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# Research Paper Review

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## Clinical Model for Pattern Recognition in Pain Assessment

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## ABSTRACT

***INTRODUCTION:*** *Common, enigmatic musculoskeletal conditions such as whiplash-associated disorder, myofascial pain syndrome, low back pain, headache, fibromyalgia, osteoarthritis, and rotator cuff pathology, account for significant social, economic, and personal burdens on a global scale. Despite their primacy (and shared sequelae) there remains a paucity of available and effective management options for patients with both acute and chronic conditions. Establishing an accurate prognostic or diagnostic profile on a patient-by-patient basis can challenge the insight of both novice and expert clinicians. Questions remain on how and when to choose the right tool(s), at the right time(s), for the right patient(s), for the right problem(s). The aim of this paper is to introduce a new clinical reasoning framework that is simple in presentation but allows interpretation of complex clinical patterns, and is adaptable across patient populations with acute or chronic, traumatic or non-traumatic pain. The concepts of clinical phenotyping (e.g. identifying observable characteristics of an individual resulting from the interaction of his/her genotype and their environment) and triangulation serve as the foundation for this framework. Based on our own clinical and research programs, we present these concepts using two patient cases; a) whiplash-associated disorder (WAD) following a motor vehicle collision and b) mechanical low back pain.*

***METHODS****: This was a clinical commentary. The authors did not perform a statistical analysis nor describe their literature search strategy.*

***DISCUSSION****: These authors published this clinical and educational tool to enable clinicians to sub-categorize pain patients into dominant pain etiologies. The point is to guide clinicians as to which biopsychosocial variables are involved in their patients' pain experience, and to guide treatment based on pain etiology. The point is not to specifically use this methodology to diagnose patients, but to identify specific pain drivers, which dictates treatment.*

# ANALYSIS

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## Background Information

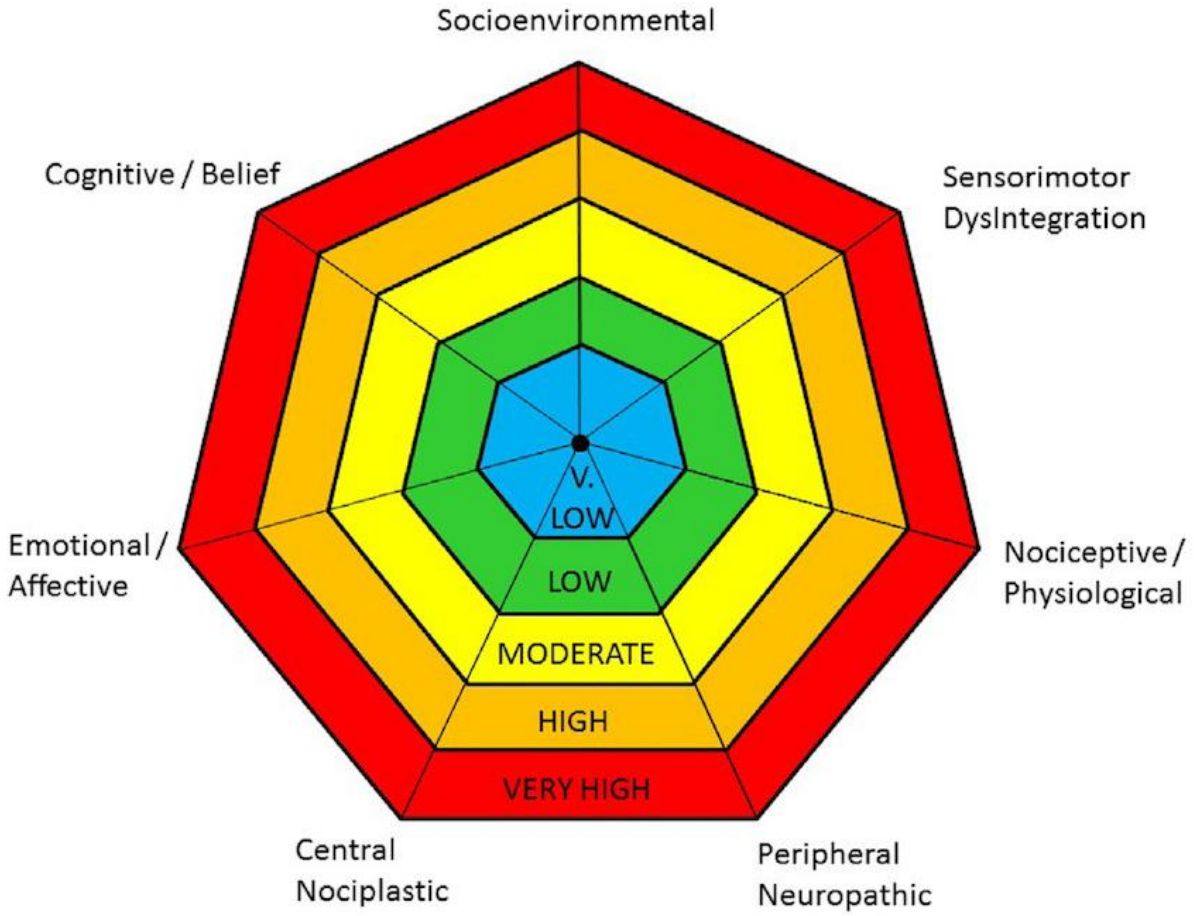
Personalized pain management is an emerging movement in clinical practice due to the inherently personal nature of the pain experience. Arguably, the most logical approach to pain management should be to: 1) implement a clinically rigorous and personalized multidimensional assessment; 2) identify the individual biopsychosocial sequelae that may drive the pain experience; and 3) structure a treatment program based on the results of the assessment. At the centre of this approach are the proverbial 3-pillars of evidence-informed practice, which are: *sound empirical evidence, clinician expertise and patient values (1).*

