THE TRANSITION FROM ACUTE TO CHRONIC MUSCULOSKELETAL PAIN: IMPLICATIONS FOR PHYSICAL THERAPY MANAGEMENT

Carol A Courtney PT, PhD, FAAOMPT

IASP definition of Pain

'a sensory or emotional experience associated with real or potential injuries'

- Individual
- Influenced by previous experience
- Pain vs Nociception

Musculoskeletal pain

- May present with myriad of symptoms
- Confusing to clinician
- Leads to non-specific diagnoses (eg: Low Back Pain)

Why devote an entire day to musculoskeletal pain?
Epidemic of Musculoskeletal Pain

- Almost 27 million U.S. adults experience some form of osteoarthritis (OA)  
  Zhang and Jordan 2010
- OA afflicts approximately one third of persons older than 65 years
- Approximately 80% of persons with OA report some degree of movement limitation; 25% cannot perform major activities of daily living, 11% of adults with knee OA need help with personal care, and 14% require help with routine needs  
  Centers for Disease Control and Prevention 2011

The Cost: Disability

- Reduces the quality of life for individuals and jeopardizes their ability to live independently  
  Hirvensalo 2000
- Increases risk of hospitalization, institutionalization, and mortality  
  Cutler 2003
- Is a major driver of health care costs resulting from arthritis  
  Lubitz 2003

MAIN TAKE HOME POINTS

- MUSCULOSKELETAL INJURY OR DISEASE and MUSCULOSKELETAL PAIN ARE SEPARATE BUT RELATED CONDITIONS
Central sensitization is a dynamic process and can happen within seconds/minutes after an injury.

There are varying degrees of central sensitization... and if not treated, may progress to regional or widespread pain and symptoms.

Regional interdependence is a clinical correlate of central nociceptive plasticity:

Understanding pain science may remove the need for new nomenclature.

Manual therapy decreases central sensitization... but we must also treat the peripheral drivers of pain.
Objectives for today

- Appreciate the neurophysiological and functional changes that may occur following joint trauma.
- Understand the role of clinical tools such as pain diagrams, pain outcome tools, and quantitative sensory testing in determining stage of chronic musculoskeletal pain.
- Apply quantitative sensory testing techniques and accurately interpret the results.

Objectives for today

- Appreciate the mechanisms behind pain flare-ups and make appropriate judgments concerning treatment dosage.
- Comprehend the value of exercise in the management of chronic musculoskeletal pain.
- Appreciate the neurophysiological effect of manual therapy and exercise following joint trauma.
- Understand the effect of manual therapy on impairments and function in individuals with chronic musculoskeletal pain.

Peripheral Receptors

- Nociceptors
- Distal end of a first order primary afferent neuron (C or Aδ fibers)

Review of pertinent neuroanatomy

- Nociceptors
- Distal end of a first order primary afferent neuron (C or Aδ fibers)

- Respond to stimulus:
  - Mechanical
  - Chemical
  - Thermal
Peripheral Nociceptive Fibers: A$\delta$ & C

- A$\delta$ – Fibers (Group III): propagate first phase or acute pain
- C – Fibers (Group IV):

Ascending: three neuron pathway

Peripheral Non-nociceptive Fibers: A$\beta$

- A$\beta$ fibers: myelinated Group II
  - Mediate sensations of light touch punctate (A$\beta$) vs deep pressure (C) Freede et al 2002, vibratory sense, joint position sense

Descending Pathways

- Regions of the brain involved in nociceptive modulation
  - Somatosensory cortex
  - Hypothalamus
  - Periaqueductal gray
  - Raphe magnus
- Fibers descend via dorsolateral funiculus and project to Lamina I and V.
Neuroplasticity

Old view: pain was hard-wired

Stimulus ➔ Response ➔ Learning

Initiation of Pain: Musculoskeletal insult

- Tearing or bruising of joint tissues results in:
  - Release of ATP & protons
  - Mast cells release histamine, 5-HT, tryptamine, & prostaglandins
  - Macrophages release cytokines & growth factors
  - Also bradykinins and COX-2
  - Primary afferent releases Substance P & CGRP

Inflammatory Soup – term first coined by CJ Woolf
(Costigan and Woolf 2000)

= primary hyperalgesia
(Increased nociceptive responses)

Peripheral Sensitization

- "stimulus evoked plasticity of the nociceptor" Woolf 2007

Inflammatory mediators bind to receptor -
- ↓ threshold
- ↑ excitability
- = hyperalgesia

Primary hyperalgesia
- restricted to the site of tissue injury
- Requires ongoing stimulus for maintenance

