CENTRAL SENSITIZATION PAIN IN PHYSICAL THERAPY PRACTICE AROUND THE WORLD

http://www.wcpt.org/wcpt2017/COURSE-11

Where the world of physical therapy meets
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- Associate Professor University of Cape Town
- Director of Train Pain Academy [www.trainpainacademy.co.za](http://www.trainpainacademy.co.za)
- chronic pain management team Groote Schuur Hospital
- President-elect of PainSA
- Chair of the Pain, Mind and Movement Special Interest Group of IASP

CENTRAL SENSITIZATION PAIN IN PHYSICAL THERAPY PRACTICE AROUND THE WORLD
http://www.wcpt.org/wcpt2017/COURSE-11
Michele Sterling

- Professor Griffith University
- Director NHMRC Centre of Research Excellence in Road Traffic Injury
- Chair Scientific Program Committee Australian Pain Society
- ranked #1 whiplash injury researcher in the world www.expertscape.com
- > $13M competitive research funding
- > 150 scientific papers
Kelly Ickmans

- Visiting & research professor
- Postdoctoral researcher
- PT - clinician
- Pain in Motion *kids*
- PhD supervisor
- > 30 papers

### Medical diagnosis

- Low back pain
- Pediatric pain
- Post-cancer pain
- Osteoarthritis
- Whiplash associated disorders
- Nontraumatic neck pain
- Shoulder pain
- Fibromyalgia
<table>
<thead>
<tr>
<th>Medical diagnosis</th>
<th>Medical discipline</th>
<th>Estimated % predominant central sensitization pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low back pain</td>
<td>Orthopedics</td>
<td>25%</td>
</tr>
<tr>
<td>Pediatric pain</td>
<td>Pediatrics</td>
<td>?</td>
</tr>
<tr>
<td>Post-cancer pain</td>
<td>Oncology</td>
<td>15%</td>
</tr>
<tr>
<td>Osteoarthritis</td>
<td>Rheumatology</td>
<td>30%</td>
</tr>
<tr>
<td>Whiplash associated disorders</td>
<td>Emergency medicine</td>
<td>90%</td>
</tr>
<tr>
<td>Nontraumatic neck pain</td>
<td>Physical medicine</td>
<td>10%</td>
</tr>
<tr>
<td>Shoulder pain</td>
<td>Physical medicine</td>
<td>10%</td>
</tr>
<tr>
<td>Fibromyalgia</td>
<td>Rheumatology</td>
<td>100%</td>
</tr>
</tbody>
</table>
Content overview

• Introduction

• Central sensitization: maladaptive neuroplasticity in patients with chronic pain (Kelly)

• Neuropathic central sensitization pain in physical therapy practice: HIV-related neuropathic pain as an example (Romy)

• Neuropathic central sensitization pain in physical therapy practice: assessment (Romy & Michele)

• Non-neuropathic central sensitization pain in physical therapy practice: Neck pain as an example (Michele & Jo)

• Non-neuropathic central sensitization pain in physical therapy practice: case study (Kelly & Jo)
Pain

Nociceptors

Nociceptive neurons and WDR neurons in DH

Thalamus

Cortical regions

Cortical output

Nociceptive pain

- Inflammation
- Tissue injury
- Growing mass
- ...
  - distension
  - rupture
  - stimulation mech. receptors

activation nociceptors
Why?

Primary hyperalgesia = adaptive response of the nervous system, preventing further damage and hence facilitating tissue healing.
Nociception vs. pain

- There’s a direct link between the amount of tissue damage and the level of pain experienced.
Pain vs. Nociception

Nociception ≠ Pain & Pain ≠ Nociception
If pain persists

• After injury → tissue sensitization

• Inflammatory mediators or strong noxious stimulation sensitize primary nociceptors (c-fibres)

⇒ Peripheral sensitization
If pain still persists

→ lack of distinct localisation
→ lack of tissue damage

• No longer adaptive function
• ≠ Prolonged acute pain

Chronic pain

• Disproportional to peripheral input
• Therapy resistant, bad recovery

Peripheral or central problem?
Sensitization

= NEUROPLASTIC PAIN:
  • Synaptic and non-synaptic changes
  • Peripheral
  • Central: spinal cord and brain

Neuroplasticity =
Planning a better response
Central sensitization

- Hyperexcitability CNS
- Hypersensitivity for all mechanical stimuli

Allodynia
Generalized hyperalgesia
Widespread pain
Chronic pain

1. Overactivation bottom-up system:
   ➤ nociceptive transmission

Central Sensitization: mechanisms

Meeus & Nijs, 2007; Nijs & Van Houdenhove 2008; Yarnitsky et al. 2010
Wind-up & LTP

C-fibres:
- prolonged discharge
- ubiquitous distribution
- Wind-up: 1/3 > 0.5 Hz
- LTP: 0.5-5 Hz (tetanic)

Injury
Peripheral sensitization
Healing
Sub P
Wind-up
Injury Peripheral sensitization

Wind-up

LTP

Healing with neuroplastic changes

Healing

with

neuroplastic

changes

CS

Dendritic Transmission
Pre-synaptic terminal
Somatic Input
Post-synaptic active
Long-term potentiation
### CS: Wind-up → LTP

Once CS, not necessarily dependent anymore of nociceptive activity.

1. C-fiber transmission
2. NMDAr-activated Ca\(^{2+}\) entry in DH neurons
3. Signaling cascades
4. ↑ synaptic excitability over long distance
5. ↓ C-fiber transmission

<table>
<thead>
<tr>
<th>Wind-up</th>
<th>LTP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low frequency (0.33 Hz-0.50 Hz)</td>
<td>High frequency (0.5-5Hz)</td>
</tr>
<tr>
<td>Up to few minutes</td>
<td>Up to months</td>
</tr>
<tr>
<td>Can lead to LTP: NMDAr activation + retrograde Sub P</td>
<td>Early phase: NMDAr activation + post-synaptic changes Late phase with protein synthesis</td>
</tr>
<tr>
<td>Rather a paradigm to test excitability</td>
<td>Source for CS</td>
</tr>
<tr>
<td>Activity-dependent</td>
<td>After installation no longer activity dependent</td>
</tr>
<tr>
<td>Homosynaptic</td>
<td>Heterosynaptic</td>
</tr>
<tr>
<td>Dorsal horn</td>
<td>Dorsal horn &amp; brain</td>
</tr>
</tbody>
</table>
Temporal summation (TS)

- Paradigm to evaluate bottom-up excitability
- Enhanced TS in CS:
  - Faster
  - More intense
  - Longer after-sensations

(Lemming et al. 2012; Staud, etc.)