Pain management research is beginning to focus on identifying subgroups of pain patients, with the intention of providing more sound and comprehensive guidelines for clinical decision making. The authors of this paper proposed a framework to sub-categorize pain patients based on dominant pain etiology, based on their combined 30-years of experience in clinical practice, teaching, mentorship, and MSK research.

## Summary:

Subgrouping patients has enabled clinicians and researchers to estimate risk of chronicity (2, 3), predict response to treatment (4, 5), and identify specific subgroups of pain mechanisms (6, 7).

These authors advocate the use of a radar plot displaying 7 domains as potential pain drivers (see below). While not exhaustive, the 7 points represent different domains of a patient's pain experience, which may improve the practitioner's ability to make clinical decisions (8).




This proposed framework is not meant to be diagnostic in nature. Rather, it is presented as a complementary tool to identify the magnitude of the primary driver of one's pain experience, without labelling the condition. The 7 sub-categories have been associated with the qualitative or qualitative experience of pain. The 7 classifications are:

*1) Nociceptive (physiological) input:* Defined as pain produced primarily through noxious chemical/thermal/mechanical input to the CNS from peripheral nociceptors. Typically, patients' complaints are well localized, and proportionate to the mechanism of injury. Patients' responses to standardized self-report evaluations (such as those for depression, anxiety, etc.) typically do not support the presence of other potential pain drivers. The clinical evaluation of these patients is consistent and predictable. These patients are also responsive to routine pharmacotherapy and/or rehabilitation. Examples include myofascial pain, mechanical low back pain, mechanical neck pain, tendinopathies, sprain/strain etc.

*2) Peripheral Neuropathy:* Defined as pain caused by a lesion or disease of the peripheral nervous system. Patients typically describe spontaneous 'ectopic' (paroxysmal) pain, allodynia, hyperalgesia, numbness or parasthesiae. Self-report evaluations such as the Leeds Assessment of Neuropathic Signs and Symptoms (LANSS), PainDETECT Questionnaire or DN4 (Doleur Neuropathique 4) are typically positive. The clinical evaluation may demonstrate signs of pain, or impaired neural transmission (i.e. sensory alterations within

the distribution of a peripheral nerve or dermatome that makes logical sense in relation to the history). These patients are typically unresponsive to NSAIDs, but may be responsive to tricyclic antidepressants (TCAs – amitriptyline, nortriptyline, doxepin, etc.), SSRIs/SNRIs (Escitalopram/Cipralex, Bupropion/Wellbutrin, Cymbalta/Duloxetine, Effexor/Venlafaxine), and gabapeninoids/anti-convulsants (pregabalin/Lyrica, gabapentin/Neurontin, etc.). Examples include radiculopathy, mononeuropathies, multiple sclerosis, diabetic neuropathy, etc.

*3) Central Nociplastic Change (9):* Defined as pain that can be traced to either a central facilitation of action potentials (amplification or disinhibition) from the periphery, or ectopic impulses generated within the CNS with no direct input from the periphery *(translation: pain emanating from functional neuroplasticity of the CNS, as opposed to pain as a response to nociceptive or neuropathic inputs)*. This definition is analogous to the International Association for the Study of Pain (IASP) definition of *central sensitization*. However, the term central nociplastic change is preferred by many groups (especially recently) as an alternative to the more ambiguous term of 'sensitization'. The implication of using the term proposed above is to imply that such neuroplastic mechanisms are potentially reversable, or plastic. *(NOTE: Interestingly, the IASP definition for 'nociplastic pain' is: '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 nervous system causing the pain'. This essentially means that the etiology of pain is centrally driven, and not from nociceptive or neuropathic sources)*.

