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A new clinical model for facilitating the development of pattern recognition skills in clinical pain assessment☆

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ABSTRACT

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.

1. Introduction

Personalized pain management is gaining momentum as a sound approach to clinical practice (Woolf, 2004). This movement is emerging from some recognizable shortcomings of rote application of evidence from clinical trials to all patients in a 'one size fits all' style (Rothwell, 2005). Randomized clinical trials (RCTs) of rehabilitation interventions have habitually struggled to adequately mimic the bespoke approach to care delivered by clinicians. Reasons for this are wide and varied, but the multifactorial and highly personal nature of the pain experience contributes to the challenges of adequate design and interpretation, of traditional RCTs. Treating every patient only as supported by evidence drawn from comparisons of group means risks under- or over-treatment of the individual person. Arguably, a more logical and achievable approach would be to 1) implement a clinically rigorous yet feasible and personalized multidimensional assessment, 2) identify multisystem patterns in the patient profile that may be driving the pain experience and 3) intervene in a targeted fashion based on the results of that assessment. At the heart of such an approach are the pillars of evidence-based practice - *sound empirical evidence, clinician experience, and patient values* (Sackett et al., 1996).

Recent years have seen an increasing focus on identifying subgroups of patients with painful musculoskeletal conditions such as neck and low back pain, intended to provide more guidance for clinical decision making. Subgroups have been described to estimate risk of chronicity (Ritchie et al., 2013; Hill et al., 2008), response to treatment (Fritz and Brennan, 2007; O'sullivan, 2005), and specific pain mechanisms (Freynhagen et al., 2006; Arendt-Nielsen and Yarnitsky, 2009; Nordin et al., 2008). In some cases this approach has shown promise; Hill and colleagues have provided preliminary evidence that prognosis-based subgrouping of patients with acute low back pain may lead to improved outcomes and treatment efficiency (Hill et al., 2011). Few other pain

☆ The model being proposed has been used to educate participants on for-profit continuing professional development courses, but the authors hold no exclusive copyright over the use of a radar plot or the concept of triangulation.

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conditions enjoy the same evidentiary base (Michaleff et al., 2014; Jull et al., 2013), and most expert clinicians would agree that basing treatment decisions on a single tool or algorithm over-simplifies the complex patterns and interactions associated with the personal experience of pain. Proponents of sub-classification have also yet to reconcile the clinical reality that few patients fit neatly within distinct homogenous 'boxes'. (Kamper et al., 2010; Sun et al., 2010; Hancock et al., 2009).

Subgrouping can be a useful tool for novice clinicians, but clinical expertise appears to be associated with a move beyond such algorithmic approaches. Benner has proposed theories on the development of clinical experts as a process through which providers rely less on structured rules and procedures, and more on past experiences, intuition, and heuristics (Benner, 1984). A recognized indicator of transition from novice to expert clinician is the growing internal reference 'archive' for recognizing patterns of clinical presentation and increased comfort with ambiguity, leading to treatment decisions that can be made without a distinct patient classification (Jones, 1992). We are choosing 'pattern recognition' to describe this competence, defining it as the perception and integration of information from multiple sources to arrive at recognizable patterns. It would seem a reasonable pursuit to develop an academic model for facilitating the development of pattern recognition skills in students and early-career clinicians to accelerate their transition to expert-level practitioners. As the field of musculoskeletal pain continues to grow in exciting scientific directions it also grows in complexity, rendering the utility of a multidimensional clinical reasoning framework even more valuable. This professional issue presents a new framework that is simple in presentation but facilitates interpretation of complex clinical patterns, and appears to be adaptable across patient populations and professional disciplines. The outcomes of such an approach should result in the exploration and development of more effective targeted interventions on a patient-by-patient basis while minimizing the likelihood that clinicians are paralyzed by too much information.

The authors are leveraging their own experiences in clinical practice (combined > 30 years), pre- and post-professional teaching, mentorship, and basic and clinical research in the field of neuromusculoskeletal pain and trauma to propose this framework. Two illustrative case examples using a patient with whiplash-associated disorder and another with mechanical low back pain are included as appendices.

