Masterclass

Diagnosis and classification of chronic low back pain disorders: Maladaptive movement and motor control impairments as underlying mechanism

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Abstract

Low back pain (LBP) is a very common but largely self-limiting condition. The problem arises however, when LBP disorders do not resolve beyond normal expected tissue healing time and become chronic. Eighty five percent of chronic low back pain (CLBP) disorders have no known diagnosis leading to a classification of 'non-specific CLBP' that leaves a diagnostic and management vacuum. Even when a specific radiological diagnosis is reached the underlying pain mechanism cannot always be assumed. It is now widely accepted that CLBP disorders are multi-factorial in nature. However the presence and dominance of the patho-anatomical, physical, neuro-physiological, psychological and social factors that can influence the disorder is different for each individual. Classification of CLBP pain disorders into sub-groups, based on the mechanism underlying the disorder, is considered critical to ensure appropriate management. It is proposed that three broad sub-groups of CLBP disorders exist. The first group of disorders present where underlying pathological processes drive the pain, and the patients' motor responses in the disorder are adaptive. A second group of disorders present where psychological and/or social factors represent the primary mechanism underlying the disorder that centrally drives pain, and where the patient's coping and motor control strategies are mal-adaptive in nature. Finally it is proposed that there is a large group of CLBP disorders where patients present with either movement impairments (characterized by pain avoidance behaviour) or control impairments (characterized by pain provocation behaviour). These pain disorders are predominantly mechanically induced and patients typically present with mal-adaptive primary physical and secondary cognitive compensations for their disorders that become a mechanism for ongoing pain. These subjects present either with an excess or deficit in spinal stability, which underlies their pain disorder. For this group, physiotherapy interventions that are specifically directed and classification based, have the potential to impact on both the physical and cognitive drivers of pain leading to resolution of the disorder. Two case studies highlight the different mechanisms involved in patients with movement and control impairment disorder outlining distinct treatment approaches involved for management. Although growing evidence exists to support this approach, further research is required to fully validate it.
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1. The need to classify CLBP disorders

Low back pain (LBP) is common with up to 80% of people reporting LBP over their life time (Dillingham, 1995). The majority of acute LBP disorders resolve within a 4 week period although recurrence is common (Croft et al., 1998). A small number of disorders (10–40%) become chronic and represent a major cost burden for society (Dillingham, 1995; Croft et al., 1998). In spite of the small number of pathological conditions that can give rise to back pain, most cases (85%) are classified as "non-specific" because a definitive diagnosis cannot be achieved by current radiological methods (Dillingham, 1995). Even when a specific diagnosis is

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made, the validity of the diagnosis can often be questioned. This leaves a diagnostic and management vacuum (Leboeuf-Yde et al., 1997). This situation commonly results in the "signs and symptoms" of the disorder being treated without consideration for the underlying basis or mechanism for the pain disorder.

It is well recognized that the classification of chronic low back pain (CLBP) disorders into homogenous groups, and the application of specific interventions tailored for these groups is likely to enhance treatment efficacy (Leboeuf-Yde et al., 1997). It is also well established that LBP is a multi-dimensional problem (Borkan et al., 2002; McCarthy et al., 2004). These dimensions consist of pathoanatomical, neurophysiological, physical and psychosocial factors (Waddell, 2004). To date, the majority of studies that relate to the classification of back pain have focused only on a single dimension of the problem, rather than consideration being given to all dimensions of LBP (Ford et al., 2003). For a classification system to be clinically useful it should be based on *identifying* the underlying mechanism(s) driving the disorder, in order to guide targeted interventions, which in turn should *predict* the outcome of the disorder.

2. Models for the diagnosis and classification of CLBP

Current approaches or models used for the diagnosis and classification of CLBP have tended to only focus on a single dimension of the disorder, limiting their validity (Ford et al., 2003). The following overview is not designed to be exhaustive, but highlights to the clinician the strengths and weaknesses of these different approaches.

2.1. Patho-anatomical model

The traditional medical approach to diagnosis of CLBP has been from a pathoanatomical perspective (Nachemson, 1999). The findings of intervertebral disc (IVD) and facet joint degeneration, annular tears, IVD prolapse, spondylolisthesis, foraminal and spinal stenosis with associated nerve pain are commonly assumed to be related to back pain (and in some cases associated neurogenic pain), with interventions provided on the basis of this assumption (Nachemson, 1999).

