Contemporary approaches to the management of chronic pain

Medicine for Psychiatrists Conference
Auckland, New Zealand
26 March 2015

Paul Vroegop
Child and Adolescent/Consultation-Liaison Psychiatrist and Pain Medicine Specialist

Chronic Pain Service
Counties Manukau Health
Contemporary approaches to the management of chronic pain - overview

- What is pain?
- Aetiology of pain
- Epidemiology
- Assessment
- Management
“What is Pain?”

• IASP 1975: "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."

• “Pain” is an
  – evolving concept
  – emotional experience
  – physical problem
  – neuro-regulatory problem
“What is Pain?”

• A challenge to dualistic approach to health
• Frequently cannot be understood purely in terms of “physical causes”
• But rarely can be understood purely as a result of “psychological causes”
• Poor recognition by healthcare professionals and the public
• Significant disability
• Little research, especially in children
“What is Pain?”
Acute vs Chronic Pain

<table>
<thead>
<tr>
<th>acute pain</th>
<th>chronic pain (arbitrarily &gt;3/12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common &gt;99% population</td>
<td>10-15% population</td>
</tr>
<tr>
<td>Tissue damage</td>
<td>No tissue damage</td>
</tr>
<tr>
<td>Diagnose</td>
<td>Multiple explanations</td>
</tr>
<tr>
<td>treat</td>
<td>Multiple treatments, variably effective</td>
</tr>
<tr>
<td>cure</td>
<td>Often no cure, chronic disease model</td>
</tr>
<tr>
<td>Return to function</td>
<td>losses</td>
</tr>
<tr>
<td>Clear sick role</td>
<td>Unclear roles</td>
</tr>
</tbody>
</table>
Aetiology: dualistic fallacies

- Pain “felt” by the brain
  = brain causes pain (it’s all “in your head”)
- “Psychological” treatments effective
  = causes of pain are “psychological”
- “Abnormal pain/illness behaviour”
  = pain is “not real”
- Parental anxiety perpetuates pain
  = parental anxiety causes pain
Aetiology: Not All Chronic Pain is the Same

• Integration of the sensory and affective/evaluative elements of the pain experience
• Identification of the multiple mechanisms responsible for production of distinct pain syndromes
  – Nociceptive pain
  – Neuropathic pain
  – Central Neural Sensitization
• Assessing patients for the specific pain mechanism(s) AND targeting treatment forms the basis for successful pain management
Neural Mechanisms of Pain

Schematic drawing of nociceptive processing:

- **Ascending pathways** (left side of diagram)
- **Descending pathways** (right side of diagram)
- “Pain Gate” in spinal cord
# Modifiers of the Pain Gate

<table>
<thead>
<tr>
<th>Open Gate</th>
<th>Close Gate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain signals</td>
<td>Anesthetics</td>
</tr>
<tr>
<td>Tired</td>
<td>Relaxed</td>
</tr>
<tr>
<td>Depressed</td>
<td>In shape</td>
</tr>
<tr>
<td>Out of shape</td>
<td>Preoccupied or busy</td>
</tr>
<tr>
<td>Negative expectations</td>
<td>Positive expectations</td>
</tr>
<tr>
<td>Loss of distractions</td>
<td>Improved sleep and mood</td>
</tr>
</tbody>
</table>
Painful Diseases and Pain Diseases

**Nociceptive pain**
Caused by activity in neural pathways in response to potentially tissue-damaging stimuli

- Postoperative pain
- Mechanical low-back pain
- Sports/exercise injuries

**Neuropathic pain**
Initiated or caused by a primary lesion or dysfunction in the nervous system

- Central post-stroke pain
- Peripheral neuropathy
- Trigeminal neuralgia
- Postherpetic neuralgia

**Central Sensitization**
Maintained by neural reorganisation at spinal cord level

**Inflammatory/Immunological Mediation**

CRPS = complex regional pain syndrome.
Nociceptive Pain (1)

