#### How Does Acute Pain Become Chronic?

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#### Disclosure

Nothing to Disclose

## **Learning Objectives**

- Describe a patient-centered approach to the formulation of the patient with acute pain
- Review risk factors/predictors of chronic pain
- Identify rational treatment approaches to reduce the risk of developing chronic pain

#### New Chronic Pain

• Who develops it?

#### **Case Example**

- 45 y/o Korean woman s/p OTJI with foot crushed by heavy equipment for depression & disability
- Immediate reconstructive surgery for stability
- Poor compliance with physical therapy
- High levels of acute pain pre- and post-op
- Treated with SAO's and acetaminophen
- Prescribed multiple agents for insomnia & anxiety
- After 6 months, referred to Orthopedics for BKA

Chapman CR, Vierck CJ. J Pain. 2017.

# **Typical Risk Factors**

- Demographic variables
- Pain characteristics
- Psychological factors
- Contextual details

Miller RM, Kaiser RS. Curr Pain Headache Rep. 2018.

# Demographics

- Age
- Gender
- Education
- Employment
- Health status

#### **Pain Characteristics**

- High pain intensity
- Long pain duration
- Radiation of pain
- Prior episodes of pain
- Multiple sites of pain
- Multiple somatic symptoms

# **Psychological Factors**

- Negative emotion
- Depression
- Anxiety
- Anger
- Fear
- Stress
- Distress

- Catastrophizing
- Hypervigilance
- Self-efficacy
- Neuroticism
- Pain sensitivity
- Somatization

#### Context

- Injured at work
- Work safety
- Work satisfaction
- Compensation
- Litigation
- Social support
- External attributions of responsibility

# **Risk Factors for New Chronic Pain**

• Why does it matter?









# **Longitudinal Relationships**

- Majority of the data support the diathesis-stress model (depression is a consequence of chronic pain)
- Treatment of depression improves pain and disability
- Directionality depends on the type of "depression" —Affective disorders (MDD, Dysthymic Disorder, Bipolar)
  - -Adjustment disorders, Grief reactions, Demoralization

Polatin P, et al. Expert Rev Clin Pharmacol. 2017; Velly AM, Mohit S. Prog Neuropsychopharmacol Biol Psychiatry. 2017; Sheng J, et al. Neural Plast. 2017.

#### **Longitudinal Relationships**

- Depressive disorders at baseline doubled the risk for new onset back pain 13 years later
- Severe depression (impairment) tripled the risk for incident back pain 12 years later
- Major depression + dysthymic disorder (excluding dysphoria) still increased risk for incident back pain 13 years later by 75%

Larson S, et al. Psychol Med. 2004.

#### **Summary of Negative Analyses**

- Current depression did not increase the risk for incident back pain; odds ratio (OR)=1.70, (0.71, 4.08)
- Depression at baseline did not increase the risk for incident back pain 1 year later
- Back pain at baseline was not associated with depression at baseline
- Back pain at baseline was not associated with incident depression at any time point

Larson S, et al. Psychol Med. 2004.

# Behaviors Drive $\rightarrow$ Choice $\rightarrow$ Learning



• Can we unlearn what we learn?



#### **Patients With Substance Use Disorder**

• What can we learn with a paradigm shift?

#### **Susceptibility To Chronic Pain**

- A history of substance use increases abuse of pain medications
- Cold pressor pain tolerance is  $\downarrow$  in current opiate and cocaine users compared with former users
- Alcoholics and families of alcoholics have  $\uparrow$  pain sensitivity and  $\uparrow$  pain reduction with EtOH

Witkiewitz K, Vowles KE. Alchol Clin Exp Res. 2018; Webster LR. Anesth Analg. 2017; Clark, et al. Can J Psychiatry. 2008.



#### **BPI Treatment**

- Receiving treatment for pain outside ATS = 14%
- Average relief provided by pain treatment = 51%
- Types of pain treatment being received:
  - -Analgesics (NSAIDs, opioids): 12% (89% of treated)
  - -Other (PT, blocks, epidurals): 7% (53% of treated)
- No one received adjuvant analgesics (ADs, AEDs)

NSAIDs. nonsteroidal anti-inflammatory drugs; PT, physical therapy; AEDs, antiepileptic drugs. Clark , et al. CPDD. 2007.

# Dimensions

 $\textbf{Potential} \rightarrow \textbf{Provocation} \rightarrow \textbf{Response}$ 

# **Pain Modulation**

• How are we different?

#### **Central Pain Modulation**

- Endogenous analgesia system (individual trait)
- Capability assessed via the Diffuse Noxious Inhibitory Control (DNIC) test paradigm
- Lower DNIC efficiency is associated with pain
  - -Healthy people with pain
  - -Chronic pain syndromes
    - Primarily those postulated to be due to central sensitization
    - Fibromyalgia syndrome, multiple sclerosis, temporomandibular disorder, migraine, tension headache, irritable bowel syndrome

Hermans L, et al. Pain Pract. 2016; Bannister K, Dickenson AH. J Physiol. 2017; Granot. Curr Opin Anes. 2009.

## **Incidence Of Post-thoracotomy Pain**

- 62 patients undergoing thoracotomy
  - -38 men, mean age = 62 +/- 14 years, multiple causes
  - -36 patients  $\rightarrow$  chronic pain, no med/surg predictors
- Mean follow-up = 29 +/- 17 weeks
- Acute post-op pain = 49 +/- 21 (0-100 NPS)
- Chronic post-op pain = 55 +/- 27 (0-100 NPS)
- Acute post-op pain correlated with chronic pain
- DNIC efficiency correlated with chronic pain

NPS, numerical pain scale. Rodriguez-Aldrete D, et al. J Cardiothorac Vasc Anesth. 2016; Yarnitsky, et al. Pain. 2008.

