Acute and Chronic Pain Mechanisms: Implications for Physical Therapy Management

Carol A Courtney PT, PhD

Epidemic

- Prevalence of chronic pain in US adults > 18 yo
  - estimated at 30.7 and 43%  
    Johannes 2010, IOM 2010

- Similar statistics in UK  
  Fayaz 2016

- Social and financial ramifications staggering
  - Disability; reduced quality of life  
    Schafer 2014
  - Increased risk of hospitalization, institutionalization, and mortality  
    Morales-Espinoza 2016
Contributing factors and risk factors to chronic pain:

**Social Factors**
- Tenuous housing status; tenuous employment status  
  Elliott 1999
- Low family income
- Low educational levels  
  Abasolo 2012
- Social isolation and recent divorce, separation, or death of a spouse  
  Hung 2016
- Physical or sexual abuse  
  Spiegel 2016

**Physiological Factors**
- Poor sleep quality  
  Bjurstrom 2016
- Menopause  
  Ancoli-Israel 2004
- Number of comorbidities  
  Coronado 2011
Contributing factors and risk factors to chronic pain: Psychological Factors

- Persons with chronic pain experienced range of psychological impairments:
  - Anxiety, depression, anger/hostility
  - Impaired self-esteem, and general emotional functioning
    Burke 2015 (Br J Clin Psychol)

- Reported that depression and chronic pain may co-occur in up to 80% of individuals suffering from those disorders
  Burke 2015 (Mod Trends Pharm)

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)

Pain Mechanisms Approach to Patient Management

1. Incorporates and builds on the biopsychosocial model
2. Defines:
   1. specific pathobiology in pain processing
   2. pain-relevant psychosocial factors
   3. movement system dysfunction
3. Includes source of pain and contributing factors
4. May have multiple aberrant pain mechanisms occurring simultaneously
IASP Curriculum Outline on Pain for Physical Therapy


Laboratory Activity #1: Mind Mapping Activity

- Use the words on the left to develop a model of a Pain Mechanisms Approach to Patient Management
- Ex: hard work, good job, study at university, happiness

- Psychosocial
- Central
- Nociceptive
- Peripheral
- Motor
- Neuropathic
- Sleep
- Inflammation
- Tissue Insult
- (source)
- (contributes to)
- (affected by)
Questions to Ask Yourself?

- Is the pain more peripherally or centrally mediated? (What %?)
- Is the pain nociceptive or neuropathic?
- Are any of the following contributing to or affected by the pain?
  1. Inflammation
  2. Sleep
  3. Psychosocial
  4. Motor
  5. Tissue Insult
- How will I assess and treat the aberrant pain mechanisms I find?

Mechanisms approach to pain management

- Example: Poor Sleep

Goal:

1. Identify faulty pain mechanism or contributing factor using clinical tools

2. Use specific Physical Therapy interventions to address specific mechanism
What is sensitization?

*Neuroplasticity*

Old view: pain was hard-wired

Stimulus ➔ Response

*The central nervous system can learn…*

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Peripheral Sensitization

Example: Ankle Sprain

Tearing or bruising of joint tissues results in:

- **Release of** ATP & protons
- Mast cells release histamine, 5H-tryptamine, & prostaglandins
- Macrophages release cytokines & growth factors
  - Also bradykinins and COX-2
- Primary afferents release Substance P & CGRP

Inflammatory Soup – term first coined by CJ Woolf

(Costigan and Woolf 2000)

= primary hyperalgesia (increased nociceptive responses)

---

Sandkuhler 2009
Peripheral Sensitization

'stimulus evoked plasticity of the nociceptor' Woolf 2007

Inflammatory mediators bind to receptor - cause:
↓ threshold
↑ excitability
⇒ primary hyperalgesia

From: Woolf 2004

Stimulus Response Curve - Hyperalgesia

Sandkühler J Physiol Rev 2009;89:707-756

Material presented at IPTA 2018 REVITALIZE Conference
Clinical Features of Peripheral Sensitization of Nociceptive Pathways

**Peripheral Sensitization = Primary hyperalgesia**

- Restricted to the site of tissue injury
- Requires ongoing stimulus for maintenance
  - Goes away as tissue heals

- Nociceptive vs. Neuropathic?

