

Chronic Pain in the Medicolegal Setting: Reframing the Narrative and the Discussion

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Introduction

This article is aimed at clinical and legal practitioners with patients/clients presenting with chronic pain. The article briefly highlights the following in chronic pain: the complexity of presentation of chronic pain; a description of the latest diagnostic classification; a brief overview of the emerging neurobiology; the integration of overlapping conditions such as PTSD, depression and mTBI, and the issue of fundamental dishonesty.

Clinical challenges associated with assessment of chronic pain:

Acute pain is much easier to understand than chronic pain. The acute pain response is present in the vast majority of higher-level organisms and serves as a protective system ensuring survival. It has developed over the billions of years that life has evolved, from the non-cognitive concept of nociception, that is the detection of noxious stimuli by a unicellular organism, e.g. from hostile environment experienced to the combined experience of pain and suffering in human beings that have a significant degree of self-awareness and social empathy.

The consequences of acute pain, say following touching a hot candle flame, lead to a reflex withdrawal response in the affected part of the body, and a more generalised response including withdrawal, guarding and learning to avoid future similar scenarios. The survival benefits are obvious. In chronic pain, however, the candle flame has now gone out, but the chronic pain persists and sometimes progresses.

It is a mistake to consider that the appearance of chronic pain is only due to the neuromusculoskeletal or a hardwired biological consequence of an injury (in an unfortunate few). By the same token, it is also a mistake to consider that the persistence of pain post injury has to be due to (prior) factors unrelated to the index event. The explanation lies in a complex interplay of a multitude of factors including: the nature and severity of the original trauma on the body and brain, its psychological interpretation by an individual, the degree of prior psychosocial vulnerability, the extent of memory formation to leading fear-avoidance and other behaviours, ongoing psychosocial factors including mood symptoms such as depression and the effects of litigation. The misattribution of the origin of chronic pain is easy to do but can be hard to correct and can be damaging to the individual.

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How do we define chronic pain currently?

The most common and indeed the least helpful view is that pain that goes on for more than three months is likely to be chronic. This temporal definition does not consider the possible persistence of significant peripheral pain sources—sometimes unrecognised—which may lead to persistent pain post injury.

It is also now clear that we may be able to predict those patients who are likely to have abnormal pain response to injury or surgery which may result in chronic pain. The mechanisms or pathways were pre-existing, they were simply waiting for an appropriate initiating event to activate the eventual presentation. This is known as a prior vulnerability and also has a firm biological basis.

Currently chronic pain cannot be objectively measured in the clinical setting, instead we rely on the reports of an individual, their behaviour patterns, an assessment of physical impairment and their disability, including what are termed “illness behaviours”. This forms a difficulty for classical diagnostic medicine as we have multiple factors which eventually give rise to the development of chronic pain. Medicine innately prefers a diagnosis based on an easily definable pathophysiological basis and easily provable with a simple test. This is not the situation for chronic pain.

Multiple factors may lead to chronic pain including ill-defined peripheral sources of nociception or adverse stimuli, for example the broken bone that is not recognised or a persistent infection along with a central brain amplification process that is unique to the individual. Chronic pain should also be regarded in part as a communication system between the individual and those others around them. Vocalisations and behaviours cause others to respond to the need. The converse is also true, that the presence of meaningful others (including family doctors, and medico-legal examiners) can enhance or diminish the symptomatology and behaviours associated with chronic pain.

ICD-11 and chronic primary pain

In recognition of the multiple factors that give rise to a diagnosis of chronic pain, the new classification for pain was produced by WHO for the revised 11th edition of the International Classification of Diseases (“ICD-11”).¹ The classification was adopted in 2019 by the World Health Assembly and is coming into effect in January 2022.² In itself, the new ICD-11 pain classification is explicitly agnostic with regard to aetiology and in particular it aims to avoid the obsolete dichotomy of physical versus psychological.³ The word “functional”, which is often used to describe symptoms or impairment that cannot be organically explained, was specifically avoided.