- Transient pain in response to a noxious stimuli
- Pain/heat receptors (Vanilloid TRPV-1&2), mechanoreceptors, inflammation receptors
- Announces the presence of a potentially damaging stimulus
- Key early warning:
  - An alarm system
  - Highly (over)sensitive
  - Affective component
  - Modifies behaviour
Nociceptive Pain (2)

- SOMATIC
  - Well-localized
  - Aching, throbbing, gnawing
- bone
- joints
- soft tissue
- muscle
- skin

- VISCERAL
  - Poorly localized
  - Deep aching, cramping, pressure, referred
  - bowel obstruction
  - biliary colic
  - liver pain
  - appendix
Nociceptive Pain (3):

- **C fibers** (afferent, unlocalised)
  - slow, unmyelinated, polymodal
  - deep pain, itch, heat, mechanoceptors, metaboreceptors (inflammation)

- **A-delta fibers** (afferent, localised)
  - Fast, myelinated, polymodal
  - Acute pain, cold, pressure, reflex arc

- **Ascending tracts:**
  - Immediate crossover to dorsal horn
  - Rostral ventral medulla (RVM), periaqueductal gray (PAG)
  - Tracts to sensory cortex, cingulate cortex, amygdala and hypothalamus
Neuropathic Pain
Injury to Peripheral Nerves and/or CNS

- Burning
- Stinging
- Shooting
- Lancinating
- Pins and needles
- Vice-like
- Electric
- Tingling

Predominantly C fibers
Central Neural Sensitization (1)

- Chronic pain often occurs in the absence of ongoing damage
- Sometimes the nervous system continues to send pain signals to the brain as though a new injury were occurring
- Spinal cord / brainstem level
  - Reorganization of neural pathways is now better understood
  - Repeat stimulation/nerve damage ➔ WDR hyperexcitability
  - 2nd order dorsal horn neurons sends ongoing signals similar to those sent in response to the original injury
  - Sensitization may spread (non-dermatomal, non PNS distribution)
Central Neural Sensitization (2)

• Not clear why these changes occur for some patients / some injuries
  – but sensitization is an adaptive component of the pain system

• Chronic pain as a functional disorder of the nervous system
  – with associated affective and behavioural components

• Arguably could be “reclassified” as a neurological disorder

• Good evidence from animal models and human observation of neuropathophysiologic changes
Central Neural Sensitization (3)

Events leading to activation, sensitization, and spread of sensitization of primary afferent nociceptor terminals.

- pain is evoked by normally non-nociceptive stimuli (allodynia)
- increased pain in response to mild nociceptive stimuli (hyperalgesia)
- referred pain - widespread recruitment of spinal cord/medullary nociceptive neurons
- Mediated by NMDA receptors; “pain windup”, temporal summation
- 2nd order Wide Dynamic Range neurons accept A and C input
Psychological theories regarding chronic pain

- *Family Systems* approach – pain maintains homeostasis in family
- *Psychodynamic* – unconscious, unacceptable need or conflict expressed as “physical” pain
- *Learning theory* - benefits of the sick role experienced or observed by the child
- *Cognitive-Behavioural model* - Cognitive Triad, Learned Helplessness
Epidemiology (1)

- Chronic pain in population prevalence at around 20% (11-51%)
- Prevalence increases with age, lower socioeconomic status, ethnicity, female gender
- NZ 17% (Dominick et al 2011)
  - 15-24 years 8% F/9% M
  - 35-44 years 14.1% F/16.1% M
  - 45-54 years 20.5% F/17.7% M
  - 75+ years 30% F/25.6% M
- SF-36: health related QoL decreases with number of pain sites
Epidemiology: sites

- Head 15%
- Neck 8%
- Upper back 5%
- Lower back 18%
- Hip 8%
- Shoulder 9%
- Hand 6%
- Leg 14%
- Knee 16%
- Joints (unspecified) 10%
- Back (unspecified) 24%
Epidemiology: specific chronic pain syndromes