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Central Sensitization

- Occurs following repetitive or intense noxious stimulus
  - [Latremoliere and Woolf 2009](#)

- Pain is no longer coupled to the noxious stimulus

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

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Adapted from Costigan 2009

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Material presented at IPTA 2018 REVITALIZE Conference
Clinical Features of Central Sensitization of Nociceptive Pathways

**Secondary hyperalgesia**
- Greater intensity of pain
- Spread of symptoms
  - Regional
  - Widespread
- Clinical Measurement: Pressure Pain Thresholds

**Allodynia**
- Often in region of most pain
- Feels like sunburn

**Secondary Hyperalgesia**
- due to change in processing of sensory input in CNS (i.e., central changes)
Bajaj et al 2001

- increased size of the peripheral receptive field

**Clinical Implication:**
Input from a wider region can induce pain
How? Heterosynaptic Facilitation

Figure 2. Mechanisms underlying allodynia and spreading of pain with central sensitization: heterosynaptic facilitation

Assessment of Pressure Pain Threshold

- Subject in a relaxed, supported position
- Applied perpendicular to tissue
- Constant rate of application
- 30 kPa/cm²/s
- 3 trials
- 30 seconds between trials

Ambite-Quesada 2017
Dynamic Mechanical Allodynia

- Innocuous stimulus
- Cotton wisp
- Standardized brush (Somedic, Sweden)
- Exerting a force of ~200–400mN
- Applied with a single stroke ~2cm over skin
- Elicits pain?

Rolke 2006

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:
  - Day 0: injected with 100 μL sterile saline into gastrocnemius
  - Day 5: same injection
  
  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

Dynamic Measures of Central Sensitization

- Conditioned Pain Modulation
  - Detects impaired descending inhibition

- Temporal Summation
  - Detects hyperexcitability of nociceptive pathways
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

**Impaired Pain Inhibition**

- Found in many chronic pain populations including knee OA
  
  **Arendt-Nielsen 2010, Courtney 2016**

- Key point: manual therapy and exercise can “normalize” impaired pain inhibition mechanisms
Exercise Induced Analgesia as a test of pain modulation

- 3-minute submaximal isometric handgrip exercise at 25% MVC
- Tested PPT and temporal summation pre- and post-exercise
- Increased thresholds indicates pain modulatory systems are functioning

Naugle et al 2014

Laboratory Activity #2: Complete the following

Assess:
1. Allodynia
   1. single stroke ~ 2cm over skin; feeling of sunburn
2. Pressure Pain Threshold
   1. Perception of the moment the sensation of pressure turns to pain
3. Exercise induced Analgesia
   1. Baseline measure of pain: PPT on forearm
   2. 3 min gripping at 25% MVC
   3. Reassess: PPT on forearm
Lab Activity #2: EIA as a test of pain modulation

First, Practice PPT:
- ⊥ to tissue
- Apply at constant rate
- 30 sec between trials

**PPT measures**

Wrist extensors:
- **Ipsilateral**
  - Pre-test: _____/Post-test: _____ = _____%
- **Contralateral**
  - Pre-test: _____/Post-test: _____ = _____%

**Tibialis Anterior**
- Pre-test: _____/Post-test: _____ = _____%

**Conditioned Pain Modulation Procedure**

- Baseline pain measures
  - Numeric Pain Rating
  - Pressure Pain Threshold
- Apply Conditioning Stimulus

**Cold Pressor Test**
- ice bath x 30 sec

**Tourniquet Test**
- Cuff applied (contralateral arm); inflated to 270 mmHg
- Time: 5 min; Pain (VAS): >50mm
- Reassess pain measures:
  - Thresholds should increase
Central Sensitization: Hyperexcitability of nociceptive pathways

Temporal Summation
- Cutaneous
- 26 g. monofilament
- Applied at 1 Hz
- Use metronome
- Apply 30 stimuli
- Every 5 sec, the subject rates pain
- Numeric pain rating scale 0-100
- Geometric mean is calculated \((a*b*c*d*e*f)^{\frac{1}{6}}\) or \((f - a)\)

Laboratory Activity #3

Conditioned Pain Modulation and Temporal Summation Activity
Laboratory #3 Worksheet

Conditioned Pain Modulation

- PPT Site  Pre_______ Post_______
  % ___

- PPT Mirror  Pre_______ Post_______
  % ___

- PPT distal  Pre_______ Post_______
  % ___

Interlude – dandelions
Nociceptive vs Neuropathic Pain

Neuropathic Pain:

*defined as injury or disease of a nerve or neural structure*  
IASP

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Contributing Factors</th>
<th>Individual Pathophysiology</th>
<th>Neuropathic Syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic Disorders</td>
<td></td>
<td>Sensory Nerve Damage</td>
<td>diabetic neuropathy</td>
</tr>
<tr>
<td>Injuries</td>
<td></td>
<td>Ectopic Activity</td>
<td>chronic diabetic neuropathy</td>
</tr>
<tr>
<td>Infections</td>
<td></td>
<td>Sensitization</td>
<td>hereditary neuropathy</td>
</tr>
<tr>
<td>Neurotropic</td>
<td></td>
<td>Peripheral &amp; Central</td>
<td>post-traumatic neuropathy</td>
</tr>
<tr>
<td>Neurotoxic</td>
<td></td>
<td>Altered Brain Connectivity</td>
<td>post-herpetic neuropathy</td>
</tr>
</tbody>
</table>

Material presented at IPTA 2018 REVITALIZE Conference
Algorithm for Diagnosing Neuropathic Pain

Finnerup 2016
Thermal changes with neuropathic pain

Mechanical Detection Threshold
Vibration Detection Threshold

Two – Point Discrimination
Receptive Fields

Note: lateral inhibition

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Neuropathic pain phenotyping by international consensus (NeuroPSIG) for genetic studies: a NeuPSIG systematic review, Delphi survey, and expert panel recommendations

Material presented at IPTA 2018 REVITALIZE Conference
### Signs

Van Hecke 2015

Delphi survey: the number of participants who ranked a clinical sign in their top 5 signs for diagnosing neuropathic pain based on the balance between validity and feasibility of measurement.

<table>
<thead>
<tr>
<th>Clinical sign*</th>
<th>Number of times a clinical sign was listed in the top 5 (%) participants</th>
<th>Median rank (QRs) in round 3</th>
<th>Good consensus on clinical sign inclusion in the top 5 (yes: ≥70%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dynamic mechanical allodynia</td>
<td>12 (81)</td>
<td>1 (1-2)</td>
<td>Yes</td>
</tr>
<tr>
<td>Abnormal sensation to punctate mechanical stimuli</td>
<td>6 (41)</td>
<td>2 (1-4.29)</td>
<td>--</td>
</tr>
<tr>
<td>Hypoesthesia to punctate mechanical stimuli</td>
<td>8 (50)</td>
<td>3 (2-4)</td>
<td>--</td>
</tr>
<tr>
<td>Punctate mechanical hyperalgesia</td>
<td>7 (44)</td>
<td>2 (2-4)</td>
<td>--</td>
</tr>
<tr>
<td>Cold allodynia</td>
<td>7 (44)</td>
<td>3 (2-4)</td>
<td>--</td>
</tr>
<tr>
<td>Thermal hypoesthesia</td>
<td>5 (31)</td>
<td>2.5 (2-4.5)</td>
<td>--</td>
</tr>
<tr>
<td>Temporal summation</td>
<td>6 (38)</td>
<td>2 (1-3.25)</td>
<td>--</td>
</tr>
<tr>
<td>Abnormal vibration sense</td>
<td>10 (61)</td>
<td>3 (2-4.29)</td>
<td>--</td>
</tr>
<tr>
<td>Thermal hyperalgesia</td>
<td>1 (4)</td>
<td>3 (2-3.5)</td>
<td>--</td>
</tr>
<tr>
<td>Allodynia reflexes</td>
<td>2 (12)</td>
<td>3 (2-4)</td>
<td>--</td>
</tr>
<tr>
<td>Static mechanical hyperalgesia</td>
<td>5 (31)</td>
<td>3 (2.5-4)</td>
<td>--</td>
</tr>
<tr>
<td>Deep mechanical hyperalgesia</td>
<td>1 (6)</td>
<td>4 (4-4)</td>
<td>--</td>
</tr>
</tbody>
</table>

* Clinical signs identified by 20 participants in round 1 in an open question to list 4 clinical signs that provide the best balance between validity and feasibility when diagnosing neuropathic pain.