The definition of chronic primary pain (“CPP”) is given as follows:

- persists or recurs for longer than three months;
- is associated with significant emotional distress (e.g. anxiety, anger, frustration, or depressed mood) and/or significant functional disability (interference in activities of daily life and participation in social roles); and
- the symptoms are not better (or completely) accounted for by another diagnosis.

ICD-11 also recognises there are biologically defined “secondary” (or known) pain syndromes. For example, an individual with a chronic neuropathic pain or the pain of bony non-union secondary to trauma can also develop, in addition, a chronic primary pain, that is an additional pain experience that is not explained by the underlying physical diagnosis. Chronic primary pain and other (known) secondary pain sources are not mutually exclusive and often co-exist. Pragmatically in pain clinics we recognise that the

¹ See <https://icd.who.int/browse11/l-m/en#/http://id.who.int/icd/entity/1326332835> [accessed 25 January 2022].

² See [https://www.who.int/news/item/18-06-2018-who-releases-new-international-classification-of-diseases-\(icd-11\)](https://www.who.int/news/item/18-06-2018-who-releases-new-international-classification-of-diseases-(icd-11)) [accessed 25 January 2022].

³ M. Nicholas, J. W. S. Vlaeyen, W. Rief, A. Barke, Q. Aziz, R. Benoliel, M. Cohen, S. Evers, M. A. Giamberardino, A. Goebel, B. Korwisi, S. Perrot, P. Svensson, S. J. Wang and R. D. Treede, “IASP Taskforce for the Classification of Chronic Pain. The IASP classification of chronic pain for ICD-11: chronic primary pain” (2019) 160(1) Pain. 2837.

pain and disability experience of some individuals is greater than would be expected from the known pathophysiology. In such a situation we would diagnose a biological source of pain—this would be known as a chronic secondary pain, as well as a chronic primary pain, that is we recognise the pain itself has become a problem.

The intention of the authors of the recent classification was to provide a diagnosis in the ICD-11 classification while avoiding the confusing, psychoanalytically driven and potentially laden terms such as “somatoform”, “non-specific” or “functional”. It avoids designating the basis of the pain as just purely “psychological” or “organic” because chronic pain includes psychological and social dimensions in addition to the known biological components. It recognises that there is a process within the central nervous system which means that the pain and disability is beyond what would have been routinely expected, for example say in an orthopaedic or neurological setting. The mechanisms of central sensitisation and nociplastic pain⁴ have been used to explain the presence of the development of CPP.

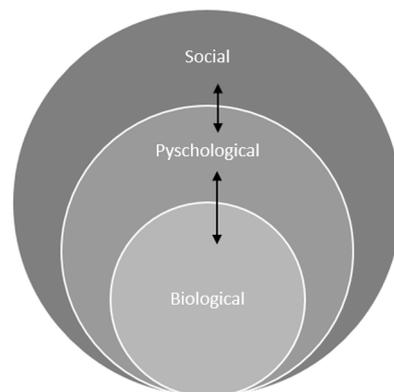
By including a distinct CPP classification within the ICD-11, the authors hoped to avoid the problems associated with previous classifications of chronic pain when the etiology is unclear, but there is often the presence of emotional distress and functional disability. In the language of the ICD classification, chronic pain has “multiple parents” or sources.

When do we consider that we need to diagnose a chronic pain?

As clinicians we recognise all diseases can have a psychological and social effect with associated distress and dysfunction. That is, the illness experienced is often greater than the disease process alone. This situation is commonly bi-directional. Not only do disease processes have a psychological and social effect but psychological and social factors affect both the experience of pain and the reporting of pain and disability. The term biopsychosocial suggests that pain must be considered in its biological form but sitting within a social and psychological context but flow of effect and affect in both directions. (See Figure 1.)

Figure 1. The biopsychosocial model of pain.