1.1. The radar plot and triangulation

The radar plot and associated concept of triangulation are being presented as emerging concepts rather than empirically derived formulae. On the contrary, the value of characterizing pain in this way is to endorse a move *away* from formulaic or algorithmic approaches that may carry unintended consequences of reducing the clinical decision making skills of practitioners (Cabitza et al., 2017). A *suggested* radar plot displaying 7 domains as potential 'pain drivers' is shown in Fig. 1. While not exhaustive, the seven points represent different domains of a patient's pain experience, offering potentially greater granularity for clinical decision-making than do contemporary models of pain (Melzack, 1999; Gifford, 1998). This framework is not meant to be diagnostic in nature, rather we present it as a complimentary tool to recent taxonomies of chronic pain, such as that endorsed by the *Analgesic, Anesthetic, and Addiction Clinical Trial Translations Innovations Opportunities Network* and the *American Pain Society* (ACTTION-APS) (Kent et al., 2017; Fillingim et al., 2014) who have described specific clinical pain diagnoses. Our new framework is presented as a tool to identify the magnitude of the primary driver(s) of a pain experience without requiring a label on the condition. We have found it applicable to a wide variety of both acute and chronic musculoskeletal conditions.

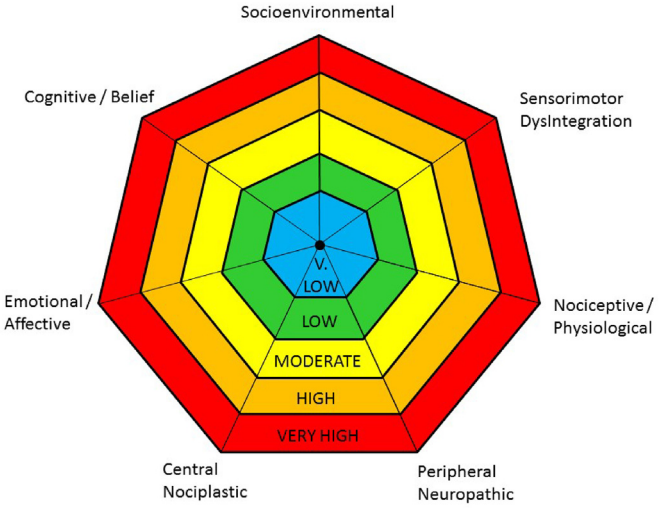


Fig. 1. Example of a radar plot with 7 distinct (but potentially overlapping) domains of the pain experience that can be useful for clinical evaluation and treatment decisions for people in pain.

The seven points have been deliberately chosen as ones that should logically be amenable to different interventions, even when a firm diagnosis cannot be reached, and have previously been associated with the qualitative or quantitative experience of pain. They are:

- **NOCICEPTIVE (PHYSIOLOGICAL) INPUT** (Perl, 1996), defined here as pain produced primarily through input from peripheral nociceptive afferents following transduction of noxious-level mechanical, thermal or chemical stimuli that leads to an action potential volley from the periphery through the central nervous system. In this case, the afferent volleys are initiated through depolarization of nociceptive end organs as a result of abnormal stress on (or injury to) peripheral tissue(s). In other contexts these have been described as 'mechanical' or 'inflammatory' pain behaviors.
- **PERIPHERAL NEUROPATHY** (Hsieh et al., 1995) defined here according to the definition from the International Association for the Study of Pain as pain caused by a lesion or disease of the peripheral nervous system (www.iasp-pain.org/Taxonomy#Neuropathicpain).
- **CENTRAL NOCIPLASTIC CHANGE** (Flor et al., 1997) defined here as pain that can be traced to either a central facilitation of action potentials (amplification or disinhibition) from the periphery, or as ectopic impulses generated within the central nervous system with no direct input from the periphery. This is analogous to the IASP definition of *central sensitization*. The term nociplastic is endorsed by Kosek and colleagues (Kosek et al., 1097) as an alternative to the more ambiguous 'sensitization', the implication being that such mechanisms are potentially reversible (e.g. 'plastic').
- **EMOTIONAL DYSREGULATION OR PATHOLOGY** (Blozik et al., 2009) defined here as diagnosable psychopathology or affective dysregulation, conditions described in the Diagnostics and Statistical Manual – V (American Psychiatric Association, 2013). These could include depression, anxiety, or other mood or personality disorders. There is a long history of association between psychopathology and pain (Dimitriadis et al., 2015), and while causal mechanisms are yet unclear it appears likely that pain magnifies negative mood while negative mood amplifies pain.
- **MALADAPTIVE COGNITIONS** (George and Hirsh, 2009) defined here as *inaccurate* or *irrational* beliefs, thoughts or behaviors about, or resulting from, the experience of pain. Similar to emotional distress, there is a long history of association between exaggerated

