Fall 2022

A Systematic Review of Chronic Pain Mechanism Differential Assessment Strategies for Physical Therapy

Kate Hovde SPT
University of Puget Sound

Kenny Li DPT
University of Puget Sound

Kylie Wong BS
University of Puget Sound

Roger Allen PhD, PT
University of Puget Sound

A Systematic Review of Chronic Pain Mechanism Differential Assessment Strategies for Physical Therapy

Kate Hovde, SPT; Kenny Li, DPT ; Kylie Wong, BS; Roger Allen, PhD, PT
University of Puget Sound - Tacoma, WA, United States of America

Original Presentation
University of Puget Sound Research Symposium
Tacoma, WA
November 05, 2022

BACKGROUND

Our understanding of pain is rapidly evolving. The International Association for the Study of Pain (IASP) redefined a new pain mechanism category (PMC) as “nociplastic pain” in 2017, IASP redefined “pain” in 2020, and new pain education guidelines were published by the Academy of Orthopaedic Physical Therapy (AOPT) in 2021.[4, 7, 8] There are currently 3 PMCs defined by the IASP: nociceptive, neuroplastic, and nociplastic pain. Debate exists about the new “nociplastic pain” terminology in contrast to “centralized sensitization” (CS), which emerged in research in 2010 and is defined, for clinical purposes, as an amplification of neural signaling within the central nervous system that elicits pain hypersensitivity.[6] Within this debate is the question of can CS be a component of each PMC or is unique to nociplastic pain. In physical therapy, assessment and treatment strategies of chronic pain are highly variable. Current pain education in doctorate of physical therapy (DPT) programs is undefined by CAPTE standards and does not require PMC education.[8]

Pain Mechanism	Definition
Nociceptive	Pain that arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors.
Neuropathic	Pain caused by a lesion or disease of the somatosensory nervous system.
Nociplastic	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.

Table 1. IASP definitions of three pain mechanism categories (PMCs) for the generation, modulation, and/or maintenance of pain. [4, 7, 9, 10]

PURPOSE

Via qualitative systematic review, identify current clinical assessment strategies and tools for differentiating between nociceptive, neuropathic, and nociplastic chronic pain, feasible for implementation by physical therapists (PTs).

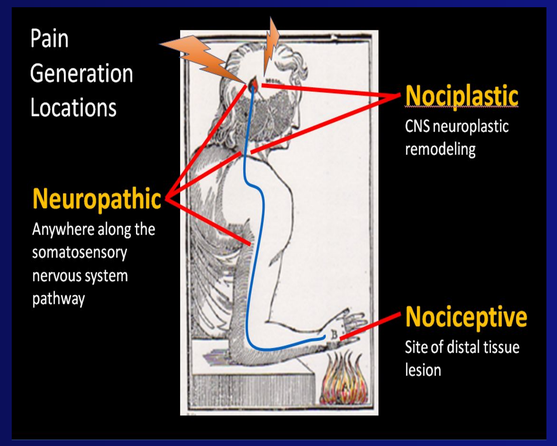

Figure 1. Pain generation and modulation locations for three Pain Mechanism Categories.

METHODS

Four investigators independently searched databases CINAHL, Cochrane Library, Google Scholar, PEDro, PubMed and SportDiscus from May-June 2020, for peer-reviewed studies pertaining to differentiating assessment methods of “nociceptive,” “neuropathic,” “central,” “mixed pain” and “central sensitization.” Upon researcher’s discovery of the term “nociplastic pain”, a follow-up search was conducted on this term for studies published between 2017-2022. Studies with undefined or exclusively psychogenic pain, not published in English, or that had a cost to access were excluded. Investigators evaluated abstracts independently using a priori criteria, voting to include 91 articles. A piloted form was used to extract the following data: pain terminology, conceptualization of pain mechanisms, assessment strategies for differential diagnosis of pain mechanisms, associated pathologies, and how the research informs physical therapy clinical practice. Extracted data demonstrated a meaningful qualitative understanding of the broad and varied nature of existing research on pain mechanism assessment.

RESULTS

Independent searches yielded 110 studies fitting inclusion criteria, with 72 selected for review. Secondary “nociplastic” search yielded additional 81 results with 19 fitting criteria (91 total studies reviewed).

