ISSN 3007-2433 (Print), 3007-2441 (Online)

EDITORIAL

Chronic Pain- Is It All in the Head

Victor Mendis

DOI:https://doi.org/10.62848/bjpain.v2i1.3449

Chronic Pain is a distressing experience with sensory, emotional, social and cognitive components and affects millions of people globally with a huge impact on the economy. It is estimated that one in five adults in the world suffers from moderate to severe chronic pain which causes distress not only to the person concerned but also to the family and has a huge impact on the economy[1]. Pain has traditionally been classified as nociceptive (from tissue injury) and neuropathic (from nerve injury) but more recently a third mechanistic descriptor has been identified and defined as nociplastic pain (from a sensitised nervous system). Nociplastic pain, therefore, is stimulus-independent and does not require inflammation or structural neuronal damage[2]. Understanding the mechanisms of pain is important and affects work-up and treatment decisions at every level. In practice however there is considerable overlap in the different types of pain mechanisms within and between patients, so many experts consider pain classification as a continuum, and this has to be considered when assessing and formulating a treatment plan.

Central sensitisation is one of the mechanisms that explain chronic persistent pain and represents an enhancement in the function of neurons and circuits in pain pathways and is a manifestation of the remarkable plasticity of the nervous system in response to soft tissue injury, inflammation, and neural injury[3,4]. Because central sensitisation results from changes in the properties of the neurons in the nervous system, the pain may be no longer coupled with (as in acute pain) the presence, intensity, or duration of ongoing noxious peripheral stimuli. Instead, central sensitisation produces pain hypersensitivity by changing the sensory response elicited by normal inputs, including those that evoke innocuous sensations long after the cessation of an injury[5]. Chronic persistent pain refers to pain that continues after an acute injury heals or after passing a period that should allow for healing. For currently unknown reasons, the nerve fibres continue to fire as if there is damage that needs attention and this continuous signal travels up the spinal column to the brain. As a result, the transmission circuits become more efficient at transmitting these signals—like a one-lane road becoming a four-lane highway. The continuous input into these circuits causes more transmission, leading to a chronic persistent pain state. At the same time, the number and array of pain-causing neurotransmitters (particularly excitatory neurotransmitters) in the nervous system increase[6]. Over time, the threshold for the pain receptors to fire is lowered, and a less intense stimulus is needed to cause the nerve to respond. What started out as a nociceptive stimulus from the site of an injury to the brain has become a self-contained feedback loop within the nervous system—a disease of the brain. Neuroplasticity, therefore, provides us with a brain that can adapt not only to

Consultant
Pain Medicine
Mid and South Essex University Hospitals, United Kingdom

Correspondence
Victor Mendis
victor.mendis@meht.nhs.uk

changes inflicted by damage but allows adaptation to any and all experiences that we may encounter[5,6].

A Pain as defined by the International Association for the study of Pain is: "An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage,"[7] and is expanded upon by the addition of six key notes and the etymology of the word pain for further valuable context:

- 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 wellbeing
- Verbal description is only one of several behaviours to express pain; the inability to communicate does not negate the possibility that a human or a nonhuman animal experiences pain

Chronic primary pain defined as per the ICD-11 classification, is pain in one or more anatomic regions that persists or recurs for longer than 3 months and is associated with significant emotional distress or significant functional disability (interference with activities of daily life and participation in social roles) and that cannot be better explained by another chronic pain condition[7]. This is a new phenomenological definition, created because the aetiology is unknown for many forms of chronic pain. The term "primary pain" was chosen in close liaison with the ICD-11 revision committee, which felt this was the most widely accepted term, in particular, from a non-specialist perspective. It is assumed that chronic primary pain has no clear underlying condition and that the mechanisms underlying it are only partially understood. This does not mean that this is a single entity, and more research is needed to gain a better understanding of the underlying mechanisms. They are also likely to be influenced by the unique biopsychosocial makeup of the patient. The ICD-11 classification allows for subdivisions of chronic primary pain when possible, into broad categories (e.g. chronic primary visceral pain, chronic primary low back or musculoskeletal) thus emphasising that this is not a single entity but a classification that provides recognition of the pain despite the underlying mechanisms being only partially understood. The mechanisms underlying chronic pain differ from those underlying acute pain. As pain persists, central nervous system factors play a more prominent role and therefore chronic pain states are understood to be centrally driven. These centrally driven pain conditions are characterised by widespread pain, non-restorative sleep, fatigue, memory problems and mood disorders[7,8].