- Headache: migraine, tension headaches, cluster headaches (autonomic), chronic daily headache, medication overuse headache
- Low Back Pain +/- radiculopathy
- Non cardiac chest pain
- Myofascial pain syndromes
- Neuropathic pain syndromes
- Musculoskeletal pain syndromes: Fibromyalgia, arthritis, bursitis, plantar fasciitis, others
- CRPS (Complex Regional Pain Syndrome)
- Visceral pain (gastrointestinal, pelvic, abdominal)
- Oral/facial pain
Epidemiology: associations

- *Remember,* psychological factors adversely affect general medical conditions in several ways:
  - close temporal relationship between psychological factors and development or exacerbations in medical condition
  - factors interfere with treatment
  - factors are an additional health risk
  - stress related physiological responses precipitate medical symptoms
Epidemiology: associations

- Affective Disorders: Depression 10-100% (cf 18-35% population), MDD 1.5-54% (cf 6.3%), Adjustment Disorder w DM 28%. Elevated suicide rates
- Anxiety disorders: GAD 15-20%, PD 11%, PTSD 7-39%, phobia 9%, social phobia 11%, PTSD (esp from CSA) may be higher
- AOD: On opioids for CNCP ADRB 11-20.4%, addiction 3.3% (recent studies suggest much higher (26%) for unselected patients
- Somatoform Disorders: Pain Disorder (little validity), Conversion Disorder (neurophysiological phenomena closely involved in generation of motor and sensory “conversion” symptoms)
- Personality Disorders: rates of 37-66%, higher than general population but similar rates to psychiatric and medical populations
Chronic Pain Services

• Multidisciplinary Teams that grew out of the knowledge of the Gate Theory
• Recognition of pain as being amenable to behavioural and psychological approaches
• Often characterized by interdisciplinary vs multidisciplinary functioning
• A number of chronic pain services in NZ, 3 FPM accredited training centres, another 6 true MD CPS’s
• 2012 - Pain Medicine was recognized as a specialist scope of practice by the MCNZ; around 30 FPM’s in NZ
• FPM (ANZCA) allows specialists from a number of Australasian medical colleges to train as Pain Medicine Specialists – now a 3 year training scheme
Assessment of the Patient with Chronic Pain

• Multidisciplinary assessment:
  – Physiotherapy / Occupational Therapy
  – Psychosocial assessment, including family assessment
  – Medical (anaesthetic, medical/paediatric, surgical)

• Assessment is where treatment starts!
Pain Assessment

1. Thorough pain history: Quality, severity, frequency, location/duration of pain
2. Relieving factors, including situational, relational, emotional, sensory, medication
3. Exacerbating factors, including situational, relational, emotional, sensory, medication
4. Psychiatric comorbidities (in patient and whanau)
5. Assessment of coping strategies (premorbid personality)
Pain Assessment 2

• Assessment of coping strategies in person and whanau

• Family reaction to pain, incl medication use

• Pain beliefs in person and whanau, family history of chronic pain and/or illness

• Family rearrangement around pain, degree of family flexibility

• Consequences of pain – degree of associated disability

• Look for development of “Pain Traps”: 
“Identification Trap”

• Very hard for family to see their loved one in pain
  – Rescue
  – Provide solutions
  – Provide hope
  – Parental concern/worry

• But increased pain with activation often necessary for improving function

• Increased family worry can lead to:
  – increased focus on pain
  – increased anxiety in patient
“Boom and Bust” Trap

1. Push myself until pain or fatigue makes me stop
2. Rest for very short time
3. Push myself until pain or fatigue makes me stop
4. Rest for short time
5. Exhausted, lots of pain
6. Take to bed, Depressed

Diagram cycle: Push → Rest → Push → Rest → Exhausted → Take to bed.
STUCK - The ‘‘Take It Easy’’ Trap

Person advised to take it easy or 'knows' they should take it easy

Avoidance of pain through avoidance of activity - "pain means damage"

Person with pain gradually gives up school, social and recreational activities

Slide towards invalid role

Increased focus on the pain

Life becomes much less fun, personally, socially, emotionally and psychologically

