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Clinical Decision -making in LBP

Case report

Evidence-informed Clinical Decision-Making for Lower Extremity Hyperalgesia and Allodynia in a 42 year-old Woman Presenting with Low Back Pain -A Case Report

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ABSTRACT

Pain with spinal cord injured patient remains difficult to understand, diagnose and treat. The presence of peripheral disorders and other (supraspinal) central nervous system pathologies always increases the complexity of clinical manifestations related to pain in patients with persistent spinal cord pathology. It is a clinician's most important responsibility to determine the underlying causes of the patient's symptoms with differential assessment based on sound multidimensional clinical reasoning that is informed by appropriate evidence. The aim of this case report is to describe the importance of implicating available evidence in clinical decision making in a patient, referred for physiotherapy, with left lower quarter hyperalgesia and allodynia and multiple central nervous system disorders associated with spinal, peripheral musculoskeletal disorders and maladaptive psychosocial behaviour. This patient had a past medical history of cervical spine myelomalacia and right parietal lobe epilepsy. During routine musculoskeletal evaluation, we found Grade I spondylolisthesis (L5-S1 level) and a symptom (skin warming) suggestive of mild type-I complex regional pain syndrome (CRPS 1). She also presented with significant positive psychosocial illness. To confirm the predominant causative mechanisms of on-going pain, an evidence search was carried out and correlated with 'diagnostic' clinical reasoning. The evidence search provided accurate validation of all clinical and evaluation findings. In this way clinical decision making regarding the predominant underlying cause for her pain and disability was facilitated. This then helped propose a realistic prognosis as well as a plan for specific physiotherapy intervention and on-going medical management. This case is an example of the vital role and importance of utilising valid evidence for mechanism-based pain diagnosis and treatment in physiotherapy practice.

Key words: Neuropathic pain, Hyperalgesia, Allodynia, Evidence-based pain diagnosis, Clinical decision making.

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INTRODUCTION

Clinical assessment is an art which involves systematic, accurate determination of the etiological factors that influences the patient's symptoms. Since clinical presentation of a symptom can be influenced by multiple contributing factors, evaluating for various etiological factors that influence the symptoms are mandatory for successful treatment. It is a clinician's most important responsibility to determine the underlying causes of the patient's symptoms with differential assessment based on sound multidimensional clinical reasoning. Clinical reasoning is a continuing thought process towards clinical decision. Absence of sound clinical reasoning makes clinical assessment and treatment as a technical operation which often require direction from a decision maker.[1]

Chronic pain is the major symptom in neuromusculoskeletal practice which often needs multidimensional rationalization for accurate judgement of physical examination, intervention and prognosis. Pattern recognition of pain symptoms plays a major

role in identifying remote pathological mechanism and helps the examiner to correlate the relationship between multiple lesions and to rule out the causes for ongoing and maintained pain symptoms. When a patient experiences pain of neuromusculoskeletal origin the possibilities of developing consequent symptom aggravation with biological, environmental and psychosocial changes are high. Along with the knowledge of multistructural involvement and extent of pathology through pattern recognition, the dynamic interrelationship of changes in quality of life due to pain, perception of disability & disease are also most important factors that influence the process of pain evaluation and management.[2] These cues about active participation of patient with their context of pain and dysfunctions are strongly recommended for clinician / physical therapist whose interventions involve direct physical contact. Also it helps physical therapists / clinicians to measure the patient's self-efficacy and enhances additional information from the patient for better evaluation and therefore appropriate intervention.[3]

Pain caused by dysfunction of the nervous system is called as neuropathic pain. In addition to ongoing pain (i.e., stimulus-independent pain), patients with neuropathic pain may have heightened pain to stimuli applied on their skin (evoked pain). This enhanced stimulus-dependent pain is called hyperalgesia. In some patients, lightly stroking the skin may evoke pain. This pain to light touch is often called allodynia.[4] Among the various types and causes, neuropathic pain that develops after brain and/or spinal cord injury or disease is one of the most excruciating and difficult to succeed with treatment unless the mechanisms underlying the pain are not fully understood.[5]

Many studies have shown the occurrences of musculoskeletal pain in spinal cord injury (SCI) patient will be common above the level lesion,[6-8] which can be solved by correction of underlying pathology. Also it is known factor that neuropathic pain commonly presented at the level, and more commonly below the level after SCI which is always refractory to treatment.[6,9-11] In cases, where the presence of musculoskeletal lesion that occur below the level of SCI with below level neuropathic pain will be most refractory to treatment and, searching for mechanism of pain in these cases will be a diagnosis of exclusion and mandatory for optimal management. Using appropriate evidence in these circumstances, or an event with more etiological factors, will play a vital role in estimation of predominant causative factors among the multiple pathologies and helps to judge the possible contributions of rarely presenting pathologies.

Research evidence indicates that standard physical therapy intervention is widely employed for the attempted management of pain in SCI patients (88% of cases).[12] This consisted mainly of strengthening exercises, mobility exercises and heat therapy (55–68% of the population), while some 35% and 42% respectively received TENS and ice. These figures are an indication of its importance and the responsible role of the physical therapist working with pain after brain/spinal cord injuries or disease. The research does, however, emphasize the urgent need for sound evidence for short and long term pain relief with physical therapy management after brain/spinal cord injury. Recent studies on clinical reasoning by musculoskeletal physiotherapists confirmed the importance of understanding various models and mechanisms of pain for effective pain diagnosis and management.[13,14] This would seem particularly relevant for complex pain syndromes such as after brain/SCI with/without other musculoskeletal involvement.

The following presents a case report of direct evaluation of a 42-years old female referred to physiotherapy with severe low back pain and burning sensation in left lower limb and left waist with multiple pathologies, worked for evidence based clinical decision making of underlying causative mechanism of pain, with possible evidence-based reasoning of each mechanism. The aim of this case report is to describe the importance of implicating available evidence in clinical decision making.

