>
<tr>
<td>SUNA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemifacial Cont</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Praoxysmal Hemicrania</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Giant cell arteritis</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Woda A et al 2005
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
Exlude 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
Menorrhagia
Vulvodynia
Pelvic pain
Back pain
Cervical pain
Atypical facial pain
Atypical odontalgia
Tension headache
BMS
Fibromyalgia
Irritable bowel
Menorrhagia
Vulvodynia
Facial arthromyalgia
Facial arthromyalgia
Pruritus
Pruritus
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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 Sinusitus Ear ache salivary gland

• Neurovascular
  – Headaches, TAC

• Neuropathic
  – BMS, TN, Secondary neuropathy

• Dysfunctional
  – FM,
  – Temporomandibular Disorder (TMD)
  – Myalgic, arthritides, dysfunctional
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Acute V dental pain

• **Odontogenic**
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  - **Inflammatory pain**
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      - Pericoronitis
    - Toothache –
      - Dental pulpitis
      - Irreversible pulpitis
      - Periapical periodontitis
  - Post surgical pain
  - **Chronic dental pain**
    - Neuropathic dental pain

Nociceptive pain

---

TN or toothache?
? TN or toothache?
TN or toothache?
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). 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 mechanisms with neurological, and pain mechanisms in pain states. 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+ 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

Post traumatic Peripheral sensory nerve injury
PTN

Alcoholism
Vitamin deficiency B 1,3,6,12, E
Radiation, Burns
Demyelination CTD, MS, GB
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

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Chemical</strong></td>
<td></td>
</tr>
</tbody>
</table>
  - **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 *endoneural edema*, increasing endoneural fluid pressure, causing *Schwann cell injury* and *axonal dystrophy* with endoneural fibrotic changes as a late consequence (Myer et al., 1986). |
| **Mechanical**    |  
  - **Epineural, endoneural, epi fascicular, endo fascicular**  
  - **Direct /Indirect**  
    - Increased perineural permeability, resultant edema, and pressures intrafascicles, the normally hypertonic endoneural 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/](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

Haemaglobin (Fe irritates nerve)

http://trigeminalnerve.org.uk/
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 Tinells 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

- >50 years
- 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

Table 2
Definitions of common features suggestive of neuropathic pain

<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paresthesia</td>
<td>An abnormal sensation, whether spontaneous or evoked</td>
</tr>
<tr>
<td>Dyesthesiain</td>
<td>An unpleasant sensation, whether spontaneous or evoked</td>
</tr>
<tr>
<td>Hypoesthesia</td>
<td>Decreased sensitivity to stimulation (tactile or thermal; both are frequent)</td>
</tr>
<tr>
<td>Hyperesthesia</td>
<td>Increased sensitivity to stimulation (tactile or thermal; both are rare)</td>
</tr>
<tr>
<td>Hypoalgesia</td>
<td>Diminished pain response to a normally painful stimulus</td>
</tr>
<tr>
<td>Hyperalgesia</td>
<td>An increased response to a stimulus that is normally painful</td>
</tr>
<tr>
<td>Allodynia</td>
<td>Pain due to a stimulus that does not normally activate the nociceptive system</td>
</tr>
</tbody>
</table>
Features of Neuropathic pain

- **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**  
Permanent: 1 in 500  
Temporary: 2%

**Local anaesthesia**  
1 in 47K

**Implants**  
0.001-3%

**Root canal**  
75% of 1 in 14K

**Orthognathic**  
BSSO 14-20%
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


- 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

teeth can be high risk when crossing Inferior Dental Canal
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

Damage to the inferior alveolar nerve as the result of root canal therapy.
Prevention of dental Implant nerve injury

Are you sure you know where the nerve is?
<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Duration</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Known/suspected nerve section</td>
<td></td>
<td>Immediate exploration</td>
</tr>
<tr>
<td>TMS IANI – retained roots</td>
<td>&lt;30 hours</td>
<td>Immediate exploration</td>
</tr>
<tr>
<td>Implant</td>
<td>&lt;30 hours</td>
<td>Remove implant</td>
</tr>
<tr>
<td>Endodontic</td>
<td>&lt;30 hours</td>
<td>Remove tooth / overfill</td>
</tr>
<tr>
<td>Implant / Endodontic</td>
<td>&gt;30 hours</td>
<td>Treat therapeutically</td>
</tr>
<tr>
<td>TMS IANI large neuropathic area, pain and disability</td>
<td>&lt;3 months</td>
<td>Consider exploration</td>
</tr>
<tr>
<td>TMS LNI – large neuropathic area, pain and disability</td>
<td>&lt;3 months</td>
<td>Consider exploration</td>
</tr>
<tr>
<td>TMS IANI –</td>
<td>&gt;6 month</td>
<td>Treat therapeutically</td>
</tr>
<tr>
<td>TMS LNI –</td>
<td>&gt;6 month</td>
<td>Treat therapeutically</td>
</tr>
<tr>
<td>LA, fracture, orthognathic</td>
<td></td>
<td>Treat therapeutically</td>
</tr>
</tbody>
</table>
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
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
- 5th-6th 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 microvascular 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.
Other Neuralgias

– Post Herpetic Neuralgia
  • 20% of patients (60%>50yrs) 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 antifungals
    – Acyclovir (Zovirax)† 800 mg orally five times daily for 7 to 10 day 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)
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

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Tmp21-I (p23) (AJ004912)
C4 methyl sterol oxidase (D50559)
Protein convertase subtilisin/kexin type 5 (U4701)
Adapter-related protein complex 2 alpha (X53773)
Calmodulin-dependent calcineurin A alpha (M29275)
Neurabin-II (AF016252)
GRG protein (L14462)
Complexin 1 (D70817)
Chromogranin A (X00832)
Preptic regulatory factor-2 (K53232)
Type III brain 4 1 (AB032227)
Palmitoyl-protein thioesterase 1 (L34262)
Chemokine receptor-like 1 (AJ002745)
ICAM-1 (H.M.012967)
Gamma-glutamyl hydrolase (U38379)
SGK (Q01624)
Cathrin heavy chain (J03583)
Fracture callus protein 1 (AF150106)
Lost on transformation (U72620)
Synaptogyrin 1 (U39549)
Testis-specific AKAP (AJ002474)
BPI protein (J39851)
Calcium-binding protein 1 (Y17048)
Synaptogyrin 3 (D28512)
Cytochrome c oxidase (X72757)
Tyrosine phosphatase-like (D38222)
Stac (AF089330)
Apolipoprotein E (J02582)
Isoferate dehydrogenase (X74125)
WAP four-disulfide core domain (AF037272)
PKC and CK substrate in neurons 1 (AF104402)
Hsp60 (AF187860)
Dihydropteridine reductase (J03481)
VQF, NGF inducible (M00025)
CGRP (L00110)
Hsp27 (M86365)
RT1.AA (Z49761)
Peripherin (AF31879)
RT1.Mb (Z49762)
C1q beta (X71127)
TIMP-1 (L31833)
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