However, the problem with pathoanatomical diagnoses for CLBP is that many 'abnormal' findings are also commonly observed in the pain free population and pathoanatomical findings correlate poorly with levels of pain and disability (Nachemson, 1999). Frequently, little consideration is given to the confounding impact of psycho-social, neuro-physiological and physical factors that may co-exist and contribute to the underlying basis

of these disorders (Nachemson, 1999). Because of this, even when a specific pathoanatomical diagnosis can been made, there is still a need to classify the disorder based on the mechanism(s) that drive the pain disorder to ensure appropriate management.

2.2. Peripheral pain generator model

More recently there has been a focus on the identification of the painful structure (peripheral pain generator) based on the patient's history, area of pain, clinical examination findings and diagnostic blocks (Donatelli and Wooden, 1989; Laslett and Williams, 1994; Schwarzer et al., 1994; Bogduk, 1995; Bogduk, 2004). This has led to studies that have reported that the majority of chronic back pain originates in the IVD (45%), with a smaller number of subjects with facet joint (20%) and sacro-iliac joint (15%) pain (Bogduk, 1995). These studies have led to diagnostic and therapeutic procedures to identify, block or denervate the nociceptive source (Bogduk, 2004). The major limitation of this treatment model is that it treats the symptom of pain without consideration for the underlying mechanism or cause of the pain generation, and these approaches frequently only result in short term pain relief and lack broad therapeutic utility (Nachemson, 1999).

2.3. Neuro-physiological model

An increased focus on the study of the nervous system and its involvement in pain disorders has documented complex biochemical and neuro-modulation changes at a peripheral, as well as at spinal cord and cortical levels (Flor and Turk, 1984; Flor et al., 1997; Moseley, 2003; Wright and Zusman, 2004). This has highlighted that pain can be generated and maintained at a peripheral level, as well as centrally at both spinal cord and cortical levels. Central sensitisation of pain which is manifest in most CLBP disorders (to varying degrees) can occur secondary to sustained peripheral noniceptive input resulting in changes at spinal cord and cortical levels (Zusman, 2002). This can be both amplified and inhibited by fore-brain descending input (see psychosocial section) (Zusman, 2002). As well as this there is growing evidence that the nervous system undergoes changes to its cortical mapping and possesses a pain 'memory' which may leave it pre-sensitized to the exacerbation and recurrence of pain (Zusman, 2002). This new knowledge has lead to an increased focus on medical interventions to inhibit both peripheral and central processing of pain (Bogduk, 2004), as well as psychological and cognitive interventions to reduce the forebrain facilitation of pain (Woby et al., 2004).

2.4. Psychosocial model

The focus on the nervous systems' role in pain modulation has coincided with increasing research investigating the impact of psychological and social factors on the modulation of pain and in particular, their capacity to increase the central nervous system mediated drive of pain via the forebrain (Linton, 2000; Zusman, 2002; Waddell, 2004). Mal-adaptive coping strategies such as negative thinking, pathological fear and abnormal anxiety regarding pain, avoidant behaviour, catastrophizing and hyper-vigilance have been shown to be associated with high levels of pain, disability and muscle guarding (Frymoyer et al., 1985; Main and Watson, 1996; Nachemson, 1999; Linton, 2000). Social factors such as the compensation system, work place disputes, work and family tensions and cultural issues affecting beliefs reinforce the psychological factors that can increase the central drive of pain (Nachemson, 1999). Despite this advanced knowledge there is debate regarding the relative contribution of these factors to pain disorders and whether these factors predispose, or are as a result of a pain disorder. In contrast positive factors such as adaptive coping strategies, appropriate pacing and distraction (reduced hypervigilance) can have a descending inhibitory effect on pain via the forebrain (Zusman, 2002). Certainly there is evidence that cognitive behavioural interventions are effective in reducing disability in specific groups with non-specific CLBP (Woby et al., 2004), however there appears to be a growing trend within physiotherapy to classify most patients with non-specific CLBP as primarily psychosocial driven due to a lack of an alternative diagnosis. Although all CLBP disorders have psychological and social impact with associated cognitive issues related to the disorder, it appears that only a small sub-group exist where these factors become the dominant or primary pathological basis for the disorder.