#### **Predictors of Post-thoracotomy Pain**

- Acute postoperative pain intensity (modifiable?)
  - -OR = 1.80 (1.28 2.77)
  - -Change of 10 units on scale of 0 to 100  $\,$
- DNIC efficiency (dynamic preoperative trait)
  - -OR = 0.52 (0.33 0.77)
  - -Change of 10 units on scale of -100 to +100  $\,$
  - -Probability of chronic post-thoracotomy pain
    - DNIC 0  $\rightarrow$  80%; DNIC 40  $\rightarrow$  23% ; DNIC 50  $\rightarrow$  12%
    - No correlation with acute postoperative pain (independent)

Humble SR, et al. Eur J Pain. 2015; Yarnitsky, et al. Pain. 2008.

#### Somatic Symptoms

How do symptoms become chronic?

# Somatization $\rightarrow$ Chronic Pain ?

- Prospective population-based follow-up survey
- 1658 people without chronic widespread pain
  (No pain = 825; Some pain = 833)
- Somatic symptoms, psychological distress, fatigue, health anxiety, illness behavior
- 1404 respondents at 12-month follow-up
- New chronic widespread pain
  - -4.4% of men; 6.8% of women
  - $-\operatorname{One-third}$  of new cases were men

Sharma MP, Manjula M. Int Rev Psychiatry. 2013; McBeth, et al. Arthritis & Rheumatism. 2001.

## **Predictors of Chronic Pain**

- 8% of people with some pain vs 2% w/o pain
- Health anxiety: NS
- Fatigue: OR = 2 (univariate only)
- Psychological distress: OR = 2 (univariate only)
- Somatic symptoms >2: OR = 4 (1.5 7.4)
- Illness behaviors: OR = 4 9 (1.8 22.2)

-Frequent healthcare visits for symptoms that disrupt normal activity

McBeth, et al. Arthritis & Rheumatism. 2001.

# **Life Stories**

# $\mathsf{Setting} \to \mathsf{Sequence} \to \mathsf{Outcome}$

#### **Post-traumatic Stress Disorder**

• What events are traumatic?

#### **PTSD and Chronic Pain**

- Criteria
  - Re-experiencing the event
  - Avoidance of reminders of the event
  - Hyperarousal
- Motor vehicle collisions → whiplash
  - Great variation across countries
  - Decreases if financial benefits are reduced
  - Rare for same magnitude collisions in other contexts
  - No dose effect of trauma intensity and probability

Siqveland J, et al. Front Psychiatry. 2017; McLean ,et al. Psychosom Med. 2005.

#### **Pain Catastrophizing**

• Why are these people so distressed?

# **Pain Catastrophizing**

- An exaggerated negative mental set brought to bear during an actual or anticipated painful experience
- An expectation or worry about major negative consequences from a situation, even one of minor importance
- Multidimensional cognitive construct
  - -Magnification: "I am afraid that something serious will happen."
  - -Rumination: "I cannot stop thinking about how much it hurts."
  - -Helplessness: "There is nothing I can do to reduce the intensity of the pain."

Schutze R, et al. J Pain. 2018; Sullivan, et al. Clin J Pain. 2001.

#### **Modifying Outcome**

- Catastrophizing predicts
  - -Acute pain intensity and sensitivity
  - -Development of chronic pain, disability,  $\downarrow$ QoL
- Treatments for catastrophizing
  - -Cognitive behavioral therapy and adaptive coping skills training
  - -Distraction, relaxation, and imagery
  - -Social support
  - —Education

Khan, et al. Am J Surg. 2011; Edwards, et al. Nat Rev Rheumatol. 2011.

# Conclusions

• What can really be done?

# **Preventing Chronic Pain**

- Diseases
  - -Repair and Cure
- Dimensions
  - —Guide and Strengthen
- Behaviors
  - -Extinguish and Expose
- Life Stories
  - -Rescript and Remoralize

#### **Treatments of Predictors**

#### Diseases

- -Neuropathic pain and major depression
  - Antidepressants
  - Anticonvulsants
  - Augmenting agents

#### Dimensions

- -Pain modulation and somatosensory amplification
  - Biofeedback and relaxation
  - Yoga, Tai Chi, Qigong
  - Cognitive-behavioral psychotherapy

#### **Treatments of Predictors**

#### Behaviors

- -Substance use disorders and fear/avoidance
  - Group-based behavioral psychotherapy
  - Desensitization
  - Active physical therapy

#### Life Stories

- -PTSD and catastrophizing
  - Support groups
  - Interpersonal psychotherapy
  - Insight-oriented psychotherapy

#### Case – Amputation was performed!

- Diseases
  - MDD: Sertraline 300 mg/d
  - PAP: Valproate 500 mg BID
- Dimensions
  - Introvert: Puppy with training
  - Amputee: Prosthetics + PT
- Behaviors
  - SUD: Opioid taper after other txs
  - F&A: Support groups (OT, Amputees, Church)
- Life Stories
  - Marital therapy  $\rightarrow$  infidelity  $\rightarrow$  divorce
  - Vocational rehabilitation  $\rightarrow$  RTW

## **Hope for Preventing Chronic Pain**

- Recognizing profiles of risk for new chronic pain
- Preventing the transition from acute to chronic pain
- Treating specific causes of new chronic pain
- Addressing the nature of barriers to restoring health