Pain → Depression

“STUCK” - The ‘‘Take It Easy’’ Trap
Inactivity

- Do less with friends, whanau

Relationship / friendship less rewarding

- whanau / friends frustrated, sad

Person in pain feels misunderstood

Relationships stressed

Focus on the pain

Danger of becoming boring, grouchy demanding

Shift/loss of duties and/or roles

Imbalance

“STUCK” - The Chronic Resentment Trap
Person with pain consults Dr, Physio, Chiro etc

Expectation of cure or significant relief

Treatment / Intervention fails

'Through frustrated with therapist

Treatment shopping

Pain labelled 'non-organic' or 'psychogenic'
Referral to OT, Psychologist or Psychiatrist

Patient tries to prove pain is 'real' - resists treatment / referral

"CURE" - The Chronic Treatment Trap
Pain Assessment (3): Psychosocial factors to consider in chronic pain

• Cognitive factors
  – Understanding, control, expectations, catastrophising

• Temperament
  – “Repressors” vs “sensitizers”

• Operant components
  – Reinforcement of pain behaviours, avoidance
  – Reinforcement of “healthy” behaviours

• Emotional factors
  – Stress, anxiety, depression, anger, frustration

• Family factors
Influence of family

- Attachment aspects
- Parental anxiety mediated by family stress secondary to chronic pain in child
- Varies with parents own responses/own experiences
- Important to educate
- Engagement with treatment team / treatment model critical
Management of chronic pain: Conceptual Shift to a Rehabilitation / Chronic Disease Management Approach

• Goals of treatment change from:
  – the identification and repair of the cause of pain
to
  – pain control, functional improvement, and decreasing suffering

• Multidisciplinary / multimodal

• Graduated
Mana / Health

PAIN

Acute

Chronic

3/6 months

LOSSES

- Activity
- Jobs
- Friends
- Sports
- Relationships
- Finances
- Worry
- Angry
- Depressed
- Anxiety
- Loss of Self Esteem

4 P's
- Pacing
- Persistence
- Passion
- Praise

Gains
- Confront the Reality
- Activation
- Regulation/Stress Management/Relaxation
- Education
- Medication Review

Stuck

Cure

Self Management

Acceptance

Relapse
The 4 ‘P’s

• Critical aspects of success are:
  – Pacing
  – Persistence
  – Passion
  – Praise

• But awareness that pain and disability may fluctuate, so we also add
  – relapse Planning
Chronic pain management is focused on developing/improving SELF MANAGEMENT skills

Core treatment is ACTIVATION – all approaches should be tailored to enhancing movement
Management: overview

• Education / Engagement

• Activation
  – Graduated physical activity
  – Re-conditioning

• Somatic management strategies

• Self-management
  – Graduated return to work/school/functioning/roles
  – Reduce “Boom and Bust”

• Treat co-morbid
  – Sleep problems
  – Psychiatric disorders

• Manage medical systems (eg ED plans, crisis plans)

• Specific psychological interventions

• Pharmacology

• Specific approaches for specific disorders
Management: Education/Engagement

- Explanations
  - “pain is pain”, not “in your head”
  - Resources: reading lists, books, websites (Hunter Valley Pain Service youtube videos - https://www.youtube.com/watch?v=4b8oB757DKc)

- Pain is not caused by psychiatric illness
  - But depression, anxiety often result of pain
  - Anxiety/worry exacerbates pain, impairs coping

- de-stigmatise
  - Education of whanau, friends, employers, teachers
  - Providing simple explanations to others
Management: Investigations

• Investigations for pain are NOT useless
  – essential to clarify whether active and modifiable disease process/injury is present
  – But also important to not continue to search for “reasons” once above has been excluded

• Over-investigation can makes pain worse
  – Invasive/surgical procedures can “stir up” pain
  – Focus on “cure” can delay return to functioning
  – Avoid “doctor-shopping” / primary physician
  – Investigate new symptoms only
Management: Core Evidence-Based Behavioural Treatments

- Distraction, relaxation, guided imagery techniques (as for acute pain)
- Activation
- Activity Scheduling
- Relaxation
- Sleep hygiene
Activity Scheduling

• Doing things differently
  – Monitoring and pacing activity
  – Scheduling in rest and relaxation, fun and enjoyment, achievement
  – Rewarding yourself
  – Physical exercise

