Clinical Pattern Recognition: Central sensitization in complex upper quarter conditions

California Chapter Annual Conference
San Diego, CA

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Special Thanks:
• Dr. Ina Diener, PT, PhD President South African Society of Physiotherapy
• Dr. Adriaan Louw, PT, PhD, CSMT

Session Learning Objectives
1. Review of literature related to central sensitization in common upper quarter conditions
2. Mechanisms underlying central sensitization
3. Clinical pattern recognition of central sensitization
4. Pain modulation for central sensitization
Review of literature related to central sensitization in common upper quarter conditions

Michael Wong, PT, DPT, OCS, FAAOMPT
Associate Professor

Clinical Examination:
Proving the local tissue:
Palpation
Resisted extension
Gripping

A more comprehensive physical examination may be necessary to identify (or rule out) coexisting pathologies or other reasons for their pain.

Severity of Pain and Disability - Lateral epicondylalgia
- Greater baseline pain and disability
- Poorer long-term prognosis
- More pronounced sensory disturbances

Interventions:
- Pharmacological therapies
- Rest and splint
- Counterforce strap
- Diamond taping

Concomitant neck or shoulder pain
- Neck pain is more common in patients with LET than age matched healthy controls
- Physical impairments at C4-C7 spinal levels
- Self-report of shoulder and neck pain in patients with LET indicative of poorer short- or long-term prognosis
Central Sensitization

- Heightened nociceptive withdrawal reflex
- Wide spread mechanical hyperalgesia
- A subgroup of patients: reporting severe levels of pain/disability
  - Cold hyperalgesia
  - Increased risk of persistent pain

Similar associated impairments in Carpal Tunnel Syndrome

- (+) correlation between function, pain intensity, depression and duration of symptoms
- (-) correlation between function and:
  - Pressure pain threshold over CS-6, Carpal tunnel, tibialis anterior muscle

These problems are clearly multifactorial!

Taking a step back....

- Of the common upper quarter conditions?
- Duration > 3 months?
- Have you seen many patients with widespread pain?
- Sensitivity to cold?
- Decreased pain free grip strength?
- Complaints of neck and shoulder pain?
- High levels of pain and disability leading to poor prognosis and persistent pain?
- Involved limb weakness?
- Altered motor control?
- Scapular weakness?

What conditions demonstrate Central sensitization?

- An increased responsiveness of nociceptive neurons in the central nervous system to normal or subthreshold afferent input leading to hyperalgesia
- Lateral epicondylalgia (Fernández-Carnero 2009)
- Carpal tunnel syndrome (Fernández-de-las-Peñas 2009)
- Thumb osteoarthritis (CMC OA) (Chiarotto 2013)
- Shoulder impingement (Gwilym 2011)
- Whiplash associated disorders (WAD) (Sterling 2008)
- Headache (Palacios Cena 2016)
- Low-back pain (Sanzarello 2016)
- Osteoarthritis (knee) (Courtney 2009)
- Patellofemoral joint pain (Pazzianato 2016, Lantz 2016)

Central sensitization → Contralateral sensitized structures

- Lateral epicondylalgia (Fernández-Carnero 2009)
- Carpal tunnel syndrome (Fernández-de-las-Peñas 2009)
- Carpometacarpal osteoarthritis (Farrell 2000)
The mechanisms and science underlying central sensitization and their implications for clinical practice

Derrick Sueki, PT, PhD, DPT, GCPT
Assistant Professor
Board Certified Specialist in Orthopaedic Physical Therapy
Fellow, American Academy of Orthopaedic Manual Physical Therapy

Goals of the module
- Physiology of Central Sensitization
- The role of the brain and memory
- Impact on upper extremity pathology
- How to apply the information in clinic

What is Sensitization?
- **Sensitization** – the process in which repeated administration of a stimulus results in the amplification or enhancement of a given response.