** Notes:**
- Signs listed according to the number of times they were listed in participants’ top 5 in round 3, and then by the median rank received by listed signs in round 3 (1: highest ranked, 5: lowest rank).
- “Yes” indicates that no consensus reached at the ≥70% threshold level after 2 rounds.

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### Screening Tools: LANSS  Bennett 2001

Leeds Assessment of Neuropathic Symptoms & Signs (LANSS)

- 5 “Yes” or “No” questions (16 pts max)
- Allodynia & Pin-Prick Threshold (8 pts max)
- Scored /24
- ≥ 12 = neuropathic component **likely**
1. Does your pain feel like strange unpleasant sensations in your skin? **(Dysesthesia)**
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? **(Alldynia)**
4. Does your pain come on suddenly for no apparent reason when you’re still? **(Spontaneous Pain)**
5. Does your pain feel as if the skin temperature in the painful area has changed abnormally? **(Thermal changes)**

1. Allodynia
2. Altered Pin-Prick Threshold  

---

**painDETECT**

- Developed for radiculopathy with low back pain Freynhagen 2006
- Sensitivity = 85% Specificity = 80%
- Classifies patients as:
  - “nociceptive”
  - “unclear”
  - “possible neuropathic”
# PAIN QUESTIONNAIRE

**Date:**

<table>
<thead>
<tr>
<th>Date</th>
<th>Patient</th>
<th>Last name</th>
<th>First name</th>
</tr>
</thead>
</table>

**How would you assess your pain now, at this moment?**

1. 0
2. 1
3. 2
4. 3
5. 4
6. 5
7. 6
8. 7
9. 8
10. 9

---

**How strong was the strongest pain during the past 4 weeks?**

1. 0
2. 1
3. 2
4. 3
5. 4
6. 5
7. 6
8. 7
9. 8
10. 9

---

**How strong was the pain during the past 4 weeks on average?**

1. 0
2. 1
3. 2
4. 3
5. 4
6. 5
7. 6
8. 7
9. 8
10. 9

---

**Mark the picture that best describes the course of your pain:**

- Persistent pain with slight fluctuations
- Persistent pain with pain attacks
- Pain attacks without pain between them
- Pain attacks with pain between them

---

**Does your pain radiate to other regions of your body?**

Yes No

---

**Do you suffer from a burning sensation (e.g., stinging nettles) in the marked areas?**

- Never
- Slightly
- Moderately
- Strongly
- Very strongly

---

**Do you have a tingling or prickling sensation in the area of your pain (like crawling ants or electrical tingling)?**

- Never
- Slightly
- Moderately
- Strongly
- Very strongly

---

**Is light touching (clothing, a blanket) in this area painful?**

- Never
- Slightly
- Moderately
- Strongly
- Very strongly

---

**Do you have sudden pain attacks in the area of your pain, like electric shocks?**

- Never
- Slightly
- Moderately
- Strongly
- Very strongly

---

**Is cold or heat (bath water) in this area occasionally painful?**

- Never
- Slightly
- Moderately
- Strongly
- Very strongly

---

**Do you suffer from a sensation of numbness in the areas that you marked?**

- Never
- Slightly
- Moderately
- Strongly
- Very strongly

---

**Does slight pressure in this area, e.g., with a finger, trigger pain?**

- Never
- Slightly
- Moderately
- Strongly
- Very strongly

---

### Scoring

- **Total score** out of 35

---

Material presented at APTA 2018 REVITALIZE Conference
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: 0
- Persistent pain with pain attacks: -1 if marked, or
- Pain attacks without pain between them: +1 if marked, or
- Pain attacks with pain between them: +1 if marked
- Radiating pains?: +2 if yes

Final score

---

Screening Result

**Final score**

<table>
<thead>
<tr>
<th>Nociceptive</th>
<th>Unclear</th>
<th>Neuropathic</th>
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</thead>
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<td>13</td>
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<tr>
<td>1</td>
<td>13</td>
<td>14</td>
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<tr>
<td>25</td>
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<td>38</td>
</tr>
</tbody>
</table>

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%)
Neuropathic Pain

- Axon Sheath degeneration
- Causes expression of eg, Sodium channels and TRPV1 receptors