negative orientation towards pain (e.g. fear, catastrophization, low self-efficacy) and pain (Sullivan et al., 2002; Crombez et al., 1999), but the causal pathways have proven elusive. The key difference between this and the prior category are that while maladaptive cognitions may be a precursor of psychopathology, there are no defined diagnostic criteria for them (i.e. there is no DSM-V entry for 'pain catastrophizer'). The practical implication, and the reason for their separation, is that, for example, a physical therapist may be well positioned to address maladaptive beliefs or cognitions about pain (Bennell et al., 2016), while addressing psychopathology should be the domain of a mental health professional.

- **SOCIOENVIRONMENTAL CONTEXT** (Raichle et al., 2011) defined here as the very wide-ranging and amorphous contextual factors that affect not only one's experience of pain but also access to appropriate care, willingness to report, and the way in which pain is described. This could include relations with important others, prevailing cultural beliefs or language about pain, socially-constructed gender roles, early life adversity, environmental demands, stressors, and many others.
- **SENSORIMOTOR DYS-INTEGRATION** (de Vries et al., 2015) defined here as discordance between the perceived self and the actual self. Alternatively described as a problem of interoception (Di Lernia et al., 2016), the driver here is one of a mismatch between two or more sensory inputs into the central nervous system, such as optical input stating the head is in one position, and cervical proprioceptive input indicating it is in a different position. While there is consistent evidence that such sensorimotor discordance exists with greater frequency in people with pain (de Vries et al., 2015), causal pathways are yet to be fully elucidated.

These points are not likely to be exhaustive, however, we believe they satisfy the ACTTION-APS criteria of being adequately exhaustive for clinical use, mutually exclusive, biologically plausible, reliable, clinically useful and simple (Fillingim et al., 2014). Readers will note that this framework is meant to be applied after the patient has passed screening for red flags or other systemic influences (comorbidities, medications) that may also contribute to their symptoms, and only after the patient has been deemed likely appropriate for assessment and care by the health practitioner.

Table 1 provides sample indicators of a patient's status on each domain. However, these are meant to be examples rather than endorse a clinical edict. Readers will also note that the radar plot tool is intended to offer adequate direction for clinical decisions but not so much that it becomes burdensome for the clinician, the patient, or other stakeholders. The status levels on each domain are limited to qualitative ranges of: *very low, low, moderate, high, and very high*. While tools exist to evaluate these domains, our belief is that few are at the stage of development to allow endorsement to greater precision than these 5 broad levels. The diagrammatic representation of *relative* patient location on each of the 7 domains should make treatment decisions easier by quickly identifying the primary drivers of the patient's pain experience, even if two different clinicians may assign different *absolute* locations on each.

A second concept is required here, that being *triangulation*, which is drawn largely from military or geographic positional science. Fig. 2 graphically depicts this concept using the contemporary example of locating the global position of a mobile phone. While one source of information can provide a very broad sense of position, two sources narrows possible position to the region of overlap, and three sources all 'pointing in the same direction' leave only one possible position. This concept of triangulation can be applied to estimate the magnitude of contribution from each of the radar plot domains, requiring use of at least 3 information sources before being confident in locating the patient on each. Especially when information gleaned from very different information sources all point in the same general direction (e.g. patient self-report, imaging, and clinical tests) does confidence in a patient's location increase. This analogy can be further understood through positive (+LR) or negative (-LR) likelihood ratios and pre- and post-test probabilities. Box 1 demonstrates an example using 3 clinical tests of low-to-moderate diagnostic validity (Sensitivities and Specificities ranging from 0.66 to 0.80). For ease, the example displays findings of all negative or all positive results, but it should be noted that negative results in one domain (e.g. central nociplastic) may be considered positive in another (e.g. nociceptive).

Through use of the multi-domain radar plot, complex data sources built on triangulated findings can be visualized. This approach encourages appropriate implementation and interpretation of sound measurement tools and tests, and a subtle but important paradigmatic shift in treatment planning where sound assessment of modifiable domains (rather than categorical labels) becomes the priority from which treatment strategies can naturally flow. As demonstrated in the sample cases below (Appendix), the patient's subjective history should also be considered a source of information for triangulation.

