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Dr. Dinesh Kumbhare is an Associate Professor and Clinician Scientist in the Department of Medicine at the University of Toronto within the Division of Physical Medicine and Rehabilitation. He is an Affiliate Scientist at the Toronto Rehabilitation Institute (TRI). He is cross-appointed to the Institute of Biomedical and Biomaterial Engineering, Faculty of Kinesiology & Physical Education and Institute of Health Policy, Management and Evaluation at the University of Toronto. He is also adjunct faculty in Engineering at McMaster University. He obtained his MSc in Health Research Management from McMaster University and his PhD in Biomedical Engineering at the University of Toronto. He is section editor for the Physiatry Reviews for Evidence in Practice and Resident, Fellow Section with the American Journal of Physical Medicine and Rehabilitation. Dr. Kumbhare was the principal author of the book, Buschbacher's Manual of Nerve Conduction Studies. According to the Neurodiagnostic Journal, this is "the gold standard in many EMG labs, this manual is a practical working reference for performing a wide variety of common nerve conduction studies. It provides both practicing clinicians and trainees with an impressive database of reference values they can use to interpret nerve conduction results with confidence". He is leading the Pain Research Institute at TRI, a newly established program that will foster a collaborative environment that brings together multidisciplinary and interprofessional constituency of researchers. TRI is committed to improving the health status of people who suffer from pain. This will be achieved through leadership and excellence in education, delivery of evidence based clinical care, and expanding the horizon of medical knowledge through fundamental science and clinical research endeavors.

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Chronic Pain: An Update

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University of Toronto

Institute of Biomaterials
& Biomedical Engineering
UNIVERSITY OF TORONTO

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FACULTY OF MEDICINE

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- Professor, Dept. Medicine, University of Toronto
- Physical Medicine and Rehabilitation
- Biomedical Engineering PhD
- Chair, Pain assessment and Rehabilitation
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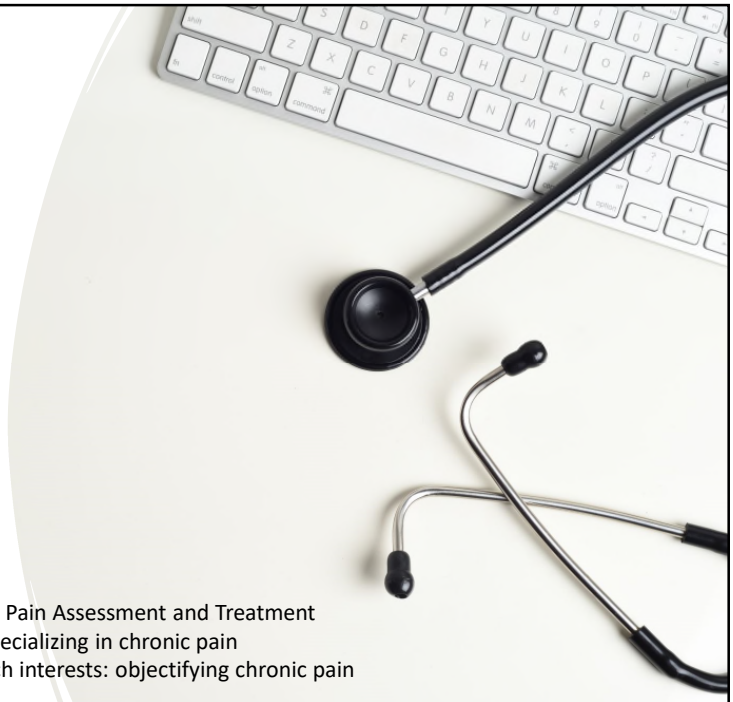


Chronic Pain Assessment and Treatment
EMG specializing in chronic pain
Research interests: objectifying chronic pain

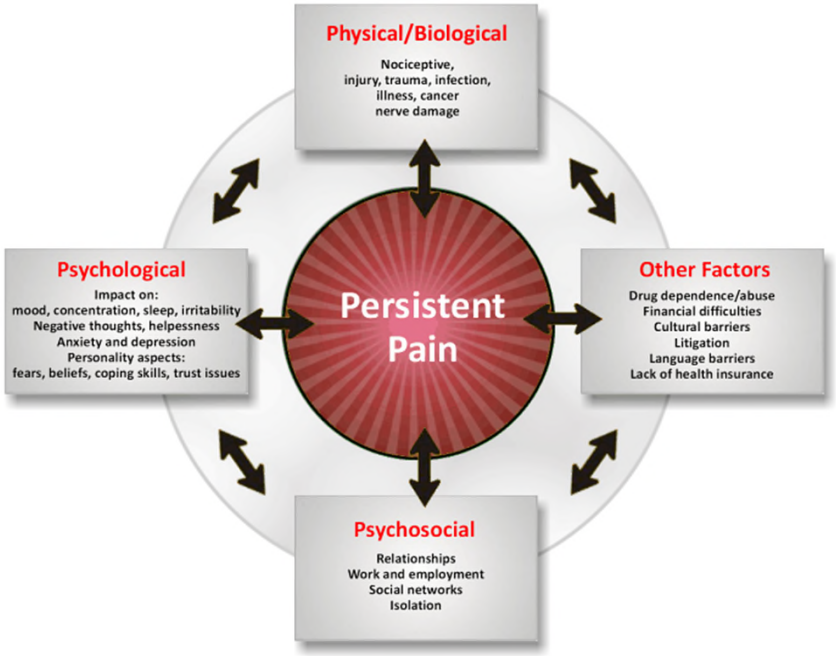
Disclosures

- The speakers do not have any conflicts of interest to declare

Agenda

1. New Definition for chronic pain
2. New Mechanism: Nociplastic Pain
    1. Definition and clinical criteria
    2. Clinical features
3. How to Objectify Chronic Pain: Imaging Evidence for chronic pain

