Painless Management of the Oral Surgical Patient: Are We There Yet?

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Love conquers all things except poverty and toothache.

Mae West
An update on pain

- An update on classification of pain
- Perioperative techniques to minimise pain
- Managing and preventing post operative pain
- Surgical methods
- What's new?
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 inevitable, suffering is optional’

(Haruki Murakami)
Pain is complex

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: Consequences UK

- 33% of UK population suffer
- 13% work force is compromised
- Diabetic and HIV neuropathy
- Accounts for £80 billion year UK
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)
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
- Can it be socially endemic?

Definition of pain

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

(IAASP, 1979)
Pain process

Nociception
Sensation
Behaviour
Suffering

TNI
Rugby player

- Nociception
- Sensation
- Behaviour
- Suffering
Pain Process

Nociception
Sensation
Behaviour
Suffering
Pain Process

- Nociception
- Sensation/perception
- Behaviour
- Suffering

Bio psycho social Model

Social / Cultural
- Age, gender, race,
peer support,
familial expectation

Cognitive / Conceptual
- Memories past
experience,
secondary gain,
threat perception

Emotional / psychological
- Depression,
anxiety, stress,
fear, anger

Do genetics influence all of these factors?
Nociception
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

Nociceptive drive
Sensation
The ‘Pain Matrix’ brain areas reactive to pain
26 areas of the brain affected so far!
Behaviour

- Phenotype
- Genotype
- Epigenetics
Affective emotional areas of Brain

- Sensory Regions
  - Primary Sensory Cortex
  - Thalamus
  - Posterior Insula

- Emotional/Affective
  - Anterior Cingulate
  - Posterior Cingulate
  - Orbitofrontal Cortex
  - Medial Prefrontal Cortex
  - Anterior Insula
  - Accumbens
  - Hippocampus
  - Thalamus
  - Amygdala
  - Caudate

- Cognitive/Integrative
  - Prefrontal Cortex
  - Temporal Lobe
  - Parietal Cortex

Pain and Empathy brain activity visualizations.
Perception of pain
Perception of pain

'I enjoy the pain'
David Beckham on tattoos

Opus Dei Priest ‘Pain is good’
Suffering

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

- Personality
- Religion
- Placebo
- Anger
- Catastrophising
- Fear
Catastrophising

Genetics of pain

- Melanocortin 1 receptor def –Mu opioid 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,”

2009 that redheads were more anxious about dental treatment and more than twice as likely to avoid a visit.

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

• An update on classification of pain
• Perioperative techniques to minimise pain
• Managing and preventing post operative pain
• Surgical methods
• Whats new?
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

Post surgical pain

Chronic neuropathic pain

Post traumatic neuropathy PDAP/PHN

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 ‘dysfunction’ with neurological, injury, and other mechanisms 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_v 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.
Acute healthy pain  →  Chronic pain = Neuromatrix disease

- **Peripheral drivers**
  - Neurotrophins
  - Altered receptor threshold

- **Central drivers**
  - Glial cells, Neurotrophins
  - Reduced downward modulation, changes in brain structure

- **Affective / behavioural drivers**

- **Genetics**

- **Social?**
The great protector......

Sensory feedback for all cranial functions

Brains- Consciousness + neural regulation

Breathing

Sight

Smell

Taste

The face...the organ that underpins communication
The trigeminal nerve

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.
Trigeminal nerve

Largest sensory nerve in the body
Trigeminal nerve

Complex region
Consequences

Social function
Eating
Drinking
Speaking
Kissing
Make up / shaving
Sleeping

IDENTITY?
An update on pain

• An update on classification of pain
• **Perioperative techniques to minimise pain**
• Managing and preventing post operative pain
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• What's new?
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?
Recommendations for prescribing antibiotics in dentistry

There are few guidelines for prescribing antibiotics in dentistry..............

- Scottish Dental Clinical Effectiveness Programme (2011) Drug Prescribing For Dentistry: Dental Clinical Guidance. 2nd Ed

- FGDP Guidance Antimicrobial prescription in dentistry 2006
  [http://www.fgdp.org.uk/content/publications/antimicrobial-prescribing-for-general-dental-pract.ashx](http://www.fgdp.org.uk/content/publications/antimicrobial-prescribing-for-general-dental-pract.ashx)

- BSAC recommendations for antimicrobial prescription for dental procedures

It is known that dentists over prescribe antibiotics rather than extract or extirpate the dental cause.