• Sleep hygiene

• Problem solving

• Goal setting
Sleep hygiene

• Establishing a sleep routine
  – maybe gradual process

• avoiding activating activities at bedtime
  – eg texting, internet, computer games

• relaxing routines to assist sleep
  – relaxation exercises, guided imagery, quiet music

• reducing daytime sleeping or resting

• pros and cons of sedatives
Management: specific psychological therapies

- Biofeedback
- Self hypnosis
- Cognitive Behavioural Therapy (CBT)
- Acceptance and Commitment Therapy (ACT)
- Family Interventions / Family Therapy
- Multidisciplinary Group Treatment (Pain Management Programmes- PMP’s) – intensive programmes with a focus on using CBT and physical activation/rehabilitation self management approaches
Biofeedback

- Monitoring and quantifying of physiological response, auditory or visual display
- Either to achieve heightened state of relaxation or to alter a physiological process
- Useful educational tool
- Biofeedback in combination with relaxation most effect treatment modality for tension-type headaches (Nestoriuc et al 2008)
Clinical hypnosis/self hypnosis

• Achieve heightened state of relaxation
• Teach mind-body link
• Demonstrate control over physiological processes
• Directly control sensation
CBT/Cognitive techniques

- Adults/ Adolescents
- Younger children – parent teaching skills
- Self talk-
  - Cognitive restructuring
  - encourage person to monitor and evaluate negative thoughts and to generate alternatives
- Coping skills training-
  - Coping self-statements
## CBT: Identifying Negative Automatic Thoughts

<table>
<thead>
<tr>
<th>Situation</th>
<th>Thoughts</th>
<th>Physical reactions</th>
<th>Feelings</th>
<th>Alternatives</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain after walking</td>
<td>I know something is wrong-its getting worse-end up in a wheelchair</td>
<td>Muscle tension</td>
<td>Fear</td>
<td>I walked much farther than normal-my dr told me to expect soreness as I start to exercise</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Frustration</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Anxiety</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Worry</td>
<td></td>
</tr>
<tr>
<td>Pain on waking</td>
<td>Its going to be a bad day-I won’t be able to do anything</td>
<td>Tension Fatigue</td>
<td>Anger</td>
<td>I don’t know for sure-sometimes better when I get going-I’ll start with small tasks</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Depression</td>
<td></td>
</tr>
</tbody>
</table>
ACT (Acceptance and Commitment Therapy) techniques

- A “3rd wave” cognitive therapy
- Involves actively embracing and accepting experiences for what they are (NOT acceptance of chronic pain or limitation)
- Techniques include:
  - Defusing techniques
  - Controlling focus of attention
  - Mindfulness
  - Identifying values
Family Interventions

- Acknowledge and address stressors
- Encourage child to take responsibility for pain management (as developmentally appropriate)
- Encourage parents to set appropriate limits
- Encourage expression of feelings
- Clarify roles and boundaries in family
- Address any unhelpful illness behaviour in family members
- Identifying and decreasing secondary reinforcers
- Family Therapy if required
“Non-psychological” approaches

• Medication (evidence base variable)
• Neurostimulation (evidence base variable)
• Interventional blocks/ablation
• Acupuncture
• Dietary modification
• Somatic treatments/distraction (“Buzzy”, heatpacks, cooling gels)
• Massage, manipulation
• Many others
Management: pharmacology

- DMARDS (Disease modifying agents) such as biologics, steroids
- Pain neuromodulators: Low dose TCA’s, and/or gabapentin, venlafaxine
  - Start low, go slow, make one change at a time
  - Allow 1-6 weeks to assess response
- Topical treatments; capsaicin, local anaesthetics, clonidine
- Management plan for exacerbations/flare-ups:
  - Paracetamol, NSAIDS/Cox-2 Inhibitors
  - Tramadol – watch for serotonergic side effects, care in <18, >65
  - Stronger opioids (morphine, oxycodone) – controversial, not recommended for CNCP. Codeine – not recommended
- Others:
  - Temporary nerve blocks, implantable spinal pumps, fentanyl patches (cancer pain)
Medication NNTs (1)