Biopsychosocial Model for Chronic Pain



New Definition Endorsed by IASP and WHO

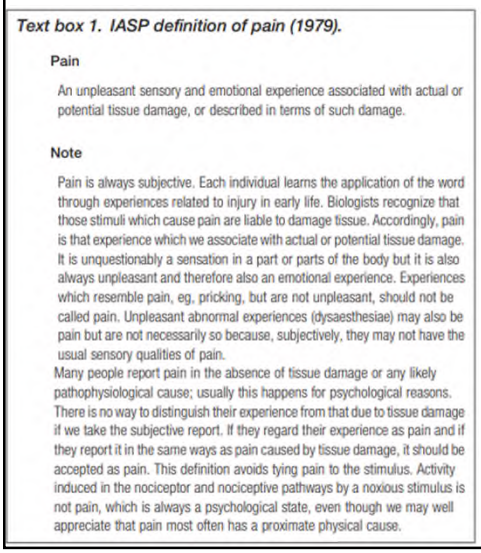
- Chronic pain recognized as a disease
- Chronic pain recognized as an experience that has multiple domains
  - Biological
  - Psychological
  - Functional
  - Societal
- Clinician needs to accept patient's experience
- "Actual Tissue Damage" demonstration no longer required


PAIN

The revised International Association for the Study of Pain definition of pain: concepts, challenges, and compromises

Srinivasa N. Raja[a,*], Daniel B. Carr[b], Milton Cohen[c], Nanna B. Finnerup[d,e], Herta Flor[f], Stephen Gibson[g], Francis J. Keefe[h], Jeffrey S. Mogil[i], Matthias Ringkamp[j], Kathleen A. Sluka[k], Xue-Jun Song[l], Bonnie Stevens[m], Mark D. Sullivan[n], Perri R. Tutelman[o], Takahiro Ushida[p], Kyle Vader[q]

New Definition for Chronic Pain 2020


Text box 1. IASP definition of pain (1979).

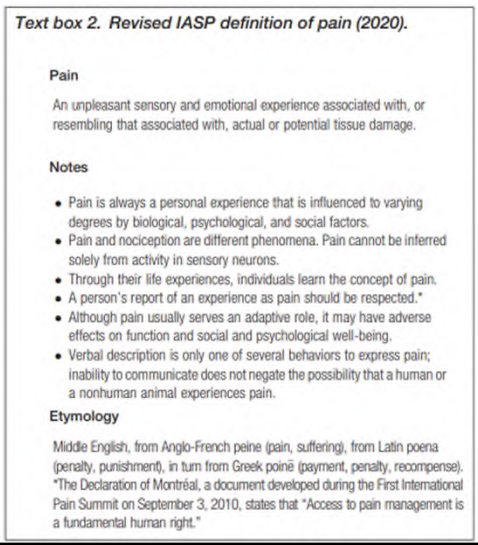
Pain

An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

Note

Pain is always subjective. Each individual learns the application of the word through experiences related to injury in early life. Biologists recognize that those stimuli which cause pain are liable to damage tissue. Accordingly, pain is that experience which we associate with actual or potential tissue damage. It is unquestionably a sensation in a part or parts of the body but it is also always unpleasant and therefore also an emotional experience. Experiences which resemble pain, eg, pricking, but are not unpleasant, should not be called pain. Unpleasant abnormal experiences (dysaesthesiae) may also be pain but are not necessarily so because, subjectively, they may not have the usual sensory qualities of pain.

Many people report pain in the absence of tissue damage or any likely pathophysiological cause; usually this happens for psychological reasons. There is no way to distinguish their experience from that due to tissue damage if we take the subjective report. If they regard their experience as pain and if they report it in the same ways as pain caused by tissue damage, it should be accepted as pain. This definition avoids tying pain to the stimulus. Activity induced in the nociceptor and nociceptive pathways by a noxious stimulus is not pain, which is always a psychological state, even though we may well appreciate that pain most often has a proximate physical cause.


Text box 2. Revised IASP definition of pain (2020).

Pain

An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage.

Notes

- Pain is always a personal experience that is influenced to varying degrees by biological, psychological, and social factors.
- Pain and nociception are different phenomena. Pain cannot be inferred solely from activity in sensory neurons.
- Through their life experiences, individuals learn the concept of pain.
- A person's report of an experience as pain should be respected.*
- Although pain usually serves an adaptive role, it may have adverse effects on function and social and psychological well-being.
- Verbal description is only one of several behaviors to express pain; inability to communicate does not negate the possibility that a human or a nonhuman animal experiences pain.

Etymology

Middle English, from Anglo-French peine (pain, suffering), from Latin poena (penalty, punishment), in turn from Greek poinē (payment, penalty, recompense).