2.5. Mechanical loading model

Both high and low levels of physical activity are reported to be risk factors for LBP while moderate levels of activity appear protective (Newcomer and Sinaki, 1996; Balague et al., 1999). Mechanical factors are usually reported to be associated with the initial development of LBP and are frequently reported to contribute to the recurrence of LBP and the exacerbation of CLBP. These factors include; sustained low load postures and movements (such as sitting, standing, bending and twisting), exposure to whole body vibration, high loading tasks (such as repeated lifting and bending), as well as sudden and repeated spinal loading in sports specific and manual work situations (Pope and Hansen, 1992; Adams et al., 1999; Nachemson, 1999; Abenhaim et al., 2000; McGill, 2004). These different mechanical exposures are also influenced by ergonomic and environmental factors (McGill, 2004), such as seating design, lifting technique, work place design and sporting equipment. Individual physical factors such as where in its range a spinal articulation is loaded (neutral zone vs. elastic zone), reduced trunk muscle strength and endurance, impaired flexibility, ligamentous laxity and motor control dysfunction as well as anthropometric considerations have also been reported to be associated with LBP (Adams et al., 1999; Abenhaim et al., 2000; McGill, 2004; Dankaerts et al., 2005b; O'Sullivan et al., 2005). Although little direct evidence supports the efficacy of ergonomic interventions for the management of LBP, there is little doubt that physical factors such as sustained end range spinal loading, lifting with flexion and rotation, exposure to vibration and specific sporting activities involving cyclical end range loading of the spine (especially combined with rotation) do negatively impact on the musculo-skeletal system and have the potential to cause ongoing peripheral nociceptor sensitization (Adams et al. 1999; Nachemson, 1999; Abenhaim et al., 2000; Burnett et al., 2004; McGill, 2004).

2.6. Signs and symptoms model

The area and nature of pain, impairments in spinal movement and function, changes in segmental spinal mobility (hyper and hypo), as well as pain responses to mechanical stress (provocation tests) and movement (peripheralisation and centralisation of pain with repeated movement) have formed the basis for classifying LBP disorders (McKenzie, 1981; Maitland, 1986; McKenzie, 2000). These approaches are based on biomechanical and pathoanatomical models and have lead to the assessment and treatment of signs and symptoms associated with CLBP (McKenzie, 1981; Maitland, 1986; McKenzie, 2000). Evidence for the efficacy of these approaches for the management of CLBP disorders remains limited (Maher et al., 1999; Abenhaim et al., 2000; Bogduk, 2004). This may in part be due to the limitations of the research design for some of these studies, as well as a neglect to account for the complex biopsychosocial nature of chronic pain disorders (Elvey and O'Sullivan, 2004).

2.7. Motor control model

There has been an increased focus on the management of CLBP from a motor control perspective (Richardson and Jull, 1995; O'Sullivan, 1997, 2000; Sahrmann, 2001). While it is well recognized that movement and motor control impairments exist with CLBP disorders, they are highly variable and their presence does not establish cause and effect. Movement and motor control impairments are known to occur secondary to the presence of pain (Hodges and Moseley, 2003; Van-Dieen et al.,

2003). Pathological processes such as neurogenic and radicular pain, neuropathic and centrally mediated pain and inflammatory disorders result in *adaptive* or protective altered motor behaviour in response to pain (Hall and Elvey, 1999; Elvey and O'Sullivan, 2004). Psychological processes such as stress, fear, anxiety, depression, hysteria, and somatisation are also known to disrupt motor behaviour (Frymoyer et al., 1985; Hodges and Moseley, 2003). Attempts to "normalize" movement or motor control impairments or treat dysfunction in the spinal muscles in many of these disorders would be inappropriate and ineffective due to the non-mechanical basis of these disorders.