From: Woolf 2004

Stimulus Response Curve - Hyperalgesia

Sandkühler J Physiol Rev 2009;89:707-758
Central Sensitization

- Occurs following repetitive or intense noxious stimulus
  - Pain is no longer coupled to the noxious stimulus

Characterized by:
- Increased excitability of nociceptive pathways
- Decreased descending inhibition

Adapted from Costigan 2009

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Clinical characteristics of centrally mediated pain:
HYPERALGESIA

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Secondary hyperalgesia

- Due to change in processing of sensory input in CNS (ie, central changes)

Bajaj et al 2001

- Increased size of the peripheral receptive field

Clinical Implication:
Input from a wider region can induce pain

Bajaj et al 2001
Facet Joint Dysfunction
Pain referral patterns after intra-articular injection of 1–3 mL of 5% saline

Mooney and Robertson 1976

Another Example: Spine related leg pain

Allodynia: another aspect of hyperalgesia

Allodynia: pain with stroking of skin ("feels like sunburn")
- Centrally mediated process
  Rolke 2006

Found in the region of most pain
  Emerson-Kavcak 2011

How Assessed?
- Dynamic Cutaneous stimulus (brushing)

Regional vs Widespread Hyperalgesia

Algometry: a means of assessing for 1º and 2º hyperalgesia

Features of centrally-mediated pain
- Heightened intensity of pain
- Altered threshold of pain outside of area of injury (2º hyperalgesia)
- Pain ramps up easily (temporal summation)
- Latent pain

Courtney et al 2010
Why do some of our patients experience latent pain?

Neurogenic Inflammation

- Efferent action of nociceptive fibers
- Release Substance P, CGRP
- Causes ↑ sensitization

Kidd 1995

Bilateral Symptoms

- Rat Model: injected with 100 μL of pH 4.0 sterile saline into the gastrocnemius muscle of the left hind limb on Day 0 (injection 1) and again on Day 5 (injection 2)
- Produced bilateral mechanical hypersensitivity of the muscle and paw that lasts up to 4 wks
  
  Da Silva 2010

- Crossed spinal pathway
  
  Radhakrishnan 2003

Bilateral Symptoms Only?

- In Rheumatoid Arthritis, CVA-affected limb is spared from arthritic changes
  
  Thompson and Bywaters 1962

- In humans: lateral epicondylalgia
  
  Fernandez-Carnero 2008
What are some laboratory methods that we assess for central sensitization?

- Spinal-mediated reflex = hyperexcitable
  - Whiplash, Fibromyalgia
    Banic 2004
  - Cervical Spine Dysfunction
    Sterling 2010
  - Knee Osteoarthritis
    Courtney 2009, 2010
  - s/p anterior cruciate rupture
    Courtney 2011

Neural plasticity at the cortical level

Sleep deprivation enhances central sensitization of nociceptive pathways
Hyperalgesia: Test sites

Assessment of musculoskeletal (deep tissue) hyperalgesia

** Algometry **

Central Sensitization: Impaired Inhibition of Pain

Inhibitory Mechanisms:
- ‘body’s adaptation of incoming nociceptive information to momentary as well as long-term circumstances and needs’
  Wilder-Smith 2011
- dynamic process
- Some evidence that impaired inhibition related to sleep impairment
  Mason 2005

Figure 10 . Factors potentially driving changes in endogenous pain modulation in visceral pain syndromes. Shifted modulatory balance may act as a central mechanism in chronic pain syndromes and may predict an individual’s pain sensitivity.
**Conditioned Pain Modulation**

- Hippocratic aphorism: ‘If two sufferings take place at the same time, but at different points, the stronger one makes the weaker silent’.  
  Willer 1999

**Impaired Pain Inhibition**

- Found in many chronic pain populations including knee OA  

- Key point: manual therapy and exercise can “normalize” impaired pain inhibition mechanisms

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**Experimental Protocol**

Courtney et al JOSPT 2015 (In press)

**Tourniquet Test**

- Cuff applied (contralateral arm); inflated to 270 mmHg  
- Weight lift reps (2-5 lbs)  
- Time: 5 min; Pain (VAS): >50mm

**Ischemic Pain/Cold Pressor Test**

**Conditioned Pain Modulation Activity**
Clinical Measurement of Pain
Written Clinical Tools for the Characterization of Pain

Are there psychosocial drivers of this chronic pain condition?
- Anxiety
- Fear
- Catastrophizing