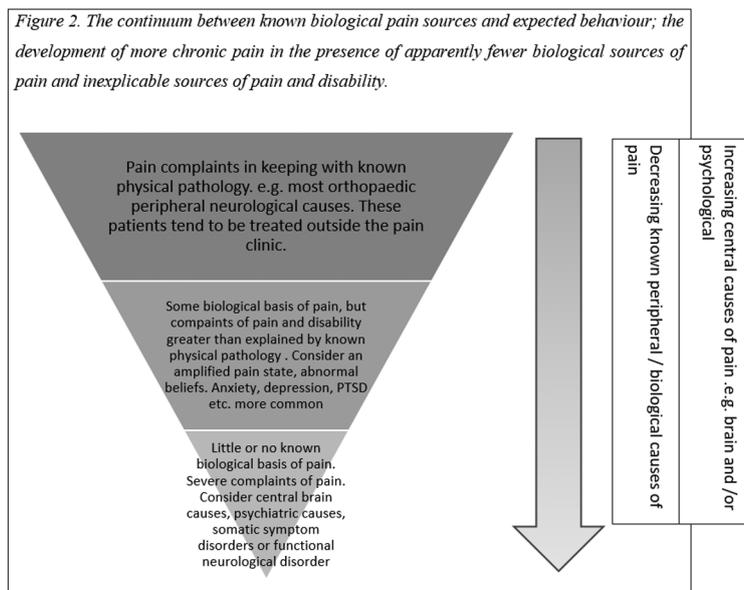
The circles are not discrete, there is a continuum and an interplay between the factors at each level.



⁴D. A. Walsh, “Nociplastic pain: helping to explain disconnect between pain and pathology” (2021) 162(11) Pain. 2627–2628.

In some clinical situations, there is no persistent discernible biological basis despite the reports of the pain and disability and so more weight will be given to the central or brain processes such as psychological/psychiatric factors, and these interplay with social factors.

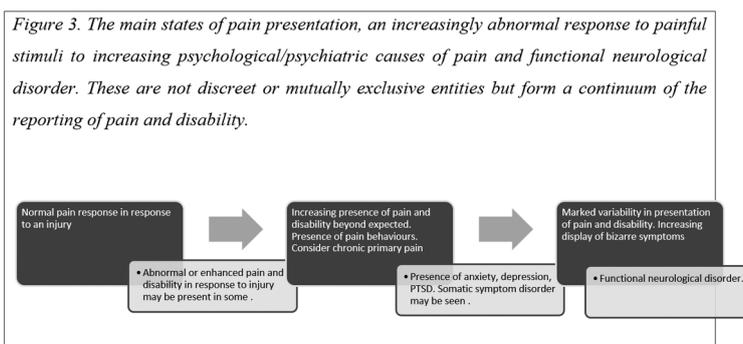
We can therefore come up with another simple schematic to represent decreasing biological basis of chronic pain along with increasing psychosocial contributions (Figure 2).



When one is considering known causes of “biological or peripheral pain” there is a certain expectation as to the reporting of level of pain disability and reasonable consistency in levels of physical performance. However, chronic pain is accompanied by more variation in physical ability and complaints of level of pain. This affects the level of activities undertaken which on a good day with little pain will be very good and often the individual will end up doing too much, followed by the need to rest for a while. This is commonly known as the “boom and bust cycle”. If the experience and reporting of pain is even more unusual and/or the variation in disability is very marked, for example some days they are walking and another day they are apparently in a wheelchair, then it is unlikely that any known physical disability will explain such a phenomenon. In such a situation, one needs then to consider psychiatric causes such as a functional neurological disorder. Such patients are often inconsistent in their reporting of pain and disability.

Many patients will have characteristics that come from all three categories in Figure 1. That is, the categories are not mutually exclusive and commonly coexist and influence each other. There is significant evidence that all such expressions of pain and disability lie on a continuum rather than being discreet clinical entities.⁵ In which case, the model can also be expressed horizontally in terms of diagnostic criteria. (See Figure 3.)