PMC assessments found across the 91 studies were broadly separated into 4 strategies of:

- pain questionnaires
- Quantitative Sensory Testing (QST)
- imaging or laboratory tests
- clinical findings

Authors often concluded that a combination of strategies was necessary for valid and clinically meaningful findings.[1, 2, 3, 9, 10]

30% of the articles reviewed analyzed PMCs in relation to a specific pathology, with notable consensus in the literature identifying the limiting nature of purely pathology-based pain phenotyping. [2, 9, 10, 11]

Feasible assessment tools providing evidence for neuropathic generation of pain include the following [1, 2, 9, 10, 11]:

- Leeds Assessment of Neuropathic Symptoms and Signs (LANSS)
- PainDETECT (PD-Q)
- Douleur Neuropathique 4 Questions (DN4)
- Identification Pain Questionnaire (ID Pain)

However, the gold standard is imaging or laboratory tests confirming a lesion or disease.[1, 2] Several pain questionnaire assessments failed to differentially diagnose pain mechanisms by only assessing for one mechanism without ruling in or out others. [1, 2, 9, 10]

Notable agreements amongst nociplastic PMC studies include: [6, 10]

- diagnostic features may be shared among PMCs
- the three PMCs are not always independent
- PMCs may evolve into a blended continuum, characterized by increasing centralized sensitization, as chronic pain develops.

DISCUSSION

While research on chronic pain is extensive, there is diverging and contradicting terminology, conceptions, and opinions of pain mechanisms and their assessments. The IASP definition of exclusion for Nociplastic Pain exacerbates confusion on the subject. As PMC differential assessment tools are emerging, now is the time to converge diverse literature, history, and opinions to help clinicians across disciplines correctly assess and treat chronic pain. This starts with education and is promoted through shared communication and actions, such as appropriate International Classification of Disease (ICD) and billing coding.[11]

Pain Mechanism	Unique Features
Nociceptive	• Responsive to NSAIDS • Signs of inflammation • Pain resolution consistent with expected timeline of tissue healing
Neuropathic	• Pain distribution pattern neurologically consistent • Characteristic signs and symptoms (e.g. numbness) • Positive diagnostic test findings for nerve damage
Nociplastic	• Pain is poorly localized, diffuse, widespread • General hypersensitivity • Multiple somatic symptoms (i.e. depression, fatigue)

Table 2. Consensus of unique clinical distinguishing features of the three pain mechanism categories. [6, 10]

Physical therapist education on pain currently averages a total of 31 hours and, as it is not standardized, is variably based on the instructor’s conceptualizations.[8] Considering this point in history, pain education is evolving to be a fundamental subject rather than an elective in DPT education. Recommendations of how to achieve this in curriculum are provided in the AOPTA Pain Education Manual.[8.] PMC education is not specifically outlined in this manual, yet is fundamental to this subject and highlighted in example curriculums. If the research and medical community agree that each PMC requires vastly different treatment approaches, then correct differential assessment matters.

CONCLUSIONS

High quality research is emerging to inform the physical therapy clinician on differential assessment strategies for pain mechanisms in chronic pain patients. The ability for clinicians to assess and treat chronic pain is challenged by the current lack of continuity within PMC education, terminology, and difficulty of in vivo research on the subject.

References

1. Colloca L, Ludman T, Bouhassira D, et al. Neuropathic pain. Nat Rev Dis Primers 2017;3:17002.
2. Fillingim RB, Loeser JD, Baron R, Edwards RR. Assessment of Chronic Pain: Domains, Methods, and Mechanisms. J Pain. 2016;17(9 Suppl):T10-T20.
3. Gierthmühlen J, Schneider U, Seemann M, et al. Can self-reported pain characteristics and bedside test be used for the assessment of pain mechanisms? An analysis of results of neuropathic pain questionnaires and quantitative sensory testing. Pain. 2019;160(9):2093-2104.
4. International Association for the Study of Pain. Task force on taxonomy. IASP terminology updated from Part III: Pain terms, A current list with definitions and notes on usage. In” Classification of Chronic Pain. 2nd ed. Seattle IASP, 2017.
5. Kosek E, Cohen M, Baron R, Gebhart GF, Mico JA, Rice AS, Rief W, Sluka AK. Do we need a third mechanistic descriptor for chronic pain states? Pain 2016;157:1382–6.
6. Nijs J, Lahousse A, Kapreli E, et al. Nociplastic pain criteria or recognition of central sensitization? Pain phenotyping in the past, present and future. J Clin Med 2021;10:3203.
7. Raja SN, Carr DB, Cohen M, et al. The revised International Association for the Study of Pain definition of pain: concepts, challenges, and compromises. Pain 2020;161:1976–82.
8. Shepard M, et al. Pain Education Manual for Physical Therapist Professional Degree Programs. AOPT. 2021:1
9. Shraim MA, Masse´-Alarie H, Hall LM, Hodges PW. Systematic review and synthesis of mechanism-based classification systems for pain experienced in the musculoskeletal system. Clin J Pain 2020;36: 793–812.
10. Shraim MA, Sluka KA, Sterling M, et al. Features and methods to discriminate between mechanism-based categories of pain experienced in the musculoskeletal system: a Delphi expert consensus study. Pain. 2022;163:1-17.
11. Treede RD, Rief W, Barke A, et al. Chronic pain as a symptom or a disease: the IASP Classification of Chronic Pain for the International Classification of Diseases (ICD-11). Pain. 2019;160(1):19-27.

Contact Information
Kate Hovde, SPT
kmerriam@pugetsound.edu