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The Pain and Movement Reasoning Model: Introduction to a simple tool for integrated pain assessment

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ABSTRACT

Pain is no longer considered to be simply the transmission of nociception, but rather an output subsequent to the complex interactions of homeostatic systems. Manual therapists' clinical reasoning needs to incorporate this complexity in order to develop individualised effective treatment plans.

Pain classification strategies attempting to assist clinical reasoning traditionally define multiple types of pain – nociceptive, neuropathic, centrally sensitised – potentially fitting elements of the pain experience to linear independent systems, rather than embracing the multiple dimensions. It is our contention that pain should not be classified unidimensionally. In all pain states consideration should be given to the combined influence of physiological, cognitive, emotional and social inputs, all of which have the potential to influence nociception.

The Pain and Movement Reasoning Model presented in this paper attempts to capture the complexity of the human pain experience by integrating these multiple dimensions into a decision making process. Three categories have been created to facilitate this – central modulation, regional influences, and local stimulation. The Model allows for the identification of a predominant element to become the focus of treatment but also for the identification of changes to clinical presentation, where new treatment targets can emerge.

1. Background

Pain is no longer considered to be simply the transmission of nociception. Current conceptions suggest pain is the most salient part of an activated body protection system; an output subsequent to the complex interaction of homeostatic systems in response to an identified threat (Fig. 1) (Jänig et al., 2006). The body protection system involves motor, autonomic, psychological, endocrine and immune systems, and pain emerges from the activation of a specific neurological network, matrix or signature (Gifford, 1998; Melzack, 2005; Moseley et al., 2012; Melzack and Katz, 2013). Pain perception takes place in a context of an individual's environment, including the physical, social and emotional contexts (Siddall and Cousins, 2004; Gatchel et al., 2007; Malenbaum et al., 2008), and then is managed in a clinical context influenced by the values and beliefs of the therapist (Foster et al., 2010; Nijs et al., 2012). Clinical reasoning requires that manual therapists integrate the multiple dimensions of pain to account for this variation and formulate effective treatment.

The Pain and Movement Reasoning Model presented in this paper attempts to capture the complexity of the human pain experience. The Model is strongly underpinned by Neuromatrix Theory and incorporates current concepts of neuroplastic determinants on the quality and nature of pain (Woolf, 2011; Moseley and Flor, 2012; Melzack and Katz, 2013). Consequently, the Model avoids the risk of simplifying elements of the pain experience into linear independent systems e.g. central sensitisation, neuropathic, nociceptive. In a similar way the Model does not separate the bio-psycho-social framework into its component parts, but instead integrates the combined influence of the physiological, cognitive, emotional and social inputs on neurophysiological mechanisms. Through consideration of this range of information, the predominant and changeable influences can be identified, leading therapists to select the most appropriate techniques.

The integration of information is facilitated by the triangular structure of the Model coordinated by the three categories located at the apices of the triangle – central modulation, regional influences and local stimulation (Fig. 2). By placing a grid in the centre

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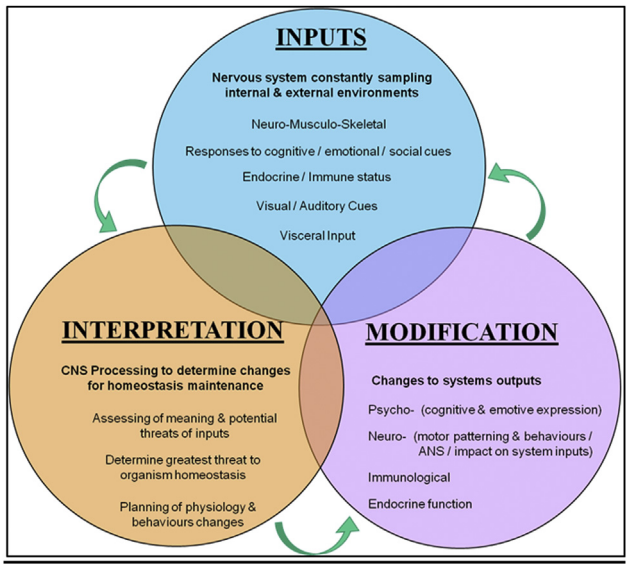