There is however growing evidence that CLBP disorders do exist where *mal-adaptive* movement and motor control impairments appear to result in ongoing abnormal tissue loading and mechanically provoked pain (Burnett et al., 2004; Dankaerts et al., 2005b; O'Sullivan et al., 2005). Following an acute episode of low back pain (when tissue healing would have normally occurred), ongoing mal-adaptive motor control behaviour provides a basis for ongoing peripherally driven nociceptor sensitisation leading to a chronic pain state. These disorders are amenable to tailored physiotherapy interventions directed at their specific physical and cognitive impairments (O'Sullivan et al., 1997a–c; Stuge et al., 2004).

2.8. Biopsychosocial model

What is clear from the scientific literature and clinical practice, is that a multi-dimensional approach to dealing with CLBP based on a biopsychosocial model is required (Elvey and O'Sullivan, 2004; McCarthy et al., 2004; Waddell, 2004). The relative contribution of the different dimensions and their dominance associated with a CLBP disorder will differ for each patient. The role of the treating clinician is to consider all dimensions of the disorder based on an interview, thorough physical examination (assessing all aspects of the neuromusculosketetal system) combined with review of radiological imaging, medical tests and screening questionnaires (Elvey and O'Sullivan, 2004; O'Sullivan, 2004; Waddell, 2004) (Fig. 1). A clinical reasoning process allows determination of which factors are dominant in the disorder and whether the patient has adapted to the disorder in a positive or negative manner. Consideration of all the factors outlined allows for a diagnosis and mechanism based classification guiding management of the disorder (Elvey and O'Sullivan, 2004) (Fig. 1).

3. Diagnosis and classification of back pain

The Quebec task force classification system provides a logical approach for the diagnosis and classification of LBP disorders within a biopsychosocial framework (Spitzer, 1987; Abenhaim et al., 2000; Waddell, 2004). Under this framework red flags are considered in a diagnostic triage. The patient is screened for yellow flags or non-organic features suggestive of psychological and/or social factors dominating in the disorder. Under this classification system, disorders can be diagnosed as specific (especially nerve root pain) or non-specific, and staged (acute, sub-acute and chronic).

3.1. Diagnosis: specific and non-specific CLBP disorders

Specific pathoanatomical diagnoses, although critical for the understanding of many disorders, require further classification. For example, a diagnosis of lumbar spine stenosis (central or foraminal/lateral—chronic stage) may be associated with an *adaptive* (protective) motor response associated with a functional reduction of the lumbar lordosis with associated lumbar multifidus inhibition, to unload sensitized neural tissue. In this case attempts to normalize the motor control impairments would result in exacerbation and deterioration of the disorder. On the other hand the same diagnosis may be associated with a *mal-adaptive* motor response, represented by a functional increase in lumbar lordosis with associated back muscle guarding, resulting in further neural compromise and direct aggravation of the disorder. In this case normalising the motor control impairments (to functionally reduce the lumbar lordosis) would be indicated and effective. This proposed classification (into adaptive/mal-adaptive motor control responses) directly influences whether the patients' specific disorder is amenable for physiotherapy management that is aimed at normalising the motor control impairments or not. Alternatively, this diagnosis may be associated with a dominance of psychosocial factors and associated dominant central nervous system sensitisation, compromising the potential success of both conservative physiotherapy and surgical interventions. In this case the same specific diagnosis may present with a different classification, reflecting a different underlying pain mechanism and therefore indicating a different intervention (Elvey and O'Sullivan, 2004).