Pain Anxiety Symptom Scale (PASS)
- Developed to evaluate importance of fear of pain in persistent pain McCracken et al 1992
- 4 subscales: cognitive anxiety, escape-avoidance behaviors, fear of pain, and physiological anxiety
- Shortened version (PASS-20) most commonly used
- Scored from 0-5; max score 100
- Good reliability, mod validity Lundberg 2011
- Higher scores correlated with higher pain and disability ratings

Pain Anxiety Symptom Scale (Short Form 20) Sample Questions
Please rate each item in terms of frequency, from 0 (Never) to 5 (Always).
1. I can't think straight when in pain
2. During painful episodes it is difficult for me to think of anything besides the pain
3. When I hurt I think about pain constantly
4. I find it hard to concentrate when I hurt
5. I worry when I am in pain
6. I go immediately to bed when I feel severe pain
Tampa Scale of Kinesiophobia (TSK)

- Developed to measure fear of movement in patients with chronic low back pain (Miller 1991)
- 17 items on 4-point scale, score ranges from 17-68
- Scores >37 indicate high degree of kinesiophobia
- Good reliability and validity (Woby 2005)

Fear Avoidance Beliefs Questionnaire

- was developed by Waddell to investigate fear-avoidance beliefs among LBP patients
- consists of 2 subscales
  - Physical Activity Subscale
  - Work Subscale

Pain Catastrophizing Scale (Sullivan 1995)

- 17 items on 4-point scale, score ranges from 0-68
- Scores >30 (75th percentile) at high risk for development of chronicity

Is this pain neuropathic or non-neuropathic?
Neuropathic Pain:

defined as injury or disease of a nerve or neural structure

- Spontaneous Pain
  - Ectopic action potential discharge at site of nerve injury and resultant neuroma
  - Hyperexcitability of the primary sensory neuron
  - Shock-like bursts and a burning quality

Screening Tools: LANSS  Bennett 2001

- Leeds Assessment of Neuropathic Symptoms & Signs (LANSS)
- 5 “Yes” or “No” questions (16 pts max)
- Sensory testing for allodynia & pin-prick threshold (8 pts max)
- Scored /24
- $\geq 12 =$ neuropathic component likely
1. Does your pain feel like strange unpleasant sensations in your skin?
2. Does your pain make the skin in the painful area look different from normal?
3. Does your pain make the skin of the affected area abnormally sensitive to touch?
4. Does your pain come on suddenly for no apparent reason when you’re still?
5. Does your pain feel as if the skin temperature in the painful area has changed abnormally?

LANSS: Sensory Testing

1. Allodynia
   - Stroke affected & unaffected areas with cotton wool
   - Positive = pain or unpleasant sensations (tingling or nausea)
2. Altered Pin-Prick Threshold
   - 23 gauge needle testing affected & unaffected
   - Positive = any difference in sensation

painDETECT

- Developed to detect the neuropathic component in patients with LBP Freynhagen et al 2006
- Sensitivity = 85% Specificity = 80%
- PPV = 83%
- Classifies patients as “nociceptive”, “unclear” & “possible neuropathic”
Neuropathic vs Nociceptive (i.e., musculoskeletal) Differential Diagnosis

Screening Result

Final score

- nociceptive
- unclear
- neuropathic

A neuropathic pain component is unlikely (> 15%)

Result is ambiguous, however a neuropathic pain component can be present

A neuropathic pain component is likely (> 90%)

Please add up the following numbers, depending on the marked pain behavior pattern and the pain radiation. Then total up the final score:

- Persistent pain with slight fluctuations: -1 if marked, or +1 if not
- Persistent pain with pain attacks: +1 if marked, or -1 if not
- Pain attacks with pain between them: +1 if marked
- Radiating pain?: +2 if yes

Final score
What are the typical culprits?

- Spinal cord (e.g., cervical myelopathy)
- Spinal nerve root (e.g., radiculopathy)
- Peripheral Nerve

Dx: Cervical myelopathy

- Due to: osteophytic or disc encroachment

- Neurological findings:
  - Hypertonia
  - Babinski
  - Clonus
  - Hoffman's sign
  - Inverted Supinator Reflex

Dx: Radiculopathy

- Due to: foraminal narrowing; chemical irritation from nucleus pulposus

- Neurological findings:
  - 1st: paresthesia
  - Next: loss of light touch
  - Next: pain
  - Next: myotomal loss &
  - Hyporeflexia
  - Anesthesia

Dx: Peripheral nerve

- Due to: eg., peripheral neuropathy; nerve impingement

- Neurological findings:
  - Dependent upon type of nerve; sensory loss within distribution of nerve

Neurological Insult: Spinal cord

Neurological Insult: Spinal n. root

Neurological Insult: Peripheral nerve
Neurological Screen

- What do you do?