Fig. 1. Central nervous system sampling, processing and modifying the psycho-neuro-immunological state in the human pain experience. (Available under licence CC BY-NC Aus 3.0 at http://latrobe.libguides.com/content.php?pid=109542&sid=825367).

of the triangle, the graphic representation of the Model becomes an interactive tool to assist the reasoning process. After considering the clinical assessment of a person's presentation, the therapist marks a point on the grid to best represent the relative contribution of each of the three categories to the person's pain. Co-ordinating this plot on the grid requires thoughtful consideration of the determinants and influences across all three categories. This judgement, about factors encompassing an individual's pain experience, then enables prioritisation of management techniques that will address the most significant elements in the clinical presentation. The Model allows for continual evaluation so that as an individual's pain presentation changes, the focus of management is able to shift.

2. Central Modulation Category

As humans appraise their personal situation, pain perception occurs within a framework of ongoing simultaneous processing at different levels of consciousness (Moseley et al., 2012; van Ryckeghem et al., 2012; Bulcke et al., 2013). For therapists, this translates into the requirement of managing a person holistically where context is important. To do this requires an understanding that the sensory component of pain, nociception, always occurs within the broader setting of an individual's situation (Weisse, 2004; Wiech et al., 2008; Wiech and Tracey, 2013) and their psycho-neuro-immunological state (Watkins and Maier, 2000; Austin and Moalem-Taylor, 2010).

The category 'Central Modulation' is representative of the factors that have been shown to influence pain through changes to higher centre processing, reducing central descending inhibitory influences, or by increasing efficacy of spinal synapses (Woolf, 2011; Smart et al., 2012a). The overall assumption is that modification of pain via these factors can be attributed to changes in nervous system function, and in persistent cases to structure, i.e. due to neuroplasticity. The resultant change in sensitivity can contribute to an enhanced pain response (i.e. sensitisation) or a diminished pain response (i.e. inhibition), depending on the nature and context of the influence (Wiech and Tracey, 2009; Ploner et al., 2011; Melzack and Katz, 2013). All experiences are processed in the psycho–neuro–immunological systems of a person, which is why

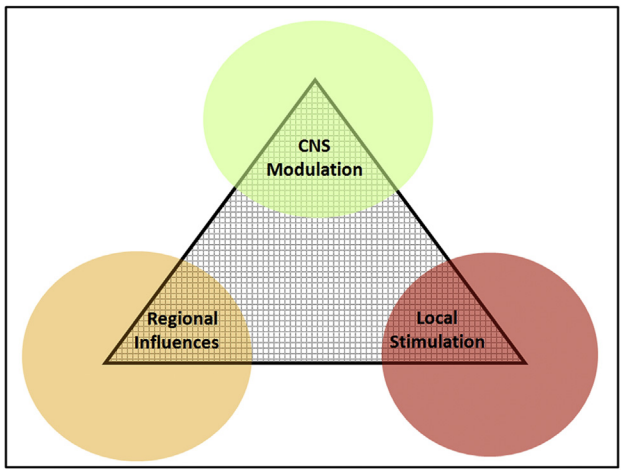

Fig. 2. Categories of the Pain and Movement Reasoning Model. (Available under licence CC BY-NC Aus 3.0 at http://latrobe.libguides.com/content.php?pid=109542&sid=825367).

in the clinical setting what is occurring within symptomatic tissue does not always relate to the pain expressed.

Therapists are encouraged to consider three subcategories to estimate the potential for central modulation; (1) predisposing factors, (2) prolonged afferent input and (3) cognitive-emotional-social state (Fig. 3).

2.1. Predisposing factors

The first subcategory recognises that prior experiences including trauma, illness and disease preset the state and structure of the nervous system. These factors may determine the baseline sensitivity of the person's nervous system to which new episodes of perceived vulnerability and threat are overlayed. Where a person has a pre-existing illness it is likely the body protection systems are activated. For example, persistent inflammatory conditions, such as inflammatory arthropathies and autoimmune diseases, can be expected to create pain sensitivity (Lee et al., 2009) through persistent chemically-generated nociception and altered immune function. Similarly, activity of the inflammatory glial cells within the CNS is increasingly seen as an important component of the pain experience (Thacker et al., 2007; Schmid et al., 2013).