TS in cancer pain

2. Changes in top-down pathways:

Central Sensitisation: mechanisms

Would this hurt?

Biopsychosocial model of pain
adapted from Loeser

Meeus & Nijs, 2007; Nijs & Van Houdenhove 2008; Yarnitsky et al. 2010
What if?

Impaired pain inhibition

Descending inhibitory pathways in dorsolateral funiculus:
- Inhibitory substances (serotonin, opioids, etc.) in synapses in dorsal horn

Experimental block or lesions of pathways → equivalent of CS
CS: Impaired pain inhibition

• Spinal block $\Rightarrow$ inhibition
  $\Rightarrow$ expansion receptive fields
  $\Rightarrow$ hypersensitivity
  $\Rightarrow$ faster Wind-up

$\Rightarrow$ Presynaptic activity not essential for CS
$\Rightarrow$ CS by failing endogenous pain inhibition
Impaired pain inhibition

Conditioned pain modulation

• Deficient in different chronic pain populations
CPM in cancer pain

CPM = % change in $PPT_{\text{counterstimulus}}$ relative to $PPT_{\text{baseline}}$

CPM in pain group < non-pain group!

Experiment (n=2)

20 x

Exercise-induced hypoalgesia
Evidence for exercise-induced hypoalgesia


Top-down & bottom-up influences on nociceptive processing
Mechanisms of EI hypoalgesia


Evidence for exercise-induced hypoalgesia

Evidence for exercise-induced hypoalgesia in chronic pain?

<table>
<thead>
<tr>
<th>Condition</th>
<th>Hypoalgesia</th>
<th>Hyperalgesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic low back pain</td>
<td>?</td>
<td>+</td>
</tr>
<tr>
<td>Shoulder myalgia</td>
<td>+</td>
<td>?</td>
</tr>
<tr>
<td>(generalized and localized when contracting painfree muscle)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rheumtoïd arthritis</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>(generalized and localized)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Osteoarthritis (hip and knee)</td>
<td>-/+</td>
<td>-/+</td>
</tr>
<tr>
<td>(depending on CPM)</td>
<td>(depending on CPM)</td>
<td></td>
</tr>
<tr>
<td>Chronic whiplash-associated disorders</td>
<td>+</td>
<td>-/=</td>
</tr>
<tr>
<td>(depending on CPM; Ex intensity??)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fibromyalgia</td>
<td>-/+</td>
<td>-/+</td>
</tr>
<tr>
<td>(depending upon ex intensity)</td>
<td>(depending upon ex intensity)</td>
<td></td>
</tr>
<tr>
<td>CFS with chronic widespread pain</td>
<td>?</td>
<td>-</td>
</tr>
</tbody>
</table>

Why exercise-induced hyperalgesia?

- ↑ NMDA receptor function in the RVM
- ↑ SERT activity

• Abnormal CPM ≈ Central Sensitization?

• Psychosocial variables?

• ↑ peripheral nociceptive input?

• ...?

Lima et al. J Physiol 2017: Epub ahaed of print

2. Changes in top-down pathways:

Central Sensitisation: mechanisms

Meeus & Nijs, 2007; Nijs & Van Houdenhove 2008; Yarnitsky et al. 2010
Catastrophizing

Catastrophic thinking about pain

- More intense pain
- Higher disability
- Increased use of health care services and medication
- Predictive of persistence of pain and disability

Sullivan et al. (2001), Crombez et al. (2003), Quartana et al. (2009), Lu et al. (2011), Edwards et al. (2009)
Stress (emotional, physical)

Tak et al. *Biol Psychol* 2011

Chronic stress

GABA neurotransmission↓

Suarez-Roca et al. 2008
Gaba, main inhibitory NT

Chronic stress

GABA neurotransmission↓
Serotonergic activity↓
Disinhibition
Hyperalgesia

Suarez-Roca et al. 2008
Pain is not over when the needle ends...

- **Children's memories for pain** may contribute to the development and maintenance of later **chronic pain** (operant and respondent learning processes and altered processing within the CNS).

- Early pain memories relate to **fear** and **avoidance** of medical care in adulthood.

- In addition to experiencing pain during medical procedures, many children also experience **fear before procedures** even begin, which can **heighten a child's pain perception**

  Cohen et al. 2002; Flor & Birbaumer 1994; Pate et al.1996; Rhudy & Meagher, 2003; Sun-Ok & Carr 1999

Psychosocial basis

Classical conditioning
- Automatic or reflexive response
- Learning through association of stimuli

Operant conditioning model
- Active response
- Learning through consequences of behavior (punishment or reward)

Biological basis

- Central sensitization entails increased synaptic efficiency / excitatory synapses ~ learning / memory (hippocampus)
- LTP in part regulated by cortisol & noradrenaline in the brain (stress!)
Overactive pain neuromatrix

Moseley, 2003
Mechanisms of CS

- ↓ descending inhibition
- ↑ descending facilitation
- Cognitive emotional sensitization
- Altered sensory processing in the brain

- Wind-up dorsal horn neurons
- ↑ neuronal receptive fields
- Persistent sensitization of WDR neurons
Table 1
Distinct reaction of microglia, astrocytes, and satellite glial cells (SGCs) in different pain conditions, as examined by upregulation of the glial markers IBA1, CD11b, and glial fibrillary acidic protein (GFAP).

<table>
<thead>
<tr>
<th>Pain conditions</th>
<th>Microglia</th>
<th>Astrocytes</th>
<th>SGCs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nerve injury</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td>Spinal cord injury</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td>Paw incision</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td>Inflammation</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td>Joint arthritis</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td>Bone cancer</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td>Skin cancer</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td>Chemotherapy</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td>Diabetes</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td>HIV neuropathy</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td>Chronic opioid</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td>Acute opioid</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
</tbody>
</table>

Detailed, with related references, in Section 2.1. Symbols: Right-upward diagonal arrow (↗) denotes upregulation; right&left horizontal arrow (↔) denotes no regulation; right-downward diagonal arrow (↘) denotes downregulation.