Medical Management Neuropathic Pain: A Pain Mechanisms Approach

<table>
<thead>
<tr>
<th>SYMPTOM</th>
<th>NEURONAL PROCESSES, MECHANISMS</th>
<th>TARGETS</th>
<th>OPTIMAL COMPOUNDS</th>
<th>AVAILABLE COMPOUNDS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous pain</td>
<td>Hyperexcitability</td>
<td>Sodium</td>
<td>Selective sodium-channel blocker</td>
<td>Lidocaine, carbamazepine, baclofen,</td>
</tr>
<tr>
<td>(shocklike)</td>
<td></td>
<td>channels</td>
<td></td>
<td>tramadol</td>
</tr>
<tr>
<td>Spontaneous pain</td>
<td>Sensitization</td>
<td>Cytokines</td>
<td></td>
<td>Neurokinin blockers</td>
</tr>
<tr>
<td>(tingling)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heat allodynia</td>
<td></td>
<td>TRPV1</td>
<td>TRPV1 receptor antagonists</td>
<td>Capsaicin cream</td>
</tr>
<tr>
<td>Cold allodynia</td>
<td></td>
<td>TRPMB</td>
<td>TRPMB receptor antagonists</td>
<td>Menthol</td>
</tr>
</tbody>
</table>

Material presented at IPTA 2018 REVITALIZE Conference
Break (15 minutes)
Please feel free to try out the tools for:
• Thermal Assessment
• Mechanical Detection Threshold
• Vibration Perception

Physical Therapy Management
Sleep Dysfunction

Prescription opioid status and dose were associated with impairment in self-reported sleep. Morasco 2014

Management

1. Stick to a sleep schedule of the same bedtime and wake up time, even on the weekends. This helps to regulate your body’s clock and could help you fall asleep and stay asleep for the night.
2. Practice a relaxing bedtime ritual. A relaxing, routine activity right before bedtime conducted away from bright lights helps separate your sleep time from activities that can cause excitement, stress or anxiety which can make it more difficult to fall asleep, get sound and deep sleep or remain asleep.
3. If you have trouble sleeping, avoid naps, especially in the afternoon. Power napping may help you get through the day, but if you find that you can’t fall asleep at bedtime, eliminating even short catnaps may help.
4. Exercise daily. Vigorous exercise is best, but even light exercise is better than no activity. Exercise at any time of day, but not at the expense of your sleep.
5. Evaluate your room. Design your sleep environment to establish the conditions you need for sleep. Your bedroom should be cool – between 60 and 67 degrees. Your bedroom should also be free from any noise that can disturb your sleep. Finally, your bedroom should be free from any light. Check your room for noises or other distractions. This includes a bed partner’s sleep disruptions such as snoring. Consider using blackout curtains, eye shades, ear plugs, “white noise” machines, humidifiers, fans and other devices.
6. Sleep on a comfortable mattress and pillows.

Sleepfoundation.org
Manual Therapy

Manual Therapy and Exercise: Mechanisms approach to pain management

Goal:
1. Identify faulty pain mechanism or contributing factor using clinical tools

2. Use specific Physical Therapy interventions to address specific mechanism
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

Heightened Flexor Withdrawal Response in Individuals With Knee Osteoarthritis Is Modulated by Joint Compression and Joint Mobilization

Carol A. Courtney, Paul O. Witte, Samuel J. Chmell, and T. George Hornby

• 2 X 3 minutes joint mobilization
• AP oscillatory technique
• Pain-free

Result: ↓ flexor withdrawal response
• = Decreased Central Sensitization
• Nociceptive reflex = flexor withdrawal reflex

• Threshold to elicit reflex increased (i.e., less sensitive) after mobilization
• Considerations:
  ▪ Immediate effects
  ▪ How does this fit into overall management?

PASSIVE JOINT EXAMINATION

Back to the basics...
With resistance at 50% of Range

Example: Resistance and Pain

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

- Patients present at different levels of central sensitization
  - Subjective report of irritability
  - Neuropathic versus nociceptive
  - Regional versus widespread hyperalgesia
  - Temporal summation
  - Conditioned pain modulation

- Manual therapy treatment effects will differ depending on level of central sensitization

---

Noxious versus Non-noxious Manual Therapy

<table>
<thead>
<tr>
<th>NOXIOUS</th>
<th>NON-NOXIOUS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry needling</td>
<td>Oscillatory manual therapy</td>
</tr>
<tr>
<td>Soft tissue techniques</td>
<td>Mobilization with Movement</td>
</tr>
<tr>
<td>Oscillatory manual therapy</td>
<td>Thrust manipulation</td>
</tr>
<tr>
<td>Electrical Stimulation</td>
<td></td>
</tr>
</tbody>
</table>