Over the last decade GMP prescription of antibiotics has significantly reduced whereas GDPs prescribe antibiotics even more!

- [http://www.rcseng.ac.uk/fds/Documents/FDJ_Vol.1_Issue%201.pdf](http://www.rcseng.ac.uk/fds/Documents/FDJ_Vol.1_Issue%201.pdf)
SDCEP prescribing in dentistry

Drug Prescribing For Dentistry

The second edition of 'Drug Prescribing For Dentistry' can be downloaded in pdf form.

'Drug Prescribing For Dentistry' is now available as an app, Dental Prescribing, for use on iPhone®, iPad® or iPod touch®.

Updates
November 2013
Managing the patient’s expectations

• Patient’s pain expectation
• Intraoperative pain experience
• Post treatment pain
• Pain complications
  – Extreme- nerve injury?
  – Persistent- neuropathic?
  – Recurrent pain- infection?
How do we minimise the pain?

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


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

Get well soon leaflet

Get Well Soon
Helping you to make a speedy recovery after removal of wisdom teeth

This leaflet is a guide to recovering from an operation to remove one or more wisdom teeth. It does not provide specific medical advice or diagnosis. Nor does it give advice about whether you should consent to an operation. All of these matters depend on individual medical advice from your consultant surgeon based on your own health, medical condition, and personal circumstances.
Managing patients expectations of surgical related pain is effective in pain relief!

Complications best avoided

- Fore arm the patient
  - Have an honest conversation about risks

**VALID CONSENT**

- Do you have the correct diagnosis?
- Can you handle the medical complexity?
- Are you able to undertake the procedure?
- DON’T overestimate your ability or talent!
  - Would you do this on your daughter/friend????????
- Can you manage the possible complications?

- **If NO to any of the above....** Ask for assistance get training or even better..........REFER?
Be Honest!

Come on!!
The suspense is killin’ me!
Which one’s ours?
Sadism
A willingness or tendency to subject others of oneself to unpleasant or trying experiences.

mas·och·ism (m s -k z m)
Psychological factors driving pain


Alternative and holistic management of pain


Clinician understanding and empathetic Good treatment planning Managing patients expectations
Distraction techniques

GOING TO THE DENTIST
more pleasant than I remember it to be

verydemotivational.com
Management – Alternative
Self empowerment  Counselling Acceptance Mindfullness

- Laughter
- Distraction
- Stress management - relaxation
- Exercise
- Social support
- Hypnosis
- Acupuncture
- Aromatherapy
- Pets
- Hobbies
Tapping into natural resources

- Maximising downward inhibition of pain
- Sleep
- Hypnotism
- Meditation
- Education...managing expectations............
So have you……

- Informed the patient? -Consent
- Identified their anxiety level?
  - Index of sedation need IoSN
- Identified if LA is contra-indicated?
  - Previous LA failed
  - Allergy to LA
  - Spreading infection making LA difficult
  - Operative area

The Indicator of Sedation Need (IOSN)

Introduction

Indications for sedation need are usually indicated because a patient's anxiety is producing undue stress for the patient, the staff or both. The use of sedation may be to assist the patient in becoming more cooperative, to reduce the need for drugs or to enable staff to perform their work more effectively. The indicators of sedation need (IOSN) were developed to help support decisions about whether a patient's anxiety, sedation or behavior is a potential problem, and whether or not a patient should be medicated for one or more of these reasons. The indicators of sedation need (IOSN) are designed to help support decisions regarding how to manage patients, but do not replace clinical judgment.

Accuracy

Some patients are more accurate than others. An initial indication of a patient's anxiety, sedation or behavior is that they are more cooperative, more calm and more relaxed than usual. This may be associated with a reduction in the need for drugs or for sedation. The indicators of sedation need (IOSN) are designed to help support decisions about whether a patient's anxiety, sedation or behavior is a potential problem, and whether or not a patient should be medicated for one or more of these reasons. The indicators of sedation need (IOSN) are designed to help support decisions regarding how to manage patients, but do not replace clinical judgment.
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So how can we prevent / manage the pain?
Main predictors of Post TMS pain

• Heat pain perception
  Thermal thresholds using QST

• Psychological vulnerability

J Orofac Pain. 2010 Spring;24(2):189-96.
Prediction of postoperative pain after mandibular third molar surgery.
Rudin A, Eriksson L, Liedholm R, List T, Werner MU
Management of Pain Process

Nociception → Sensation → Behaviour → Suffering

Use Local anaesthesia to block off nociceptive pain!