SUBJECTIVE EXAMINATION

Chief Complaints

A 42-years old female presented with primary

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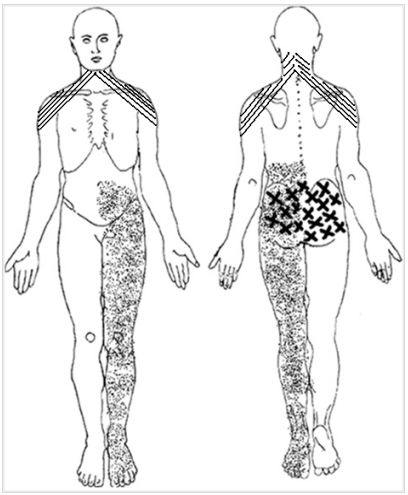
Figure 1: Body Chart – Pain distribution



- **LBP**
  - VAS score – 6 **(XXX)**
  - Extremely tiring & Aggravation with sitting, standing, walking. She was not able to lie prone due to pain. More in Night.

- **Burning pain** (Hyperalgesia)

No significant relieving factor for her low back and left lower limb pain.

- History of pain in bilateral neck-shoulder region a month back which has been resolved with medication at that time.

complaint of low back pain of eight months duration, weakness of right leg (7 months) and progressive burning sensation over left leg (3 months) with acute exacerbation of these symptoms following a fall 15 days earlier. The patient also complained of frequent falls over the past seven months due to epilepsy and frequent dizziness over the past week. She also had a minor complaint of diffuse multisite pain over various regions of body.

The low back pain was diffuse, poorly located and aching in type, presented bilaterally over lumbosacral and buttock region (Rt > Lt) with unilaterally distributed burning pain over left leg on L_3, L_4, L_5, S_1 & S_2 dermatomes and left side waist below to T_{12} spinous process. Low back pain was 6 on the VAS scale and it was extremely tiring and irritating. Her back pain was severely restricting her activities of daily living. She complained of aggravation of pain during sitting, standing, walking and while turning (either side) in bed; she was not able to lie prone due to pain. She reported burning pain in the leg (*Hyperalgesia*) that was aggravated with light touch, pinprick and hot weather. No burning was stated with cold weather. Both back pain and leg pain were more at night and was disturbing her sleep. There was no significant relieving factor for her low back and left lower limb pain. She had a history of bilateral neck-shoulder region pain a month back which had been resolved with medication at that time (Figure: 1).

History:

Her old history of fall was associated with sudden onset of loss of all limb movements (acute quadriparesis). MRI-cervical spine & CT-brain showed C2-3-4-5 diffuse disc bulge compressing bilateral neural foramina at C2-3 level with myelomalacic changes and right parietal region gliotic changes. Finally she had been medically diagnosed with pericentral cord syndrome with epilepsy. Her limb movements spontaneously recovered, but her right leg remained weaker. She continued with episodes of seizures following discharge after 18 days. She had relevant history of tubectomy and miscarriage 20 years back. There was no history of pelvic inflammatory diseases, DM, HTN and her bowel & bladder behaviour were normal. Her socioeconomic history was poor; she was highly disturbed with her husband's unemployment and lack of care from him Since the present & past history of the patient's complaints suggested both hyperalgesia & allodynia, we had administered the LANSS pain scale which also showed positive signs of neuropathic pain (LANSS Score >12).

The patient's presentation of chronic pain with confirmative neurological involvement and poor socioeconomic factor led us to concentrate on the psychological aspects of her pain. She reported highly 'irritable' and worsening pain,

and presented with significant depression, drowsiness, worsening mood, loss of appetite, feelings of self blame, loss of interest in enjoyable activities, dissatisfied family and sexual relationship and lack of sleep. She believed (*Cognition*) that her pain and disabilities were caused by untreatable brain and spinal cord damage / disease. She was very concerned about the progressive burning pain and thought that it was irreversible (an extremely poor prognosis). Her worries of low economic status, dissatisfied family life and compulsive resumption of ADL with severe pain and disability due to lack of social & family support were significantly associated with her anger. This may have contributed to greater pain 'irritability' and its impact on function. She appeared to have a significant lack of interest in living.

Her premorbid personality was poor with a history of low self-esteem due to inability to have children; her husband's drinking addiction and lack of socioeconomic support. She had poor coping strategies with which to accept the uncontrolled pain and disability. The patient was under medical treatment managed with Epsolin, Liofen, Evion, Elmecob, Voitm, Dolonex and Tizan. We suspect that, along with the ongoing pathology, her drowsiness may have been an added side effect of these drugs.

Physical Examination:

With well-constructed history we initiated a physical examination, evaluating firstly the neurological system to rule out the red-flag signs of movement examination. During sensory evaluations her right side upper and lower limbs showed mild proprioceptive deficit, whereas the left lower limb sensory examination showed hyperalgesia / allodynic responses to pinprick and light touch. Higher functions and vital examination were normal. Motor screening showed right side spasticity (Ashworth scale 1), brisk DTR bilaterally with equivocal plantar response, but no presence of clonus. Non equilibrium co-ordination testing was positive bilaterally. Dynamic sitting balance was good but painful. However, dynamic standing was severely affected due to pain. There were no signs of autonomic changes over her left & right lower limb and other regions (oedema, trophic changes on skin, nail & soft tissues). She reported mild warmth in her left leg compared to other regions of the body.

Palpation showed positive bilateral tenderness over L_{4-5}-S_1 and T_{11} – T_{12} interspinous process and paraspinal region, sacroiliac joint line, PSIS, long dorsal sacroiliac ligament, gluteus medius, piriformis, calf muscles, iliopsoas, groin and bilateral anterior iliac fossa. Left lower limb and waist (up to T_{12}) responding with a burning sensation to light touch and pinprick. Left lower limb was warm compared to right.