Symptoms of central sensitization

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Neuropathic central sensitization pain in physical therapy practice

HIV-related neuropathic pain as an example

A/Prof Romy Parker\textsuperscript{PhD}
Director: Pain Management Unit
Neuropathic Pain

• **What is neuropathic pain?**
  – pain that arises as a ‘direct consequence of a lesion or disease affecting the somatosensory system’ (Jensen et al, 2011)

• **What conditions do you treat that involve neuropathic pain?**
  – HIV, diabetes, alcohol abuse, spinal injuries, trigeminal neuralgia

PAIN IN HIV/AIDS

• Pain is recorded as the second most commonly reported symptom in several populations of People Living With HIV/AIDS (PLWHA)

• Systematic review (Parker et al, 2014a)
  – 60 studies reporting on prevalence of pain
  – Samples predominantly
    • Male
    • Homosexual
    • Developed countries
PAIN IN HIV/AIDS

• But what is the prevalence of pain in developing countries where people living with HIV are predominantly female and have contracted the virus through heterosexual contact?

• Cross-sectional study of amaXhosa women in Cape Town, South Africa (Parker et al, 2017)
The sample

- 229 amaXhosa women living with HIV/AIDS
- Mean age 30 yrs (± 4.83)
- Able to speak and write a mean of 2 different languages
- 65.5% (150) unemployed.
- Completed 10 ± 1.69 years of school
- 58% single, 36% married or living with a partner

Disease markers

- CD4+ count
  - 213 ± 185 on diagnosis
  - 330 ± 211 most recent
- Clinical Stage:
  - 58% stage III or IV
- 79% on first line ARV’s
Pain

• Prevalence of pain – 74% (95%CI 68–79%).
  – 170 of the women interviewed had pain in the previous week

• Median of 2 different painful areas (1 – 6)

Sites of Pain

- Thorax: 33
- Flank: 23
- Bilateral Hands: 3
- Bilateral Lower limbs: 32
- Bilateral Feet: 31
- Full body pain: 7

Parker et al, 2017
Pain

- Pain Severity Score 5.06 ± 1.57
- Pain Interference Score 6.39 ± 1.96
  - Greatest interference was with the category "enjoyment of life" (7.07 ± 2.46)
Predictors of pain?

• Those with pain had significantly worse scores on
  – Self-efficacy (p < 0.05)
  – HRQoL (p < 0.01)
  – Depression (p < 0.01)
  – Likelihood of PTSD (p < 0.05)

Predictors of pain in HIV/AIDS

• People with pain have
  – Higher levels of unemployment (p < 0.05)
  – Fewer number of years in school (p < 0.01)

• There are no links between any disease markers and pain in PLWHA
Pain in HIV/AIDS

What does this suggest about pain in PLWHA?

Neuropathic Pain in HIV

- Painful neuropathies in HIV include:
  - Distal Symmetrical Polyneuropathy (DSP) and Antiretroviral Toxic Neuropathy (ATN)
  - Herpes
    - Acute and postherpetic neuralgia
    - Mononeuritis multiplex
Neuropathic Pain

- Treatment guidelines suggest:
  1. Pregabalin or Gabapentin
  2. Tricyclic antidepressants
  3. SNRI’s

  - But none of these are effective in HIV neuropathies

Neuropathic Pain

- *Does Central Sensitization contribute to Neuropathic pain in PLWHA?*
  - Multiple pain sites
  - No links between pain and disease processes
  - Large placebo responses to treatment
Placebo or Meaning Responses

- PLWHA with neuropathic pain appear to have significant placebo responses to treatment:
  - Six-week peer-led exercise and education
  - Pregabalin

Non-pharma Treatment of Pain in PLWHA

- What effect a six-week peer-led exercise and education intervention on pain in PLWHA?
Testing the intervention

• A single blind randomised controlled trial exploring the effects of a 6-week peer-led exercise and education intervention in amaXhosa women living with HIV.

• Participants identified from a previous study determining the prevalence of pain.

The intervention

• A six-week peer-led exercise and education intervention
  – Six-weeks
  – Group work
  – Peer-leaders
  – Education
  – Exercise
The intervention

• “Positive Living” workbook
  – Self-management
  – Exercise
  – Managing common symptoms
  – Pain
  – Nutrition

• All linked with problem solving tasks and goal setting activities

Testing the intervention

• Experimental group: attended intervention programme once a week for 6-weeks (2hours)
  – Exercise
  – Education and discussion
  – Weekly goal setting
  – Relaxation

• Control group: provided with information workbook used to guide intervention programme.
Testing the intervention

• Participants were interviewed at weeks 0, 4, 8, 12 and 16
  – Demographic and disease history
  – Pain (BPI-Xhosa)
  – Self efficacy (SE-6-Xhosa)
  – Health related quality of life (EQ5D-Xhosa)
  – Risk for depression (BDI-Xhosa)

Results

• Week 0 measures vs. prevalence of pain measures (15 months previously - a period of normal care with no interventions)
  – No changes in pain, self-efficacy, or depression.
  – Improvement in HRQoL

  – i.e. routine care had no effect on their pain
What was the effect?

• Both the experimental and control groups had clinically meaningful improvements in pain

• Why
  – The “care effect” or meaning response
  – The South African health care setting has been described as “hostile”
  
  *What effect might this have on someone with a chronic illness?*
Meaning Responses elsewhere?

**Pregabalin for painful HIV neuropathy**
A randomized, double-blind, placebo-controlled trial

D. M. Simpson, MD; G. Schiffito, MD; D. B. Clifford, MD; T. K. Murphy, PhD; E. Durso-De Cruz, PhD; P. Glue, MD; PhD; F. Whalen, PhD; B. Fmrir, PhD; G. N. Scott, PharmD; R. Freeman, MD; and On behalf of the 1666 HIV Neuropathy Study Group

**Conclusions:** Pregabalin was well-tolerated, but not superior to placebo in the treatment of painful HIV neuropathy. Factors predicting analgesic response in HIV neuropathy warrant additional research.