EASY

Social / cultural
Age, gender, race, peer support, familial expectation

Cognitive / conceptual
Memories past experience, secondary gain, threat perception

Emotional / psychological
Depression, anxiety, stress, fear, anger
Is that it?

- Sadly not........

- Patient is still conscious
- Anxious
- Fearful
- Compliant?
Management of Pain Process

Nociception

Sensation

Behaviour

Suffering

Depends upon Clinician role

communication
diagnosis
surgery
medical management

Patient factors
Pre operative
Information
Anxiolysis
Analgesia
LA

Social / cultural
Age, gender, race, peer support, familial expectation

Cognitive / conceptual
Memories past experience, secondary gain, threat perception

Emotional / psychological
Depression, anxiety, stress, fear, anger
Pre-emptive analgesia

• **Some benefit**

• **No benefit**
Pain after surgery: Can protective analgesia reduce pain? randomised clinical trial

Sin Leong Yong, Paul Coulthard
School of Dentistry, The University of Manchester, Higher Cambridge Street, Manchester M13 9PL, UK

Received: May 19, 2009; Received in revised form: February 15, 2010; Accepted: March 3, 2010; Published Online 12 Sep 2010

DOI: 10.1002/14651858.CD008392.pub2

Results
122 patients entered the study providing 98 evaluable patients for analysis. Patients in the protective analgesia group reported more pain than those in the conventional group at 30 min, 1, 6 and 48 h following surgery, although this difference was only statistically significant at the 30 min time point. 62.4% of patients required rescue analgesia after surgery. The median time for patients who had to take rescue analgesia was 3.1 h. Patients in the protective analgesia group reported a longer time to rescue analgesia compared with those in conventional analgesia. Overall, 91.7% of patients were at least satisfied with their pain control.

Conclusion
There was no difference in the protective analgesia group compared with conventional analgesia group in improving postoperative pain experience. A different protective analgesia regime may be necessary, which employs a more aggressive and multimodal strategy for postoperative pain management.

Keywords:
Manage the Pain Process

Bio psycho social Model

- Social / cultural
  - Age, gender, race, peer support, familial expectation

- Emotional / psychological
  - Depression, anxiety, stress, fear, anger

- Cognitive / conceptual
  - Memories past experience, secondary gain, threat perception

LA, Spinal Block
Antiinflammatory analgesics
Membrane stabilising drug

Sedation, CBT

CBT
Where do drugs work?

- Opiates central block of NMDA receptors
- Tramadol is a very weak $\mu$-opioid receptor agonist, induces serotonin release, and inhibits the reuptake of norepinephrine
- Sedation blocks central GABA receptor pathway = anxiolytic
- Paracetamol: cyclooxygenase inhib, induces serotonin release,
- NSAIDS peripheral block - cyclooxygenase
- Local analgesia blocks all sodium nerve channels (motor and sensory)
Actual LA nerve injury incidence

- GDP restorative procedures
  - 1 in 14K
  - 25% permanent

- Oral surgery
  - 1 in 3.3K
  - 29% permanent

Compare this with anaesthetic LA block procedures. NAP3 reports the estimated that nerve injury resulting from neuroaxial blocks (epidurals, spinals and combined epidural with spinals) resulted in sensory or motor nerve injury in 1 in 24-54K patients (and paraplegia or death in 1 in 50-140K patients)
Local anaesthesia

Smart LA

- Articaine 4% Buccal Infiltration
- +/- IDB Lidocaine 2%

- Articaine 4% Buccal Infiltration
- Post + ant near Mental foramen
- +/- Lingual Inf Lidocaine 2%

- Buccal infiltration + Lingual both Lidocaine 2%

No palatal blocks required!

- Anesth Prog. 2013 Summer;60(2):42-5. doi: 10.2344/0003-3006-60.2.42. Comparison of buccal infiltration of 4% articaine with 1 : 100,000 and 1 : 200,000 epinephrine for extraction of maxillary third molars with pericoronitis: a pilot study.