Range of motion was good in the upper limb, whereas active lower limb mobility was restricted due to pain (Rt > Lt). Both right and left active hip flexion (SLR & leg bent) caused pain at lumbosacral, sacroiliac and groin region and in the anterior thigh. Passive ROM was normal bilaterally but all with pain and a mild 'catch' at the end range. SLRT was positive bilaterally (70^{o}) with pain over low back region without any sign of sciatica on either side (there was no sign of neural mobility restriction). Faber's test showed positive sign bilaterally with initial range; she complained of pain over the groin region and lower back. Femoral nerve testing was not possible, because she was not able to lie prone. Her cervical spine mobility was normal and pain free, whereas lumbar spine mobility was restricted due to pain. Since the patient's neurological and ROM showed no significant neural claudication with movement, we tested muscle power in lower limbs using the precautionary measure of testing distal muscles first. Muscle power in left upper and lower limb was 'near normal'; whereas all muscle groups at right side were listed as 'fair'. She was unbalanced during standing and bore more weight on her left lower limb with trunk lateral shift / tilt towards the left. Significant forward propulsion of trunk was positive on standing. Her gait was independent with a pronounced limp and buckling to the right side.

Radiological Evaluation:

X-Ray lumbar spine lateral view showed Spondylolisthesis (Grade I) at L5-S1 level. Physical examination did not show a

'step sign' during evaluation. With regards to the cervical spine we took into consideration her earlier MRI report.

Diagnostic formulation:

Following completion of the evaluation the identified contributing factors for pain were listed based on the biopsychosocial model using the International Classification of Functioning *(ICF)* (Refer Table: 1). Biological factors were progressive SCI with central dysaesthesia syndrome due to myelomalacia (confirmation needed for its nature of progression), parietal lobe gliotic changes with epilepsy, musculoskeletal nociception and mechanical instability due to spondylolisthesis Grade I at L_5-S_1 level with mild autonomic trophic changes, type I complex regional pain syndrome (CRPS-II).

Among psychosocial contributing factors, psychological causes were her poor coping strategy to accept the uncontrolled pain and disabilities, depression, feelings of self-blaming, loss of interest in enjoyable activities, fear of irreversible pathologies that were causing pain and disability and having significant lack of interest in living with a willingness to die. The social and environmental factors were low self-esteem due to inability to have children, dissatisfied family life and the compulsive resumption of ADL with severe pain and disability, her husband's drinking addiction and lack of socioeconomic support.

With this number of contributing etiological factors it was extremely difficult to estimate, which of them were the actual/possible source of ongoing pain. The possibility exists that all may make a contribution. Hence, in order to appropriately estimate possible contributions to the current pain manifestations, evidence was sought for all identified contributing factors. Table:2 provides evidence for all identified bio-psychosocial factors we estimated were able to maintaining progressive pain of more than 3 months with a high susceptibility for becoming 'irritable'.

Based on the available evidence for informed mechanisms of ongoing pain and its possible correlations with present biomedical problems, the predominant contributing factors were tabulated (Table: 3). Together this evidence indicated a comprehensive final diagnosis of central neurogenic pain with a significant psychosocial contribution, peripheral neurogenic nociceptive pain with autonomic influence (CRPS-I), and inflammatory musculoskeletal pain. Based on IASP guidelines and classification of pain related to SCI, the case findings were consistent with below level central neuropathic pain, and below level musculoskeletal nociceptive pain related to SCI.

Discussion:

The present case study involved a chronic pain patient whose history, investigations and examination findings indicated multiple potential sources and mechanisms for her pain and disability. These multiple contributing factors are commonly cause nervous system 'sensitization' as a result of peripheral and central neuropathy and peripheral inflammation (including neurogenic inflammation), with an accompanying cognitive-emotional contribution. The following discusses the mechanisms likely to be involved in the contribution each of these factors make to the clinical picture. Preliminary changes in the patient's condition in response to appropriate mechanisms-based treatment are noted.

Spontaneous, continuous and intermittent pain as well as pain evoked by non-noxious stimulation (allodynia) can be indicative of central 'dysaesthetic' pain.[15, 16] Though the presence of pain, hyperalgesia, allodynia and other sensory symptoms like tingling, prickling, numbness are directly associated with dysfunctions / disease of nervous system,[17] these manifestations may also occur without clear nerve injury (eg type-I complex regional pain syndrome CRPS 1).[18] It is also believed that spinothalamocortical pathway abnormalities and 'hyperexcitability' of nociceptive spinothalamic tract neurons are common in patients with central dysaesthetic pain. Interestingly, clinical manifestations of diseases of the dorsal column/medial lemniscal system may occur without pain and related manifestations.[19] This is

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Table-1: Description of assessment findings based on the "*International Classification of Functioning* (*ICF*)"

Medical Diagnosis	$C_{2-3-4-5}$ diffuse disc bulge with neural foramina compression at C_{2-3} Myelomalacia at C_{2-3}(Spinal cord infarction) Pericentral cord syndrome Right parietal region gliosis with epilepsy
Structural Impairment	Quadriparesis, Forward headed posture & Forward propulsion of trunk Spondylolisthesis Grade I at L_5-S_1
Functional Impairment	PAIN DYSFUNCTION: Hyperalgesia & Allodynia - Over left lower limb and waist Mild warmth in left leg compared to other regions of body Type II complex regional pain syndrome (CRPS-I) Joint Tenderness - T_{11} – T_{12} & L_{4-5}-S_1 region, PSIS, Sacroiliac joint line Muscle Tenderness - Gluteus medius, Piriformis, Iliopsoas, Calf muscles SENSORY DEFICIT: Right upper & lower limbs mild proprioceptive deficit Right side spasticity (Ashworth scale 1) Brisk DTR bilaterally with equivocal plantar response Impaired non equilibrium co-ordination bilaterally ROM: Active lower limb and lumbopelvic mobility restriction with pain Passive ROM normal bilaterally with pain SLRT was positive bilaterally (70^{o}) without sciatica Faber's test showed positive sign bilaterally MUSCLE POWER: Left side - near Normal & Right side – Fair
Activity Limitation	Unable to sit, stand, walk, turn in bed & lie prone Unable to carry out ADL
Participation Restriction	Not involving in Household works Not willing to work due to pain
Personal Factors	Thinking and Fear about major pathologies or diseases Highly worried about resting & progressing burning pain Feelings of self-blaming and poor coping with pain Depression, worsened mood, loss of appetite Lack of sleep & Dissatisfied sexual relationship Low self-esteem due to inability to have children Dissatisfaction about quality of active life and ADL Lack of interest in living and willingness to die
Environmental Factor	Dissatisfied family and social support Husband's drinking addiction Low economic back ground

reinforced by Boivie et al in their statement[20] "…a rule without apparent exception is that the lesion leading to pain must directly involve the nociceptive pathways". Thus, altered sensitivity to thermal and nociceptive stimuli within the painful area would appear to endorse a (not yet fully understood) role for spinothalamic tract lesions in

central neuropathic pain. Nevertheless, some studies have found no difference in altered sensitivity to thermal and nociceptive stimuli in patients with and without pain after lesions of the central nervous system.[21 -24] It would appear that abnormalities of central nervous system structure alone may not always be a sufficient causal mechanism for pain related clinical manifestation. At least with peripheral neuropathic pain pain-related abnormalities (discharges) develop in nearby uninjured, as well as (later) injured, peripheral afferents supplying the affected region.