In these cases, patients have great difficulty drawing connections between the mechanism of injury and current complaints. They also complain of resting local or widespread pains that may be related to the patient's mood or emotional status. A score of > 40 points on the the Central Sensitivity Index (CSI) can be used to strengthen a suspicion of central sensitization/nociplastic pain. The clinical evaluation may reveal non-mechanical and non-predictable patterns of pain and may present with or without impairment in descending pain modulation. These patients are typically unresponsive to routine front-line therapies, but may be responsive to opioids, TCA's and/or SSRI's. Examples include fibromyalgia, irritable bowel syndrome, and in some cases chronic low back pain and chronic neck pain.

*4) Emotional Dysregulation:* Defined as diagnosable psychopathology or affective dysregulation, as described by the DSM-5. These may include depression, anxiety or any other mood/personality disorders. While the causal mechanism connecting mood and pain are unclear, it is apparent that pain and mood are bidirectionally related. These patients have a history of psychopathology, which may be temporally consistent with symptom onset. The mood-related symptoms are consistent with DSM-5 criteria. Examples include PTSD, major depressive disorder, illness anxiety disorder, adjustment disorder, somatic symptom disorder, etc. The presence and severity are confirmed using validated tools such as the PHQ-9, GAD-7, post-traumatic distress scale checklist, etc. Typically, the patient's pain is not consistent with predictable mechanical patterns. Front-line pharmacotherapies are not often useful. Medications such as TCAs, SSRI/SNRI's may have some analgesic or mood effects.

*5) Maladaptive Cognitions:* Defined as *irrational or inaccurate* beliefs, thoughts or behaviours about, or resulting from, the experience of pain. These patients have an exaggerated negative orientation towards pain. While maladaptive cognitions may be a precursor for specific psychopathology, the patients do not fulfill any specific DSM-5 criteria. A rehabilitation professional may be well-positioned to address maladaptive beliefs or cognitions about pain, while psychopathology should be the domain of a mental health professional. These patients typically do not demonstrate a definitive pattern of pain. Their pain can be acute, chronic, traumatic or non-traumatic. Patients often report that hurt is proportional to harm, and that they must be 100% recovered prior to returning to activity. These patients typically prefer passive coping methods and demonstrate 'all-or-none thinking'. Examples include kinesiophobia, catastrophization and low pain self-efficacy. Self-report measures such as the Pain Catastrophizing Scale, Tampa Scale of Kinesiophobia, Fear Avoidance Beliefs Questionnaire or Pain Self-Efficacy Questionnaire can be used to identify and quantify the severity of maladaptive cognitions.

*6) Socioenvironmental Context:* This category includes the wide-ranging contextual factors that affect the pain experience, such as access to appropriate care, willingness to report pain-related cultural beliefs, language, socially-constructed gender roles, environmental demands, and psychoemotional stressors. These patients may be more likely to present when pathologies have occurred within, or in association with, compensation, medicolegal claims, tort, or in association with other stressors. Self-report evaluative tools such as the Spousal Response Inventory, or Injustice Experience Questionnaire (which has been validated in medicolegal, workplace and MVA related injury contexts) can confirm clinical suspicion. The patients may display signs of intentional exaggeration and may have been counselled to avoid 'straining' until after a case is completed.

*7) Sensorimotor Dys-integration:* Defined as discordance between the perceived self and the actual self, or as a problem of interoception. The driver of pain is a mismatch between two or more sensory inputs into the CNS, such as optical input or cervical proprioceptive input. This pattern of pain is more likely to manifest in chronic pain patients. Patients will often describe the body region as if it is detached from their physical self *(NOTE: in chronic pain settings, we typically ask the patient if their injured area feels as if it belongs to them)*. Patients may also struggle to identify painful areas on a body diagram. On examination, clinicians may find signs of somatosensory reorganization (i.e. 2-point discrimination, left/right discrimination, joint position sense error). Examples include Complex Regional Pain Syndrome/Reflex Sympathetic Dystrophy, chronic low back pain, chronic neck pain, post-stroke syndrome, phantom limb pain etc. CNS disorders should be ruled out in this population. The exact neurophysiological etiology has not been fully elucidated.

It should be explicitly stated that this framework is meant to be applied after excluding red flags and other systemic comorbidities. Each patient is graded in terms of how well they fit into these categories, using qualitative ranges of: *very low, low, moderate, high and very high.*

Patients are slotted into the various classifications via the concept of *triangulation.* Put

simply, while one source of information can provide a very broad sense of position, two sources narrows the possible position to a more specific region, while three sources all pointing in the same direction and offering similar conclusions leave only one possible action. Using this concept, triangulation can be used to estimate the magnitude of contribution from each domain. Clinicians are required to utilize at least 3 information sources (i.e. subjective history, patient narrative, questionnaires, clinical evaluation and signs, and other observations) before concluding where the patient fits into any specific category. *NOTE: While these categories are clearly outlined in this article, patients will often embody features of multiple domains at the same time. The trick is to identify which classifications/domains are MOST important to address at the present time for the patient in front of you.*

The authors' experience suggests that this method resonates with students and novice, or mid-career clinicians across professional disciplines. This methodology also functions well as a teaching tool. However, experienced clinicians may believe this classification system to be too reductionistic.