⁵ J. Maggio, P. R. Alluri, S. Paredes-Echeverri, A. G. Larson, P. Sojka, B. H. Price, S. Aybek and D. L. Perez, “Briquet syndrome revisited: implications for functional neurological disorder” (2020) 2(2) Brain Commun.



Many such patients, prior to the injury in question, will have already shown an abnormal pain response to previous situations or the presence of medically unexplained symptoms, irritable bowel syndrome, migraines etc and prior psychological/psychiatric difficulty.⁶ The situation may be compounded by existence of prior psychosocial stressors⁷ and subsequently unavoidable concurrent stressors such as the presence of financial distress, perceived injustice and the medicolegal process itself.⁸ Illness belief systems and how they are constantly updated in light of new experiences is an important factor to consider whilst developing a formulation or treatment plan.⁹ An allowance should be given for all these factors, as often the stressors, even if acknowledged by the individuals concerned, will still play a significant role in the presentation and prognosis of the individual. The presence of consistent pain behaviours is a poor prognostic factor.¹⁰ The presence of pre-existing vulnerability may be such that there is always a significant chance that absent a major injury a patient would have developed chronic pain in any event. Often it is the combined burden of pre-existing known disease as well as psychosocial factors which helps to predict this. Indeed, the chances of an individual developing a chronic pain picture at some point in the future can now be relatively accurately predicted from population studies.¹¹

The (clinical and legal) situation can be very complex and only clinicians who have spent a long time studying such patients will be able to recognise the appropriate models, true extent of complex interplay of relevant factors leading to accurate diagnoses and treatments to use in clinical practice, and to opine upon them in medico-legal settings.

Central neurological aspects of chronic pain

Pain is defined as:¹²

⁶ M. B. Yunus, “The prevalence of fibromyalgia in other chronic pain conditions” [2012] *Pain Res. Treat.* 584573. A. Burri, S. Ogata, J. Vehof and F. Williams, “Chronic widespread pain: clinical comorbidities and psychological correlates” (2015) 156(8) *Pain*. 1458–1464. L. A. McWilliams, “Adult attachment insecurity is positively associated with medically unexplained chronic pain” (2017) 21(8) *Eur. J. Pain*. 1378–1383.

⁷ B. I. Nicholl, G. J. Macfarlane, K. A. Davies, R. Morriss, C. Dickens and J. McBeth, “Premorbid psychosocial factors are associated with poor health-related quality of life in subjects with new onset of chronic widespread pain—results from the EPIFUND study” (2009) 141(1–2) *Pain*. 119–26.

⁸ M. J. L. Sullivan, “Perceptions of Injustice and Problematic Pain Outcomes” (2020) 21(7) *Pain Med.* 1315–1336. D. A. Fishbain, “Re: Secondary loss and pain-associated disability: theoretical overview and treatment implications” (2003) 13(3) *J. Occup. Rehabil.* 197–8. D. A. Fishbain, B. Cole, R. B. Cutler, J. Lewis, H. L. Rosomoff and R. S. Rosomoff, “A structured evidence-based review on the meaning of nonorganic physical signs: Waddell signs” (2003) 4(2) *Pain Med.* 141–81.

⁹ T. Kube and L. Rozenkrantz, “When beliefs face reality: An integrative review of belief updating in mental health and illness” (2021) 16(2) *Perspectives on Psychological Science* 247–274.

¹⁰ P. Thibault, P. Loisel, M. J. Durand, R. Catchlove and M. J. L. Sullivan, “Psychological predictors of pain expression and activity intolerance in chronic pain patients” (2008) 139(1) *Pain*. 47–54.

¹¹ C. H. Dominick, F. M. Blyth, M. K. Nicholas, “Unpacking the burden: understanding the relationships between chronic pain and comorbidity in the general population” (2012) 153(2) *Pain*. 293–304. K. A. Davies, G. J. Macfarlane, J. McBeth, R. Morriss and C. Dickens, “Insecure attachment style is associated with chronic widespread pain” (2009) 143(3) *Pain*. 200–205. J. McBeth, G. J. Macfarlane, I. M. Hunt and A. J. Silman, “Risk factors for persistent chronic widespread pain: a community-based study” (2001) 470(1) *Rheumatology (Oxford)* 95–101.