- Lima JL Jr, Dias-Ribeiro E, Ferreira-Rocha J, Soares R, Costa FW, Fan S, Sant'ana E. Prospective, double-blind, controlled clinical trial involved 30 patients between the ages of 15 and 46 years who desired extraction of a partially impacted upper third molar with pericoronitis
Successful extractions in

Incisors-premolars 90%
M1Ms 60%
M2Ms 75%

Prospective audit 280 extractions by dental UGs
-no palatal blocks given
- Articaine infiltration
- Lidocaine IDB rescue

87% success!
Medical pain management

- Anxiolysis
  - Chairside manner- Education & reassurance
  - Hypnosis
  - Acupuncture
  - Indication for sedation need
    - Oral, inhalational, IV sedation

- Analgesia
  - Pre-operative analgesia
  - Intraoperative- LA
  - Post operative
    - paracetamol + ibuprofen **GOLD standard** for third molar surgery / extractions

- Review/ Homecheck
Post surgical pain

- **Getting the pain you expect: mechanisms of placebo, nocebo and reappraisal effects in humans.**
- **Tracey I.**
- **Author information**
- **Abstract**
- The perception of pain is subject to powerful influences. Understanding how these are mediated at a neuroanatomical and neurobiological level provides us with valuable information that has a direct impact on our ability to harness positive and minimize negative effects therapeutically, as well as optimize clinical trial designs when developing new analgesics. This is particularly relevant for placebo and nocebo effects. New research findings have directly contributed to an increased understanding of how placebo and nocebo effects are produced and what biological and psychological factors influence variances in the magnitude of the effect. The findings have relevance for chronic pain states and other disorders, where abnormal functioning of crucial brain regions might affect analgesic outcome even in the normal therapeutic setting.
Post surgical pain
Patients get the pain they expect
Pain and the context

Elisa Carlino, Elisa Frisaldi & Fabrizio Benedetti
Affiliations
Contributions
Corresponding author
doi:10.1038/nrrheum.2014.17
Published online
Post surgical pain
Patients get the pain they expect

Getting the pain you expect: mechanisms of placebo, nocebo and reappraisal effects in humans
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Getting the pain you expect: mechanisms of placebo, nocebo and reappraisal effects in humans
Irene Tracey
Placebo effect-hypnosis, meditation, suggestion

Getting the pain you expect: mechanisms of placebo, nocebo and reappraisal effects in humans
Irene Tracey
Analgesics

- Pre prandial
- During
- Post prandial
Optimal timing for analgesia
worst pain happens at 2-4am after surgery

Seymour J. J One Day Surg 1997
Anatomy revisited

• Additional amygdala, hippocampus, brainstem, and V5 ROIs
Level of pain

Drives analgesic selection
What is the level of Post TMS pain?

UK reported pain levels
3-5

USA reported pain levels
>7

**Medical analgesics**

**WHO analgesic ladder**

**Rescue medication**
- NSAIDs
- Tramadol
- Pethidine
- Morphine
- IV lidocaine/LA blocks
Medical- analgesics

WHO analgesic ladder

- Rescue medication
- NSAIDs
- Tramadol
- Pethidine
- Morphine
- IV lidocaine/LA blocks
Single dose paracetamol (1g) 50% post op pain relief for 4 hours

Codeine not effective as NSAIDs or paracetamol for TMS pain

Aspirin better than paracetamol for Post TMS pain

Review for TMS


• 400mg ibuprofen
• With 1000mg
• Paracetamol

• Lowest re-medication rate

Andrew Moore a,⇑, Sebastian Straube b, Jocelyn Paine c, Sheena Derry a, Henry J. McQuay  M. PAIN 152 (2011) 982–989

Minimum efficacy criteria for comparisons between treatments using individual patient meta-analysis of acute pain trials: Examples of etoricoxib, paracetamol, ibuprofen, and ibuprofen/paracetamol combinations after third molar extraction
Most commonly prescribed analgesia for TMS

• 75% US Oral surgeons prescribe Ibuprofen 400mg

  Moore PA, Nahouraii HS, Zovko JG, Wisniewski SR. Dental therapeutic practice patterns in the U.S. II. Analgesics, corticosteroids, and antibiotics. Gen Dent. 2006 May-Jun;54(3):201-7; quiz 208, 221-2

• Combined is better- Synergistic effect Ibuprofen+Paracetamol

Perfalgen- IV paracetamol

No difference between start up does 2g vs 1g

No difference Pre op Oral vs IV

Soluble ibuprofen provides earlier pain relief than tablets.
Ibuprofen and/or paracetamol (acetaminophen) for pain relief after surgical removal of lower wisdom teeth (Review)

Ibuprofen is more effective than paracetamol at all doses studied in this review.