Assessment: Pain Diagram

**Importance:** Symptoms may seem more neuropathic than musculoskeletal
- e.g., numbness, tingling, allodynia
- Pain Pattern
  - Spread of symptoms?
- Mapping of Sensory Symptoms
  - Nerve root/peripheral nerve pattern?
- Number of Areas of Pain

The Body Chart (pain diagram)

- To be completed by the physical therapist
- Essential to get overall picture of patient’s symptoms
  - Arendt-Nielsen and Yarnitsky 2009
- Why so important?
  - Pattern Recognition
  - Diagnostic Reasoning
- Pain Pattern
  - Spread of symptoms?
- Mapping of Sensory Symptoms
  - Nerve Root? Peripheral Nerve Pattern?
- Number of Areas of Pain

Pattern Recognition

- We don’t store our knowledge in the form of biomedical or clinical textbook
  - Ex: Anatomy muscle cards; Magee
- We store our knowledge in **clinical patterns or schema**
- With ↑ **expertise**, patterns become >vivid
- Expert will cue into a pattern > quickly
Diagnostic Reasoning

- Diagnosis (by the PT) may be made on several levels
  - Medical Diagnosis
  - Movement Dysfunction Diagnosis
  - Pain Diagnosis

Diagnosis by definition should direct treatment

Subjective Examination

(should help you plan objective/physical exam)

Three main components
- Main complaint
- Pain Diagram
- Behavior of symptoms:
  What aggravates? Eases?
  (inflammatory vs mechanical basis of sx)
- History

Pain Diagram Activity

(Video)

What are some other characteristics of centrally mediated pain?
Quantitative Sensory Testing (QST)

Psychophysical means for examining patency of the somatosensory system

Clinical Assessment: Sensory Exam

- **Thermal**
  - Less reported in lower extremity musculoskeletal conditions

Types of assessment
- Hot/Cold Detection Threshold
- Heat/Cold Pain Detection Threshold

Hypoesthesia?

- C-fiber excitation presynaptically inhibits non-noxious processing from primary afferent fibers to spinal projection neurons
  - Geber C 2008, Sluka 1993 (review), Willis 1999 (review)
- Capsaicin-sensitive nociceptors provide an inhibitory control on cortical excitability of non-nociceptive somatosensory cortical neurons
  - Calford and Tweedale 1991
- Touch-gate
  - Apkarian 1994
- Vibrotactile threshold ↑ in TMD patients
  - Hollins 1996

Clinical Implication: diminished mechanical detection; vibration sense

Hyperalgesia
(We’ve discussed this)
Clinical Assessment: Sensory Exam

- **Hypoesthesia**
  - Decreased cutaneous sensation (light touch)
    - Assessed with cotton tipped applicator or monofilaments
  - Decreased vibration detection threshold
    - Clinically often reported as Yes/No

- **Hyperalgesia and Hypoesthesia?**
  - A paradox
    - ↑ sensitivity to pain + ↓ sensitivity to light touch/vibration
  - Independent Mechanisms
    - both triggered by nociceptive input
    - both indicate central plasticity
    - Geber 2008
  - Possible clinical importance?

Instability during functional activities

Pain and Instability

- Vibratory sense may be a marker for ↓ knee proprioceptive acuity
  - Shakoor 2005

- Pain associated with ↓ knee proprioceptive acuity
  - Felson 2009

Pain and Instability

- Relationship between QST measures and instability during a step task

- Found ↑ PPT sensitivity and hypoesthesia (↓ light touch and vibration) at the knee

- Demonstrated a correlation between ↓ vibration sense and instability while negotiating a step
  - Kavchok et al 2011
Key Point:

- Some research suggests that pain may have effect on proprioception

- Hypoesthesia/Proprioception loss may have effect on stability during gait/stance activities

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Quantitative sensory testing changes in the successful management of chronic low back pain

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Acute and Chronic Knee Pain

My question:
Why did functional outcomes differ so widely following ACL rupture?

- **Laxity:** Not found to be a factor. Snyder-Mackler 1997
- Heightened hamstring activation as a compensatory mechanism. Boerboom 2001

Unexpected platform perturbations
- Reflexive hamstring activation is augmented following ACL rupture (DiFabio 1992, Courtney 2006)
  - At latencies of 100 ± 20 ms

Anterior Cruciate Ligament Rupture
- Increased (and earlier) hamstring activation... that's a good thing - right?
Knee Osteoarthritis

Gait Pattern:
- Increased hamstring and gastrocnemius activity

Childs et al 2004 Clin Biomech

Could augmented hamstring responses in ACL deficient and knee OA subjects be due to facilitated flexor withdrawal reflexes (FWR)?