*The Declaration of Montréal, a document developed during the First International Pain Summit on September 3, 2010, states that "Access to pain management is a fundamental human right."

Subjective→personal experience

Person's report of pain should be respected

Verbal description is only one way to communicate pain

Actual tissue damage→associated with/resembling

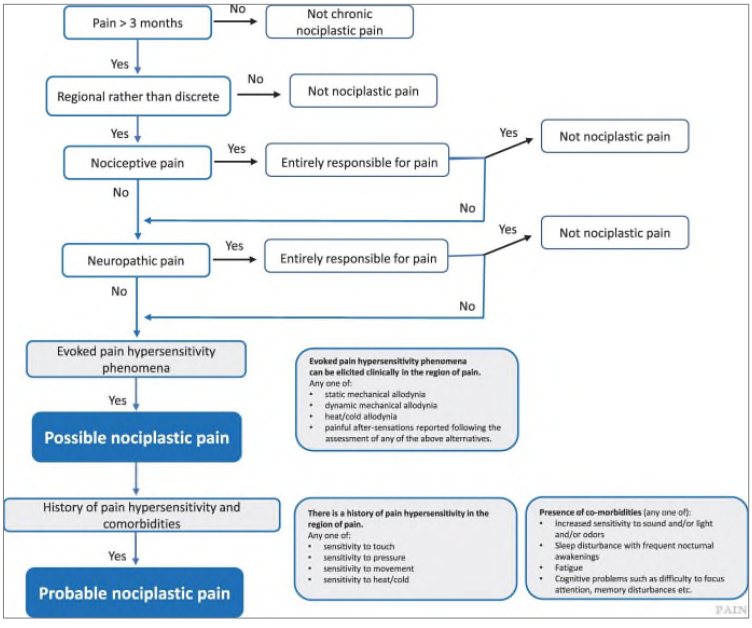
Pain causes adaptation of function, social and psychological well-being

Pain Mechanisms

- NOCICEPTIVE PAIN
- Pain that arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors.
- NEUROPATHIC PAIN
- Pain caused by a lesion or disease of the somatosensory nervous system.
- NOCIPLASTIC PAIN
- Pain that arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain.

IASP
INTERNATIONAL ASSOCIATION FOR THE STUDY OF PAIN
CELEBRATING | 1974–2024

Chronic nociplastic pain affecting the musculoskeletal system: clinical criteria and grading system



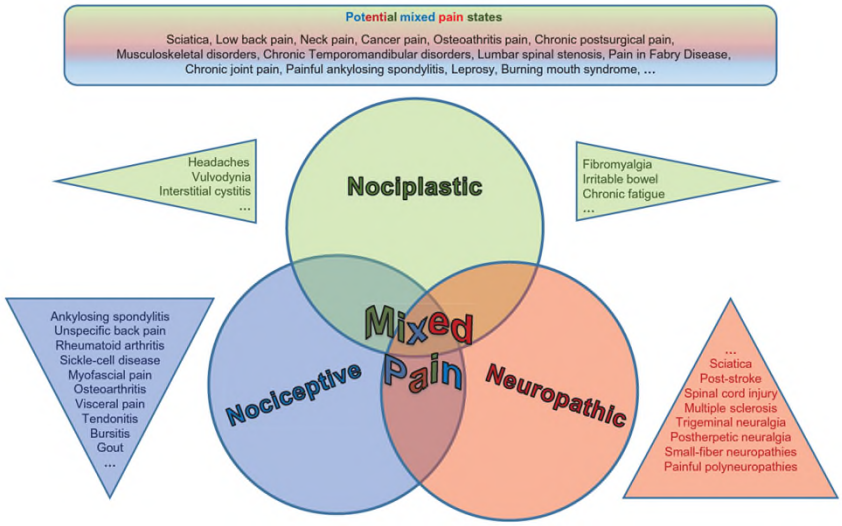
Kosek, Eva; Clauw, Daniel; Nijs, Jo; Baron, Ralf; Gilron, Ian; Harris, Richard E.; Mico, Juan-Antonio; Rice, Andrew S.C.; Sterling, Michele

PAIN162(11):2629-2634, November 2021.

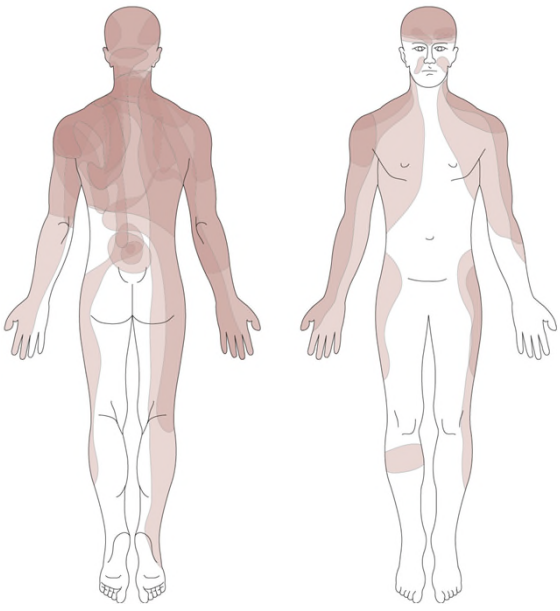
doi: 10.1097/j.pain.0000000000002324

Flow chart of identifying and grading nociplastic pain affecting the musculoskeletal system. Musculoskeletal pain is deep, rather than cutaneous and regional, multifocal, or widespread in distribution (rather than discrete). In case of multifocal pain states that can be caused by different chronic pain conditions (eg, shoulder myalgia and knee osteoarthritis), each chronic pain condition or pain region must be assessed separately.