People who have experienced trauma or torture have been shown to have an enhanced pain response (Linton, 2002; Granot et al., 2011; Fleischman et al., 2014; Williams and van der Merwe, 2013). This could be due to reduced central inhibition, especially where there is ongoing distress and perceived vulnerability and

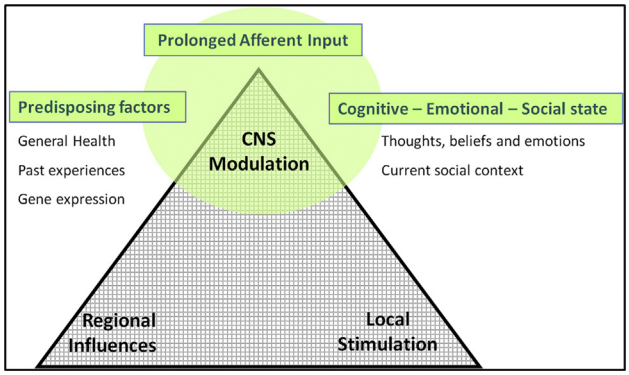

Fig. 3. Central Modulation Category. (Available under licence CC BY-NC Aus 3.0 at http://latrobe.libguides.com/content.php?pid=109542&sid=825367).

threat (Linton and Shaw, 2011), but also could be due to enhanced efficacy of nociceptive transmission (Woolf, 2011).

The genetics of pain also needs to be considered. A person may be born with a predisposition for pain (Nielsen et al., 2008; Mogil, 2012). More commonly, previous nervous system experiences can modify phenotypes expression through genetic transcription, enhancing neural transmission (Woolf, 2011; Hush et al., 2013). As a result conditions such as persistent neck and back pain, tension-type headache, orofacial pain, irritable bowel syndrome and fibromyalgia frequently coexist (Woolf, 2011). Gene expression also contributes to the large variation of response to analgesic medications (Diatchenko et al., 2011).

2.2. Prolonged afferent input

It is well established that prolonged noxious stimulation leading to ongoing afferent activity can enhance synaptic transmission (Woolf, 2011) and this subcategory directs therapists to consider evidence of this in the patient's history. Persistent pain leads to changes in the spinal cord and higher centres, not only in processing of pain-related inputs, but also in the outputs seen in an individual's psychological state, and their immune, endocrine and motor systems (Siddall and Cousins, 2004). In various persistent pain states, imaging suggests neural organisational changes and degeneration (Apkarian et al., 2004; Wand et al., 2011).

In the spinal cord there are chemical changes affecting signal transmission, processing and genetic transcription. This leads to inhibitory inter-neurone loss, sprouting of cells between laminae, sustained activation of descending facilitatory circuits and dysfunction of descending inhibition paths (Gifford and Butler, 1997; Siddall and Cousins, 2004; Winkelstein, 2004; Tracey and Mantyh, 2007). Such neural changes can result in the spreading of symptoms and an enhanced response to both noxious and non-noxious stimuli (Woolf, 2011; Schmid et al., 2013).

The modifying capabilities of prolonged stimulation at both the spinal cord and cortical levels demonstrate activity-dependent neuroplasticity. This process needs to be incorporated into reasoning and challenges the labelling of pain as acute or chronic. Clearly, time alone is not responsible for these higher centre changes seen in imaging studies for those with persistent pain. Recognising the transformation from an acute to an ongoing pain state as a transitory neuroplastic process affecting certain individuals, and valuing the inter-relatedness of certain psycho−social factors, would seem more appropriate.

2.3. Cognitive-emotional-social

Psychological and social factors are regarded as important for predicting long term pain-related disability (Watson and Kendall, 2000; Hill and Fritz, 2011; Linton and Shaw, 2011; Nicholas et al., 2011). Their role in determining or modulating the individual's pain experience, as postulated in Neuromatrix Theory, is now well established (Carter et al., 2002; Campbell and Edwards, 2009; Main and George, 2011; van Ryckeghem et al., 2012; Moore et al., 2013; Ruscheweyh et al., 2013; Taylor et al., 2013). Psychosocial factors are important in the clinical presentations of people with ongoing pain states but also of people with a recent onset of pain.