FWR (on ipsilateral limb) =
- Inhibition of the extensor muscles
- Excitation of the flexor muscles

Spinally-mediated reflex responses?
- Conditioning of spinal reflexes
  - sensitized with joint inflammation (He et al. 1998)
  - Hyperexcitable FWR = Central Sensitization

Heightened Flexor Withdrawal Responses in Subjects With Knee Osteoarthritis
Carol A. Courtney,* Michael D. Lowek,1 Paul O. Witte,* Samuel J. Chinell,* and T. George Honiby1

- Found FWR excitability in individuals with knee OA vs. healthy age matched control subjects
- However, FWR excitability not necessarily correlated to resting pain
What about in Sport?

In subjects with ACL deficiency

- Question: Could instability and 'giving way' be related to central sensitization?

- Nociceptive Reflex: hyperexcitable on injured limb

In supine (relaxed):
- Applied passive (painfree) tibial translation = significant ↑ in excitability of the reflex

Key Point:

Following a traumatic joint injury:

- Although painfree, central sensitization may be present

- Painfree passive stretch of joint intensified the reflex response

  - Serves to maintain central sensitization?
  - Functional consequences

Courtney 2011
Accelerated Rehabilitation?
Early return to Sport?

Modulation (“treatment”) of central sensitization (increased pain sensitivity)

Cognitive Behavioral Model

Psychological frameworks for managing chronic pain

- dominant current psychological framework and treatment approach to chronic pain
- key concept is that human emotions and behavior are determined largely by how one views the world

Pincus and McCracken 2013
Cognitive Behavioral Model

The model proposes:
- thoughts, beliefs, and behavior patterns are important in understanding adjustment to chronic pain
- thoughts, beliefs, emotions, and behaviors interact with each other and with the situations where they occur
- thoughts, beliefs, and behavior patterns can be targeted for change by specific methods of skills training and learning.

Pincus and McCracken 2013

Other model: Fear Avoidance Model

- Two routes are available when one has an acute painful injury
  1. normal activity re-engagement and recovery
  2. catastrophizing about pain, fear, avoidance, inactivity, possible physical deconditioning, possible depression, persisting pain and becoming stuck in a fear and avoidance cycle

Pincus and McCracken 2013

Fear Avoidance Model Vlaeyen 2000

Key psychological processes

- Depression
  - strongest predictor of long-term disability Pincus 2002
- Fear and avoidance
  - Fearful beliefs about movement, exercise, and activity have been linked to poor outcomes
- Catastrophic cognitions and anxiety
  - characterized by the tendency to magnify the threat value of the pain stimulus and to feel helpless in the context of pain and by a relative inability to inhibit pain-related thoughts
  - related to anxiety, fear, avoidance, and depression
Key psychological processes

- Acceptance
  - Acceptance of pain, flexible present-focused attention, cognitive defusion and committed action, are correlated with reduced distress and disability, with less health-care use, with better general functioning and better work status in groups with chronic pain.

- Pain perceptions and expectations
  - Patients who hold negative expectations for recovery may be less likely to recover.

Treatment

1. Pain Education
2. Cognitive Behavioral Therapy
3. Graded Activity Exposure
4. Graded Motor Imagery
5. Manual Therapy
6. Exercise

Pain Education

- https://www.youtube.com/watch?v=pbqrM8W7D48
- https://www.youtube.com/watch?v=4b8a8757DKc

Cognitive Behavioral Therapy

- Use of a variety of techniques designed to modify the dysfunctional beliefs and faulty information processing characteristic of each disorder.
**Cognitive Distortions**

- Overgeneralization: I hurt myself once, therefore doing the same thing will hurt me every time
- Dichotomous thinking: all-or-nothing
- Maximization: catastrophizing, “awfulizing”
- Discounting the positive: holding on to negative beliefs even if daily experience contradicts them
- Personalization: being a victim or self-blaming

**Mindfulness-Based Therapy**

- Attempts to increase a focused, purposeful awareness of the present moment and relating to one’s experiences in an open, nonjudgmental, and accepting manner
- Awareness of the present moment is thought to increase sensitivity to important features of the environment and one’s own reactions, and thus enhance self-management and successful coping
- Mindfulness-Based Stress Reduction may include sitting meditation, yoga, body scans, and mindfulness during everyday activities