The manifestation of clinical pain is known to vary with the type and combinations of disease, the structures involved as well as throughout the time course of a given disease. It is therefore possible that the mechanisms producing pain will be different and multiple in different individuals. Understanding and identifying these differential mechanisms of pain may play a vital role in improving outcomes in suitable patients with appropriate treatment methods.

A basic understanding of central neuropathic pain may be obtained from an understanding of the mechanisms of secondary (mechanical) hyperalgesia and central sensitization, since the (spontaneous) pain, allodynia and hyperalgesia of neuropathic pain are consistent with these mechanisms.[25] The reported possible key mechanisms[22,26-28] of neuropathic pain were, neuronal hyper excitability due to inflammatory process, loss of intraspinal and descending inhibitory pathways, and convergence of tactile and nociceptive fibres on to dorsal horn neurons, enhancement of synaptic efficacy of tactile fibres,[29 -32] sensitization of wide-dynamic range neurons. The role of primary afferent and dorsal horn neuron with pain provocation is now reasonably well established; however, little is known about mechanism of brain, cognitive and perceptual aspects of pain. Nevertheless, it is believed that abnormalities of supraspinal and subcortical structures may cause considerable 'plastic' changes at the sensory cortical level which undoubtedly play a major role in shaping the pain experience**.**

Ischemic disease of the spinal cord is defined as myelomalacia. The causes of cord ischemia and its clinical consequences include a spinal cord cyst, spinal column instability with spinal cord compression, spinal cord tethering, microcystic spinal cord degeneration or gliosis[33,34.] There is a reported 0.3% to 3.2% prevalence rate.[35] The most predominant feature of myelomalacia is tethering of spinal cord due to scar formation. It is also believed that this tethering may be associated with local hemorrhage due to recumbent postural strain that leads to chronic ischemia.[36-38] All of these factors contribute to adhesion formation of the spinal cord and nerve roots over dorsal and lateral aspects of the dural sac. This presents with signs and symptoms that include sensorimotor deterioration, local and/or radicular pain, increased spasticity, autonomic dysreflexia, and sphincter dysfunction.

Pain manifestation in epilepsy is most unusual. Pain in epilepsy is commonly associated with structural lesions of parital lobe like tumours, gliosis and scaring.[39,40] The occurrence of partial lobe epilepsy is very rare, with a prevalence of 1.4%. It falls into a somatosensory type of seizure, which often presents with contralateral (rarely ipsilaterally/bilateral) sensory alteration that includes tingling, numbness, pricking, tickling, crawling and/or electric shock like sensations. Pain is the second most common somatosensory experience in parietal lobe epilepsy, often described as stabbing, intense, and torturing. A burning sensation is more common than feelings of cold.[41,42]

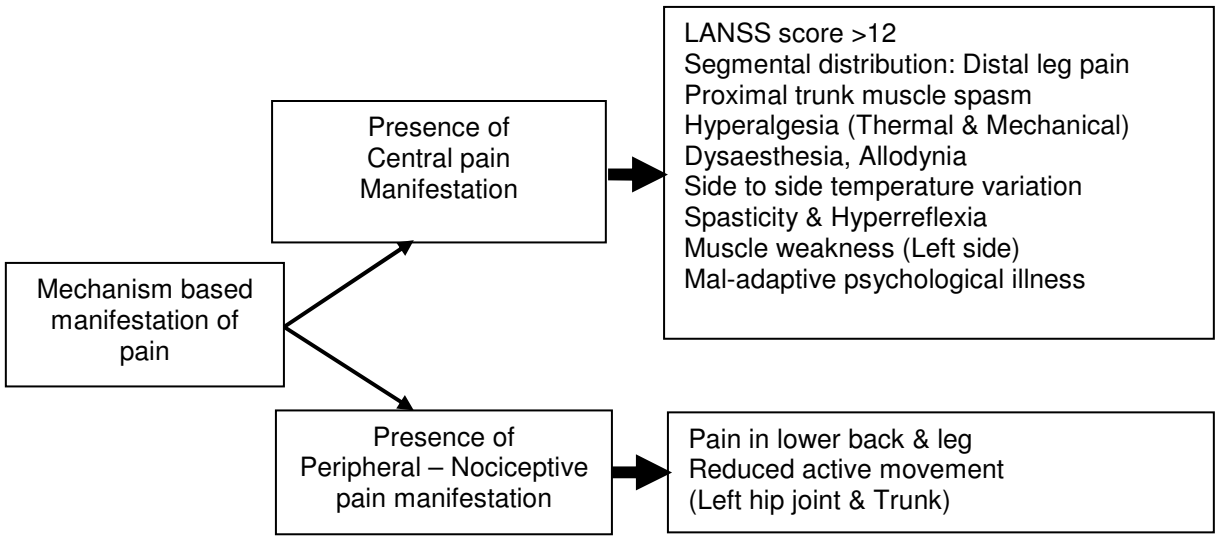
Complex regional pain syndrome (CRPS) is a (disputed) neuropathic pain disorder occurring after musculoskeletal trauma (type I) or direct injury to nerve (type II). The clinical manifestation involves autonomic, trophic, motor and sensory disturbances along with pain related changes.[18,43] The proposed diagnostic criteria[43] of CRPS from IASP are the presence of continuing pain and at least one or more of the following: pain to light touch (allodynia) or pinprick, hyperaesthesia, temperature asymmetry, skin colour changes / asymmetry, oedema, weakness, restricted movements, and trophic changes. Sensory abnormalities in CRPS always spread in a hemisensory

manner,[44] ipsilaterally and even contralaterally.[45] The sequence[46,47] of CRPS symptoms are characterized by the presence of regional inflammation followed by functional atrophy. However, the actual time course for occurrences of sensory abnormalities remains unclear.[48] Peripheral neurogenic inflammation (likely with this patient), small-fibre axonal degeneration and cortical reorganization are hypothetical pathophysiological mechanisms of CRPS, which is now recognized as having a significant central nervous system mechanism.[49,50] Paradoxical heat sensation is a sensation of hot or burning pain to mild cold stimulation following a preceding mild warm stimulus.[51] In patients with a positive paradoxical heat sensation a combination of inflammation and small fibre degeneration are considered as a two major pathomechanisms acting in acute CRPS.[51]