## CLINICAL APPLICATION & CONCLUSIONS

These authors published this clinical and educational tool to enable clinicians to sub-categorize pain patients into dominant pain etiologies. The point is to guide clinicians as to which biopsychosocial variables are involved in their patients' pain experience, and to guide treatment based on pain etiology.

The point is not to specifically use this methodology to diagnose patients, but to identify specific pain drivers, which dictates treatment. For instance, a patient with chronic low back pain demonstrating little to no pain at rest, difficulty with transitioning, flexion intolerance and extension tolerance, with reactive depression may score *very high, and high* in the nociceptive (physiological) and emotional dysregulation domains, and *low and very low* in the sensorimotor dys-integration and nociplastic domains. However, another patient with chronic low back pain with similar superficial complaints, but demonstrating poor joint position sense/proprioception (i.e. inability to smoothly perform spine/pelvis dissociation movements like cat-camel), constant high pain ratings, high levels of disability, unpredictable patterns of pain, poor 2-point discrimination sense and lack of response to all pharmacological/conservative treatment modalities may score *high and very high* in the sensorimotor dys-integration and central nociplastic domains, and *low and very low* in nociceptive and neuropathic domains. In these cases, the treatments for each patient may be different. Patient 1 may require biomechanical education, optimization of movement quality (i.e. temporary flexion avoidance), positions of relief/McKenzie therapies into extension, core endurance training and CBT for depression. On the other hand, Patient 2 may require alternative treatments such as pain neuroscience education, 2-point discrimination training, sensory localization/discrimination, and graded motor imagery prior to introducing more classical rehabilitation techniques. Simply put, these patients present very similar pain qualities superficially, yet require very different treatments. The

concept of *triangulation* (using at least 3 data points) enables clinicians to classify the patient in to the *most dominant pain classification(s)*, which dictates future management. More complicated patients can be re-evaluated after several treatments to determine if other categories are now dominant and warrant different treatment.

*NOTE: I personally have seen several patients who initially present like Patient 2 above, whom after several treatments begin to present much like Patient 1. In these cases, the treatment methodologies and rehabilitation strategies change to suit the patient's needs at that time.*

## STUDY METHODS

This was a clinical commentary. The authors did not perform a statistical analysis nor describe their literature search strategy.

## STUDY STRENGTHS/WEAKNESSES

Some research groups have shown preliminary evidence that prognosis-based subgrouping of patients with acute LBP may lead to improved outcomes and treatment efficacy (10). However, some clinicians have criticized such approaches, reminding us that making decisions based on a single tool or algorithm oversimplifies the complex patterns and interactions associated with the personal experience of pain. Unfortunately, proponents of the sub-classification methodology have not been able to reconcile the clinical reality that few patients fit within distinct, homogenous 'boxes' (so to speak).

Sub-grouping pain patients into a dominant pain etiology can be a very useful tool for novice clinicians. However, expert clinicians tend to rely less on structured rules and procedures, and more so on past experiences, intuition and heuristics (i.e. problem solving and discovery). Expert level clinicians are also known to embody greater pattern recognition skills and are more comfortable with ambiguity.

Many of these domains interact, and as such, patients may fall into multiple categories simultaneously. The concept of triangulation enables clinicians to identify which 2 or 3 classifications are dominant in any specific case, to enable treatment. Of course, access to other healthcare providers specializing in CBT, sensorimotor reorganization training, mindfulness meditation, etc. may be limited in smaller communities.

Clinicians familiar with treating chronic pain understand that bidirectional associations exist between one's mood/social status/cognitions and the pain experience. There is one unfortunate, but large and important, flaw in this methodology: while the severity of various clinical entities have been identified (i.e. mild/moderate/severe depression, anxiety, joint position sense error, scores on questionnaires etc.), the *strength* of the association with a specific individual's pain experience remains to be elucidated. For instance, you may have

a chronic low back pain patient with large nociceptive and emotional dysregulation qualities, whose nociceptive pains respond well to conservative and rehabilitation treatments, while remaining severely depressed, despite pain alleviation. With this being said, the described methodology serves as a treatment guideline and teaching/learning tool, rather than a solid framework. It does not work with complete certainty in EVERY patient, yet may enable clinicians to identify previously unseen variables that are correlated with chronicity, and direct clinicians towards the most appropriate treatment for their individual patient at that time.

It should also be noted that the lead author is an Associate Editor for the journal the article was published in, which introduces the possibility of publication bias!

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