In these circumstances, pain is no longer protective. The pain in these situations arises spontaneously, can be elicited by normally innocuous stimuli, is exaggerated and prolonged in response to noxious stimuli, and spreads beyond the site of injury. Central sensitization has provided a mechanistic explanation for many of the temporal, spatial, and threshold changes in pain sensibility in acute and chronic clinical pain settings and has highlighted the fundamental contribution of changes in the central nervous system to the generation of abnormal pain sensitivity. Features of central sensitisation have been identified in many chronic pain disorders such as fibromyalgia, chronic whiplash- associated disorders, temporomandibular dysfunction, irritable bowel syndrome and low back pain. Patients presenting with symptoms of central sensitisation typically show higher pain ratings, and greater disability, are more likely to develop future musculoskeletal conditions and show poorer outcomes and increased likelihood of developing chronic persistent pain[9,10,11].

Chronic pain is complex, that can be outside of awareness and control and it is important to understand that nociception is neither sufficient nor necessary for pain and that neural networks that produce pain become more sensitive when pain persists. Many clinicians, healthcare professionals and patients lack an understanding of how complex chronic pain is, and some rely on medications alone, and others on the use of costly interventions, despite limited evidence and this may be spurred by aggressive industry and marketing. Proper access to multidisciplinary services within an established pain

clinic is vital to offer patients a multidisciplinary and multimodal approach to managing chronic persistent pain which can be defined as a complex disorder of the nervous system.

References

1 Cohen SP, Vase L, Hooten W M. Chronic pain: an update on burden, best practices, and new advances. Lancet. 2021 May 29;397(10289):2082-2097. doi: 10.1016 /S01 40-6736(21)00393-7. PMID: 34062143.

2 Latremoliere A, Woolf CJ. Central sensitization: a generator of pain hypersensitivity by central neural plasticity. J Pain. 2009 Sep;10(9): 895-926. doi: 10.1016/j.jpain.2009.06.012. PMID: 19712899; PMCID: PMC2750819.

3 Phillips K, Clauw D J. Central pain mechanisms in chronic pain states--maybe it is all in their head. Best Pract Res Clin Rheumatol. 2011 Apr;25(2):141-54. doi: 10.1016/j.berh.2011.02.005. PMID: 22094191; PMCID: PMC3220875.

4 Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. Pain 2011;152:S2-S15

5 Pusch D. Central Sensitization: A generator of Pain Hypersensitivity by Central Neural Plasticity. Jour of molecular pathophysiology 2021, vol 10, No 6

6 Rethinking chronic pain. Lancet. 2021 May 29;397(10289):2023. doi: 10.1016/S0140-6736 (21)01194-6. PMID: 34062132.

7 IASP, publication July 2020: IASP announces revised definition of pain

8 Detlef R, Rief W, Barke A, Aziz Q et al. A classification of chronic pain for ICD-11.Pain 2015 Jun;156(6) :1003-1007

9 Smart K M, Blake C, Staines A et al. Mechanism- based classifications of musculoskeletal pain: part 1 of 3: symptoms and signs of central sensitisation in patients with low back pain(leg pain). Man Ther 2012;17:336-44

10 Van Oosterwijck J, Nijs J, Meeus M et al. Evidence for central sensitisation in chronic whiplash: a systemic literature review. Eur J Pain 2013; 17:299-312

11 Fingleton C, Smart K, Moloney N, et al Pain sensitization in people with knee osteoarthritis: a systemic review and meta-analysis. Osteoarthritis Cartilage 2015; 23:1043-56