On limited evidence, the combination of ibuprofen and paracetamol appeared to be no more effective than the single drugs when measured two hours after surgery.

On limited evidence, it was found to be more effective than the drugs taken singly when measured at six hours after surgery. Participants taking the combined drug also had a smaller chance of requiring rescue medication.

The information available regarding adverse events from the studies (including nausea, vomiting, headaches and dizziness) indicated that they were comparable between the treatment groups. However, review authors could not formally analyse the data as it was not possible to work out how many adverse events there were in total.
What do I do?

- Frank consent
- Repeat consent on day of surgery
- No pre-emptive analgesia
- LA +/- sedation
- Post op
  - 4 hourly ibuprofen (600mgs) and Paracetamol (1g) orally on day of surgery
  - 2pm, 6pm and 10pm
- Homecheck
- If analgesia required 6 hourly ibuprofen with paracetamol half or full dose
An update on pain
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• **What's new?**
  – Investigating pain
    • Interactions
    • How drugs work?
      • Long term irreversible consequences of pain
  – Neurostimulation
  – Imaging peripheral branches
  – Surgical methods
What’s new?
Structural Brain Imaging: A Window into Chronic Pain.

Arne May. The Neuroscientist 17(2) 209-220:2011

- Neuroplasticity
- Cortical reorganisation
- Gray matter changes
- Central sensitisation
- Maladaptive plasticity
- Downward facilitation
- Downward inhibition
(Placebo effect)
Facilitation
Augmentation
Potentiation
Amplification
=hypersensitivity

Alban Latremoliere & Clifford J. Woolf
Central Sensitization: A Generator of Pain Hypersensitivity by Central Neural Plasticity.
Continuous Arterial Spin Labelling (cASL)

- Novel technique→ quantitative measure of cerebral blood perfusion throughout the brain.
- Examine changes in regional cerebral blood flow (rCBF) to determine physiological perturbation of pathways stimulated by stimulus.
- Arterial blood water protons are magnetically labelled (endogenous tracer) continuously via a continuous 180 continuous RF inversion pulse.

1. **Tag** inflowing arterial blood by magnetic inversion
2. Acquire the **tag image**
3. Repeat experiment **without tag**
4. Acquire the **control image**

Subtract: **Control image** magnetization - **Tag Image** magnetization = rCBF
What’s new?

A window into acute pain brain 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
Anatomy revisited

- Additional amygdala, hippocampus, brainstem, and V5 ROIs
What’s new?
How do routine analgesic drugs work?
### What’s new?

**Why does pain become chronic?**

<table>
<thead>
<tr>
<th>Reason</th>
<th>Description</th>
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<tbody>
<tr>
<td>Persistent acute stimulus</td>
<td>- Increased sensitivity of CNS to peripheral stimulus</td>
</tr>
<tr>
<td>Neuroplasticity</td>
<td>- Interaction between PNS and CNS results permanent changes in system</td>
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<tr>
<td>Memory of pain</td>
<td>- Somatosensory cortex changes</td>
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<tr>
<td>Genetic predisposition</td>
<td></td>
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</tbody>
</table>

**Genetic predisposition**
What’s new?

Peripheral and central interaction: The ‘neuromatrix’

[Diagram showing brain areas with labels for Sensory, Affective, Cognitive, and functional MRI responses to painful stimulation.]

Functional measures

A. Brain areas functionally related to pain processing.

B. Example of functional MRI response to painful stimulation.
What’s new?

The genetic basis of V pain
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)
- **CYP2D6** (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)
What’s new?

Brain activity

affective vs neurophysiological

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
What’s new?

Structural Brain Changes:
Cause or Consequence of Chronic Pain?
Reorganisation
Loss of Gray matter

- Numerous modulatory mechanisms have been postulated and altogether addressed as “neuronal plasticity” (Woolf and Salter 2000), and structural changes of the brain need to be added to this list (May 2008).
- Gray matter changes. The key message of all three studies is that the main difference in the brain structure between pain patients and controls may recede when the pain is cured.
- The impact of pain killers and other medications on morphometric findings is simply not known.
- Chronic nociceptive input leads to intra cortical remodelling.
- We need to improve our understanding of experience-dependent changes in cortical plasticity as this will have vast clinical implications for the treatment of chronic pain.
What’s new?
Cortical reorganisation

A recent study suggests that brain changes in amputees may be pain-induced, questioning maladaptive plasticity as a neural basis of phantom pain. These findings add valuable information on cortical reorganization after amputation.