¹² R. D. Treede, “The International Association for the Study of Pain definition of pain: as valid in 2018 as in 1979, but in need of regularly updated footnotes” (2018) 3(2) *Pain Rep.* e643.

“An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.”

This means that chronic pain can also be the result of only a brain processing abnormality but feel just as real as a pain generated in the periphery. One needs to consider perception, subjectivity and consciousness. There is also a distinction between central nociceptive processing v the eventual conscious experience of pain.¹³ These mechanisms are now included in the term “nociceptive pain”.¹⁴ Consideration of the supraspinal aspects support the chronic primary pain classification.¹⁵ Biologically, the inflammation and immune systems which have long been known to play a fundamental role in peripheral pain stimuli also seem to play a significant role centrally.¹⁶ These insights provide an explanation for the daily clinical observation that pain experience is often poorly correlated to the simple amount of likely peripheral nociceptive input and, in fact, can be “centrally”—generated *even* with normal nociceptive input.

The brain structures and processes which are involved in the central processing and eventual perception of pain include the limbic-emotional circuitry, the effects of emotional-learning mechanisms based upon predispositions and injury-related nociceptive inputs to the nervous system. There are now clear fMRI differences shown between acute and chronic pain brain activity.¹⁷ Chronic pain can be considered a specific brain state, with distinct anatomical and functional properties. It is now much easier to appreciate how pre- and post-accident neuropsychiatric conditions can amplify pain presentations. mTBI is commonly associated with a number of neurological and neuropsychiatric factors which interact with each other and influence perception of pain.

Role of neuropsychiatric and other maintaining factors

Central processing of any peripheral pain stimuli involves the limbic system which is the part of the brain involved in the processing of emotions including sadness, despair, fear and threats. Hence pain is always influenced by a range of negative emotions including worry, sadness and fear associated commonly with psychiatric conditions such as depression, anxiety or PTSD. Additionally, experience and reporting of pain is commonly influenced by negative emotional states and physiological factors such as poor sleep and fatigue. It is widely recognised that brain injury is commonly associated with a range of neuropsychiatric conditions such as depression, anxiety disorder, emotional and behavioral dysregulation, all of which can affect pain perception and perpetuate chronic pain.

A careful neuropsychiatric evaluation of a claimant in a clinical or medico-legal setting would involve comprehensive evaluation and consideration of interaction between prior vulnerability, impact of the index injury in precipitating/triggering a range of symptoms and the role of concomitant psychiatric and psychosocial factors in maintaining the pain. The presence of significant prior vulnerability is not itself enough to conclude that the index event is noncontributory aetiologically. Subsequent significant physical, psychiatric or psychosocial factors could still interact with and modulate significant prior vulnerability, resulting in greater symptomatology, distress or dysfunction with poorer prognosis. Comprehensive consideration of the “but for” scenario and role of post injury triggering and maintaining factors is important.

¹³ M. N. Baliki and A. V. Apkarian, “Nociception, Pain, Negative Moods, and Behavior Selection” (2015) 87(3) *Neuron*. 474–91 at <https://pubmed.ncbi.nlm.nih.gov/26247858/> [accessed 25 January 2022].

¹⁴ D. A. Walsh, “Nociceptive pain: helping to explain disconnect between pain and pathology” (2021) 162 *PAIN* 2627–2628.

¹⁵ I. Diez, B. Williams, M. R. Kubicki, N. Makris and D. L. Perez, “Reduced limbic microstructural integrity in functional neurological disorder” (2021) 51(3) *Psychol. Med.* 485–493.