While anecdotal, our experience suggests the radar plot and concept of triangulation appear to resonate with students and novice or mid-career clinicians across professional disciplines. It appears to function adequately well as a teaching tool, but experienced readers will recognize that, like any such approach, it is arguably too reductionistic. For example, it assumes clear distinctions between domains of the pain experience that likely overlap (e.g. central nociplastic and sensorimotor dysintegration). Further, consistent with the current state of research in the field, the separate domains ignore interactions between, for example, nociceptive input and sensorimotor dysintegration in the presence of maladaptive cognitions of, say, middle-aged East-Asian females. As the research progresses, so too can this teaching tool evolve.

We hope that the radar plot will facilitate communication across professional disciplines, between patients and providers, and with third-party payors. Three tools per domain, not all of which need to be exhaustive measures (e.g. a well-formed single direct question could serve as a useful discriminatory tool for one or more domain(s)) appear to be reasonable educational targets for trainees and educators, offering a scaffold for education in pain rehabilitation disciplines.

The intention of publishing this tool is to permit further development by educators, researchers, and clinicians in the spirit of facilitating professional development towards optimizing patient outcomes. Whether use of this tool expedites transition towards expert-level clinical reasoning or improves clinical behaviors/outcomes is a reasonable direction for future study. We hope others will find value in this line of reasoning by substituting the domains in our example with those that are relevant for fields other than pain, and that it helps novice clinicians across any number of disciplines to make sense of a highly complex and often confusing field of research.

Disclosure

The model being proposed has been used to educate participants on for-profit continuing professional development courses, but the authors hold no exclusive copyright over the use of a radar plot or the concept of triangulation.

Table 1
Examples of tools or clinical signs currently available for estimating magnitude of dysfunction/impact in each of the 7 domains described by the sample radar plot. NSAIDs = Non-Steroidal Anti-Inflammatories, TCA = Tricyclic Antidepressants, SSRI = Selective Serotonin Reuptake Inhibitors, SNRI = Serotonin & Norepinephrine Reuptake Inhibitors.

Assessment Domain	Nociceptive (Physiological) input	Peripheral Neuropathy	Central Nociplastic Mechanisms	Emotional Dysregulation	Maladaptive Cognitions or Beliefs	Socioenvironmental Context	Sensorimotor Dys-integration
History of the complaint	• Complaints are proportionate to the mechanism	• Mechanism of onset consistent with trauma of a peripheral nerve	• More difficult to draw connection between mechanism of onset and current complaints	• History of psychopathology especially if temporally related to other symptom onset	• No defined pattern, can be acute or chronic, traumatic or non-traumatic	• May be more likely when pathogenesis has occurred in a compensable environment or linked to other stressors	• More likely to manifest in chronic problems
Patient narrative	• Well-localized pain complaints	• Spontaneous or 'ectopic' pain, allodynia and local hyperalgesia	• Resting pain (local or widespread), may be related to mood or emotional status	• Symptoms consistent with psychopathology (e.g. DSM-V criteria)	• Examples: Belief that hurt = harm, or that 100% relief is required before resuming activity	• Feels under constant scrutiny or surveillance (e.g. medicolegal involvement)	• Describes the injured body region as though it is detached from self
Standardized self-report evaluations	• Responses do not support other drivers in the framework	• Self-report diagnostic tools (e.g. SLANSS[a])	• Self-report diagnostic tools (e.g. CSI[d])	• Self-report diagnostic tools (e.g. PHQ-9[f], PCL[g])	• Self-report evaluative tools (e.g. PCS[i], TSK[j], FABQ[k])	• Self-report evaluative tools (e.g. SRI[l], IEQ[m])	• Few available, but may struggle to identify painful areas on a body diagram
Standardized clinical evaluations and signs	• Consistent and predictable movement-related pain behaviour	• Clinical signs of pain or impaired neural transmission along the course of a known sensory nerve	• Non-mechanical and non-predictable patterns of pain reproduction, with/without dysfunctional descending pain modulation[e]	• Pain not consistent with predictable mechanical patterns	• Exaggerated or inconsistent pain behaviours out of proportion to magnitude of testing	• Signs suggestive of intentional exaggeration may provide a clue, but careful interpretation is encouraged	• Signs of somatosensory reorganization (e.g. 2PD[n], JPSE[o])
Other observations	• Responsive to routine front-line pharmacotherapy	• Not responsive to NSAIDs, may be responsive to TCAs[b], SNRIs[c], pregabalin or gabapentin	• Not responsive to routine front-line therapies, nay be responsive to opioids, TCAs and/or SSRIs	• Small to no effect on pain from front-line pharmacotherapy, may see effect from TCAs[b] or SSRIs[h]/SNRIs[c]	• Preference for avoidant or passive coping methods, 'all or none'-type thinking	• Counseled to avoid activity or 'straining' until after case is settled	• May require exploration and exclusion of a CNS disorder