The presence of aseptic inflammation is considered a valuable critical feature of acute CRPS.[52] Since a neurogenic mechanism of pain strongly contributes to aseptic inflammation, neurogenic inflammation might be possible starting point of an inflammatory process with acute CRPS.[53] Further hallmarks of an of inflammatory process such as the presence of oedema, side to side difference in skin temperature and heat hyperalgesia seen with acute CRPS patients may indicate peripheral sensitization of heat sensitive (small fibre) C-fibre nociceptors.[54-56] There is also evidence that heat hyperalgesia is absent in chronic stages of CRPS.

The pain related clinical manifestations with this patient are consistent with the evidence based literature discussed above for neuropathic pain and spinal cord pathology such as myelomalacia, parietal lobe gliotic changes, and type-I complex regional pain syndrome (Refer Table: 2).

Table-2: Evidence informed mechanism based manifestations related to current ongoing neuropathic & nociceptive pain



The evidence supports the possible contributions of multiple structural pathologies present in this patient and helps explain the mechanism(s) of ongoing pain and profound related problems (Refer Table: 3). The possible presence of an inflammatory musculoskeletal lesion (spondylolisthesis) is proposed as a factor contributing to an acute exacerbation of neuropathic pain related manifestations such as heat hyperalgesia and side to side skin temperature variation. Though the presence of allodynia and hyperalgesia was predominant finding from the initial examination, the subsequent information and evaluation provided a sound rationale for possible

biomedical causes for ongoing pain related manifestations.

It is now well recognized that the presence of chronic pain with increased irritability both triggers and in turn is exacerbated by alterations in biobehavioral factors.[57,58] The literature confirms that the presence of chronic pain with psychological illness is associated with depression, anxiety, activity limitation, catastrophic and erroneous perceptions.[59,60] Together these initiate a vicious cycle of fear regarding the meaning and prognosis of pain and reinjury and safety-seeking (avoidance) behaviors and hyper vigilance. This serves to help keep the central nervous system sensitised[61] with on-going (mis)perceptions, beliefs, attributions and continuous development of disability.[62,63] The literature cited is also consistent with this patient's premorbid findings, psychological illness and undesirable environmental factors.

The evidence reviewed provided an informed understanding of the underlying causes of pain with this patient and helped with decisions concerning likely prognosis and current medical management. Clinical reasoning based on scientific evidence not only helped with determining the predominant basis for ongoing pain, but also provided valuable information concerning a possible contributing role for silent pathological entities like parietal lobe gliotic changes. Together these helped to reasonably estimate the probable contribution of various etiologies to ongoing pain-related manifestations and optimally weight the multiple pathologies.

Among the measures designed to discriminate between neuropathic and non-neuropathic pain, currently the full LANSS and self-administered S-LANSS are described as valid choices. In keeping with IASP taxonomy, the use of pain intensity measures (using various rating scales) along with the presence/absence of mechanical and thermal hyperalgesia and allodynia are recommended measures for the assessment of neuropathic and chronic pain.[64] Though LANSS has not been regularly used for persons with SCI this scale was selected on the grounds that the patient showed a non-traumatic history of spinal cord pathology with intact sensation at and below the level of injury. The clinical reasoning and decision making process is explained in Figure: 2 (*clinical reasoning and decision making algorithm).*

Table-3: Predominant contributing / etiological factor and its evidence informed estimation with ongoing pain mechanism