Mixed Pain



Freynhagen et al CMRO 35:6, 1011-1018; 2019

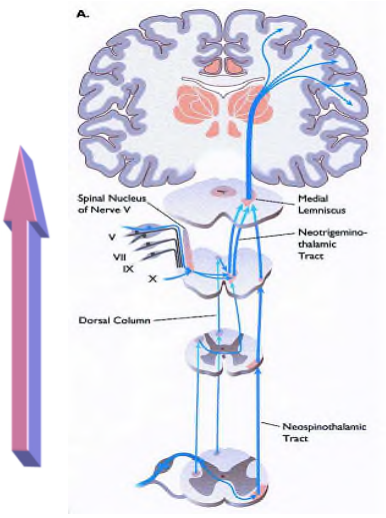


CNS Changes

- Pain inhibition; Descending neural inhibitory control (5HT, NA, EnK, …)
- Spinal Changes: **wide dynamic range neuron** (WDR) neurons prioritize pain signals; Ephaptic crosstalk occur; Interneurons opioid receptors downgrade; Reduced activity of Diffuse Noxious Inhibitory Controls (DNIC)
- Brain Changes: regions, not previously involved, are now recruited, brain volume lost, central glial cells become activated

Pathophysiology: pain sensing and processing abnormalities

Pain Sensing



Acute pain:

Pain-sensing signals are initiated in response to a stimulus

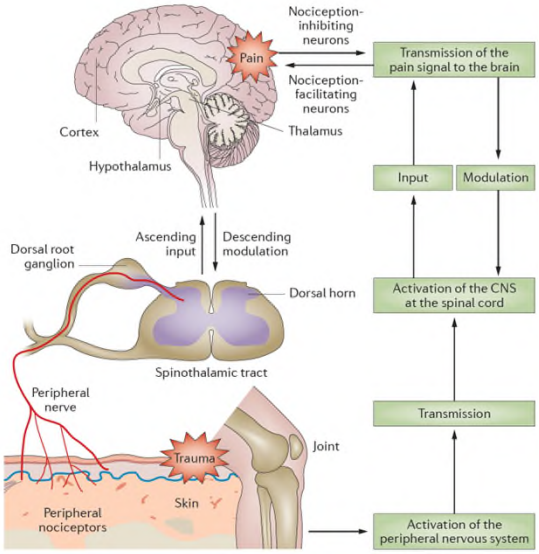
Chronic pain:

Pain signals are generated for no reason and may be intensified

- **Pain-relieving mechanisms may be defective or deactivated**

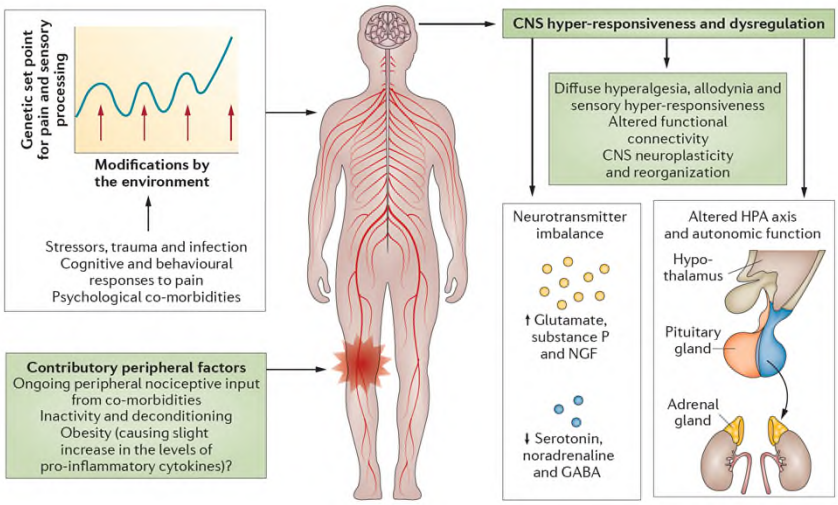
(Illustration: Seward Hung, 2000)

Pain Processing and its Modulation

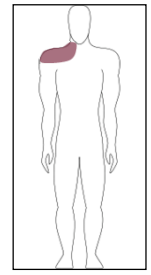


Hauser et al. Nature Reviews, 2015 doi 10.1038

Pathophysiological Processes in Nociplastic Pain

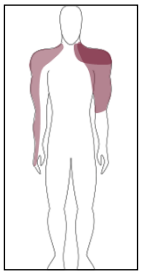


1. Central Sensitization
2. Genetic Set Point for sensory regulation
3. Peripheral Factors like persistent nociceptive input produced by co-morbidities
4. Modified by: psychosocial factors (eg. Anxiety, depression), catastrophizing, biopsychosocial stress



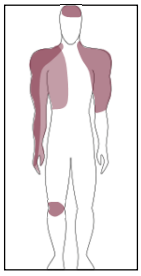
Mild

- 1 limb
- Mainly peripheral
- 1 segment centrally
- 0-25% BSA
- QST?
- Ultrasound
- River's criteria