**Attentional control**

**Example:**

- Metacognitive Therapy emphasizes changing attentional processes to alter the relation to thoughts instead of attempting to change thoughts themselves

**Cognitive Disputation**

- Is that really true?
- What evidence do you have for that belief?
- When you think that way, how does it feel?
- Where does thinking that get you?
- In-vivo exposure along with graded exposure
  - exposes you to what elicits your fears
  - helps you realize that terrible things won’t happen
Other considerations

- Technology-assisted delivery
- Pain diary

Motivation and behavioral activation methods (ex: Motivational Interviewing)

- a broad, client-centered, directive clinical method that enhances readiness for change by reducing resistance and ambivalence within the context of a supportive and empathic therapeutic relationship
  
  Hayes 2011

- six components of MI are summarized by the acronym FRAMES: Feedback, an emphasis on personal Responsibility, Advice, a Menu of options, an Empathic counseling style, and support for Self-efficacy

Confrontational therapy (Graded Exposure)

- exposure of patient to the feared stimuli (activity) without any danger
- graded or hierarchical approach
- the most mildly feared activities are targeted first
- Gradual exposure to more intense anxiety provoking stimuli
- therapist and client collaboratively develop an exposure hierarchy in which feared stimuli are ranked accordingly
- http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2737053/figure/ta1/
### Graded Exposure example

<table>
<thead>
<tr>
<th>Fearful activity</th>
<th>Session 1</th>
<th>Session 2</th>
<th>Session 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Walking up steep hill</td>
<td>60/100</td>
<td>20/100</td>
<td>20/100</td>
</tr>
<tr>
<td>Preactivity fear level</td>
<td>60/100</td>
<td>20/100</td>
<td>20/100</td>
</tr>
<tr>
<td>Clinic activity</td>
<td>Walking 5 min, 3% incline, 1.5 mph, 3 mins</td>
<td>Walking 5 min, 3% incline, 1.5 mph, 4 mins</td>
<td>Walking 5 min, 3% incline, 1.5 mph, 4 mins</td>
</tr>
<tr>
<td>Post-activity fear level</td>
<td>10/100</td>
<td>30/100</td>
<td>10/100</td>
</tr>
<tr>
<td>Fear reduced?</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Plan</td>
<td>Increase exposure time</td>
<td>Increase exposure time and distance</td>
<td>Increase exposure time and distance</td>
</tr>
</tbody>
</table>

### Fearful activity 2

<table>
<thead>
<tr>
<th>Standing activity 30 mins to cook meal</th>
<th>Session 1</th>
<th>Session 2</th>
<th>Session 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preactivity fear level</td>
<td>70/100</td>
<td>50/100</td>
<td>30/100</td>
</tr>
<tr>
<td>Clinic activity</td>
<td>Standing assembly activity 5 mins</td>
<td>Standing assembly activity 7 mins</td>
<td>Standing assembly activity 10 mins</td>
</tr>
<tr>
<td>Post-activity fear level</td>
<td>30/100</td>
<td>20/100</td>
<td>40/100</td>
</tr>
<tr>
<td>Fear reduced?</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Plan</td>
<td>Increase exposure time</td>
<td>Increase exposure time</td>
<td>Increase exposure time</td>
</tr>
</tbody>
</table>

### Fearful activity 3

<table>
<thead>
<tr>
<th>Lifting groceries from floor 20#</th>
<th>Session 1</th>
<th>Session 2</th>
<th>Session 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preactivity fear level</td>
<td>80/100</td>
<td>60/100</td>
<td>50/100</td>
</tr>
<tr>
<td>Clinic activity</td>
<td>Knee to table lift 5# 10 reps</td>
<td>Knee to table lift 8# 10 reps</td>
<td>Floor to waist lift 8# 10 reps</td>
</tr>
<tr>
<td>Post-activity fear level</td>
<td>20/100</td>
<td>10/100</td>
<td>30/100</td>
</tr>
<tr>
<td>Fear reduced?</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Plan</td>
<td>Increase exposure weight</td>
<td>Increase exposure distance</td>
<td>Increase exposure weight</td>
</tr>
</tbody>
</table>

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### Laterality Recognition Exercise

- Laterality testing: left/right judgments
- Patient views an image of a limb and judges whether the image is of a right or left limb. Also done with rotation of neck and back
- Ten minutes every waking hour
- fMRI of brains of healthy subjects show this task selectively activates the premotor cortex without activating primary motor areas.
Laterality Recognition Exercise

- Right
- Left
- Left
- Left
- Right
- Right
- Right
- Right
- Right
- Left
- Right