Pharmacological Management of Neuropathic Pain in HIV

**Figure 3** Mean change from baseline in Numeric Pain Rating Scale score
Meaning Responses

- Why do PLWHA and pain respond so well in studies?
  - Pain is a response to threat – even neuropathic pain
  - People with HIV (and other chronic illnesses?) suffer from:
    - Persistent traumatic stress (Frenkel et al, 2017)
    - Stigma (Wadley et al, 2016)
    - Hostile treatment settings (Parker et al, 2017)

Pathological Peripheral Nerve
  - Spontaneous firing of action potentials
    - Ectopic pacemaker-like activity
    - Central Sensitization
      - SC
        - Expression of synaptic transmitters, receptors and other genes that modify transmission and responsiveness change.
        - Upregulation of α2δ subunit of voltage-gated calcium channels
      - Central Sensitization
        - Cortex
          - Alterations in cortical function, loss of grey matter
          - Altered connections between SC and cortex - increase in descending facilitation and reduced descending inhibition

Neuroimmune interactions:
1. Inflammation in periphery – TNFα increases ectopic activity
2. Microglia – act on dorsal horn neurones further sensitizing
Neuropathic Pain and Central Sensitization

• Treatments that target the peripheral nerve are limited
• Treatments that target the spinal cord are limited
• Treatments need to target all the mechanisms
  – We need to include the cortex in our assessment, reasoning and treatment of neuropathic pain

Tea Break

Romy.parker@uct.ac.za
Neuropathic central sensitization pain in physical therapy practice

Skills training

Assessing for Neuropathic Pain

• Diagnosing neuropathic pain is based on:
  – History
  – Clinical examination

• Some useful tools
  – DN4
  – LANSS
  – PainDETECT
DN4
(Bouhassira et al, 2005)

- 10 item clinician-administered
- Seven items related to pain quality (i.e. sensory and pain descriptors) are based on history,
- 3 items based on the clinical examination.
- A score of ≥4 positive for neuropathic pain.

LANSS
(Bennett, 2001)

- Leeds Assessment of Neuropathic Symptoms and Signs
  - Self-report
- Score of ≥12 indicates pain of neuropathic origin
Pain DETECT  
(Freynhagen et al 2006)

- Designed to assess for neuropathic pain in LBP
- Self-report
- Interpretation
  - ≤12 neuropathic pain unlikely
  - ≥19 neuropathic pain likely
  - In between – further examination recommended

Workshop

- In groups of three
  - Each person complete one of the instruments as the patient
  - As a group check you can score them
  - Discuss the clinical utility of each instrument for your setting
Treating Neuropathic Pain

• **Workshop**
  – In groups of 6 discuss
    • What treatments do we have a physiotherapists which target Neuropathic mechanisms?
    • What are the barriers to these treatments in your setting?
    • What facilitators are there to using these treatments?
Treating Neuropathic Pain

• How can we enhance the meaning effect?

Lunch Time

Romy.parker@uct.ac.za
Clinical assessment of central sensitisation - physiotherapy

Michele Sterling
BPhy, MPhty, Grad Dip Manip Physio, FACP, PhD
Director NHMRC CRE in Road Traffic Injury
Associate Director, Recover Injury Research Centre
Menzies Health Institute Qld, Griffith University
Adjunct Professor, Centre for Advanced Imaging, UQ

Central Sensitisation
There is no Gold Standard of measurement

How do we recognize it in the clinic:

• Patient history/interview
• Questionnaires
• Physical examination

THINK PAIN MECHANISMS!!

Sterling M. JMPT (2008), 31(7)
Patient Interview (subjective examination) is Important

- May also have higher levels of pain & disability – use validated measure

Patient Interview (subjective examination) is Important

- Detailing of patient’s symptoms
  - pain area
  - nature of the pain
  - irritability
  - sleep disturbance
- Cold hyperalgesia
  - pain with cold
- Mechanical hyperalgesia/allodynia
- Autonomic disturbances
Findings from patient interview that may be suggestive of central sensitisation

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Example of patient report</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mechanical allodynia/hyperalgesia</td>
<td>Pain with touch</td>
</tr>
<tr>
<td></td>
<td>Pain from clothes or bedclothes</td>
</tr>
<tr>
<td>Thermal allodynia/hyperalgesia</td>
<td>Pain with cold (e.g., ice, cold weather)</td>
</tr>
<tr>
<td>Irritable condition</td>
<td>Pain is easily aggravated but difficult to settle infers the presence of sensitisation</td>
</tr>
<tr>
<td>Sleep disturbances</td>
<td>Difficulty sleeping due to pain</td>
</tr>
</tbody>
</table>

Questionnaires


- **Central Sensitisation Inventory (CSS)**
Physical Examination

<table>
<thead>
<tr>
<th>Clinical tests</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manual examination of the affected area</td>
<td>Presence of aldynia (pain with light touch) infers central sensitisation</td>
</tr>
<tr>
<td>Manual examination of structures away from the affected area eg UL and LL in patient with neck pain</td>
<td>Presence of aldynia/hyperalgesia infers central sensitisation</td>
</tr>
<tr>
<td>Pressure pain thresholds</td>
<td>Decreased pain thresholds at sites away from the neck may indicate central sensitisation</td>
</tr>
<tr>
<td>Cold sensitivity</td>
<td>Pain with ice application - cold hyperalgesia</td>
</tr>
<tr>
<td>Neural tissue provocation test eg ULT, SLR</td>
<td>Bilaterally reduced elbow extension infers central hyperexcitability of motor responses</td>
</tr>
</tbody>
</table>

Types of Quantitative Sensory Testing (QST)

- Mechanical
  - Pressure Detection Threshold
  - Two-point Discrimination
  - Pressure Pain Detection Threshold
  - Pressure Pain Tolerance

- Electrical
  - Vibratory
  - Chemical

- Thermal
  - Cold/Warm Detection Threshold
  - Cold/Warm Pain Detection Threshold
  - Cold/Warm Pain Tolerance
  - Cold/Warm Endurance
Pressure Pain Thresholds (PPTs)

Local – over site of injury/pain
• could be peripheral sensitisation

Remote – away from site of injury/pain
• likely indicates CNS changes

How to apply

• Instructions:
  • ‘I’m going to slowly apply pressure to the skin over top of your muscle. Please [tell me] the moment the sensation changes from pressure to pain.’

• Application tips:
  • Screen facing away from you
  • Increase force ~5N/s
  • Wait at least 30 seconds b/w applications
What is sensitive?