Maladaptive plasticity, memory for pain


- Phantom limb pain is associated with plastic changes along the neuraxis
- Changes in the cortical representation of the affected limb
- Mechanisms underlying these maladaptive plastic changes are related to a loss of GABAergic inhibition, glutamate-mediated long-term potentiation-like changes and structural alterations such as axonal sprouting
- Behavioral interventions, stimulation, feedback and pharmacological interventions that are designed to reverse these maladaptive memory traces
What’s new?
Tractography

White Matter Connections Obtained with MRI Tractography.png Diffuser tension imaging DTi
What’s new?
Loss gray matter

- It is indeed remarkable that the alterations (i.e., decrease in gray matter) seen in the ACC in migraine patients are similar to a decrease in this region in tension-type headache (Schmidt-Wilcke et al., 2005), posttraumatic headache (Obermann et al., 2009), idiopathic facial pain (Schmidt-Wilcke et al., 2010), chronic back pain (Schmidt-Wilcke et al 2006), and chronic phantom pain (Draganski, Moseret al 2006).


**Figure 3.** Gray matter decrease in 30 studies including a total of 839 patients. Compared with controls, 30 areas in the brain have been identified (increase and decrease of gray matter). Most areas are only cited by one or two studies. Only the brain areas being cited by at least five independent manuscripts are displayed (n corresponds to number of studies citing this brain area), and the percentages correspond to these nine structures. The most prominent findings are decreases in the cingulate cortex, the insular cortex, the temporal lobe, the frontal cortex, and the prefrontal cortex. DLPFC = dorsolateral prefrontal cortex.
What’s new?

Trigeminal nerve

- Reduction gray + white matter TMD

White matter brain and trigeminal nerve abnormalities in temporomandibular disorder. Massieh Moayedi. PAIN Vol 153,7,2012,1467-1477

- Gray matter reduction in TN

Neurostimulation Advances

• Frequency
• Indwelling electrodes
• External remote control

• Sites
  – Sphenopalatine ganglion
  – Trigeminal sensory roots
What’s new?

High-Resolution Magnetic Resonance Imaging
inferior alveolar lingual nerve
An update on pain
• An update on classification of pain
• Perioperative techniques to minimise pain
• Managing and preventing post operative pain
• **What’s new?**
  – Surgical methods
Modified Surgery no brainer-
minimise soft and hard tissue damage

OLD                                        NEW
Division of fractured roots
Tooth Section
Canine surgery
Lingual split..the old
Buccal technique the new
Buccal technique..for all
Prevention of lingual nerve injury in

Spot the lingual nerve!
Prevention of lingual nerve injury in...
Tailor your surgery minimise harm!

Coronectomy
Prevention of nerve injury

Tailored treatment
Less than 10% of high risk M3Ms need coronectomy.

Prevention of IAN injury
Prevention of IAN injury
Simple recommendation for OS pain management

• Homecheck
  – telephone call 24 hours after surgery by the surgeon
• Instruct patient to start to take ibuprofen with paracetamol as soon a LA is wearing off (3.5 hours post surgery)
• Take analgesics
  – Ibuprofen (max 600mg) + 1g Paracetamol
  – 4 hourly first day (2pm-6pm 10pm)

• Less than 50% of my patients need any Pain relief the day after surgery!!!!!
Summary

• Understanding of pain and recent developments
• Strategy for managing /preventing pain in OS
  – Preoperative
    • Care compassion consent competency clear
    • Analgesics/ steroids?
  – Intra operative methods to minimise pain and morbidity
    • LA
    • Minimal access Surgical methods
    • Analgesics / steroids?
  – Post surgical
    • Good instructions expectancies
    • Appropriate analgesic regime……no codeine
    • Home check
Remember

• If there is NO response to anti inflammatory drugs
• Consider neuropathic pain
Baffled?????

• Does the patient reaction seem disproportionate to the cause?