Central (cortical)
Peripheral
Central (spinal cord)

Pain as a set hardwired system
- We use to believe that injury elicited a set hardwired response.
- Ever time we were injured, a similar electrophysiological response was triggered.
- Responses were seen as stable and fixed. Suggesting that a given stimulus would always result in a predictable response.

Responses are not fixed and stable.
- Since the 1960’s, we have begun to see that nociceptive responses are not stable.
- They can be modulated
  - Increased – sensitization
  - Decreased – habituation

The many causes of sensitization

**Peripheral**
- Receptor
- Peripheral Nerve

**Spinal Cord**
- Substantia Gel
- Glial Cells
- Neurotransmitters

**Brain**
- Activity
- Hypertrophy
- Plasticity
What type of sensitization are we looking at?

- In the studies presented, there are likely multiple mechanisms at play, but we are primarily looking at two types:
  - 1. Wind up
  - 2. Long Term Potentiation

What is wind up?

- In 1965, Lorne Mendel working Patrick Wall's lab found that small subthreshold stimulus, if given in quick repetition could activate C-fibers.
- We later determined that C-fibers have a very synaptic potential (20 seconds) compared to A-beta fibers (3 milliseconds)
- Therefore, small charges would add up and build on each other causing nociceptive responses
- This is also called temporal summation
- In studies, it is generally found that stimulus need to be delivered at frequencies less than 3 seconds to elicit a nociceptive response

What is long term potentiation?

- Long term potentiation is different then wind up
- It is uses a supra-threshold stimulus generated at lower frequencies to activate nociceptive fibers
- But once activated, these neural interactions become facilitated
- So that it is easier for subsequent stimulus to trigger the pathway
- LTP is the means by which we learn and generate memories

What do wind up and long term potentiation have in common?

- They are both forms of synaptic and neuroplasticity
- NMDA receptors dependent responses
- Both can be influenced or modulated by local and supraspinal mechanisms
- These mechanisms can include:
  - Chemical
  - Electrophysiological

Top Down vs Bottom Up Modulation

- Top Down
  - Descending Pain Modulation
  - Cortically Driven
- Bottom Up
  - Locally Driven
  - Stimulus Driven
  - Nociceptive Driven

What does all this mean

- Cortical and local mechanisms are capable of altering the amount of nociceptive information received in the brain
- Central sensitization is an ingredient in the pain experience, but it is not pain.
- This is an important concept because many equate central sensitization to chronic pain. They are not the same.
- Other things need to happen in order for central sensitization to become pain.
Sensitization does not equal pain

- The experience of pain involves the inputting of information to the brain for processing.
- Once processed, a number of behavioral responses are enacted.
- One of which is pain.

Processing leads to pain

- The experience of pain involves the inputting of information to the brain for processing.
- Once processed, a number of behavioral responses are enacted.
- One of which is pain.

It’s about what happens in the brain . . .

- The last three decades has seen an amazing amount of research involving the brain and pain.
- While the ingredients such as sensitization are incredibly important aspects of pain, it is the brain that puts the information together to form pain.

Pain is in the Brain

- And along with these studies and research have come a significant number of generalizations and platitudes about the role of the brain in the formation of pain.
Why is the brain important and what makes humans different?

<table>
<thead>
<tr>
<th>Animal</th>
<th>Brain Wt.</th>
<th>Brain Neurons</th>
<th>Cortex Neurons</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cat</td>
<td>25.6 g</td>
<td>760 million</td>
<td>20 million</td>
</tr>
<tr>
<td>Chimp</td>
<td>440 g</td>
<td>6.2 billion</td>
<td>40 billion</td>
</tr>
<tr>
<td>Human</td>
<td>1320 g</td>
<td>86 billion</td>
<td>65 billion</td>
</tr>
<tr>
<td>Animal</td>
<td>1320 g</td>
<td>86 billion</td>
<td>65 billion</td>
</tr>
<tr>
<td>Elephant</td>
<td>5712 g</td>
<td>257 billion</td>
<td>5.6 billion</td>
</tr>
</tbody>
</table>

Humans have the greatest percentage of neurons in the cerebral cortex.