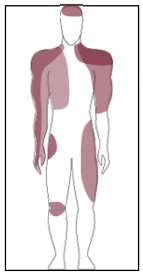
Moderate

- 2-3 limbs peripherally
- 2 segments centrally
- 26-50% BSA
- QST?
- Ultrasound



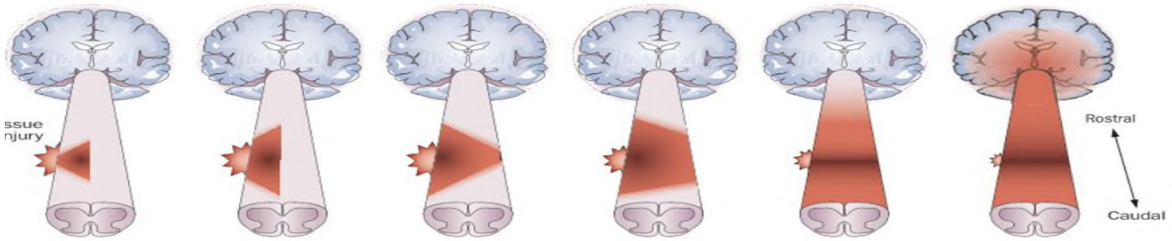
Severe

- 2-3 limbs peripherally
- Multi segmental central sensitization
- 51-75% BSA
- QST
- Ultrasound

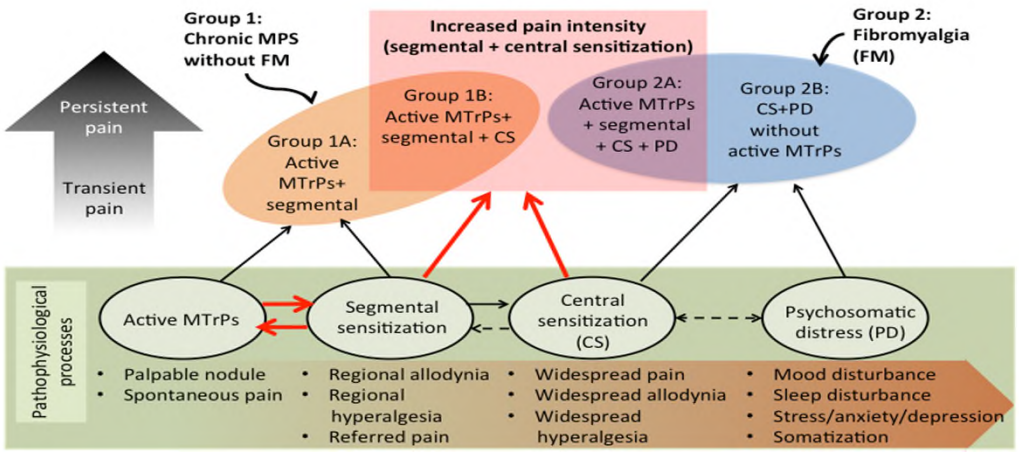


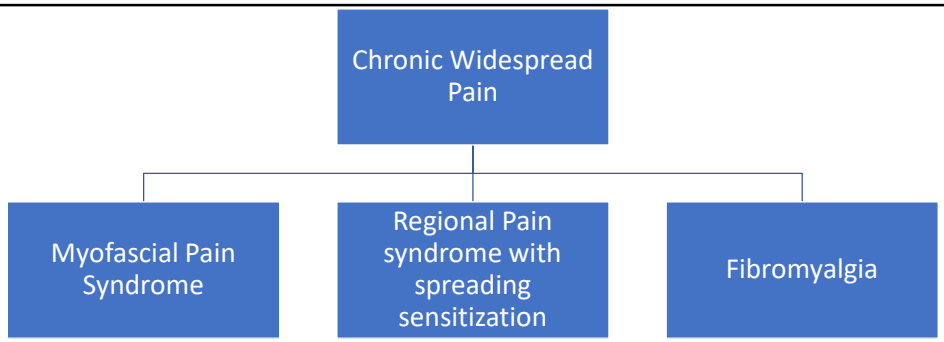
Extreme

- 4 limbs peripherally
- Full central sensitization
- 76-100% BSA
- QST
- Ultrasound
- 2016 FM criteria (Wolfe et al., 2016)



New Trends in Clinical Diagnosis





Chronic Pain is a continuum with 3 major clinically relevant groups

Assessment of a Chronic Pain Patient

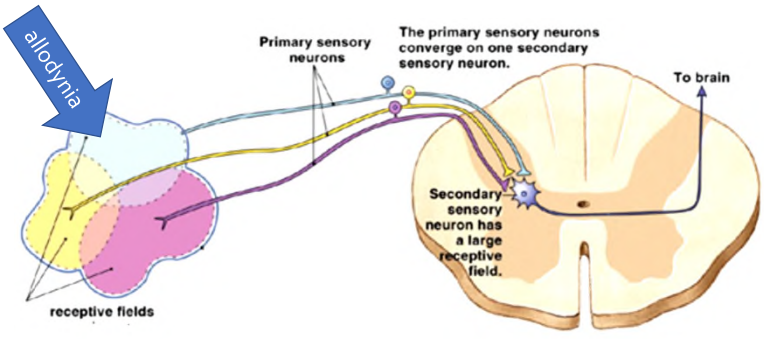
Can we objectify?