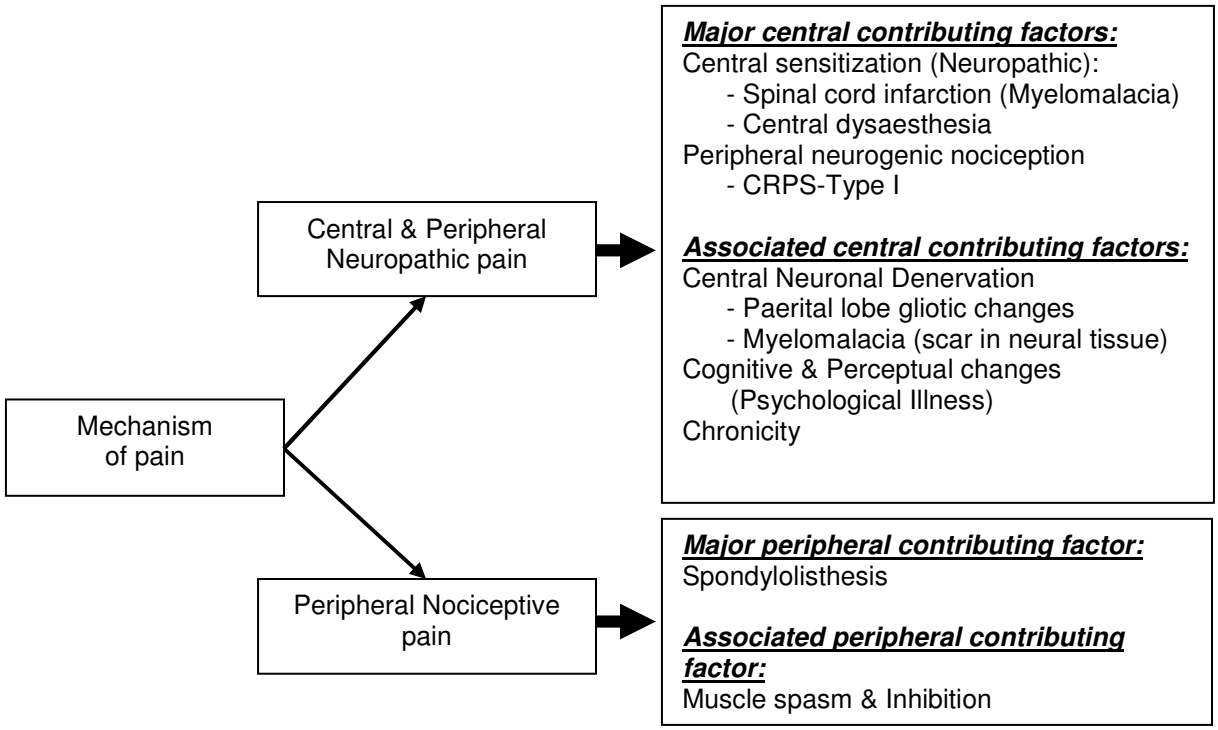


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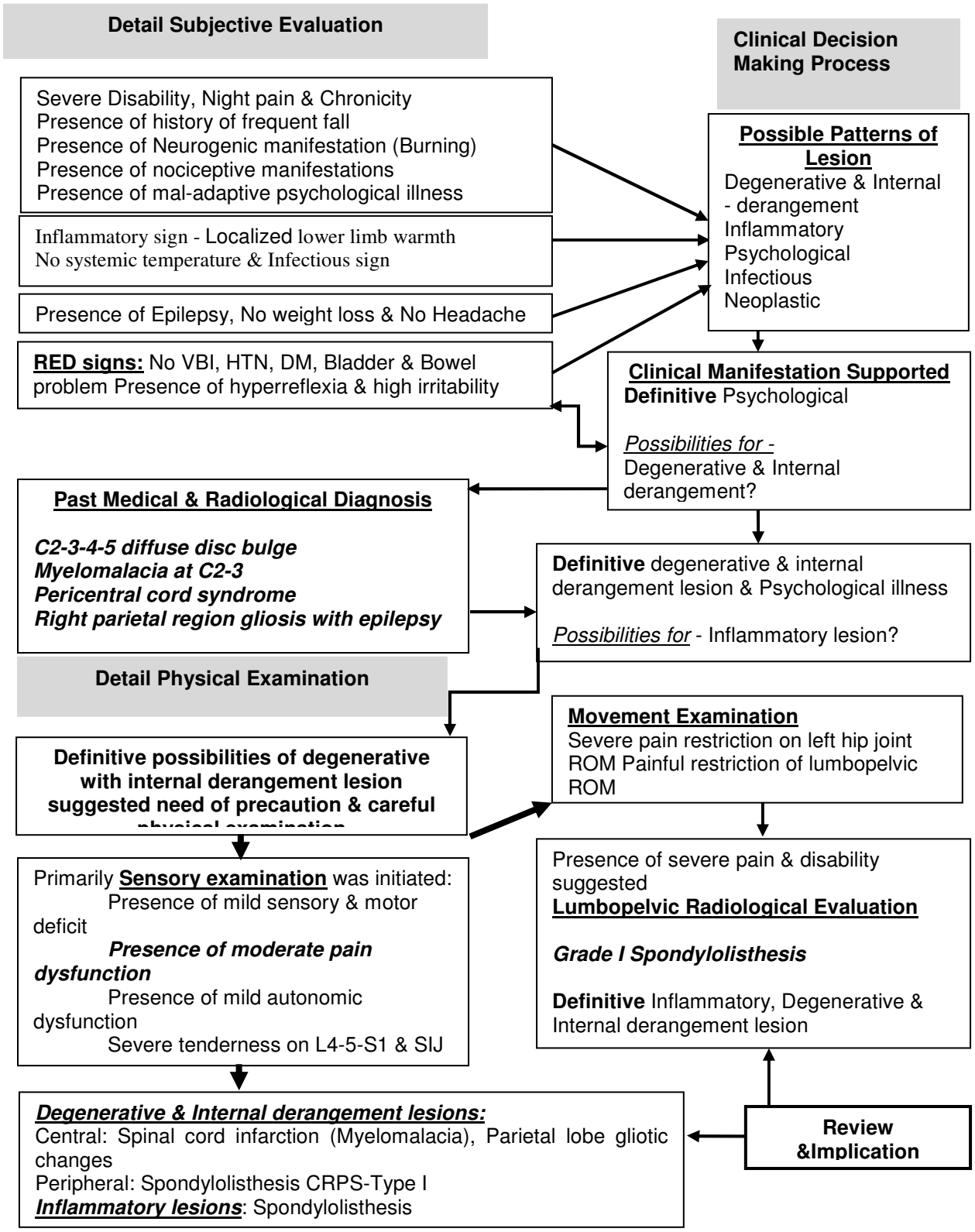
Figure -2: Clinical reasoning and decision making algorithm



Clinical decision-making and treatment planning:

Evidence supported clinical reasoning for causative mechanisms of ongoing pain and disability provided guidance when it came to the prescription of suitable rehabilitative measures within the now widely recognized bio-psychosocial framework (eg cognitive behavioral motor learning). Thus, the patient received appropriate information concerning the causes of pain and nature of the involved pathological factors. Further (self-help, functionally oriented) advice and reassurance aided the patient in developing a good rapport with the physiotherapist. As a result she began listening and thinking positively about recommended interventions. Specific soft tissue manipulations, specific peripheral articular mobilization, specific segmental stabilization procedures and TENS were then planned. Cognitive behavioural therapy with specialized professionals was recommended. Thought might also be given to the use of recent interventions such as mindfulness and acceptance of pain with this patient. It is worth noting that the immediate effect following the use of TENS and specific manual therapy was positive.

Conclusion:

This case provides an example of the opportunity for clinical physiotherapists involved in the management of complex pain problems that include combinations of musculoskeletal and multilevel nervous system pathology to use evidence based rationalization in order to gain an in-depth understanding of the different causal mechanisms. It is hoped that such insight would then lead to informed decisions regarding appropriate treatment and realistic outcome expectations.