Overall:
- Amitriptyline 5.1
- Desipramine 2.1
- Imipramine 1.1
- Venlafaxine 3.1, NNH 20+
- Duloxetine 5.1
- Gabapentin 5.8,
- Pregabalin 3.9 Carbamazepine 1.7, NNH 2.6
- Anticonvulsants PDN 2.7, PHN 2.9, minor NNH 2.7
- Topical NSAIDs NNT 3.1 2 weeks

Peripheral / neuropathic pain
- Capsaicin topical DPN 4.2, OA 3.3, mixed evidence PHN
- TCA’s 3.3, PDN 3.4, PHN 2.1
- NNH minor 2.7, major 17

FMS
- Duloxetine 6 (50% reduction)
- Pregabalin 6 (50% reduction)
- Amitriptyline 25

IBS
- Peppermint oil 3.1
Medication NNT’s (2)

Headache:
- Valproate 4.5
- Topiramate 3.5
- Carbamazepine 2.1 (1.6 for migraines)
- Gabapentin 3.3
- Antidepressants 3.2

Migraines:
- Sumatriptan sc 2.0, po 2.6
- Aspirin/maxalon 3.1
- Excedrin (paracetamol/aspirin/metaclopramide) 3.9

Headache Prevention:
- topiramate 4.1
- valproate 3.5

Trigeminal neuralgia:
- carbamazepine 1.4-2.1
- baclofen nnt 1.4
- lamotrigine small trial
- oxcarbazepine

Less evidence!
- Clonidine (a2, imidazoline agonist)
  - Opioids (for CNCP)
  - Benzodiazepines
  - Cannabinoids (HIV peripheral neuropathy)
Antidepressants (1)

- Tricyclic’s considered gold standard
  - amitriptyline, nortriptyline good evidence
  - separate to anti-depressant effect, ceiling 30-50mg/day
  - Much lower dosage, and usually faster onset than anti-depressant action
    - helpful for sleep (amitrip>nortrip) but hangover effect, take early
    - QTc prolongation, falls in elderly, weight gain,
    - action via predominantly noradrenaline reuptake inhibition
      - NNT of 2.0 (1.7 to 2.5)
      - NNH (minor) of 4.6 (3.3 to 6.7)
      - NNH (major) of 16 (10 to 45)
  - dose of 0.2 to 1mg/kg nocte
Antidepressants (2)

– Venlafaxine
  • No evidence in <18, not funded for pain in NZ

– Duloxetine
  • No evidence in <18, not funded in NZ

– SSRI’s, others
  • No evidence in chronic pain
Neurostimulation

- **TENS (Transcutaneous Electrical Nerve Stimulation)** – cheap, safe, evidence base reasonable, variable (NNT 7), includes Cefaly (migraines)
- **Spinal Cord Stimulators** – expensive, some risks, evidence base small but positive in selected groups (extensively used in Australia and US)
- **tcDCS (Transcranial Direct Current Stimulation)** – cheaper than TMS, safe, evidence base minimal (CRPS)
- **TMS (Transcranial Magnetic Stimulation)** – very expensive, safe, evidence base minimal
- **Deep Brain Stimulation** – very experimental, very expensive, risky
Specific management approaches to specific presentations

- CRPS - activation of affected limb, Graded Motor Imagery (aimed at addressing cortical blurring), clonididine patches
- IBS/RAP (Recurrent Abdominal Pain) – CBT, parent training
- FMS/CFS – graduated activation programme + CBT
- Migraines – triptans
- Headaches – topiramate, relaxation, ?CBT, propranolol
Barriers to Treatment

- **potential barriers to a rehabilitation approach**
  - clinician/whanau differences
    - in focus on causation
    - in belief around meaning of pain
  - clinicians
    - from different services/disciplines/approaches not developing joint approaches
    - failure to recognize family/whanau’s lack of engagement
  - Systems of care
    - Lack of coordinated funding / “silo’s”
    - Lack of political interest
    - Unavailability of clinical components of even “virtual treatment teams”
Questions