Eighty-five percent of CLBP disorders do not have a specific diagnosis (Dillingham, 1995). These disorders are labelled 'non-specific CLBP' disorders and represent a large group of 'tissue strains' and 'sprains' that have not resolved beyond normal tissue healing time (Abenhaim et al., 2000). This group has been broadly classified based on the area of pain and defined as somatic referred or radicular in nature (Abenhaim et al., 2000). However this diagnostic/classification system is of limited clinical value as it does not identify the underlying mechanism driving the pain disorder, and consequently there is no clear direction for specific management (Padfield and Butler, 2002).
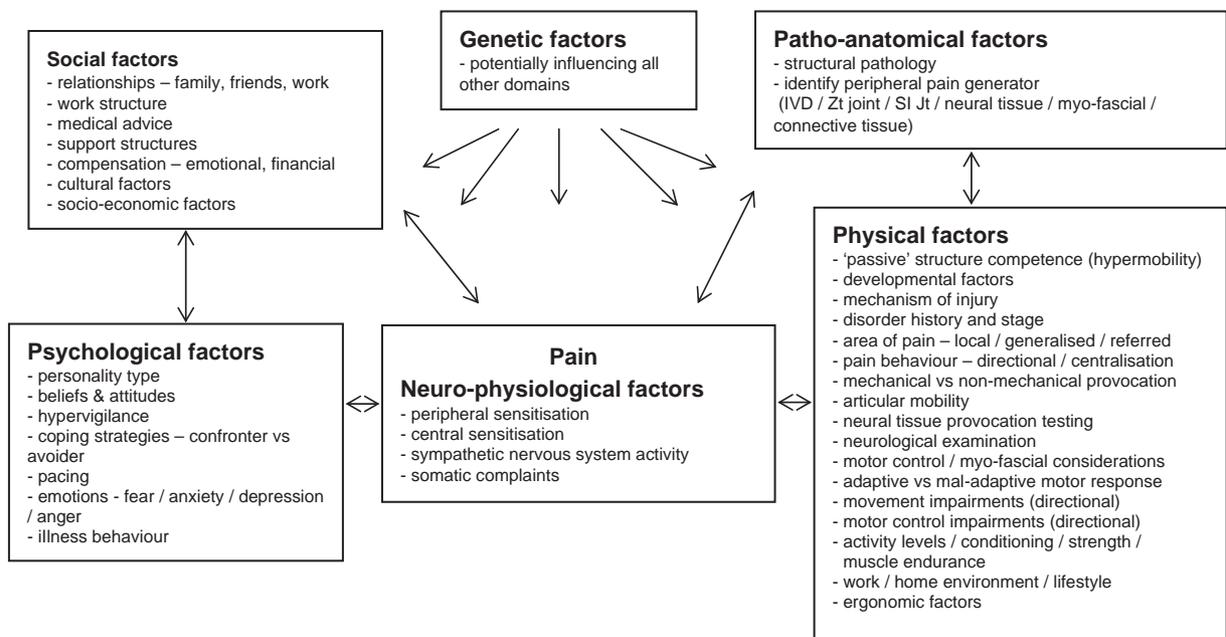
Fig. 1. Factors that need consideration within a biopsychosocial framework, for the diagnosis and classification of CLBP disorders.

3.2. Classification of CLBP

Due to the shortcomings of the current models, it is clear that both specific and non-specific CLBP disorders require further classification based on a biopsychosocial construct. There are a number of key clinical indicators regarding pain area and behaviour, which provide an important insight into the different mechanisms underlying and driving a pain disorder, allowing classification to be made. Considered simplistically, the presence of localized and anatomically defined pain associated with specific and consistent mechanical aggravating and easing factors, suggest that physical/mechanical factors are likely to dominate the disorder resulting in a primary peripheral nociceptive drive. Correlation between clinical examination and pathoanatomical findings is critical to determine their significance and relationship to the disorder. If pain is constant, non-remitting, widespread and is not greatly influenced by mechanical factors (or minor mechanical factors result in an exaggerated and disproportionate pain response), then inflammatory or centrally driven neurophysiological factors (such as altered central pain processing) are likely to dominate the disorder. High levels of anxiety, hypervigilance, fear and emotional stress presenting as primary aggravating or precipitating factors in the disorder, highlight the influence of psychological and in some cases social factors indicating the dominant forebrain drive of pain in a disorder (Linton, 2000). Understanding a patient's social circumstances, work environment, lifestyle factors and beliefs regarding their disorder is also critical (Waddell, 2004). Whether the patient has active or passive coping strategies in managing their disorder, and whether they pace themselves is important in understanding their capacity to actively manage their pain (Bergstrom et al., 2001). In reality most disorders will be associated with a combination of these factors, and the role of the clinician is to consider the balance and dominance of them in the disorder (Fig. 1).

It is proposed that there are three broad sub-groups of patients that present with disabling CLBP associated with movement and control impairments (Fig. 4).