[a] SLANSS = Self-report version of the Leeds Assessment of Neuropathic Signs and Symptoms.
[b] TCAs = Tricyclic Antidepressants.
[c] SNRIs = Serotonin and Norepinephrine Reuptake Inhibitors. For a review of pharmacotherapy in neuropathic pain see Finnerup and colleagues 2015 (Woolf, 2004).
[d] CSI = Central Sensitivity Index.
[e] Dysfunctional pain modulation may manifest as exercise-induced hyperalgesia or abnormal conditioned pain modulation.
[f] PHQ-9 = Patient Health Questionnaire 9-item version.
[g] PCL = Post-traumatic distress scale checklist.
[h] SSRIs = Selective Serotonin Reuptake Inhibitors.
[i] PCS = Pain Catastrophizing Scale.
[j] TSK = Tampa Scale for Kinesiophobia.
[k] FABQ = Fear Avoidance Beliefs Questionnaire.
[l] SRI = Spousal Response Inventory.
[m] IEQ = Injustice Experience Questionnaire.
[n] 2PD = two-point discrimination.
[o] JPSE = Joint Position Sense Error.

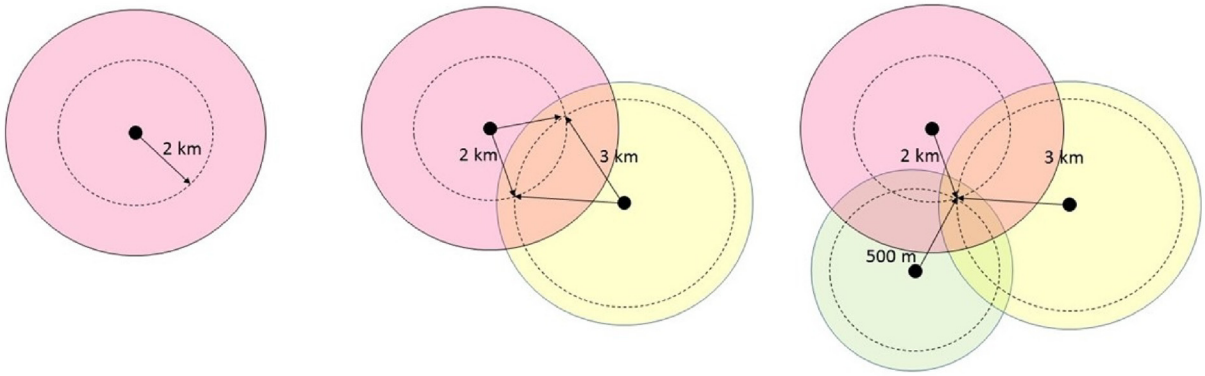


Fig. 2. A graphical depiction of the concept of triangulation. Starting from left to right, if the distance from a center (e.g. a cell phone tower) is known to be 2 km, the object (e.g. a phone) can be anywhere within that 2 km radius. When a second tower, 3 km away is also able to 'see' the device, there are two possible points that those two radii overlap at which the device can be located. If a third tower, in this example a less powerful one, can see the device 500 m away, there is only one possible location that all 3 radii overlap where the device could be located.

Box 1
Sample Triangulation using Likelihood Ratios.