PPT (neck):
< 185 kPa; 1.8 kgF (females)
< 210 kPa; 2.1 kgF (males)

PPT (med N):
< 210 kPa, 2.1 kgF (females)
< 250 kPa; 2.5 kgF (males)

Tib Ant:
< 230 kPa, 2.3 kgF (females)
< 360 kPa; 3.6 kgF (males)

Need the most sensitive unit

Cold Sensitivity

• ‘Gold standard’
  • Medoc TSA-II Neurosensory Analyzer
  • ~$20,000

• Other options
  • Ice
  • Cold nail
  • Different materials
The ‘cheap’ method – apply something cold

An investigation of the use of a numeric pain rating scale with ice application to the neck to determine cold hyperalgesia
Samuel Maxwell, Michele Sterling

10 seconds of ice application

5 seconds of cold iron nail application

Measurement of cold pain threshold

- No clinical device available to quantify cold pain threshold
- \( N=63 \) chronic WAD
- testing with lab equipment
  - Cold hyperalgesic \( \geq 13 \) degrees C
  - Not Cold hyperalgesic < 13 degrees C
- Application of ice to neck, 10 seconds, NRS pain
- ROC analysis

(Maxwell & Sterling 2012, Manual Therapy.)
Measurement of cold pain threshold

- No clinical device available to quantify cold pain threshold
- Application of ice to neck, 10 seconds  (Maxwell & Sterling 2012, Manual Therapy)

<table>
<thead>
<tr>
<th>NRS score</th>
<th>Sensitivity (95% CI)</th>
<th>Specificity (95% CI)</th>
<th>Positive Likelihood Ratio (95% CI)</th>
<th>Negative Likelihood Ratio (95% CI)</th>
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<tbody>
<tr>
<td>=0</td>
<td>100.0(94.4-100.0)</td>
<td>0.0(0.0-6.0)</td>
<td>1.00</td>
<td></td>
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<tr>
<td>&gt;0</td>
<td>93.75(84.8-98.3)</td>
<td>48.33(35.2-61.6)</td>
<td>1.81(1.4-2.4)</td>
<td>0.13(0.05-0.3)</td>
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<tr>
<td>&gt;1</td>
<td>89.06(78.8-95.5)</td>
<td>60.00(46.5-72.4)</td>
<td>2.23(1.8-2.8)</td>
<td>0.18(0.08-0.4)</td>
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<tr>
<td>&gt;2.3</td>
<td>76.56(64.3-86.2)</td>
<td>71.67(58.6-82.5)</td>
<td>2.55(2.1-3.2)</td>
<td>0.33(0.2-0.6)</td>
</tr>
<tr>
<td>&gt;3</td>
<td>64.06(51.1-75.7)</td>
<td>83.33(71.5-91.7)</td>
<td>3.84(3.1-4.8)</td>
<td>0.43(0.2-0.8)</td>
</tr>
<tr>
<td>&gt;4</td>
<td>54.69(41.7-67.2)</td>
<td>88.33(77.4-95.2)</td>
<td>4.69(3.7-6.0)</td>
<td>0.51(0.2-1.1)</td>
</tr>
<tr>
<td>&gt;5</td>
<td>42.19(29.9-55.2)</td>
<td>95.00(86.1-99.0)</td>
<td>8.44(6.3-11.3)</td>
<td>0.61(0.2-1.9)</td>
</tr>
<tr>
<td>&gt;6.33</td>
<td>25.00(15.0-37.4)</td>
<td>95.00(86.1-99.0)</td>
<td>5.00(3.3-7.7)</td>
<td>0.79(0.3-2.4)</td>
</tr>
<tr>
<td>&gt;7</td>
<td>23.44(13.8-35.7)</td>
<td>100.0(94.0-100.0)</td>
<td>0.77</td>
<td></td>
</tr>
</tbody>
</table>

Dynamic Tests: Does the system work?

- Conditioned Pain Modulation (CPM)
  - Test pain threshold
  - Apply a ‘conditioning stimulus’
  - Re-test pain threshold after 30 seconds
  - Positive test: <10% increase in pain threshold on the re-test
  - Indicates dysfunctional descending nociceptive inhibitory control (DNIC)
CPM: Conditioning Stimuli

- Ice water immersion x 1-2 mins
- Inflation of a BP cuff x 30 – 60 sec
- High-intensity exercise or isometric holds (e.g. wall squat, plank, neck flexion in supine)

Temporal Summation

- To add
Summary

• QST is a psychophysical test that tells you something different about a patient’s pain condition than other clinical tests or PROs
• Can help to develop a prognostic or theranostic phenotype
• PPDT is accessible now and reasonably well-supported
• CPDT is emerging, mechanism still unclear

Thank you

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www.recover.edu.au
Content overview

• Introduction

• Central sensitization: maladaptive neuroplasticity in patients with chronic pain (Kelly)

• Neuropathic central sensitization pain in physical therapy practice: HIV-related neuropathic pain as an example (Romy)

• Neuropathic central sensitization pain in physical therapy practice (Romy & Michele)

• **Non-neuropathic central sensitization pain in physical therapy practice: Neck pain as an example** (Michele & Jo)

• Non-neuropathic central sensitization pain in physical therapy practice: case study (Kelly & Jo)

*Pain in Motion*

Aberrant glia activity & central sensitization

activated glia in PFC + amygdala + hippocampus

IL-1↑ + IL-6↑ = neuroinflammation
The role of physical trauma

chronic non-specific neck pain

whiplash

no whiplash exposure

generalized hyperalgesia

local hyperalgesia

Malflie et al. Pain Physician 2015
Van Oosterwijck et al. Eur J Pain 2013
Stone et al. Man Ther 2013

Dysfunctional endogenous analgesia in response to exercise

Cognitive-emotional factors, including maladaptive pain cognitions

Central sensitisation (ie, hypersensitivity of the nervous system)

Impaired cervical neuromuscular control

Post-traumatic stress

Dysfunctional stress response systems

Whiplash-associated disorders

Nijs & Ickmans The Lancet 2014;384(9938):109-111.
Chronic stress activates the glia

- Adrenaline
- Cortisol

Activated glia in PFC + amygdala + hippocampus

Stress & central sensitization

- Chronic stress
- IL-1↑ + IL-6↑ = neuroinflammation

Activated glia in PFC + amygdala + hippocampus
Pain education → Stress management → Sleep management

PAIN IN MOTION

Pain education → Stress management → Sleep management

Recovery ← Retraining pain memories ← Graded activity

PAIN IN MOTION
Stress & sleep interconnected

stress ↑ ————> sleep ↓

Sleep deprivation triggers brain inflammation

Sleep deprivation triggers brain inflammation


What if 100 people >60y take sleep drugs for 1 week?