(1) The first sub-group is represented by disorders where high levels of pain and disability, as well as movement and/or control impairments are secondary and *adaptive* to an underlying pathological process. These include red flag disorders, specific pathoanatomical disorders in some circumstances (such as IVD prolapse, spinal and foraminal stenosis with associated radicular pain ± neurological deficits, internal disc disruption with associated inflammatory pain, 'unstable' grade 2–4 spondylolisthesis), inflammatory pain disorders, neuropathic and centrally or sympathetically mediated pain disorders. These patients present with antalgic movement patterns and altered motor control that is driven directly by the pain disorder. The therapist will quickly determine this as attempts to 'normalize' these motor control and movement impairments results in exacerbation or non-resolution of the disorder, as these impairments are adaptive and driven by pathological processes. If the pathological process resolves with time or secondary to specifically targeted interventions (i.e. appropriate medical and/or surgical management when indicated), the signs and symptoms (e.g. motor

control and movement impairments) related to the disorder resolve.

Specifically targeted therapy management may be indicated for some of these disorders in conjunction with other primary medical interventions with full knowledge of the non-mechanical underlying basis of the disorder (Elvey and O'Sullivan, 2004). These disorders represent a small but severely disabled group within the CLBP population.

(2) A second small sub-group exists where the drive of the pain disorder is from the forebrain, secondary to a dominance of psychological and/or social (non-organic) factors. Although psychological and social impact occurs with all chronic disabling pain disorders, it appears that for a small group of patients it represents the dominant central drive of their disorder. This results in high levels of disability, altered central pain processing, amplified non-remitting pain, and resultant disordered movement and motor control impairments. These disorders commonly present with dominant psycho-social features, including pathological anxiety, fear, anger, depression, negative beliefs, un-resolved emotional issues, poor coping strategies (lack of pacing resulting in pain provocation or excessive avoidance of activity as means of controlling pain) as well as negative social and inter-personal circumstances (Linton, 2000; Bergstrom et al., 2001; Waddell, 2004). These psychological and social stresses present as dominant co-existing, precipitating and primary aggravating factors for the disorder (Linton, 2000).

The key feature of these disorders is the absence of an organic basis to the disorder, and lack of clear and consistent mechanical provocation or relieving patterns (absence of peripheral nociceptor drive). When mechanical factors are provocative they are inconsistent and tend to result in abnormal and disproportionate pain, disability and emotional responses. These patients commonly present with high levels of dependence on strong analgesic medication and passive forms of health care provision by multiple practitioners, even though they report a poor response to these interventions (Waddell, 2004). It is important to note that a therapist should not arrive at this classification without consultation and confirmation by either a treating clinical psychologist or psychiatrist.

In this sub-group, attempts to simply treat the 'signs and symptoms' of the disorder directly (e.g. movement and control impairments) does not result in their resolution, as the underlying mechanism driving the pain is not addressed. Management of these disorders requires multi-disciplinary management with a primary focus on cognitive behavioural therapy (Bergstrom et al., 2001) and psychiatric management. Physiotherapy management can play a specialized role in reinforcing graded functional recovery while reducing the focus on pain, however it cannot be seen as the primary treatment for these disorders (Elvey and O'Sullivan, 2004).

(3) It is proposed that a large third sub-group exists where *mal-adaptive* movement or control impairments and associated faulty coping strategies result in chronic abnormal tissue loading (associated with either excessive or reduced spinal stability), pain, disability and distress. This group is classified on the basis that the 'movement' impairments (characterized by pain avoidance behaviour) or 'control' impairments (characterized by pain provocation behaviour) act as the underlying mechanism that drives the CLBP state. Normalisation of the movement or control impairments based on a cognitive behavioural approach results in resolution and/or control of these disorders. Disorders with a 'movement' and 'control' impairment classification present commonly in clinical practice, and they appear to have different underlying pain mechanisms from each other and therefore their management is distinctly different (Figs. 2 and 3). These disorders may present as specific (associated with a pathoanatomical diagnosis) or non-specific CLBP disorders, and are commonly associated with psychological, social, neurophysiological (central sensitisation) factors, that may contribute to but do not dominate or drive the disorder. The classification of these disorders leaves them amenable to therapy intervention directed at the primary physical (movement and control) impairments while addressing the secondary cognitive aspects of the disorder (see Fig. 4).