Assumptions for this example:

Pre-test probability that condition exists (odds) 50% (1:2)

	Discriminative Validity	Likelihood Ratios	Post-test probability All tests positive	Post-test probability All tests negative
Clinical test 1	Sn 0.80, Sp 0.80	+**LR 4.00, -LR 0.25**	67% (2:1)	11% (1:8)
Clinical test 2	Sn 0.75, Sp 0.75	+**LR 3.00, -LR 0.33**	86% (6:1)	4% (1:25)

Box 1 (*continued*)

Assumptions for this example:

Pre-test probability that condition exists (odds) 50% (1:2)

	Discriminative Validity	Likelihood Ratios	Post-test probability All tests positive	Post-test probability All tests negative
Clinical test 3	Sn 0.66, Sp 0.67	+**LR 2.00, -LR 0.51**	92% (12:1)	2% (1:50)

With 3 tests, each of modest to low discriminative validity, but each positive, likelihood that a condition exists (or that a particular mechanism on the radar plot is important) goes from 50% to 92%, suggesting that domain should be in the high to very high range of the plot. If all 3 tests are negative, the likelihood a domain is a strong driver goes from 50% to 2%, moving that domain to low or very low. Both calculations are conducted accepting some likely inflation due to ignoring the prior odds fallacy.

Appendices. Sample triangulation approach[1]

Case 1: Jan

Jan is a 39 year-old Caucasian female with a history of persistent whiplash-associated disorder arising as a result of a motor vehicle crash 11 months prior. She was the belted driver of a 4-door sedan that was stopped when it was impacted from behind on the right side by a sport utility vehicle (SUV) traveling an estimated 30 km/h (~19 mph). Her headrest was well adjusted. She denies loss of consciousness. Imaging in the Emergency Department has ruled out significant pathology. Neck pain, stiffness and headaches began the following day and worsened within 48 h motivating her to see her family physician. She received a diagnosis of whiplash associated disorder grade II. She has received physical therapy (modalities and unsupervised exercise), massage, NSAIDs (acetaminophen) and wage indemnity benefits since that time. She has yet to return to her pre-collision job of floor supervisor for an auto parts plant, complaining of ongoing neck pain and headaches as well as sensitivity to light and difficulty concentrating that worsens after a couple hours of work. She is married with one teenaged son and prior to her injury she contributed equally to the family's finances.

On clinical examination cervical mobility is limited in all planes with no other obvious mechanical pattern. She appears exquisitely tender to palpate anywhere in the neck or shoulder girdle region, even flinching at times in response to light touch. Pressure pain detection threshold testing reveals widespread sensory hypersensitivity (local to the neck and over tibialis anterior) and she is hypersensitive to cold stimuli. Her pain thresholds decrease (more sensitive) following 3 min of moderately vigorous stationary cycling. Joint position sense error (nominating the center of a target after returning from cervical rotation with the eyes closed) and two-point discrimination are both impaired compared to population norms but still within the high ends of normal. Smooth pursuit neck torsion reveals no signs of saccadic eye movements.

Her self-report measures indicate poorly-localized widespread pain on a body diagram, severe disability according the Neck Disability Index score of 35/50, a Pain Catastrophizing Scale score of 32/52 (high), a score on the self-report version of the Leeds Assessment of Neuropathic Signs and Symptoms of 11/24 (one point under the cut score of 12/24), and scores on the Patient Health Questionnarie-9 are over-threshold for a potential

[1] This is meant to be an illustrative exercise and the tests and their interpretation described are based on the authors own experience and expertise in the field. They are not meant to be an endorsement of those specific tests or interpretations. Such questions are better left for formal systematic reviews.

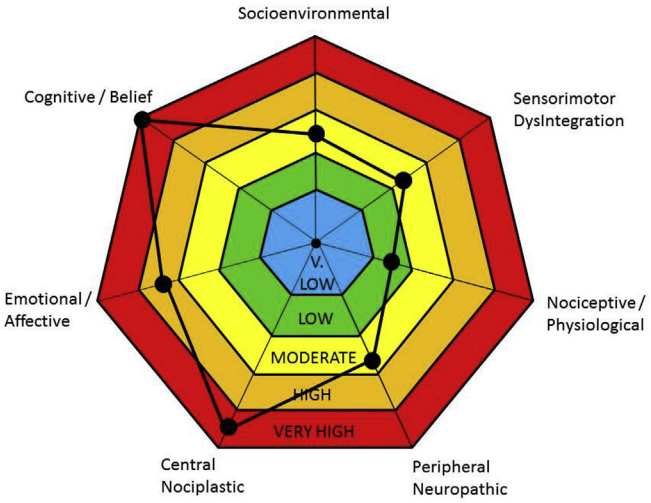
depressive disorder.