- n=7: 25min more sleep + wake up 1x less per 2 nights
- n=76: no change
- n=17: side effects

Do patients need to buy an expensive mattress?

How can we improve sleep in chronic pain patients?

1) Cognitive behavioural therapy
2) Acceptance & commitment therapy
3) Exercise therapy

History taking about sleep

- Sleeping hours
- Sleeping at daytime
- Sleep quality & quantity
- Recovering sleep?
- Premorbid sleep
- Activities & food intake hours before going to bed
- Sleep perceptions
- Sleep medication

PAIN IN MOTION

Sleep management

changing negative thoughts about sleep
Because of your poor sleep, your central nervous system becomes inflamed ...

Sleep management
- changing negative thoughts about sleep
- sleep hygiene
The brain should (re)connect bedroom + sleep
**Sleep education**

- Daytime sleeping
- Hourglass metaphor
- Jetlag metaphor
- Secretion melatonin epiphysce
  ~ sleep-waking rhythm

**Sleep management**

- changing negative thoughts about sleep
- sleep hygiene
- sleep restriction therapy
Sleep restriction therapy

4 hours of sleep / night
11:00 pm
7:00 am
= 50% sleep efficiency

12:00 pm
5:00 am
= 80% sleep efficiency

Sleep restriction therapy

12:00 pm - 5:00 am (4 hours sleep)
= 80% sleep efficiency

12:00 pm – 6:00 am (5 hours sleep)
= 83% sleep efficiency

11:00 pm – 6:00 am (6 hours sleep)
= 86% sleep efficiency
Sleep management

- changing negative thoughts about sleep
- sleep hygiene
- sleep restriction therapy
- teaching relaxation skills

Non-neuropathic central sensitization pain in physical therapy practice: Neck pain as an example

**Michele Sterling**
BPhy, MPhty, Grad Dip Manip Physio, FACP, PhD
Director NHMRC CRE in Road Traffic Injury
Associate Director, Recover
Menzies Health Institute Qld, Griffith University
Adjunct Professor, Centre for Advanced Imaging, UQ
Stress Related Responses

- PTSD symptoms predict poor recovery

Original Investigation

Relationship Between Stressfulness of Claiming for Injury Compensation and Long-term Recovery
A Prospective Cohort Study

- 34% high levels of stress understanding claim
- 30.4% with claim delays
- 27% with number medico-legal assessment
- 26% with amount of compensation
- Predicted disability:
  - WHODAS (+6.94 pts); HADS (+2.61)
  - Lower QOL – WHODAS (-0.73 pts)

Recovery Pathways

Predicted disability trajectories & predicted probability of membership (%).

N=155
Group based trajectory modeling
2-3 months important

Sterling, Hendrikz, Kenardy 2010 Pain 150:22-28
Predictors of Disability Trajectories

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coeff (SE)</th>
<th>OR (95% CI)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cold Pain TH &gt; 13°C</td>
<td>3.27 (0.85)</td>
<td>26.3 (4.98, 139)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Pain (VAS) &gt; 5/10</td>
<td>1.46 (0.27)</td>
<td>4.31 (2.55, 7.28)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Age (&gt; 37)</td>
<td>0.103 (0.03)</td>
<td>1.11 (1.04, 1.18)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Sterling, Hendrikz, Kenardy 2010 Pain 150:22-28

Posttraumatic stress symptoms

Sterling, Hendrikz, Kenardy 2010 Pain 150:22-28
Predictors of posttraumatic stress Trajectories

Sterling, Hendrikz, Kenardy 2010 Pain 150:22-28

New Clinically Significant Pain 6 Weeks after trauma

MVC

Sexual Assault

McLean et al 2014 PAIN

Ulirsch et al 2013 Eur J Pain
Striking similarity in proportion of trauma survivors with new neck or back pain at six weeks, despite radically different types of tissue trauma (<1/3 of sexual assault survivors report any physical assault), suggests that no specific tissue injury is necessary or sufficient to cause posttraumatic neck or back pain.

Recall of traumatic event

Pressure Pain thresholds

Thermal Pain thresholds

Dunne-Proctor, Kenardy, Sterling Clin J Pain 2015
Implications for Management

- Stress comes from a variety of sources
  - The event/accident/injury
  - Interactions with health care providers
  - Interactions with compensation process

- Stress factors influence ‘biological’ processes
  - Sensory thresholds/pain processing
  - Possibly healing processes

- Treatment may need to address stress related factors
  - Acute vs chronic
  - Target those most at risk; many recover well
  - Improve compensation procedures

Targeting stress responses & central sensitisation
Whiplash Grade II
No psychopathology – PHQ-9, ASDS, past history
Medium/high risk based on CPR
6 week intervention & 6wk, 6 and 12 month follow-up

Targeting psychological factors in acute whiplash

Potential to prevent later sequelae
  Central neuroplastic changes may be irreversible

Target vulnerable and ‘at risk’ patients

Treatment based on peripheral pathology models are not very effective
  Exercise/MT interventions only small effects
  (Southerst et al 2014, The Spine Journal)

  Too much might even be iatrogenic
  (Skillgate et al, Arch Phys Med & Rehab, 2016)
The case for using physiotherapists

• Patients not keen on seeing a psychologist

"GP and/or insurance company sent me to a psychologist – that was worthless; I have whiplash."