Graded Motor Imagery Stage 2

- Motor imagery: imagined movement of the area
- Using photos, imagine movements, 10 minutes every waking hour
- Imagined movements activate motor cortical areas similar to those activated in actual movement
- Motor imagery should not be used in isolation, as it may increase pain intensity

Graded Motor Imagery Stage 3

- Mirror therapy: patient places affected limb inside a mirror box and watches movements of non-affected limb in the mirror
- 20 positions, 10 reps with both hands, every waking hour
- Gives illusion of a moving, but pain-free, affected limb
- Activates the motor cortex in healthy subjects
- No studies to date showing cortical activation in pain patients

Bowering 2013
Graded Motor Imagery

- GMI has moderate to large effects Moseley 2004, 2008
- Ordering important; unordered GMI may actually increase pain Johnson et al 2012

Progression of Graded Motor Imagery Priganc 2011

<table>
<thead>
<tr>
<th>Step in Program</th>
<th>Start by</th>
<th>Progress by</th>
<th>What if</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Laterality training</td>
<td>Using 12 easy laterality cards. Ask the patient to quickly identify whether the images of a right or left hand. Record the number of correct answers and the time.</td>
<td>Improving the speed, accuracy, number, and difficulty of the images until you feel as though the patient has established laterality</td>
<td>The patient experiences pain when looking at an image? Find images that do not evoke pain and use those images.</td>
</tr>
<tr>
<td>2 Imagined hand movements</td>
<td>Having the patient visualize the affected hand in various positions.</td>
<td>Having the patient visualize actually moving the hand to these various positions</td>
<td>The patient experiences pain? Go back to laterality training. Find images that do not evoke pain.</td>
</tr>
<tr>
<td>3 Motor visual feedback</td>
<td>Having the patient watch the mirrored image of the unaffected hand moving in the mirror.</td>
<td>Having the patient move the affected hand while still in the mirr or box and watching the mirrored image of the unaffected hand</td>
<td>The patient experiences pain? Go back to having the patient watch the movements without performing the movements. Find a movement pattern that does not evoke pain. Go back to imagined hand movements.</td>
</tr>
</tbody>
</table>

Addressing Pain-Related Fear

- Reviewed:
  - Graded in vivo exposure (GivE)
  - Graded activity (GA)
  - Acceptance and commitment therapy (ACT)
  - Mixed cognitive behavioural protocols

GivE and ACT have best results for treating fear-avoidance beliefs in chronic musculoskeletal pain Bailey 2010

Acceptance Commitment Therapy

- Intensive residential Rx of 171 patients with chronic pain Vowles 2008
- ACT model - to promote flexible and effective daily functioning, not to reduce or change pain or other symptoms
- Included mindfulness training, values clarification, exposure-based techniques, and cognitive defusion exercises
- Significant reductions in depression, pain-related anxiety, disability, and healthcare use and significant improvements in physical performance measures at 3 month follow-up
- Effect size post-treatment 1.07 (range: 0.67–1.76)
- Effect size at follow-up 0.89 (range: 0.48–1.51)
Is Manual Therapy Effective in Treating Centrally Mediated Pain?

In a rat model:
- Induced injury at the ankle
- Mobilized rat’s knee (flexion/extension + AP) 3 X 3 minutes  
  Sluka & Wright 2003

Result: ↓ flexor withdrawal response
- = Decreased Central Sensitization

What would be the effect of 2 X 3 minute bouts of joint mobilization on the Flexor Withdrawal Response?

Result: ↓ flexor withdrawal response
- = Decreased Central Sensitization

- Nociceptive reflex = flexor withdrawal reflex
- Threshold to elicit reflex increased (ie, less sensitive) after mobilization
Passive Movement Assessment

- Consider...
  - range of movement
  - onset & quality of resistance
  - onset & behavior of pain
  - relationship between tissue resistance and pain

Old Concept of Grades of Oscillatory Movement

Back to the basics...
New Concept of Grades of Oscillatory Movement

With resistance at 50% of Range

Example: Resistance and Pain

Studies addressing effects of manual therapy

How do you decide whether or not to push into pain?
What else should be considered?