Relevant psycho-social influences on pain include: levels of anxiety; fear, and the accompanying avoidant behaviour related to pain, functional changes, and fear itself; depression; anger; self-efficacy; catastrophising; acceptance; attention; coping strategies; social support; fatigue and work circumstances (Hill and Fritz, 2011; Linton and Shaw, 2011; Schuh-Hofer et al., 2013). These relationships with pain have been identified in both recent-onset and persistent low back pain, whiplash, osteoarthritis and rheumatoid arthritis and other complex pain states (Giardino et al., 2003; Bradley et al., 2004; Gatchel et al., 2007; Lee et al., 2009; Somers et al., 2009; Arendt-Nielsen et al., 2010).

Imaging studies have demonstrated the relevance of psychosocial influences on higher- centre processing of pain (Loggia et al., 2008; Wiech et al., 2008; Tracey, 2010; Villemure and Schweinhardt, 2010; Simons et al., 2014). Notably, studies suggest pain and social distress or rejection activate similar parts of the brain, raising the possibility that people who are socially unsupported or disadvantaged may be predisposed to pain (Eisenberger, 2012; Meerwijk et al., 2012). This reflects the initial premise that pain is the most salient expression of a centrally evoked body protection response.

2.4. The importance of the Central Modulation Category

All presentations of pain will have an element of central sensitisation or inhibition that needs to be incorporated into the reasoning process. While it has been convention to regard 'acute' and 'chronic' pain as different, this model regards all 'types' of pain as one, and acknowledges the capacity of the central nervous system for plasticity. The variation in presentation can largely be explained by the state and structure of the nervous system; that is, how sensitive it is. Common conceptions of acute pain assume a naive nervous system or at least an unsensitised one that has no experience in processing previous pain and threatening situations.

Accordingly, if attributions of central sensitisation are reserved for chronic presentations, the clinical reasoning of acute presentations becomes simplistic, risking poor decision making with regard to treatment. For example, a person who is anxious about the seriousness of their acute injury will likely have reduced central inhibitory influences, modulated through attentional and emotional synaptic networks, leading to a sensitised state of the nervous system (Villemure and Schweinhardt, 2010). The therapist needs to incorporate knowledge of this enhanced pain state into any clinical reasoning process, especially in response to the person's pain report. Failure to address anxiety or distress in acute presentations risks a poorer outcome for the person (Hill and Fritz, 2011; Nicholas et al., 2011).

Consideration of the Central Modulation Category provides an understanding of the potential influences on central processing including the impact of learning and memory abilities (Flor, 2012; Zusman, 2012). The estimation of a significant contribution from factors known to modulate the sensitivity of the nervous system may lead the therapist to explore psycho-neuro-immunological retraining approaches including education, stress management, cognitive reframing, body awareness, graded motor imagery and graded exposure (Flor, 2012; Moseley and Flor, 2012).

3. Regional Influences category

The 'Regional Influences' category reflects biomechanical principles and neurological influences on pain that suggest dysfunction remote to the site of reported pain (Smart et al., 2012b; Schmid et al., 2013). The sub-categories identified include 'Kinetic Chain', 'Patho-neuro-dynamics' and 'Convergence' (Fig. 4).

3.1. Kinetic chain

Biomechanically-related issues such as proprioception, hypermobility and hypo-mobility are represented by the 'Kinetic chain' sub-category. The assumption is that when elements of the 'chain' are not providing normal support, or alternatively flexibility, then movement along the 'chain' is adversely affected (Slipman et al., 2000b; Winkelstein, 2004; Eygendaal et al., 2007; Johnston et al.,

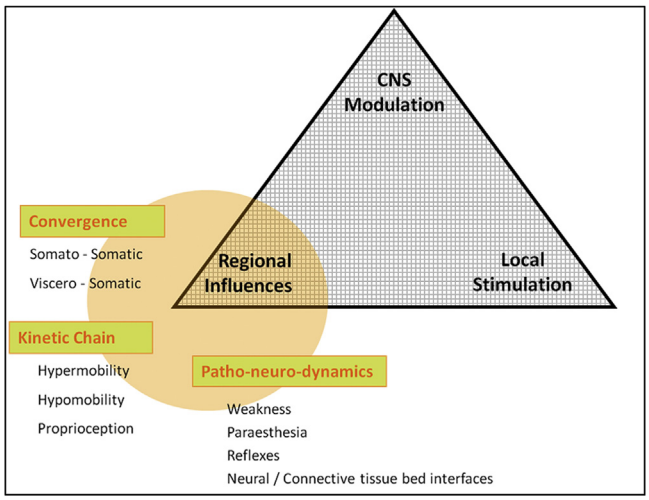

Fig. 4. Regional Influences Category. (Available under licence CC BY-NC Aus 3.0 at http://latrobe.libguides.com/content.php?pid=109542&sid=825367).