• Consider
  – Potential nerve injury
  – Psychometrics..........................................................
    • Liaison Psychiatry
    • Clinical psychology
<table>
<thead>
<tr>
<th>Pain Level</th>
<th>Recommended</th>
<th>Alternative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild pain &lt;3</td>
<td>Ibuprofen 200/400mg TDS</td>
<td>Paracetamol 1g QDS reduce to PRN</td>
</tr>
<tr>
<td></td>
<td>With paracetamol 1g QDS then PRN</td>
<td></td>
</tr>
<tr>
<td>Moderate pain &gt;3</td>
<td>Ibuprofen 400/600mg TDS + paracetamol 1g QDS PRN</td>
<td>Paracetamol 1g QDS + codeine 30mg QDS</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe pain &gt;7</td>
<td>Ibuprofen 400/600mg QDS + paracetamol 1g QDS + codeine 30mg</td>
<td>Paracetamol 1g QDS + codeine 60mg QDS</td>
</tr>
<tr>
<td></td>
<td>Diclofenac 25/50mg TDS + paracetamol 1g QDS + codeine 30mg QDS reduce to ibuprofen 400mg + paracetamol 1g QDS reduce to paracetamol 1g QDS with ibuprofen PRN</td>
<td>Tramadol</td>
</tr>
<tr>
<td>Rescue medication</td>
<td>Tramadol?</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: TDS, 3 times/day; QDS, 4 times/day; PRN, as needed
## Suggested analgesic regimes for acute trigeminal pain.

<table>
<thead>
<tr>
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<th>Recommended</th>
<th>Alternative</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mild pain &lt;3</strong></td>
<td>Ibuprofen 200/400mg TDS With paracetamol 1g QDS then PRN</td>
<td>Paracetamol 1g QDS reduce to PRN</td>
</tr>
<tr>
<td>(e.g. routine restorative dental work, routine extraction, routine endodontic treatment, scaling)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Moderate pain &gt;3</strong></td>
<td>Ibuprofen 400/600mg TDS + paracetamol 1g QDS PRN</td>
<td>Paracetamol 1g QDS + codeine 30mg QDS</td>
</tr>
<tr>
<td>(e.g. surgical dental extractions, implant surgery,)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Severe pain &gt;7</strong></td>
<td>Ibuprofen 200/400mg QDS + paracetamol 1g QDS + codeine 30mg</td>
<td>Paracetamol 1g QDS + codeine 60mg QDS</td>
</tr>
<tr>
<td>(e.g. osteotomies, open reduction internal fixation of jaws, autologous bone graft)</td>
<td>Diclofenac 25/50mg TDS + paracetamol 1g QDS + codeine 30mg QDS reduce to ibuprofen 400mg + paracetamol 1g QDS reduce to paracetamol 1g QDS with ibuprofen PRN</td>
<td>Reduce to paracetamol 1g QDS then PRN</td>
</tr>
</tbody>
</table>

**Rescue medication**

*Tramadol? Or Tapentadol*

---

**Abbreviations:** *TDS, 3 times/day; QDS, 4 times/day; PRN, as needed*
Launching June 2015
Orofacialpain.org.uk
Remember in order to manage your patient’s expectations you need to know your patient!
Thank you
Problems with oral analgesics

- **NSAIDs**
  - allergy to aspirin
  - history of asthma
  - under 12 years - Reyes syndrome
  - history peptic ulceration or GI bleeding
    - DU 3-4 % PU 4-7%
      (risk factors >75yr/history PU or GI bleed and heart disease/ + H pylori 27%)
  - bleeding disorders - reduced platelet adhesion
  - pregnancy / breast feeding
  - renal impairment
  - Decreases effectiveness of anti-hypertensives

- **Paracetamol**
  - no inflammatory action
    - side effects are rare
    - Decreases liver function (CI AZT therapy)
    - irreversible hepatic impairment
      10-15g within 24 hours (ONLY 20-30 tablets)
    - renal impairment

- **Opioids**
  - respiratory depression
  - constipation
  - overdose
  - Dependency
  - Pethidine can cause convulsions with repeated doses
## Deaths associated OTC analgesics (pts/year)

