IMMEDIATE INFLUENCE OF PSYCHOSOCIAL VARIABLES AND CENTRALLY MEDIATED PAIN ON ACUTE MUSCULOSKELETAL INJURY

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WHO ARE WE?

• Karin Townson, DPT, MTC, CSCS
• Clinical Associate Professor at the University of Puget Sound
• Physical Therapist
  • Primary patient population – local performing artists
    - Dancers
    - Circus artists
    - Musicians
  • Paciﬁc Northwest Ballet

• Jason Steere, PT, DPT, ATC, OCS, FAAOMPT
• Evidence In Motion faculty
• Physical Therapist
  • Current patient population – US Army Special Operations unit
  • Prior professional experience:
    - Professional baseball
    - Collegiate athletics
    - General outpatient orthopedics

IMMEDIATE PHYSICAL MANIFESTATIONS

• Inflammatory process
• Local changes vs. central changes
  - Prostaglandins, glutamate, aspartate
  - Bradykinins
  - Cortisol
  - Thyroxine
**CENTRAL SENSITIZATION**
- Defined as "an augmentation of responsiveness of central neurons to input from unimodal and polymodal receptors."\(^1\)
- Increased sensitivity of CNS nociceptors that receive afferent information from body\(^2\)
- Increased responsiveness to exogenous stimuli
  - Heat, cold, pressure, sound, light
  - Increased cerebration of pain
  - Decreased tolerance to stimuli

**PRIMARY V. SECONDARY HYPERALGESIA**
- Released substances cause primary hyperalgesia
  - Primary sensitization of nociception
  - Sensory-discriminative component
  - Prevents further injury
- Secondary hyperalgesia
  - Increased reactivity of dorsal horn
  - Altered sensory processing in the brain
  - Affective-motivation component

**SIGNS AND SYMPTOMS OF CENTRAL SENSITIZATION IN CHRONIC POPULATION**
- Seemingly disproportional pain associated with less than expected injury or pathology
- Hypersensitivity to various stimuli in the absence of inflammation or neural lesion
- Generalized rather than local decrease in pain pressure threshold
- Heightened bilateral response to neurodynamic testing
- Increased pain perception in response to exercise

**CENTRAL “SENSITIZATION” VS. CENTRALLY MEDIATED PAIN**
- “Sensitization” involves neurons becoming more reactive due to afferent stimuli
- Central “mediation” encompasses central processing of the pain and disability experience
- Indissoluble connection between brain and periphery
- Life experiences (past trauma, coping strategies, etc) influence connection
- Locus of control – external v. internal

**CENTRAL MEDIATION OF PAIN**
- Under normal circumstances:
  - Excitatory and inhibitory stimuli balanced
- Central mediation affects the perception of pain immediately
  - Descending pain inhibitory mechanisms
- Pain may be amplified or inhibited to detriment
  - Altered pain neuromatrix 2° neuroplasticity
- Activity in descending pathways modulated by vigilance, attention, and stress

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\(^1\) Meyer et al., 1995; \(^2\) Shacklock, 1999

Nijs, 2010; Nijs, 2011; Rygh, 2002
"People feel they understand complex phenomena with far greater precision, coherence, and depth than they really do; they are subject to an illusion—an illusion of explanatory depth."

Rozenblit 2002

"Can we see the forest for the trees?"
**BINARY THINKING**

- Peripheral OR Central
- Nociceptive OR Central Sensitization
- Biomechanical OR Psychosocial

**“OR”**

- Manual therapy
- Mechanical effects
- Hands on
- TBC
- Graded exposure
- Neuropysiological effects
- Hands off
- Desensitization

**“AND”**

**TIME FACTOR**

- Centrally mediated pain often only considered when primary injury has "healed"
- Brain connection is resolute, so central mediation of pain begins immediately
- Stimulation of one group of afferent nociceptor fibers results in amplified responses to other non-stimulated nociceptor or non-nociceptor fibers
- High degree of variability across populations
  - Both in time and intensity

Woolf, 2011
CENTRAL MEDIATION IN ACUTE INJURIES

- Centrally mediating factors can be present in non-chronic injuries
  - LBP, fibromyalgia, WAD, RA, TMD
- Presence of central symptoms can be a negative predictor of outcomes
  - E.g., WAD
- Acute inflammatory process can trigger peripheral and central sensitization

BRING IT TO COGNITION

- Recognition is often intuitive
- Measures to help us recognize
  - Depression screen, FABQ, TSK, PCS, FOTO, etc.
- Need to acknowledge the central component in EVERY patient
- Meta-cognition to acknowledge YOUR own central component

TREATMENT INTERVENTION

- Nociception from tissue injury can be part of initial intervention
- Treat acute/subacute inflammatory dysfunction
- Consider contribution of centrally mediating factors
- Education on the neurophysiology of pain
  - Timed appropriately
  - Dosed appropriately
- Consider neurophysiologic effects of manual therapy/exercise

COMPREHENSIVE TREATMENT MODEL

CASE STUDIES

- BG 27 y.o. professional ballet dancer
- Landed from a large jump, rolled ankle and heard a pop
- Attempted to ambulate with 7/10 pain
- Immediate swelling and ecchymosis
- Despite significant inflammatory response, pt did not display apprehension or fear of pain
CASE STUDIES

- JA 24 y.o. Army Ranger PFC
- Fell 12-15 ft off of a repel rope, landing on his back
- Severe pain throughout the spine and unable to walk
- Medivaced to ED
- Imaging read as negative by radiology, possible T2 fracture later
- Initial PT examination revealed chief complaint at the TL region
- No change in pain and disability for 6 months
- Discharging

CASE STUDIES

- LM is a 24 y.o. professional ballet dancer
- During a performance, was performing a rotational jump
- Felt R low back "tighten up"
- Was "unable to bend in any direction" and had to discontinue show
- At evaluation 2 days later, was initially very guarded but able to perform nearly full AROM

CASE STUDIES

- BR 28 y.o. Army Ranger PFC
- Began to have hip "soreness" in RASP
- 5 mile run when he arrived at Battalion, last 3 miles on dead leg
- Limped into PT office and refused crutches
- Initial x-rays read as negative by radiology
- No change after 3 weeks, sent for MRA
- Severe femoral neck stress fracture, immediate surgery
- Continues to soldier

REFERENCES

- www.india-forums.com
- www.radiopaedic.org

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