How are human brains different?

- Greater amount in the prefrontal cortex
- Allow integration of context and emotion
- Has several unique structures including anterior cingulate cortex and the anterior aspect of the insular cortex

What areas of the brain are affected with persistent pain?

- Medial Prefrontal Cortex (mPFC)
- Anterior Cingulate Cortex (ACC)
- Insular Cortex (IC)
- Amygdala
- Hippocampus

What are the roles of these areas?

- Amygdala and Hippocampus
  - Fear and Fear Memory
- Medial Prefrontal Cortex, Anterior Cingulate Cortex, and Insular Cortex
  - Memory and Emotions

How do these protective responses function during injury? – An Integrative System of Protection

- Loops of activity
  - Immediate Loop – Quick Responses
    - Brain Stem
  - Secondary Loop – Access Fear Memories
    - Amygdala and Hippocampus
  - Tertiary Loop – Access Long Term Contextual Memory
    - ACC, mPFC, IC

Protection. Survival.
How are these areas altered with persistent pain?

- **Activity Level**
  - Increased amount of activity
  - Increased duration of activity

- **Structural Changes**
  - Atrophy of four out of five
  - Only structure that hypertrophies is the amygdala

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So... When we talk about pain in the brain we are really talking about pain is about **memory**...
We are not injured in a vacuum...

**Sights**
The things we see during an injury can trigger pain

**Sounds**
The things we hear during an injury can also trigger pain

**Smells**
Smells can be the most powerful trigger of pain memory

**Emotions**
The emotional status of the patient during injury can trigger memories of injury

**Sensations**
Touch and temperature can trigger pain memories

**Proprioception**
Proprioceptive information received during injury can trigger pain

**Interoception**
Internal markers such as blood pressure or hormone levels can be associated with injury

Back to the brain

• Now that we have this background, we can begin to see the significance of the areas impacted by pain and those altered in people with persistent pain

The role of context, contextual learning, and contextual cueing cannot be overstated. They are critical for associative learning and key to top down nociceptive sensitization.

Models of Fear and Pain

• **Fear Avoidance Model**
  - Fear of Pain
  - Fear of Movement

Key Concept – Fear has a potentially significant role in persistent pain and in central sensitization.

Effects of Negative Emotion on PPT

• Negative emotions, thoughts, and stimulus have been shown to decrease pressure pain thresholds.
• This suggests that negative stimulus can make an individual more sensitive to nociceptive stimulus.
• On the positive side, positive images and stimulus can make pain more difficult to elicit
  - (Rhudy 2002)

Effects of Anxiety on PPT

• Anxiety decreases pressure pain thresholds making it more easy for pain to be elicited
  - (Cornwall 1988, Vedolin 2009)
• Functionally, anxiety preps the body for quick behavioral responses including pain.
• Anxiety is highly dependent upon memory and past experience
Effects of Fear on PPT

- The effect of fear on pain thresholds has been less studied.
- Preliminary research suggests that fear responses are different than anxiety responses
  - (Rhudy 2000)
- Anxiety decreases pain thresholds making it easier to elicit pain, whereas fear increases pain thresholds and makes it harder to elicit pain.
- This has the functional purpose of allowing an organism to escape threat without being hampered by pain.

Elbow, Wrist, and Hand Rehabilitation Considerations

- There are very few studies that specifically address the issue of fear in pain rehabilitation.
- The majority of research addresses fear avoidance models.
- There is a model of fear rehabilitation that has significant literature behind it.
  - Post Traumatic Stress Disorder (PTSD)

Is Chronic Pain a form of PTSD?

