Pain Mechanism Classification Clinical Pearl
Part 1

Pain, acute or chronic, is a leading reason patients seek physical therapy care.[1] How do we as skilled practitioners and specialists in treating pain navigate the complexity that is our patient's lived experience with persistent pain? How do we effectively determine the factors that influence their pain and how does that information subsequently guide their treatment? The goals of this outline are to present a practical approach to the application of the current evidence in pain mechanism classification, how it can influence our clinical decision making, and ultimately guide treatment.

The first part of this series will look to simplify the evidence explaining what pain mechanism classification is and how to apply it. Recognizing pain mechanisms as an important component of diagnosis is not novel, as it is common in pharmaceutical management, however it has not been widely implemented in physical therapy practice.[1] There is a large diversity of opinions on how certain components of pain can be categorized and the need that continues to exist for continued research and validation. I am simply sharing my experience in utilizing this framework in my clinical practice, in the hopes that it may help to better guide and refine your clinical decision making.

The International Association for the Study of Pain (IASP) suggests three main pain mechanism classifications including: nociceptive, neuropathic, and nociplastic. Shraim et al[2] does a nice job in synthesizing the three main categories succinctly. Nociceptive pain can be categorized as pain that is primarily triggered by peripheral nociceptive input from at the level of the tissue. Pain that is a response to peripheral noxious stimuli, involves damage to non-neural tissue, is provoked by movements and postures, and is localized. This category can be teased out even further to include nociceptive mechanical pain, nociceptive ischemic pain, and nociceptive inflammatory pain. I like to think of this as simply "issues with the tissues," for example an acute ankle sprain or a case of shoulder impingement.

Next, Shraim et al[2], outlines neuropathic pain that can be further subdivided into peripheral and central neuropathic pain. Peripheral neuropathic pain can be classified as pain that is associated with a history of or evidence of damage, lesion, or disease to

the peripheral nervous system. Peripheral neuropathic pain is thought to be aggravated by movement and activity that loads the peripheral neural tissue, for example carpal tunnel syndrome or lumbar radiculopathy. Central neuropathic pain is considered to involve damage, lesion, or disease to the central nervous system (CNS).

The third category, nociplastic pain involves pain maintained by altered nociceptive processing and represents a dysfunction of the central nervous system. Previously known as central sensitization, nociplastic pain is a "state of amplified or increased excitability or neural signaling within the CNS in response to normal or subthreshold afferent input."[2] Autonomic pain, also known as sympathetic pain, commonly seen in conditions such as CRPS type 1 and 2 is a more disputed category. Some authors recommend it as a subtype of central neuropathic pain, while others argue it can fall under nociplastic pain.[2]

Since nocioplastic pain is defined as abnormal processing not due to a disease state, psychological factors usually play a role in nociplastic pain. Psychological factors, such as anxiety, kinesiophobia, pain catastrophizing, and depression should be considered as they are a part of the biopsychosocial model of pain. Stress and emotions should not be ignored, as they can continue to drive pain well after the initial injury.

Chimenti et al[1] states that evaluation of pain mechanisms can help individualize care to a patient rather than a diagnosis, and is a step toward providing precision medicine to patients with pain. When faced with a complex patient with an extensive medical history and multiple persisting pain reports, I think it is helpful to first ask the question of whether this patient is showing signs and symptoms of nociplastic pain. Kolski and O'Connor[3], state that early identification of patients with central mechanisms can facilitate realistic expectations for outcomes, appropriate allocation of resources, and where appropriate, redirection to other services such as psychology. I find a screen of yellow flags, presence of hypersensitivity and/or allodynia, fear avoidance behaviors, and comorbidities such as fatigue and sleep disturbances can help rule in or rule out if components of my patient's pain are centrally dominated. A clinical outcome measure that can be useful for this includes the Tampa Scale of Kinesiophobia.

Applying the mechanism-based approach during an evaluation and subsequent follow ups requires clinical reasoning skills that allows the clinician to differentiate the clinical signs and symptoms of the nociceptive, peripheral neurogenic, and central categories.[3] The limitation here, unfortunately, is identification of these mechanisms are not directly measurable, but must be inferred from indirect assessment.[3] A thorough subjective and movement exam is a tool already in our scope of practice that can assist with this.

It is also important to acknowledge that a patient can and likely will experience a combination of multiple pain mechanisms. I have found in clinical practice, focusing initial interventions on the dominating nociplastic mechanism first is a good place to start. For example, if a patient is experiencing a combination of peripheral neurogenic pain from a lumbar radiculopathy AND nociplastic pain resulting in hypersensitivity to movement and sleep disturbances; treatment would best be focused on interventions that help with downregulation first. Once a patient's sensitivity begins to reduce, the mechanical interventions in our PT arsenal to treat the radiculopathy can then be effectively utilized.

Overall, when faced with the complexities of patients with persistent pain, applying the pain mechanism classification framework helps to:

- implement the biopsychosocial model of care
- assist with clinical decision making
- guide individualized treatment.

Look out for part two of this clinical pearls series that will focus primarily on the assessment and treatment of the nociceptive pain mechanism.

This Clinical Pearl was provided by Mary Zalinger PT, DPT. Mary is a full time clinician in the Pain Management Center at the Shirley Ryan AbilityLab in Chicago, IL. Her clinical practice is focused on improving quality of life and functional outcomes for individuals living with persistent pain.

References

1. Chimenti RL, Frey-Law LA, Sluka KA. A mechanism-based approach to physical therapist management of pain. *Physical Therapy*. 2018;98(5):302-314. doi:10.1093/ptj/pzy030
2. Shraim MA, Massé-Alarie H, Hall LM, Hodges PW. Systematic review and synthesis of mechanism-based classification systems for pain experienced in the musculoskeletal system. *The Clinical Journal of Pain*. 2020;36(10):793-812. doi:10.1097/ajp.0000000000000860
3. Kolski MC, O'Connor A, Van Der Laan K, Lee J, Kozlowski AJ, Deutsch A. Validation of a pain mechanism classification system (PMCS) in Physical therapy practice. *Journal of Manual & Manipulative Therapy*. 2016;24(4):192-199. doi:10.1179/2042618614y.0000000090