• Not feasible to see a psychologist

Psychological debriefing non recommended post trauma (Aust PTSD Guidelines)

• Physiotherapists are commonly involved

“GP not listening and not believing that I am in pain”; “feel let down by lawyers and GPs”

“Start Physiotherapy as soon as possible”

“Physiotherapy is very good as soon as possible. Doing the exercises the Physio gives you. I also used warmth on my neck to ease pain twice a day”

• Using current primary care resources

• Funding/compensation implications

Interventions

• SIT + physiotherapy exercise

• Physiotherapy exercise alone

<table>
<thead>
<tr>
<th>Week</th>
<th>Sessions/week</th>
<th>SIT and Physiotherapy Exercise</th>
<th>Physiotherapy Exercise</th>
</tr>
</thead>
</table>
| 1    | 2             | Session 1: Intro to SIT, Physiotherapy Exercise  
Session 1b: Physiotherapy Exercise | Session 1: Physiotherapy Exercise  
Session 1b: Physiotherapy Exercise |
| 2    | 2             | Session 2: SIT/Physiotherapy Exercise  
Session 2b: Physiotherapy Exercise. | Session 2: Physiotherapy Exercise.  
Session 2b: Physiotherapy Exercise. |
| 3    | 2             | Session 3: SIT/Physiotherapy Exercise.  
Session 3b: Physiotherapy Exercise  | Session 3: Physiotherapy Exercise  
Session 3b: Physiotherapy Exercise |
| 4    | 2             | Session 4: SIT/Physiotherapy Exercise.  
Session 4b: Physiotherapy Exercise | Session 4: Physiotherapy Exercise  
Session 4b: Physiotherapy Exercise |
| 5    | 1             | Session 5: SIT/Physiotherapy Exercise | Session 5: Physiotherapy Exercise |
| 6    | 1             | Session 6: SIT/Physiotherapy Exercise | Session 6: Physiotherapy Exercise |
Exercise Program

Specific Exercise – Low load movement/control & sensorimotor training
Progression to higher loads
Progression to functional activities
Return to usual enjoyable activities

Aerobic exercise

SIT + Physiotherapy Exercise

Stress Inoculation Training:

3 phases

Identifying and understanding stress
- Education about the influence of stress on nociception/pain
- What thoughts, feeling, actions have you noticed increase or decrease your whiplash pain?

Developing skills
- Relaxation
- Problem solving
- Helpful coping self statements

Applying skills in various stressful situations
- Identify specific stressor
- Prepare for stress
- Plan into action and review
- Cannot move all anxiety, just keep it manageable
Outline of SIT Sessions

<table>
<thead>
<tr>
<th>Session</th>
<th>Overview</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Introduction to Stress Inoculation Training, why it is important, theories of pain and abdominal breathing exercise</td>
</tr>
<tr>
<td>2</td>
<td>Body Scans</td>
</tr>
<tr>
<td>3</td>
<td>Problem Solving</td>
</tr>
<tr>
<td>4</td>
<td>Coping Statements</td>
</tr>
<tr>
<td>5</td>
<td>Applying SIT to the real world</td>
</tr>
<tr>
<td>6</td>
<td>Coping Skills Maintenance: Early warning signs, coping plans, relapse prevention and maintenance</td>
</tr>
</tbody>
</table>

Preliminary Results

- Intervention is acceptable to patients and physiotherapists
  - Credibility/expectancy questionnaire
  - Physios (n=11) ranked credibility as 20±2
  - Patients (n=57) ranked credibility as 19.6±2.5/10

- Physiotherapists can successfully deliver the intervention
  - Audit of recorded sessions by clinical psych
  - 2 day training + accreditation
  - Random follow-up audits
Preliminary Data

Musculoskeletal Pain

- Exercise based interventions are common
- Exercise recommended in clinical guidelines
Chronic WAD

Comprehensive physiotherapy exercise program or advice for chronic whiplash (PROMISE): a pragmatic randomised controlled trial (ACTRN12609000825257)

Michaleff, Maher, Lin, Rebbeck, Jull, Connelly, Sterling

The Lancet (2014)
Exercise

? DOSE

? TYPE

? INTENSITY

? Duration
Different mechanisms seem to underlie different MSK conditions

Elliott et al Clinical Radiology 2008
Chien, Eliav, Sterling 2009 Manual Therapy

Hypoalgesia & Exercise

Sub-Maximal Exercise 75% MHR, 15 mins

Self-paced, physiologically limited, aerobic threshold >80% MHR

Exercise induced hypoalgesia is elicited by isometric, but not aerobic exercise in chronic WAD.

Ashley Smith, Carrie Ritchie, Ashley Pedler, Kaitlin McCamley, Kathryn Roberts, Michele Sterling

Only upper body exercise significantly raised pain thresholds in the knee OA group, with variable non-significant effects following lower body exercise.

Hypoalgesia After Exercise and the Cold Pressor Test is Reduced in Chronic Musculoskeletal Pain Patients With High Pain Sensitivity

Howell B. Vaqtor, MSc;** Gitte Randborg, MD,*
and Thomas Graven-Nielsen, DMSc, PhD†

- Inflamed Knee/ankle
- Voluntary exercise
- 2 hours/day, 4 days/week for 3 weeks in running wheel cages

Figure 2A. Voluntary exercise is anti-nociceptive. (A) While static weight bearing on the CFA-injected paw remained significantly impaired in the CFA-SED group over the course of the study, the CFA-RUN group improved from week 1 to be indistinguishable from shams by week 3.
Who Responds to exercise?

Low Back Pain

- Self-reported clinical instability predicted response to motor control exercises (Macedo et al, Phys Ther. 2014 Nov;94(11):1543-54)

- Baseline pain, pain with movement, leg pain, constant pain, pain with flexion, expectations of good effect did not predict response to McKenzie exercises (Sheets et al, Eur Spine J. 2012 Jul;21(7):1250-6)

Who Responds to exercise?

- Psychological factors did not predict response to exercise and advice in LBP (Smeets et al, Arthritis Rheum. 2009 Sep 15;61(9):1202-9)

- SES, education, and number of pain medications as treatment effect modifiers of prognostic stratified care delivered in the STarT Back Trial (Benecuik J, J Pain. 2017 Jan;18(1):54-65)