- Patients present at different levels of central sensitization
- Manual therapy treatment effects will differ depending on level of central sensitization

Exercise Induced Analgesia

Manual Therapy Pain Modulation Activity
Animal Studies

- Extended swimming exercise reduces inflammatory and peripheral neuropathic pain in rodents Kuphal 2007
- Treadmill or swimming attenuates neuropathic pain in rats Chen 2012
- Aerobic exercise alters analgesia and neurotrophin-3 synthesis in an animal model of chronic widespread pain Sharma 2010

Human Studies

- Specific cranio-cervical flexion exercises result in a greater increase in PPTs than general cervical flexion exercises O'Leary 2007
- Eccentric exercise reduces PPTs at first session of exercise but not a week later Hosseinzadeh 2013
- 30’ of treadmill exercise reduces PPTs but not thermal pain thresholds (TPTs) in healthy subjects Ruble 2005
- Treadmill exercise reduces distal PPTs in individuals with CLBP for at least 30’ Hoffman 2005

Exercise Induced Analgesia (EIA)

- Sensitivity to noxious stimulus shown to decrease after exercise
- In young healthy adults, exercise of higher intensity (60%–75% V O2max) most consistently produces EIA after aerobic exercise Hoffman 2004, Koltyn 2002
- With isometric contraction, greatest ↓ in sensitivity to noxious stimulus occurs after low-intensity contractions (25%–50% MVC) held for longer duration Hoeger Bement 2008, Naugle 2012

Exercise induced Analgesia (EIA)

- Dynamic resistance exercise (Bench press, leg press, pull downs, arm ext – 45 min)
- No significant changes pre to post exercise (immediate), or 15 minutes post exercise Koltyn and Arbogast 1998, Focht and Koltyn 2009

Conclusion: Dynamic resistance exercise less effective at EIA
Is there something to learn from endurance athletes?

What is somewhat agreed upon...
- Increased mechanical pain thresholds? Johnson 2012, Tesarz 2013
- Increased mechanical & cold pain tolerance Johnson 2012, Tesarz 2013, Geva 2013, Freund 2013
- Lower pain ratings at suprathreshold stimuli Geva 2013
- Enhanced conditioned pain modulation? Geva 2013

Why Can They Take More Pain?

Psychological or Physiological

Psychological
- Associative vs Dissociative Coping Strategies Morgan 1977, Sporting 1993
- “Feelers” vs “Floaters”
- Internal vs External Stevinson 1998
Psychological

- Less fear of pain? Rhudy 2013
- More self-transcendent & more self-centered Freund 2013
- Elite runners have more flexible cognitive strategies Silva 1989
- Associative strategies are associated with greater pain tolerance Johnson 2012

Physiological

- More effective endogenous opioid inhibitory processes? Rhudy 2013

Electrical Stimulation Induced Analgesia

Kathleen Sluka PT, PhD

on Electrical Stimulation:

‘If it’s used for pain modulation, it is called TENS …

no matter what the parameters’

Sluka 2003
How does it work? (central effects)

- High Frequency TENS increases β-endorphins in bloodstream and cerebrospinal fluid
  - Decreases spinal excitability
  - Enhances descending inhibition
  Leonard 2010

- Opioid-mediated analgesia
  Vance 2014

- Manual Therapy and Exercise = non-opioid mediated analgesia
  Skyba 2003, Koltyn 2013

How does it work? (peripheral effects)

- Both High Frequency and Low Frequency TENS have local effect at the site of stimulation

- In animal model, reduces Substance P
  Rokugo 2002

- Substance P:
  - Is increased in tissue of ganglia after tissue injury
  - Promotes central sensitization

Why does it work?

Dosing:
- Stimulation amplitude must be of sufficient strength to produce an analgesic effect
- Repeated use dampens central excitability and enhances descending inhibition

Interpretation:
1. Turn it up
2. Use it often
   Sluka 2013
Why does it not work?

Another US Epidemic:
- Opioid-related abuse and addiction

Opioid Prescriptions Dispensed

# Opioid Prescriptions Dispensed vs Date

Opioid Tolerance
- Opioid medications (eg, Morphine, Vicodin) and Low Frequency TENS produce pain relief by activating same receptor (µ-opioid)
- High Frequency TENS activates different opioid receptor (δ-opioid)
- **Recommendation: Use High Frequency TENS in patients using opioid medications**
  Leonard 2011

Non-pharmaceutical approaches to pain modulation
- Manipulation Induced Analgesia
- Exercise Induced Analgesia
- Noxious Electrical Stimulation Induced Analgesia
- Dry Needling/Acupuncture Induced Analgesia

Exercise and Electrical Stimulation Induced Analgesia Activity
Conclusions

- Typical analgesic methods include pharmacologic agents
- Rehabilitative practice in chronic musculoskeletal conditions serves > purpose than strengthening, ROM and endurance
- Exercise, electrical stimulation and manual therapy have some evidence for analgesic effects, likely occurring at the central level