REFERENCES

1. Higgs J, Jones M. *Clinical Reasoning in the Health Profession.* Butterworth-Heinemann, 1995, London.

2. Edwards. I. *Unpublished paper submitted in partial fulfillment of the Graduate Certificate in Orthopedics*, School of Physiotherapy, University of South Australia, 1995.

3. Mattingly C. What is clinical reasoning? *Am J Occup Ther.* 1991;45:979-86.

4. Treede RD, Meyer RA, Raja SN, Campbell JN. Peripheral and central mechanisms of cutaneous hyperalgesia. Prog Neurobiol. 1992; 38:397–421.

5. Finnerup NB, Biering-Sorensen F, Johannesen IL, et al. Intravenous lidocaine relieves spinal cord injury pain: a randomized controlled trial. *Anesthesiology.* 2005;102:1023–30.

6. Donovan WH, Dimitrijevic MR, Dahm L, et al. Neurophysiologic approach to chronic pain following spinal cord injury. *Paraplegia.* 1982; 20:135–46.

7. Frisbie JH, Aguilera EJ. Chronic pain after spinal cord injury: an expedient diagnostic approach. *Paraplegia.* 1990;28:460–5.

8. Siddall PJ, Yezierski RP, Loeser JD. *Pain following spinal cord injury: clinical features, prevalence, and taxonomy*. IASP Newsletter. 2000:3–7.

9. Britell CW, Mariano AJ. *Chronic pain in spinal cord injury*. In: Walsh NE, ed. Physical Medicine and Rehabilitation: State of the Art Reviews. Philadelphia, PA: Hanley and Belfus. 1991:71–82.

10. Turner JA, Cardenas DD, Warms CA, McClelland JM. Chronic pain associated with spinal cord injuries: a community survey. *Arch Phys Med Rehabil.* 2001;82:501–8.

11. Cardenas DD, Turner JA, Warms CA, Marshall H. Classification of chronic pain associated with spinal cord injuries. *Arch Phys Med Rehabil.* 2002; 83:1708–14.

12. Cardenas DD, Jensen MP. Treatments for Chronic Pain in Persons with Spinal Cord Injury: A Survey Study. *J Spinal Cord Med.* 2006; 29:109–17.

13. Smart K, Doody C. Mechanisms-based clinical reasoning of pain by experienced musculoskeletal physiotherapists. *Physiotherapy.* 2006;92:171–8.

14. Smart K, Doody C. The clinical reasoning of pain by experienced musculoskeletal physiotherapists. *Man Ther.* 2007;12:40–9.

15. Davidoff G, Roth EJ. *Clinical characteristics of central (dysaesthetic) pain in spinal cord injury patients.* In: Casey KL, ed. Pain and central nervous system disease: the central pain syndromes. New York: Raven Press. 1991;77-83.

16. Boivie J. *Central pain.* In: McMahon, SB.; Koltzenburg, M., editors. Wall and Melzack's Textbook of Pain. London: Elsevier; 2006;1057-74.

17. Scadding, JW, Koltzenburg, M. *Painful peripheral neuropathies.* In: McMahon, SB.; Koltzenburg, M. Editors: Wall and Melzack's Textbook of Pain. London: Elsevier; 2006; 973-99.

18. Stanton-Hicks M, Janig W, Hassenbusch S, Haddox JD, Boas R, Wilson P. Reflex sympathetic dystrophy: changing concepts and taxonomy. *Pain.* 1995; 63:127–33.

19. Cook AW, Browder EJ. Function of Posterior columns in man. *Arch Neurol.* 1965;12:72–9.

20. Boivie J, Leijon G, Johansson I. Central post-stroke pain–a study of the mechanisms through analyses of the sensory abnormalities. *Pain.* 1989;37:173–85.

21. Defrin R, Ohry A, Blumen N, Urca G. Characterization of chronic pain and somatosensory function in spinal cord injury subjects. *Pain.* 2001;89:253–63.

22. Finnerup NB, Jensen TS. Spinal

33. Schneider RC, Knighton R.

44. Rommel O, Malin JP, Zenz M,

cord injury pain–mechanisms and treatment. *Eur J Neurol.* 2004; 11: 73–82.

23. Ducreux D, Attal N, Parker F, Bouhassira D. Mechanisms of central neuropathic pain: a combined psychophysical and MRI study in syringomyelia. *Brain.* 2006;129:963–76.

24. Finnerup NB, Pedersen LH, Terkelsen AJ, Johannesen IL, Jensen TS. Reaction to topical capsaicin in spinal cord injury patients with and without central pain. *Exp Neurol.* 2007;205:190–200.

25. Meyer RA, Ringkamp M. Campbell JN, Raja SN. *Peripheral mechanisms of cutaneous nociception.* In: McMahon SB, Koltzenburg M., editors. Wall and Melzack's Textbook of Pain. London: Elsevier. 2006;3-34.

26. Yezierski RP. Spinal cord injury: a model of central neuropathic pain. *Neurosignals.* 2005;14:182–93.

27. Waxman SG, Hains BC. Fire and phantoms after spinal cord injury: Na+ channels and central pain. *Trends Neurosci.* 2006;29:207–15.

28. Finnerup NB, Sorensen L, Biering-Sorensen F, Johannesen IL, Jensen TS. Segmental hypersensitivity and spinothalamic function in spinal cord injury pain. *Exp Neurol.* 2007;207:139–49.

29. Campbell JN, Raja SN, Meyer RA, Mackinnon SE. Myelinated afferents signal the hyperalgesia associated with nerve injury. *Pain.* 1988;32:89–94.

30. LaMotte RH, Shain CN, Simone DA, Tsai EFP. Neurogenic hyperalgesia: Psychophysical studies of underlying mechanisms. *J Neurophysiol.* 1991; 66:190–211.

31. Torebjörk HE, Lundberg LER, LaMotte RH. Central changes in processing of mechanoreceptive input in capsaicin-induced secondary hyperalgesia in humans. *J Physiol.* 1992;448:765–80.