2008; Mitchell et al., 2008; Hodges and Tucker, 2011; Cook and Purdam, 2012; Hodges and Tucker, 2011 and Tobias et al., 2013). This may mean tissue remote to the biomechanical insufficiency is compressed or distracted or distended, with the result that mechanical nociceptors are triggered. This also takes into consideration that muscles, joints and connective tissues are actually a continual matrix and their movements are inter-related. Mechanics of the lumbo–pelvic complex involves the movement of numerous joints, muscles and their related fascia, as well as the connective tissue of adjacent visceral organs (Barker and Briggs, 1999; Robertson, 2001; Willard et al., 2012).

Recent reviews indicate biomechanical influences on pain are not straightforward. Assumptions about the influences of hyper- and hypo-mobility, the role of load in tendinopathy, and biomechanical explanations for neck and back pain have been challenged (Hetsroni et al., 2006; Cook and Purdam, 2012; McCluskey et al., 2012; Baster, 2013; Beinert and Taube, 2013; Littlewood et al., 2013; Mulvey et al., 2013). Therefore any discussion about the kinetic chain should not be independent of neurological influences, including cognitive, emotional and social modifiers.

3.2. Patho–neuro–dynamics (PND)

PND can be defined as when a stimulus of a position or movement exceeds the capability of a compromised nerve bed (Nee and Butler, 2006). For example if a nerve bed has altered functioning and accompanying inability to slide in relation to adjacent tissue, movement that may normally be benign, leads to transmission of nociceptive information along the nerve bed, and the pain may be perceived as arising from a different body part. Such compressive or entrapment neuropathic results in inflammatory changes, which produce altered functioning at the free end of the nociceptor, along the length of the nerve, at cell bodies and in addition leads to changes within the CNS (Nee and Butler, 2006; Zusman, 2009; Schmid et al., 2013).

Literature suggests it is movement of sensitised neural connective tissue that is involved in this response (Coppieters et al., 2005, 2006; Nee et al., 2012; Schmid et al., 2012). However it has also been considered that stretching of compromised blood vessels, lymphatics, fascia or other multi-segmental tissue may produce a similar effect of pain arising from limited movement of distant body parts (Wilson, 1994; Kelley and Jull, 1998; Walsh, 2005).

Nerves are considered to be vulnerable when passing through tunnels, when branching, and when passing through fascia or adjacent to bony surfaces (Walsh, 2005). As a result there exist a variety of interfacing places where nerve root and peripheral nerve PND is implicated in various disorders of both upper and lower quadrants (Pratt, 2005; Nee and Butler, 2006).

3.3. Convergence

Convergence relates to the so-called 'referral of symptoms' based upon shared neural structures and mis-location of the tissue source i.e. attributed to an area without nociceptor activity. Lumbar spine research has demonstrated referral to the back, pelvic and leg regions can occur, in the absence of peripheral nerve dysfunction, when various different tissues are stimulated. This includes the intervertebral discs, muscles, zygapophysial joints, sacroiliac joints, tendons, ligaments and periosteum (Slipman et al., 2000a; Cornwall et al., 2006; van der Wurff et al., 2006; Graven-Nielsen and Arendt-Nielsen, 2010). The referral patterns of different tissues are difficult to distinguish. It has become evident that using symptomatic presentations and clinical signs alone, it is very difficult to distinguish pain arising for example from an intervetebral disc or zygapophysial joint (Schwarzer et al., 1994, 1995; O'Neill et al., 2002; DePalma, 2012; Bogduk et al., 2013).