- No effect modifiers were found in rehabilitation trial for chronic WAD (Michaleff, Maher, Lin, Rebbeck, Jull, Connelly, Sterling, The Lancet (2014) 384(9938):133-41)
Sensory hypersensitivity moderates the effects of multimodal physiotherapy.
<table>
<thead>
<tr>
<th></th>
<th>1 Responder (n=38)</th>
<th>0 Non-responder (n=36)</th>
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<tbody>
<tr>
<td>PPT Tibialis Anterior</td>
<td>381.0</td>
<td>346.9</td>
<td>.368</td>
</tr>
<tr>
<td>PPT Neck</td>
<td>199.8</td>
<td>190.6</td>
<td>.702</td>
</tr>
<tr>
<td>Cx Cold Hyperalgesia</td>
<td>13.6</td>
<td>12.6</td>
<td>.578</td>
</tr>
<tr>
<td>ROMtotal - Neck</td>
<td>188.8</td>
<td>184.3</td>
<td>.747</td>
</tr>
<tr>
<td>SF36MHO</td>
<td>67.5</td>
<td>57.8</td>
<td>.042*</td>
</tr>
<tr>
<td>PTSD : Re-experiencing</td>
<td>2.2</td>
<td>3.7</td>
<td>.045*</td>
</tr>
<tr>
<td>PTSD: Avoidance</td>
<td>2.7</td>
<td>5.3</td>
<td>.020*</td>
</tr>
<tr>
<td>PTSD: Arousal</td>
<td>3.6</td>
<td>5.9</td>
<td>.015*</td>
</tr>
<tr>
<td>PTSD: Total</td>
<td>8.5</td>
<td>14.9</td>
<td>.015*</td>
</tr>
<tr>
<td>PCS rumination</td>
<td>5.6</td>
<td>7.0</td>
<td>.210</td>
</tr>
<tr>
<td>PCS magnification</td>
<td>2.7</td>
<td>3.6</td>
<td>.147</td>
</tr>
<tr>
<td>PCS helplessness</td>
<td>7.3</td>
<td>8.6</td>
<td>.376</td>
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<tr>
<td>PCSTotal - catastrophising</td>
<td>15.6</td>
<td>19.1</td>
<td>.217</td>
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<tr>
<td>SLANSStotal</td>
<td>9.9</td>
<td>10.7</td>
<td>.576</td>
</tr>
<tr>
<td>Compensation claim settled</td>
<td>Yes: (37%)</td>
<td>Yes: (37%)</td>
<td></td>
</tr>
</tbody>
</table>

Thank you

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@micheleSterlin7

www.recover.edu.au
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• Neuropathic central sensitization pain in physical therapy practice: HIV-related neuropathic pain as an example (Romy)
• Neuropathic central sensitization pain in physical therapy practice (Romy & Michele)
• Non-neuropathic central sensitization pain in physical therapy practice: Neck pain as an example (Michele & Jo)
• Non-neuropathic central sensitization pain in physical therapy practice: case study (Kelly & Jo)

Case study knee osteoarthritis

Discuss in small groups (n=3):
1) Does Mrs. Ni presents a predominant nociceptive, neuropathic or central sensitization type of knee pain?

2) What options do we have for treating Mrs. Ni’s knee pain? “Bottom-up” or “top-down” oriented interventions or a combination? Rationale behind the selection of interventions?
3) Pick an order for the selected interventions.
4) Can we treat her in a monodisciplinary PT setting?
Central sensitization predicts pain following surgery

Shoulder impingement syndrome
Total knee replacement
Thoracotomy
Spinal fusion

Baert et al. Osteoarthritis Cartilage 2016
Bennet et al. World Surgery 2017
Yarnistky et al. Pain 2008

Case study knee osteoarthritis

1) Does Mrs. Ni presents a predominant nociceptive, neuropathic or central sensitization type of knee pain?
Musculoskeletal pain

Is neuropathic pain present & able to explain the clinical picture?

YES

Disproportionate pain experience?

YES

predominant neuropathic pain

NO

no central sensitization

NO
Musculoskeletal pain

Is neuropathic pain present & able to explain the clinical picture?

- YES
  - predominant neuropathic pain

- NO
  - Disproportionate pain experience?
    - YES
      - Diffuse pain distribution?
        - YES
          - predominant central sensitization pain
        - NO
          - no central sensitization
    - NO
      - no central sensitization
Musculoskeletal pain

Is neuropathic pain present & able to explain the clinical picture?

YES

Disproportionate pain experience?

YES

Diffuse pain distribution?

YES

predominant neuropathic pain

NO

no central sensitization

NO

predominant central sensitization pain

NO

Central Sensitization Inventory ≥ 40 ?

YES

predominant central sensitization pain

NO

no central sensitization

Case study knee osteoarthritis

Discuss in small groups (n=3):

1) Does Mrs. Ni presents a predominant nociceptive, neuropathic or central sensitization type of knee pain?

2) What options do we have for treating Mrs. Ni’s knee pain? “Bottom-up” or “top-down” oriented interventions or a combination? Rationale behind the selection of interventions?
Case study knee osteoarthritis

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3) Pick an order for the selected interventions.

4) Can we treat her in a monodisciplinary PT setting?
Tailored pain neuroscience education

- Chronic pain
- Nociceptive
  - Explain source of nociception + role of the brain (pain matrix)
- Neuropathic
  - Explain pain neuroscience underlying neuropathic pain mechanisms
- Central sensitization
  - Explain pain neuroscience underlying central sensitization pain

Spam filter metaphor

Pain in Motion
‘Most people don’t understand how severe my condition is’

‘No one should have to live this way’

‘I worry that my condition is not being taken seriously’

To grade or not to grade daily activities?

Activity limitations

Avoidance behaviour

Persistance behaviour

Graded activity / graded exercise therapy

Graded exposure in vivo

Acceptance-based interventions / pacing
To grade or not to grade daily activities?

Knee pain Pt reports physical activity limitations

- Stopped cycling & swimming
  - Graded activity / graded exercise therapy
  - 40/100 fear scale

- Graded exposure in vivo

- Continued ironing
  - Acceptance-based interventions / pacing
  - 83/100 fear scale

40/100 fear scale 83/100 fear scale

? 40/100 fear scale 83/100 fear scale

To grade or not to grade daily activities?
Boy your back muscles and spinal joints feel very stiff – luckily you didn’t wait longer to come and see me!

I’m now activating the spam filter in your brain, to prevent danger messages to enter your brain.
Balancing hands-on with hands-off interventions

Hands-on treatment:
• Following pain neuroscience education
• Explain brain effects
• Do not ↑ pain anticipation
• Do not rely on pain self-report

Lluch et al. Manual Therapy 2015

Combining pain education with Mulligan joint mobilisation in knee osteoarthritis

RCT – total knee replacement surgery for OA

pain education + Mulligan vs. biomedical education + Mulligan

Pain education + Mulligan:
  • Pain catastrophizing ↓
  • Pain hypervigilance ↓
  • Fear of movement ↓
  • Global rating of change >>

Stay connected

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