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Material presented at IPTA 2018 REVITALIZE Conference
Wrong choice of manual technique = temporal summation
Laboratory Activity #5 Manual Therapy to Modulate Pain

- PPT Site  Pre_______ Post_____ % ___
- PPT Mirror  Pre _____ Post_____ % ___
- PPT distal  Pre _____ Post_____ % ___

Manual Techniques (nox. vs non-nox.)
1. Rx Upper trapezius Trigger Point
2. Central PA at C2 (Grade IIII)
3. Thrust Technique T5-6
4. Nerve glides Median Nerve
5. AP at tibiofemoral joint (Grade IV)

Exercise to Manage Pain
Inoculation for chronic pain?

- Regular physical activity prevents development of chronic muscle pain and exercise-induced muscle pain
  - Lima 2017
- How?
- Reducing phosphorylation of NR1 subunit of NMDA receptor in CNS (a component of central sensitization)
- Regular physical activity = no effect on development of acute pain
- But prevents development of central sensitization
- **Physical inactivity is a risk factor for development of chronic pain**

Exercise induced Analgesia: peripheral effects

- Muscle contraction disperses inflammation
- Exercise restores joint normal movement
  - removes mechanical driver of pain

Also
- Increases expression of endogenous analgesic substances in exercising muscle
  - Lima 2017
Exercise induced Analgesia: central effects

**Opioid Mechanisms**
- At central level, β-endorphin release activates descending inhibitory pathways  
  
  Stagg 2011
- Positive effects of exercise on mental health
  - Mood elevation
  - Reduction of stress and depression  
  
  Janal 1984

**Non-Opioid Mechanisms**
- Serotonergic Inhibition  
  
  Bobinski 2015
- Exercise increases serum concentrations of endocannabinoids  
  
  Dietrich & McDaniel 2004

Material presented at IPTA 2018 REVITALIZE Conference

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**Resistance training**

- In healthy controls  
  
  Koltyn and Arbogast 1998
- In knee Osteoarthritis  
  
  Burrows 2014

Material presented at IPTA 2018 REVITALIZE Conference
Aerobic Exercise: Dose Response

- Recommend intensity >50% VO₂max and duration >10 min to elicit exercise analgesia
  
  Hoffman 2004

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**ISOMETRIC CONTRACTION: DOSE RESPONSE**

**low level contraction** (elbow flexion) for long duration = greatest decrease in pain

Hoeger-Bement 2008
Isometric protocols

<table>
<thead>
<tr>
<th>QUADRICEPS MUSCLE</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Exhaustion (~12 min)</td>
<td>1 kg on ankle</td>
<td>(healthy controls) Lundberg &amp; Kosek 2003</td>
</tr>
<tr>
<td>Exhaustion (~10 min)</td>
<td>39 N; ~ 10% MVC</td>
<td>(healthy controls) Katetoff &amp; Kosek 2007</td>
</tr>
<tr>
<td>5 min</td>
<td>20-25% MVC</td>
<td>(healthy controls) Lannersten &amp; Kosek 2010</td>
</tr>
<tr>
<td>4 X 45 s</td>
<td>70% MVIC</td>
<td>(patellar tendinopathy) Rio 2015</td>
</tr>
</tbody>
</table>

“hypoalgesic effect larger for contractions at a **low to moderate intensity** held for **longer durations**.” — Naugle 2012

Laboratory Activity #6 Exercise to Modulate Pain

- PPT Site  Pre_______ Post_______ % ___
- PPT Mirror  Pre ____Post_______ % ___
- PPT distal  Pre ______Post_______ % ___

**Isometric:**
- 25% contraction to exhaustion
- Quadriceps with 1 Kg weight
- Deep neck flexor training
- Muscle energy technique (SIJ)
- Mobilization with movement

**Resistance exercise**
- Push-ups

**Aerobic exercise**
- ≥ 50% VO2 max for ≥ 10 minutes
Conclusions

• Typical analgesic methods include pharmacologic agents

• Rehabilitative practice serves greater purpose than simply strengthening, ROM and endurance

• Exercise, electrical stimulation and manual therapy have some evidence for analgesic effects, likely occurring at the central level