She reports a generally good relationship with her insurer and family doctor, and her husband and son are supportive. However, she also describes feeling pressured and scrutinized by her employer and coworkers during an earlier failed attempt to return to work. She is also experiencing increasing financial hardship due to medical expenses and lost wages.

Triangulating:

Domain	Level
Nociceptive	Low
Peripheral Neuropathic	Low-Moderate
Central Nociplastic	High
Cognitive	High
Emotional	Moderate-High
Socioenvironmental	Mod
Sensorimotor	Low-Moderate

Create pain profile:



Case 2: Alex

Alex is a 31 year-old African-American male with a history of intermittent low back pain that he attributes to his job standing 7.5 h per day in a retail electronics store. The pain has been present for the 3 or 4 months with no definable etiology. He is otherwise healthy and enjoys playing tennis twice weekly. He has remained at work but reports that he requires non-steroidal anti-inflammatories to help manage the low back pain on average 3 out of 5 shifts per week. He describes pain and stiffness that is worse in the morning, improves through the mid-part of the day but worsens again in the last few hours of most shifts. This is his first time seeking formal rehabilitation care on the recommendation of a coworker. He is also the primary breadwinner for his family that includes a wife and one young son.

On clinical examination lumbar mobility is nearly full in all planes though he describes a 'pinching' type pain during extension and combined multiplanar movements of extension/side-bend especially to the right side. He describes local tenderness to palpation over the right lower lumbar/lumbosacral region. Pressure pain threshold testing reveals mechanical hypersensitivity in that same right lumbar area but normal sensitivity elsewhere. Neurological testing appears normal with no obvious signs of motor weakness or fatigue, though straight leg raising is somewhat limited on the right side. Two-point discrimination is within normal limits and he is easily able to complete a line drawing of his back that is proportionate to objective reality.

His self-report measures indicate well-defined localized pain over the right lower lumbar/lumbosacral region on a body diagram with no indication of numbness or paraesthesia. Scores on the Roland Morris Disability Questionnaire (RMDQ) are 6/24, noting issues with standing for long period, occasionally bending over, rising in the morning, and sometimes moving more slowly. His Pain Catastrophizing Scale score is 4/52 or low, and he shows no clinical indications of an emotional pathology so formal screening is not conducted.

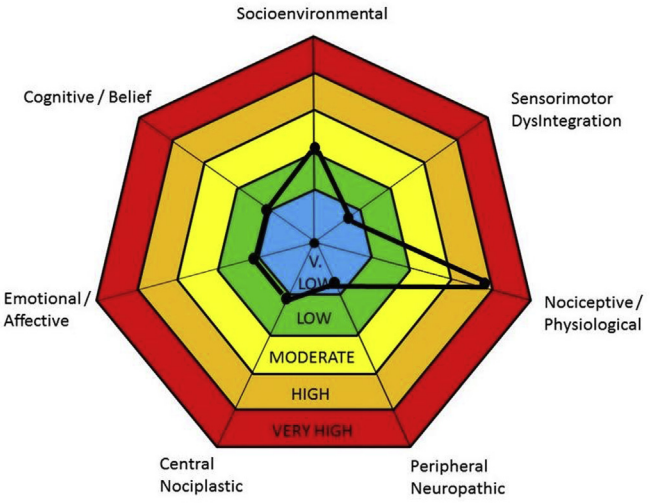
He reports that he generally enjoys his job and has a good group of coworkers who tend to have fun at work, though his manager is not always supportive of his need to sit down at times. His family doctor is the one that suggested NSAIDs but otherwise Alex feels as though she sort of 'waived-off' his questions about his low back. He is not one to let on that he is in pain when around home as he feels compelled to be the 'man of the house' and a 'good father' when not at work.

Triangulating:

Domain	Level
Nociceptive	High
Peripheral Neuropathic	Very Low

Central Nociplastic	Low
Cognitive	Low
Emotional	Low
Socioenvironmental	Low-Moderate
Sensorimotor	Very Low

Create pain profile:



Resultant radar plots from example data obtained from clinical examinations of two cases detailed in Appendix.

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