Chronic Pain & Post Traumatic Stress

The link between PTSD and Persistent Pain

Similarities between PTSD and persistent pain

<table>
<thead>
<tr>
<th></th>
<th>PTSD</th>
<th>Chronic Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>The Shared Neuroanatomy and Neurobiology of Comorbid Chronic Pain and PTSD: Therapeutic Implications</td>
<td>Post Traumatic Stress Disorder (PTSD)</td>
<td>Chronic Pain</td>
</tr>
<tr>
<td>Brain activity returns to normal in PTSD</td>
<td>Chronic Pain</td>
<td>Post Traumatic Stress Disorder (PTSD)</td>
</tr>
<tr>
<td>Atrophied regions of the brain can regenerate</td>
<td>Chronic Pain</td>
<td>Post Traumatic Stress Disorder (PTSD)</td>
</tr>
<tr>
<td>Fear and pain memories are not changed or erased, they are overwritten by new memory pathways</td>
<td>Post Traumatic Stress Disorder (PTSD)</td>
<td>Chronic Pain</td>
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</table>

Using PTSD as a model of rehabilitation for persistent pain

- Physical evidence: The role of associative learning and fear in the development of chronic pain – a comparison of chronic pain and post-traumatic stress disorder
  - (Suarez, K. D. et al., 2004; Pinnegar, M. J., 2000; Spinella, M. S., 2000)

Reshaping pain requires reshaping memories

- Fear and Pain Extinction – this process occurs when a conditioned stimulus (contextual cue) no longer triggers an associated response (fear or pain).
  - Brain activity returns to normal in PTSD
  - Brain activity returns to normal in persistent pain
  - Atrophied regions of the brain can regenerate
  - We believe that extinction uses different pathways.
  - We also believe that fear and pain memories are not changed or erased, they are overwritten by new memory pathways.
Breaking the pain and sensitization cycle

- Breaking the pain cycle involves changing pain and fear memories
- Changing fear processing can alter top down regulation of nociceptors
- Decreasing central sensitization and decreasing nociceptive input

New memories over ride old memories

The 30 Day Window - Early Intervention

- Fear is processed initially in the amygdala and then routed to the hippocampus for short term storage
- PTSD literature suggests that after 30 days these memories are shifted to the longer term memory regions of the brain (ACC, mPFC, and IC) for more permanent storage.
- In PTSD and in pain, this suggest that there is a 30 day window of opportunity to reduce fear and prevent pain from becoming encoded in long term memory

Don’t throw out the tool box, repurpose the tools . . .

- Many of the common interventions and techniques used to treat patients are appropriate in this new model
- What changes is the intent. The purpose is to reduce fears and break associations made with these fear.
- The goal being to reshape and re-encode new positive memory engrams to overlay the old script that are contributing to the persistent upper extremity pain.

What can we learn from the research from other regions?

Clinical descriptors for the recognition of central sensitization pain in patients with knee osteoarthritis

Subjective exam:

- Self report of moderate to severe levels of pain >5/10 may be a first indicator (Not to be used in isolation)
- Inconsistencies between degree of structural damage and pain/disability
Pain distribution:

- Widespread, non-anatomical distribution of pain - strong indicator of Central sensitization
- Aggravation and expansion of existing symptoms to sites around and remote from the source region - indicator of central sensitization

Can you think of patients with elbow/wrist/hand pain that has spread beyond the initial tissue injury?

Increased sensitivity to physical activity

- Disproportionate self-reported increase in pain after physical activity tests or activity-based interventions

Unclear, disproportionate, non-mechanical

- Aggravating and easing factors don’t make sense
- They are disproportionate
- They do not demonstrate a mechanical nature
- They are unpredictable

Presence of neuropathic-like or centrally mediated symptoms

- Sleep disturbances
- Memory changes
- General fatigue
- (Frequently seen in central sensitization)