There is also complex integration of information between the somatic systems and the viscera and their adjacent connective tissue (Robertson, 1999; Gerwin, 2002). There can be changes related to processing occurring with visceral dysfunction, including: an increase in somatic nociceptor response and spontaneous neural activity; neurogenic inflammation, an expansion of normal somatic referral fields; and altered activity and oedema in muscles (Al-Chaer and Traub, 2002; Foreman, 2004; Giamberardino et al., 2010; van den Wijngaard et al., 2010). The afferent information can result from dysfunction of the organ(s), mechanical stimulation of the mesenteric connective tissue, or due to PND of the autonomic nervous system (Robertson, 1999).

3.4. The importance of the Regional Influences category

Subjective reports of distant yet related symptoms, biomechanical insufficiencies in adjacent tissue, associated paraesthesia, or changes in objective neurological signs and PND tests will suggest there are regional factors to be addressed.

Examples of assessment of the relative contribution of kinetic chain influences can include a presentation of knee pain requiring examination of proprioceptive abilities, muscle length and stability and joint play at both the foot and ankle and the lumbo-pelvic-hip complexes. Similarly an assessment of shoulder pain and movement dysfunction can include assessing the proprioceptive abilities at the gleno-humeral joint, tests of length and stability of the muscles of the upper quadrant and movement of the scapula-cervico-thoracic region.

Caution needs to be taken when linking regional influences to pain, especially neurological tissues in the physical examination. Identifying the involvement of specific nerve roots or peripheral nerves is clinically limited by the presence of any mechanisms sensitising the CNS as per the category of Central Modulation. Clinical reasoning as to the location of neural compromise is also difficult due to referral patterns overlapping, and the potential for bilateral referral of symptoms (Fukui et al., 1997; Cooper et al., 2007; Littlewood et al., 2013) In addition, referral patterns differ greatly between healthy individuals, and this variation becomes magnified for people with various pain presentations (Coderre and Katz, 1997; Bajaj et al., 2001; Slater et al., 2005; Graven-Nielsen, 2006; Fernández-de-las-Peñas et al., 2007).

Assessing the movement patterns and neural integrity of distal parts will highlight where connective tissue, joints or muscles are altered. These altered tissues become the targets for manual treatments or exercises. Alleviating regional influences will allow for the clearer identification of any matters occurring local to the site of symptoms.

4. Local stimulation category

The third category, local stimulation, further emphasises a pain mechanisms approach by identifying the nociceptive triggers that might occur within damaged or diseased tissue, or indeed healthy tissue being loaded in a manner which threatens damage (Fig. 5) (Gifford and Butler, 1997; Smart et al., 2012c). Of course, nociception, like central sensitisation, is not pain.

4.1. Chemical stimulation

It can be expected that when manual therapists are faced with predominantly nociceptive presentations in the clinical setting, they will be dealing with the chemical sequalae associated with tissue damage. By the time the injured person arrives at a clinic, tissues are generally not still being subjected to supra-threshold mechanical stressors e.g. the stressors sustained by a ligament at time of injury are no longer at play. The chemical nociceptive contribution to pain can be explained by the increased concentration of inflammatory substances (sometimes called the 'sensitising soup') stimulating free nerve endings of nociceptors and lowering thresholds of activation (Siddall and Cousins, 1997; Schmelz et al., 2003; Bove, 2008; Richards and McMahon, 2013). This peripheral sensitisation is most useful, as the resultant tenderness promotes protection of vulnerable injured tissue and is utilised by therapists to identify the potential location of superficial tissue pathology with reasonable accuracy. Evidence of inflammation and tissue disease or damage can help with estimations of the contribution of local stimulation to the person's pain experience (Smart et al., 2012c). However it is important the therapist is making judgements based on the prevailing pain mechanisms i.e. prevalence of sensitising chemicals, not simply that tissue damage equals pain.

An additional challenge to the chemical milieu is ischaemia. This may be due to circulatory disorders, or postural or movement limitations, altering tissue pH that in turn triggers chemical nociceptors (Steen and Reeh, 1993; Hodges and Tucker, 2011).