<table>
<thead>
<tr>
<th></th>
<th>UK</th>
<th>USA</th>
<th>Canada</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Opiates</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Addiction</td>
<td>30,000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>100</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>NSAIDs</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prescription</td>
<td>20m</td>
<td>70m</td>
<td>10m</td>
</tr>
<tr>
<td>Death</td>
<td>150</td>
<td>7,600</td>
<td>365</td>
</tr>
<tr>
<td>Admissions</td>
<td>12-35K</td>
<td>76K</td>
<td>39K</td>
</tr>
<tr>
<td><strong>Paracetamol</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>600</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Responsible for 1 in 10 liver transplant patients</td>
<td></td>
</tr>
</tbody>
</table>
Possible neural pathways of cognitive pain modulation. Cognitive modulations of pain are related to activation of prefrontal brain areas such as the dorsolateral prefrontal cortex (DLPFC), ventrolateral prefrontal cortex (VLPFC), and to the anterior cingulate cortex (ACC); shown in orange. These regions may modulate activation in afferent pain regions in the cortex (ACC, primary- and secondary somatosensory cortex, insula and thalamus), as well as the periaqueductal gray (PAG) and dorsal horns of the spinal cord; shown in blue. The DLPFC and VLPFC are connected to the ACC, which, in turn, projects to thalamus and the PAG, a core component of the descending pain modulatory system.
Relative efficacy of oral analgesics after third molar extraction

J Barden1, J E Edwards2, H J McQuay2, P J Wiffen2 & R A Moore2

This paper reviews the available high quality information on analgesics commonly prescribed by dentists, including COX-2 selective inhibitors.

Problems related to chance effects are avoided by combining multiple trials in a meta-analysis.

There is good evidence of efficacy for most commonly-prescribed analgesics.
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Central Sensitization: A Generator of Pain Hypersensitivity by Central Neural Plasticity

- Facilitation
- Augmentation
- Potentiation
- Amplification

=hypersensitivity

Genetics to blame?
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Protective analgesia for postoperative pain following third molar surgery

Sin Leong Yong¹,², Tanya Walsh², Paul Coulthard¹

Editorial Group: Cochrane Oral Health Group

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Additional Information (Show All)

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Sin Leong Yong, Oral and Maxillofacial Surgery, School of Dentistry, The University of Manchester, Higher Cambridge Street, Manchester, M15 5FH, UK. sin.l.yong@manchester.ac.uk.
Evidence for analgesic effect in acute pain – 50 years on

Henry J. McQuay, Sheena Derry, Christopher Eccleston, Phillip J. Wiffen, R. Andrew Moore

Topical review

1. Introduction

The basic design of studies to measure the analgesic effect of drugs in acute pain was worked out in the 1960s and 1970s, was rigorously tested at the time, and established randomisation and double blinding as essential standards for objective assessment of analgesic efficacy [7]. The design became the conventional way to establish analgesic efficacy, typically performed early in the development of new pain-relieving drugs. Several individual patient analyses have confirmed the validity of methods for evaluating efficacy [2,3,6,18,19], but not adverse events [6].

A recent change has been the way in which outcomes are studied. While the original use of average summed pain intensity difference or total pain relief over 4-hour has triglyceride value, analysis is now based on the individual patient's experience. This is an important change in the way in which outcomes are studied.

2. The evidence

To the Cochrane overview [15] we have added results from non-Cochrane reviews on tramadol [16], tramadol plus paracetamol [6], and ketorolac plus paracetamol [19], all using identical methods. Collectively they provide a wealth of information:

- Seven drugs had no useful trials, including meclozine, naxolone, nefopam, and sufentanil.
- There was good evidence of no analgesic benefit for aspirin 500 mg, oxycodone 5 mg, and acetaminophen 150 mg.
- Twenty-five drug and dose combinations either had very limited data (fewer than 2 trials and 200 patients) or had more extensive data, but where small numbers of patients were involved, small effect sizes combined to make the results insignificantly different.

Sponsorships or competing interests that may be relevant to content are disclosed at the end of the article.
Interventions for the treatment of keratocystic odontogenic tumours (KCOT, odontogenic keratocysts (OKC))

Fyeza Nj Shafi, Richard Oliver, Christopher Street, Mohammad O Shafi

Editorial Group: Cochrane Oral Health Group
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Abstract

Background...
Dental extractions prior to radiotherapy to the jaws for reducing post-radiotherapy dental complications

Shiyana Eliyas¹,¹, Ahmed Al-Khayatt², Richard WJ Porter², Peter Briggs²

Editorial Group: Cochrane Oral Health Group
Published Online: 28 FEB 2013
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