Use of validated questionnaires

- PainDETECT
- Central Sensitization Inventory

<table>
<thead>
<tr>
<th>CENTRAL SENSITIZATION INVENTORY: PART A</th>
</tr>
</thead>
<tbody>
<tr>
<td>Name:</td>
</tr>
<tr>
<td>Please circle the best response to the right of each statement.</td>
</tr>
<tr>
<td>1 I feel tired and overwhelmed when I wake first thing in the morning.</td>
</tr>
<tr>
<td>2 I find it hard to keep my eyes open.</td>
</tr>
<tr>
<td>3 I feel anxious attacks.</td>
</tr>
<tr>
<td>4 I have pain or stiffness in the hands.</td>
</tr>
<tr>
<td>5 I have problems with dizziness or unsteadiness.</td>
</tr>
<tr>
<td>6 I feel pain while performing my daily activities.</td>
</tr>
<tr>
<td>7 I feel sensitive to bright lights.</td>
</tr>
<tr>
<td>8 I feel tired even when I am physically active.</td>
</tr>
<tr>
<td>9 I feel pain all over my body.</td>
</tr>
<tr>
<td>10 I feel helpless.</td>
</tr>
<tr>
<td>11 I feel dependent on my medication or to help me function.</td>
</tr>
</tbody>
</table>
Psychosocial factors

- Clinical practice guidelines: Low back pain
  - Pain with cognitive and affective tendencies
  - Generalized pain

Relationship between psychosocial factors and central sensitization

- Pain hypervigilance
- Pessimism
- Catastrophizing
- Poor coping strategies

Cognitive and affective tendencies

Responsiveness to previous treatment

- Inconsistent
- Unpredictable
- Unsuccessful response to nociception-targeted treatments
  - Or
  - Strong exacerbation of symptoms severity post-treatment

Less responsiveness to analgesic or non-steroidal anti-inflammatory pain medications

Persistent post-surgical pain

- 20% of patients have persistent pain after knee replacement
- Linked to central sensitization
Physical exam:
• Widespread mechanical hyperalgesia

Can I use manual pressure instead of algometer?

Ice pain test
• Moderate to good correlations between ice pain test and cold pain thresholds

Clinical pearl:
• Apply ice to local tissue pain region
  • 5 seconds
  • Pain >5/10
  • 90% likelihood of cold hyperalgesia
Quantitative Sensory Testing: Pressure Pain Threshold

Quantitative Sensory Testing: Allodynia

Modulating pain
Effectiveness of Pain Neurophysiology Education

- Studied in many populations:
  - In chronic whiplash associated disorders
    - Improvements in illness perceptions, pain thresholds and movement perception (Van Oosterwijck et al., 2011)
  - Chronic low back pain (Moseley, 2002, 2003b, 2004, 2005; Moseley et al., 2004; Ryan et al., 2010)
    - Alters pain perceptions
    - In conjunction with physiotherapy, improves functional and symptomatic outcomes

Chronic fatigue syndrome

- Pain neurophysiology education
- Alters pain perceptions such as catastrophizing and pain behavior

Is a booklet on pain education enough?

- Simply providing a detailed pain education booklet
- Did Not change illness perceptions or health status in patients with fibromyalgia
- BUT
  - Same booklet combined with two educational sessions (1 face to face, 1 by telephone)
  - Individually tailored pain physiology education
  - Vitality, physical functioning, mental and general health all improved

Patient’s need us to walk side by side with them in this pain journey...

Clinical Guidelines for Pain Neurophysiology Education...
Are maladaptive pain cognitions present?

Assessment of illness perceptions:
- What do you think are the causes of your pain?
- What have been the consequences?
- What has been done to manage the problem? How was the success?
- What is the timeline for this pain experience?

Assessment of illness perceptions:
- Observe for maladaptive pain cognitions:
  - Ruminating about pain
  - Hypervigilance to somatic signs
  - Use of Pain Catastrophizing scale

Pain Catastrophizing Scale

Brief Illness Perceptions: Questionnaire or Query

Can you think of what you might say for Each of these questions?
Remember! Pain physiology education aims to re-conceptualize pain!

- Education session 1

PNE gives you a window of opportunity....

- Decrease the threat value of pain through understanding how the nervous system works....