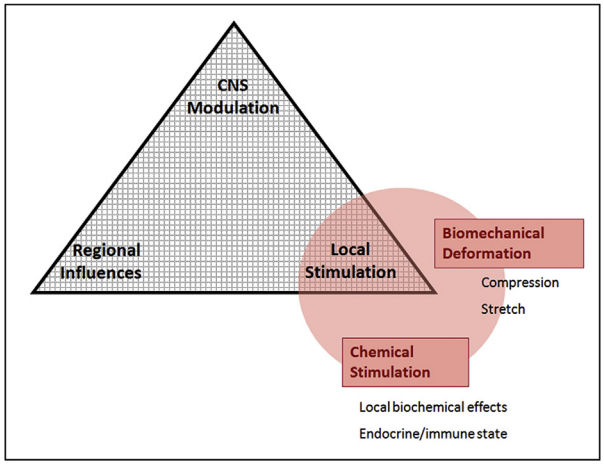

Fig. 5. Local Stimulation Category. (Available under licence CC BY-NC Aus 3.0 at http://latrobe.libguides.com/content.php?pid=109542&sid=825367).

The inclusion of 'endocrine and immune state' in this subcategory arises as hormonal and chemical influences are likely to have an enhanced affect locally to tissue that is already exposed to some degree of inflammation, ischaemia or other tissue threat (Jänig et al., 2006; Watkins et al., 2007).

4.2. Mechanical deformation

Mechanical nociceptors will respond to above-threshold distortion or derangement of tissues (Gifford and Butler, 1997; Smart et al., 2012c). Mostly, by the time the person presents at the clinic a guarding and protective posture means the original mechanical trigger – a supra-threshold distortion of tissue - is no longer relevant. The affected body part is postured for safety and protection but other mechanical stimuli may still be contributing to a person's pain report. The distortion of tissue – distraction and compression - caused by swelling or subluxation or altered mechanics, may still be adequate to trigger mechanical nociceptors, especially if these have been sensitised by the inflammation process. The painful movements often experienced with tissue changes are likely to be better explained as due to chemically-triggered sensitising mechanisms, not the mechanically-triggered danger signals indicative of potential tissue damage (Littlewood et al., 2013; Richards and McMahon, 2013).

4.3. The importance of the local stimulation category

Identifying this category as the predominant mechanism to address in treatment will be based on assessment findings that infer nociceptors are being triggered by chemical or mechanical stimulation, or peripheral sensitisation is influencing the transmission of danger messages. This may also include finding pain responses that are predictable to specific postural and/or movement patterns, and especially when occurring in the absence of symptoms and signs indicative of neurogenic involvement, CNS sensitisation or PND (Smart et al., 2012c).

In response to the estimation of a significant contribution from local stimulation factors, the treatment aims would be to manage the inflammation response, rectify any tissue distortion and normalise circulation, and address any mechanics, local or distal, impacting on the local stimulation.

5. Example of application of the model

Pain-provoking gleno-humeral movement provides a good example of how the Model could be applied. Central processes, such as the enhancement of signals at the dorsal horn of the spinal cord and reduced central inhibitory processes due to the perception of tissue vulnerability (i.e. central modulation), influence the pain output. Causal behaviours may include abnormal scapula-thoracic movement or posture, in parallel with regional patho–neuro–dynamics (i.e. regional influences). Another mechanism is likely to be altered pH triggering chemical nociceptors (i.e. local stimulation). Valuing these multiple contributors directs treatment to where the greater proportion of pain is arising from, and offers an increased number of management approaches. This reasoning can ensure a more comprehensive approach to addressing pain.

Apart from the clinical application, there may also be value in using the Model in pain research. Future researchers may consider the Model when designing methodology, identifying the similar or divergent contextual influences on pain for different sub-groups of subjects and accordingly, identifying appropriate outcome measures, and as a way to develop a shared understanding of pain amongst the research team. The application of the Model to research

design should encourage the selection of more sophisticated measures of the human pain experience, over simple pain ratings.

6. Conclusion

The Pain and Movement Reasoning Model is a simple tool to assist manual therapists reason through the complexities of the human pain experience. As an introduction to clinical reasoning, it highlights the need to address all dimensions of the pain experience and allows the therapist to document his or her estimate of the relevant contributions of the identified categories. By highlighting the range of contributors to an individual's pain experience, the Model also has the potential to increase the number of treatment options considered by a therapist, which will lead to the effective treatment of pain.

Reasoning for pain presentations and movement dysfunction will always be a complex process for therapists. The Model presented aims to provide structure to this work by acknowledging that central, regional and local factors can co-exist, yet management needs to be directed to what is the principal influence in the clinical presentation.

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