32. Magerl W, Fuchs PN, Meyer RA, Treede RD. Roles of capsaicin-insensitive nociceptors in cutaneous pain and secondary hyperalgesia. *Brain.* 2001;124:1754–64.

Chronic neurological sequelae of acute trauma to the spine and spinal cord. Part III. The syndrome of chronic injury to the cervical spinal cord in the region of the central canal. *J Bone Joint Surg (Am).* 1959;41:905–19.

34. Ragnarsson TS, Durward QJ, Nordgren RE: Spinal cord tethering after traumatic paraplegia with late neurological deterioration. *J Neurosurg.* 1986; 64:397–401.

35. Lee TT, Arias JM, Andrus HL, et al. Progressive posttraumatic myelomalacic myelopathy: treatment with un tethering and expansive duraplasty. *J Neurosurg.* 1997; 86:624–8.

36. Adams CBT, Logue V. Studies in cervical spondylotic myelopathy. I. Movement of the cervical roots, dura and cord, and their relation to the course of the extrathecal roots. *Brain.* 1971;94:557–68.

37. Pang D, Wilberger JE Jr: Tethered cord syndrome in adults. *J Neurosurg.*1982;57:32–47.

38. Hoffman HJ, Hendrick EB, and Humphreys RP: The tethered spinal cord: its protean manifestations, diagnosis and surgical correction. *Childs Brain.* 1976;2:145–55.

39. Wilkinson HA. Epileptic Pain. An uncommon manifestation with localizing value. *Neurology (Minneap).* 1973;23:518-20.

40. Salanova V, Andermann F, Rasmussen T, et al. Tumoural parietal lobe epilepsy. *Brain.* 1995;118:1289-304.

41. Williamson, PD, Spencer S. Clinical and EEG features of complex partial seizures of extra temporal origin. *Epilepsia*. 1986; 27(Suppl. 2):546-63.

42. Siegel AM, Williamson PD. Parietal lobe epilepsy. *Adv Neurol.* 2000;84:189-99.

43. Bruehl S, Harden RN, Galer BS, et al. External validation of IASP diagnostic criteria for Complex Regional Pain Syndrome and proposed research diagnostic criteria. International Association for the Study of Pain. *Pain.* 1999;81:147–54.

Janig W. Quantitative sensory testing, neurophysiological and psychological examination in patients with complex regional pain syndrome and hemisensory deficits. *Pain.* 2001;93:279–93.

45. Thimineur M, Sood P, Kravitz E, Gudin J, Kitaj M. Central nervous system abnormalities in complex regional pain syndrome (CRPS): clinical and quantitative evidence of medullary dysfunction. *Clin J Pain.* 1998;14:256–67.

46. Bonica JJ. *Causalgia and other reflex sympathetic dystrophies.* In: Bonica JJ, ed. Management of pain. Philadelphia, PA: Lea and Feibinger.1990;220–43.

47. Veldman PH, Reynen HM, Arntz IE, Goris RJ. Signs and symptoms of reflex sympathetic dystrophy: prospective study of 829 patients. *Lancet.* 1993;342:1012–6.

48. Birklein F, Riedl B, Sieweke N, Weber M, Neundorfer B. Neurological findings in complex regional pain syndromes–analysis of 145 cases. *Acta Neurol Scand.* 2000;101:262–9.

49. Huge V, Lauchart M, Fo¨rderreuther S, et al. Interaction of Hyperalgesia and Sensory Loss in Complex Regional Pain Syndrome Type I (CRPS I). *PLoS ONE* 2008;3(7):e2742.

50. Naleschinski D, Baron R. Complex regional pain syndrome type 1: neuropathic or not? *Curr Pain Headache Rep.* 2010;14:196-202.

51. Rolke R, Baron R, Maier C, Tolle TR, Treede RD, et al. Quantitative sensory testing in the German Research Network on Neuropathic Pain (DFNS): Standardized protocol and reference values. *Pain.* 2006;123:231–43.

52. Veldman PH, Reynen HM, Arntz IE, Goris RJ. Signs and symptoms of reflex sympathetic dystrophy: prospective study of 829 patients. *Lancet.* 1993;342:1012–6.

53. Taha R, Blaise G. Is complex regional pain syndrome an inflammatory process? Theories and therapeutic implications. *Can J Anesth.* 2007;54:249-53.

54. Blumberg H, Hoffmann U, Mohadjer M, Scheremet R. Sympathetic nervous system and pain: a clinical reappraisal. *Behav Brain Sci.* 1997;20:426–34.

55. Birklein F, Riedl B, Claus D, Neundorfer B. Pattern of autonomic dysfunction in time course of complex regional pain syndrome. *Clin Auton Res.* 1998;8:79–85.

56. Treede RD, Meyer RA, Raja SN, Campbell JN. Peripheral and central mechanisms of cutaneous hyperalgesia. *Prog Neurobiol.* 1992;38:397–421.

57. Main CJ, Wood PL, Hollis S, et al. The Distress and Risk Assessment Method: a simple patient classification to identify and evaluate the risk of poor outcome. *Spine.* 1992;17:42–52.

58. Feuerstein M, Beattie P. Biobehavioural factors affecting pain and disability in low back pain: mechanisms and assessment. *Phys Ther.*1995;75:267–81.

59. Crombez G, Vlaeyan JWS, Heuts PHTG, et al. Pain-related fear is more disabling than pain itself: evidence on the role of pain –related fear in chronic back pain disability. *Pain.* 1999;80:329-39.

60. Waddell G. Chronic low back pain, psychological stress, and illness behavior. *Spine.* 1984;9:209.

61. Wall PD. Interview: ready for action (ed: McCrone J). *New Scientist.* 1999;5:48-51.

62. Main CJ, Watson PJ. Psychological aspects of pain *Man Ther.* 1999;4:203-15.

63. Moseley GL, Nicholas MK, Hodges PW. A randomized controlled trial of intensive neurophysiology education in chronic low back pain. *Clin J Pain.* 2004;20:324–30.

64. Bryce TN. Pain after spinal cord injury: an evidence-based review for clinical practice and research. *J Spinal Cord Med.* 2007;30:421–40.

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CONFLICTS OF INTEREST

None identified or declared.