Pain Neuroscience Education: Tips

- Likely more effective as part of physical therapy
- Incorporate into movement therapy
- Not a separate psychological intervention

Aims of Pain Neuroscience Education

- Decrease fear
- Positively change patient’s perception of their pain
- Improve attitudes about pain
- Improve pain, cognition, and physical performance

Aims of Pain Neuroscience Education

- Increase pain thresholds during physical tasks
- Improve outcomes of therapeutic exercises
- Reduced widespread brain activity
- Reduce health care costs and shorten return to work after surgery

Education session 1: What is pain?

- Acute vs. Chronic pain
- Why acute pain is normal and useful
- How acute pain originates in the nervous system
- How pain become chronic (plasticity of nervous system)
How typical nerves work

Your heightened nervous system

Images from: Adriaan Louw WHY DO I HURT? 2013

Nosy neighbors- widespread pain

Factors keeping nerves sensitive

Knowledge calm nerves down

Blood and Oxygen calm nerves down.

• Aerobic exercise
**Medicine Calms Nerves Down, but…..**

Reinforcing the conversation....

<table>
<thead>
<tr>
<th>Homework!</th>
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<tbody>
<tr>
<td>Homework between sessions 1 &amp; 2</td>
</tr>
<tr>
<td>Educational information buffet</td>
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<tr>
<td>2nd educational session</td>
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<tr>
<td>Neurotransmission of pain</td>
</tr>
<tr>
<td>Application during daily life situations</td>
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<td>Application during treatments</td>
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**Take a test! 1 day before coming back to clinic...**

<table>
<thead>
<tr>
<th>Revised Neurophysiology of Pain Questionnaire</th>
<th>T</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. It is possible to have pain and not know about it.</td>
<td>☑️</td>
<td>☑️</td>
</tr>
<tr>
<td>2. When part of your body is injured, special pain receptors convey the pain message to your brain.</td>
<td>☑️</td>
<td>☑️</td>
</tr>
<tr>
<td>3. Pain only occurs when you are injured or at risk of being injured.</td>
<td>☑️</td>
<td>☑️</td>
</tr>
<tr>
<td>4. When you are injured, special receptors convey the danger message to your spinal cord.</td>
<td>☑️</td>
<td>☑️</td>
</tr>
<tr>
<td>5. Special nerves in your spinal cord convey &quot;danger&quot; messages to your brain.</td>
<td>☑️</td>
<td>☑️</td>
</tr>
<tr>
<td>6. Nerves adapt by increasing their resting level of excitement.</td>
<td>☑️</td>
<td>☑️</td>
</tr>
</tbody>
</table>

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**2nd Education session**

<table>
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<tr>
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</tr>
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</table>

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- **7.** Chronic pain means that an injury hasn’t healed properly.
- **8.** The body tells the brain when it is in pain.
- **9.** Nerves adapt by making ion channels stay open longer.
- **10.** Descending neurons are always inhibitory.
- **11.** Pain occurs whenever you are injured.
- **12.** When you injure yourself, the environment that you are in will not affect the amount of pain you experience, as long as the injury is exactly the same.
- **13.** The brain decides when you will experience pain.
Questionnaire can guide conversations...

Key discussion:
- Discuss the existence of sensitization related to the patient
- Give insight into:
  - Somatic, psychosocial
  - Behavioral factors associated with pain

Discuss how this information can be applied to daily life
- Sets the stage for:
  - Application of adaptive pain coping strategies
  - Self management programs
  - Graded activity/exercise therapy

Application of pain physiology education during treatment
- Pain physiology education is a continuous process
- Introduce treatments like stress management, graded activity/exercise during educational sessions
- Explain how it helps to calm the nervous system
- Watch for re-emergence of maladaptive cognitions
- Redirect patient via conversations and readings

Shifting the focus from somatic signs towards adaptive coping strategies...
Immediate Effects on Pressure Pain Threshold Following a Single Cervical Spine Manipulation in Healthy Subjects