Diagnosing Nociplastic Pain…..

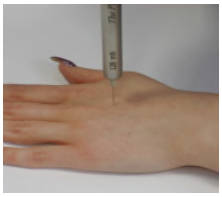
Intention of Physical Examination:

- Establish trust and rapport
- Standard MSK and neurological examination to assess for treatable causes of CWP
- Assess for the differential diagnoses from the history: confirm or refute
- Central sensitization assessment:
  - Sensory assessment: nondermatomal, non-peripheral nerve distribution; hyperpathia, hypoesthesia or allodynia
  - Wind-up phenomenon
  - Lingering sensory phenomenon
  - Pinch and roll technique
  - Wartenburg phenomenon assessment

Assessing Central Sensitization in the Clinic: Brush Allodynia

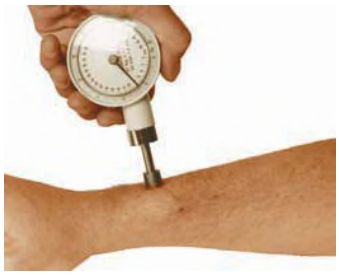


Assessing central sensitization in the clinic
Weighted Pinpricks



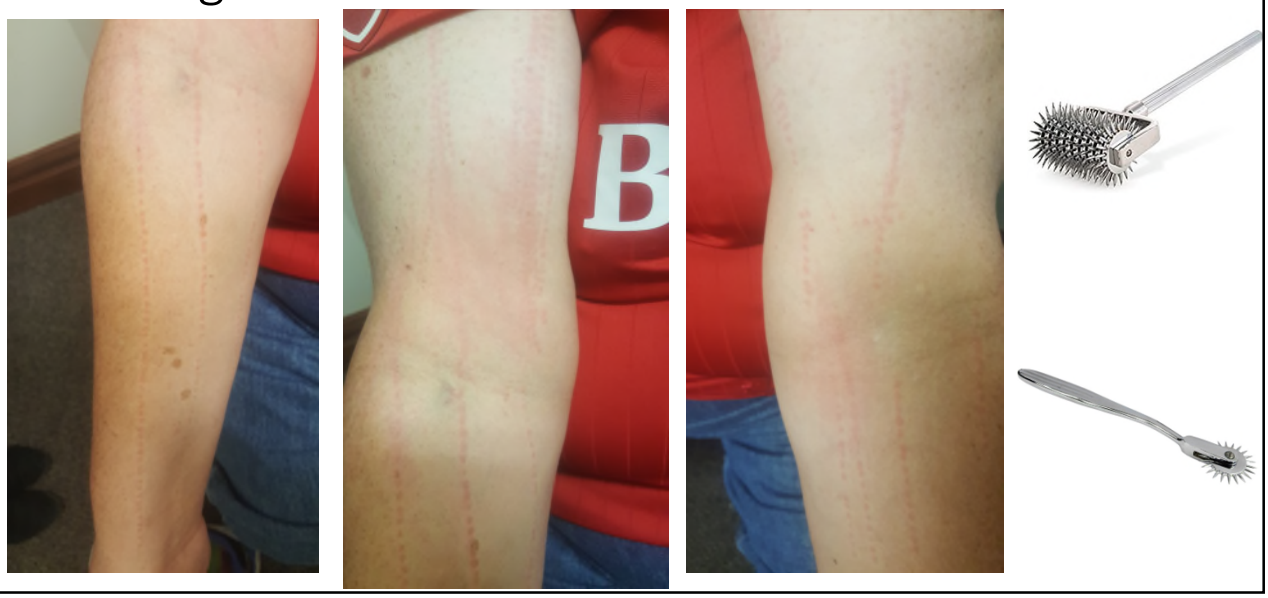
- 7 weighted pinpricks between 8-512 mN
- "Mechanical Pain Threshold" (mN)
- "Windup Ratio"
  - 256 mN pinprick (Rolke 2006)
  - Baseline
  - 10 stimuli applied at 1Hz
  - VAS (baseline):VAS (10^{th} application)

Assessing central sensitization in the clinic
Pain Pressure Threshold



- Pressure algometer
- Applied over the MTrP region
- Constant rate of application
- Highly reliable clinical outcome
  - Very low COVA