¹⁶ S. K. Totsch and R. E. Sorge, “Immune System Involvement in Specific Pain Conditions” (2017) 13 *Mol Pain*. 1744806917724559. A. Ellis and D. L. Bennett, “Neuroinflammation and the generation of neuropathic pain” (2013) 111(1) *Br. J. Anaesth.* 26–37. D. Yoon, Y. Xu, P. Cipriano, V. Tawfik, C. Curtin, I. Carroll and S. Biswal, “Musculoskeletal changes on [18F]FDG PET/MRI from complex regional pain syndrome in foot” [2019] *SOC NUCLEAR MEDICINE INC.*

¹⁷ D. Reckziegel, E. Vachon-Preseau, B. Petre, T. J. Schnitzer and M. N. Baliki MN, “Deconstructing biomarkers for chronic pain: context- and hypothesis-dependent biomarker types in relation to chronic pain” (2019) 160(1) *Pain*. S37-S48 at <https://www.youtube.com/watch?v=Ex1dxoXdnMg> [accessed 25 January 2022].

Intention motivation and credibility

Ultimately, there is no test which will help us to recognise conscious exaggeration or malingering. This can occur in any of the clinical scenarios, which themselves are difficult to define.¹⁸ Clinicians need to consider issues such as intention and motivation and these are not routine practice for most clinicians apart from neuropsychiatrists. Clinicians generally tend to be very accepting of their patients' symptomology, it is the only way to form a therapeutic relationship. However, in the medico-legal setting, it is often at the invitation of the lawyers that we are asked to comment on issues around credibility. In such a situation, doctors do not do well and can open themselves up to criticism.¹⁹ However, they can advise on whether the pattern and presentation of pain and disability is consistent within a repeated observation (including that seen on surveillance) of a claimant and also consistent with what they have observed in other patients outside the medico-legal context. Clinicians or experts in medico-legal settings can evaluate all the relevant aetiological factors and whether they are consistent with the observed behaviours or reported symptoms. When the evidence is not consistent, then it is entirely appropriate to re-examine the basis for the original diagnosis. However, inconsistency itself is not sufficient to conclude lack of credibility or even malingering, as inconsistent behaviours and symptoms are commonly noted in conditions driven by subconscious processes such as functional neurological disorders.

We do have to recognise that if you have a condition like pain which is entirely subjective then it is so much easier to simply report that one is in severe pain. It is an unverifiable condition. However, somebody who reports a pain score of 10/10 sitting in front of you smiling is unlikely to have unbearable pain. Such a pain score and incongruent behaviour of itself will not immediately raise the issue of credibility. For many patients, a score of 10/10 does not mean intensity but a continuity of pain which they find unbearable. It is important to discuss in detail with the claimant and point out that doctors might interpret the scale differently from the way the patient has reported the pain. Emotional states, illness beliefs, level of sleep and fatigue, amongst other psychosocial factors, will also influence the reporting of level of pain.

Furthermore, if one accepts the biopsychosocial model of pain, it follows that social factors including the medico-legal process will tend to enhance the experience of pain. Patients' pain and disability in our experience is invariably much worse in the context of the examination room, and often appears much less when, for example, sitting in a coffee shop or going shopping or going to and from appointments. The role of social media is particularly difficult as this is often put on for a show rather than to express to the world how difficult a patient is finding their problems.²⁰ Therefore, not only does one need to ask about good and bad days, but also less and more stressful environments. By identifying these specific social factors and the way they influence pain behaviours one can help not only the patient understand their pain but also help the court understand patient disability and behaviour in different contexts.

¹⁸ N. L. Tuck, M. H. Johnson and D. Y. Bean, "You'd Better Believe It: The Conceptual and Practical Challenges of Assessing Malingering in Patients With Chronic Pain" (2019) 20(2) *J Pain*. 133–145.

¹⁹ K. J. Weiss and L. Van Dell, "Liability for Diagnosing Malingering" (2017) 45(3) *J. Am. Acad. Psychiatry Law*. 339–347.

²⁰ *Palmer v Mantas* [2022] EWHC 90 (QB).