CÉSAR FERNÁNDEZ DE LAS PEÑAS, PT, FMP(OPT)†, MARTA PÉREZ DE HEREDIA, OT†, MIGUEL BREDA-NERYO, OT†, JOAN C. MANZANARRA-PÁGNO, MD, FICP†

JOURNAL OF ORTHOPAEDIC & SPORTS PHYSICAL THERAPY
VOLUME 37 | NUMBER 6 | JUNE 2007 | 325

• 15 healthy subjects
• 3 treatments sessions
  • Manipulation - C5-C6 level
  • Placebo
  • Control
  • Assessment via Pain pressure threshold or PPT

| Post manipulation change in pressure pain threshold demonstrates mechanical hypoalgesic effect |

| Supporting evidence |
• Vincenzino et al: Lateral glide of cervical spine ⇒ immediate increase in PPT with lateral epicondylalgia
• Paungmali et al, Vicenzino et al: MWM (mobilization with movement) ⇒ immediate increase in PPT with lateral epicondylalgia

| Initial Effects of Elbow Taping on Pain-Free Grip Strength and Pressure Pain Threshold |

• 12 Men
• 4 Women
• 13.1 +/− 9.9 months duration of lateral elbow pain
Diamond tape improved pain-free grip strength immediately and 30 min after application, making pain-free implementation of exercise possible.

In healthy participants, mean effect sizes for isometric exercise ($d_{thr} = 1.02$, $d_{int} = 0.72$) and dynamic resistance exercise ($d_{thr} = 0.83$, $d_{int} = 0.75$) were large. Meeus et al. — Cycle ergometry had increased PPTs at multiple body sites. Hoffman et al. demonstrated large effect even 30 minutes post exercise. Isometric quadriceps had large hypoalgesic effects for shoulder myalgia.

Moderate submaximal isometrics and vigorous aerobic exercises—moderate to large effect on experimental pain in FMS. Submaximal isometrics at low intensity (10%) increased PPT of deltoids in FMS with large effect. Generally, low to moderate intensity exercise may be the way to modulate pain in FMS patients.

What dosage?

- First use dominant hand to determine maximum handgrip contraction strength
- 5 second max contraction
- 1 minute rest
- 40-50% of max contraction for 2 minutes
- 1 minute rest
- 40-50% of max contraction for 2 minutes

Contralateral Attenuation of Pain After Short-Duration Submaximal Isometric Exercise

Kati F. Koltyn and Masatuki Umeda
Department of Kinesiology, University of Wisconsin-Madison, Madison, Wisconsin.

Decrease in pain ratings...

Increase in pain threshold bilaterally...

Clinical implications:

- What are your thoughts?
- Use of low level isometrics... in non-painful extremity
- As a precursor to daily function
- As a precursor to therapy

Joint mobilizations for Pain modulation?

Overall demographics of patients very similar

Musculoskeletal Science and Practice

Elevated systolic blood pressure in impaired group

Longer duration of pain in impaired group
Conditioned pain modulation enhanced!

Looking back....making connections

- Duration > 3 months
- Have you seen many patients with widespread pain?
- High levels of pain and disability leading to poor prognosis and persistent pain?
- Sensitivity to cold?
- Decreased pain free grip strength?
- Complaints of neck and shoulder pain?
- The longer duration pain correlates with decreased PPT and impaired conditioned pain modulation
- Modulating wide spread pain and pain sensitivity via mobilization, isometric exercises, manipulation, neural mobilization, dynamic exercise
- Early focus on modulating pain?

Clinical application:

- Recognize signs of Central sensitization
- Apply a systematic yet customized approach to Pain Neuroscience Education (Injuries do not occur in a vacuum)
- Create a window of opportunity to “calm” the brain using:
  - Isometric exercises
  - Joint mobilizations/manipulations
  - Taping

Questions?

- Thank you!
  - Dsueki@apu.edu
  - msuong@apu.edu
- Videos available at www.Youtube.com
- Search: PhysioU Quantitative sensory testing