Wartenburg Phenomenon using Pinwheel: Neurogenic Inflammation



Clinical Features Associated with Neurogenic Inflammation

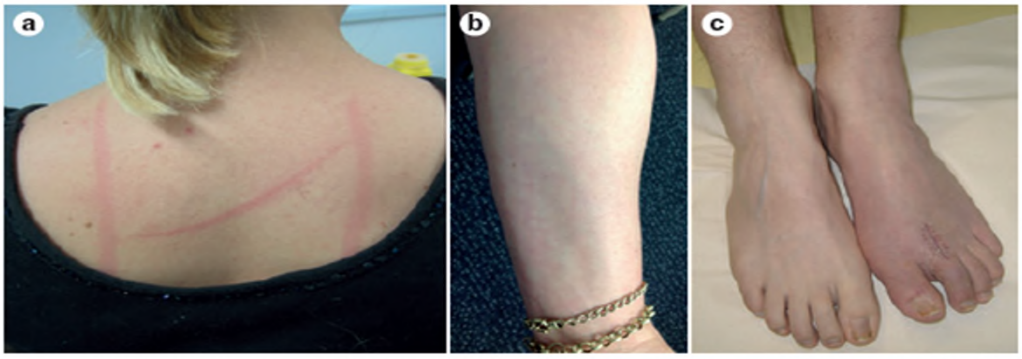
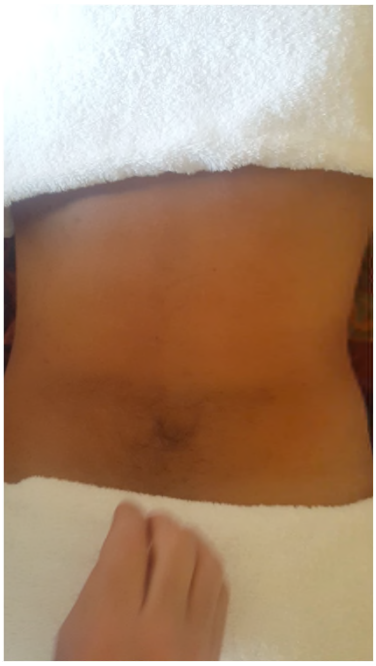


Figure 2 | Clinical features of neurogenic inflammation in fibromyalgia and complex regional pain syndrome. **a** | Dermatographia elicited after gentle stroking of skin in a patient with fibromyalgia. **b** | Reticular skin discolouration in forearm of patient with fibromyalgia. **c** | Redness, swelling and allodynia of the left foot and ankle in a patient who developed complex regional pain syndrome after undergoing surgery for a metatarsal bone fracture.

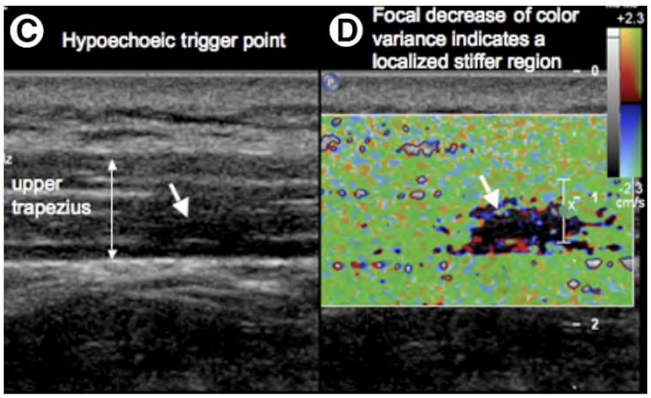
Pinch and Roll Technique

Pinch skin (without pressure on underlying tissues) then Roll the skin between fingers and thumb then move slightly up

Positive: increased pain + pressure (usual sensation)



B-mode and Elastography



US guidance for better localization!

Sikdar, Arch PMR 2009

Functional MRI of Patient with Knee OA

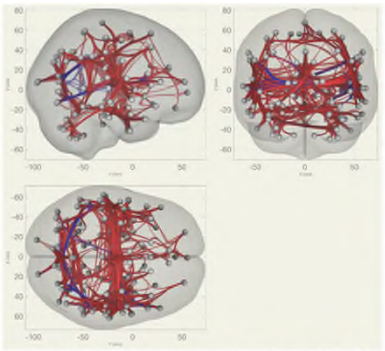


Figure 2. The ROI-to-ROI connectivity analysis of the single knee OA participant comparing resting baseline to the controlled pain modulation (CPM). This was a threshold free cluster enhancement (TFCE) analysis that included the Default Mode Network (DMN) and the SensoriMotor Network (SMN), along with 131 ROIs. Connections in red represent significantly increased connectivity at CPM compared to baseline, and connections in blue represent significantly decreased connectivity. Significance was p<0.05 p-family wise error corrected.

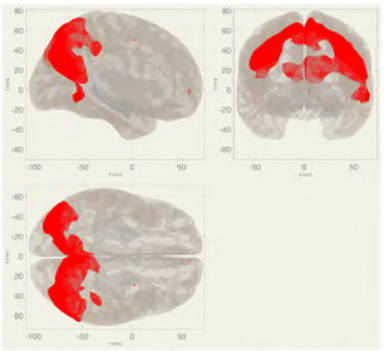


Figure 3. The seed-to-voxel analysis in CONN that compared baseline and CPM fMRI data in a person with localized knee pain and knee OA. This analysis found 5 clusters of significantly increased connectivity, with the major cluster containing 9699 voxels that spanned the superior occipital lobe and posterior parietal lobe. This analysis was a threshold free cluster enhancement (TFCE) analysis with 2000 simulations, and significance was p<0.05 p-family wise error corrected.

- THANKS VERY MUCH!
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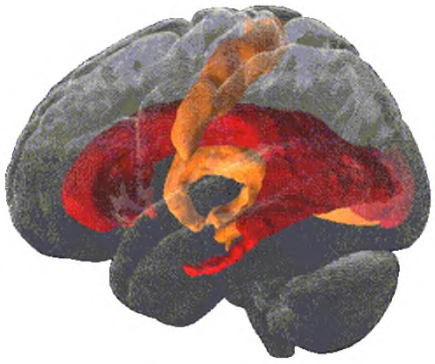
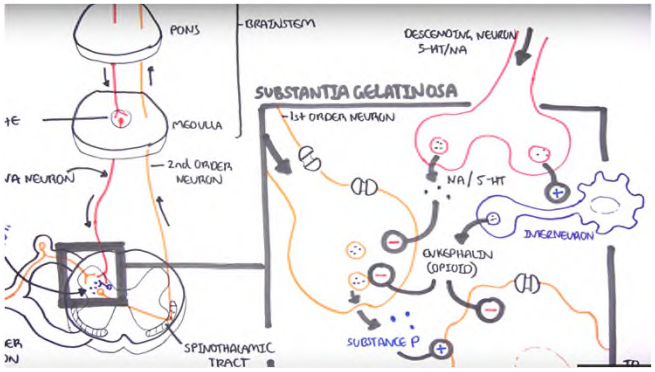




Diagnosing Nociplastic Pain

Use QST (PPT and temporal Summation)

Features of chief complaint and history:

1. Features of catastrophization
2. Central sensitization
3. Features of response to treatment
4. Past history attributes of importance

1. Features of catastrophization:

- Discordance b/t injury mechanism and complaints
- High level of analgesics for physiological injury mechanism
- Personality traits: over inclusive, judgmental of medical system,
- High healthcare utilization rate
- Failure to accept the explanation from competent MDs

2. Central sensitization:

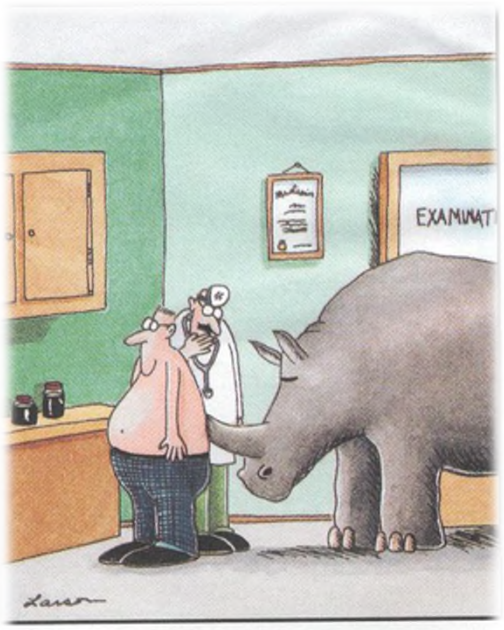
- Sensory complaints that are not within the dermatome or peripheral nerve territory
- Character of pain complaint: multiple descriptors used, high severity, long lasting, wind up phenomenon
- Spread of pain over time to parts of body that do not have any obvious anatomical or pathophysiological connection
- Provocation of pain by multiple nociceptive inputs: physical, emotional, cognitive

3. Features of response to treatment:

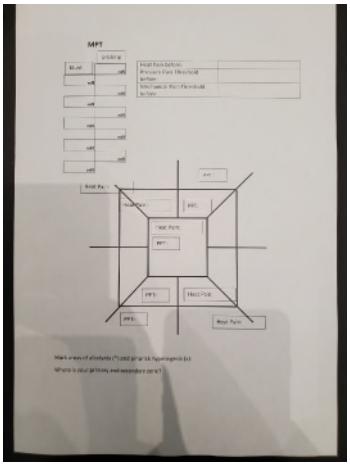
- Noncompliance with strategies that they do not believe in
- Loss of locus of control
- Failure to respond to the usual care-path; for example, “no” help with PT, chiropractic, medications
- Medical investigations thus far do not offer explanation for the pain complaint
- “I came to you doctor because you are the BEST”

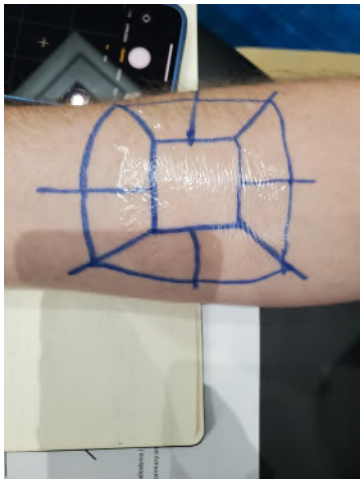
4. Past history attributes of importance:

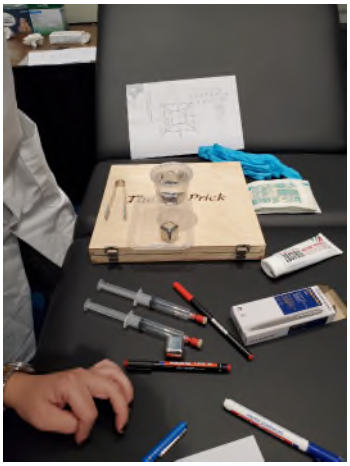
- History of taking a very long time to get back to work or sports after a simple MSK injury
- History of chronic pain complaint
- History of multiple investigations for a pain problem with no obvious explanation or solution
- History of emotional or physical abuse in the past
- Large analgesic intake for the medical pathophysiology
- Concomitant psycho-pathology: eg depression, anxiety



"Wait a minute here, Mr. Crumbley. ... Maybe it isn't kidney stones after all."






Prick