3.2.1. Movement impairment classification

CLBP disorders classified as 'movement impairment' present with a painful loss or impairment of normal (active and passive) physiological movement in one or more directions (Figs. 2, 3 and 5a). These disorders are associated with abnormally high levels of muscle guarding and co-contraction of lumbo-pelvic muscles when moving into the painful and impaired range. This appears to be driven by an exaggerated withdrawal motor response to pain. This leads to high levels of compressive loading across articulations, movement restriction and rigidity (excessive stability), resulting in a mechanism for tissue strain and ongoing peripheral nociceptor sensitisation. These patients are usually acutely aware of their pain and are fearful of moving into the painful movement direction as they perceive that pain provocation is damaging. The fear of movement appears to develop from the patients' initial experience of severe acute pain, as well as their beliefs (reinforced by sympathetic family members and treatment providers) that pain is harmful. Movement related fear, hyper-vigilance and anxiety associated with the pain reinforces the faulty cognitive coping strategies and beliefs, further amplifying the pain centrally and reinforcing their muscle guarding. This represents a *mal-adaptive* response to the pain disorder, as the compensations for the pain in turn becomes the mechanism that drives the disorder. These disorders

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(A) Movement impairment classification

Nature and mechanism of pain:
Localised pain +/- referral
Severe pain of rapid onset
Movement impairment in direction of pain
Hyper-awareness of pain
Exaggerated reflex withdrawal motor
response
Muscle guarding and abnormal tissue
loading (↑spinal stability)
Avoidance of movement into painful range
Disability
Directional (flexion, extension, rotation,
lateral shift, loading)
Multi-directional

Result: Peripheral pain sensitisation

Anxiety related to movement pain
Fear avoidance when moving in direction
of pain (pathological)
Hyper-vigilence
Belief that pain is damaging (pathological)

Result: Central pain sensitisation

Normalisation of movement impairment
leads to resolution / control of disorder

</td><td>

(B) Control impairment classification

Nature and mechanism of pain:
Localised pain +/- referral
Gradual onset of pain from repeated or sustained
strain
No impaired movement in direction of pain
Lack of awareness of pain triggers
Poor lumbo-pelvic position sense
Absence of reflex withdrawal motor response
Ongoing tissue strain (↑or↓ spinal stability)
Provocation into painful range
Avoidance of painful activity
Disability
Directional (flexion, extension, rotation, lateral
shift, loading)
Multi-directional

Result: Peripheral pain sensitisation

Anxiety related to chronic disabling pain
Fear of activity (non-pathological)
Lack of control and awareness of disorder
Belief that activity is damaging (non-pathological)

Result: Central pain sensitisation

Normalisation of control impairment leads to
resolution / control of disorder

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Fig. 2. The nature and mechanism associated with mal-adaptive motor control disorders with: (A) Movement impairment classification and (B) control impairment classification (italics represent common features of the disorders / normal text highlights differences between the disorders).

may present in a directional manner (flexion, extension, side bending and rotational impairments) as well as combinations of these movements (multi-directional movement impairments).

Management of this patient sub-group is directed at both the dominant physical and associated cognitive factors that underlie the disorder. The aim is first to educate the patient that their pain is not damaging and they have developed faulty compensations to their pain, which now act to maintain their disorder. Restoration of the painful impaired movement is critical for the resolution of the disorder. The aim of the intervention is to desensitize the nervous system by restoring normal movement, reducing the fear of movement into pain and associated muscle guarding. This is facilitated by graded movement exposure into the painful range in a relaxed and normal manner based on the individual patient presentation. The cognitive strategies of reducing fear and changing beliefs regarding pain is augmented by manual therapy 'treatment' to restore the movement impairment (articular mobilisation/manipulation and soft tissue techniques). This is combined with active 'management' approaches directed to restore the movement impairment (muscle relaxation, breathing control, postural adjustments, graded movement exposure ex-

ercises, cardio-vascular exercise and most importantly graded functional restoration to normalize motor control). As the movement impairment and associated movement-based fear reduces, so too does the disability and pain related to the disorder. Stabilising exercise programs and treatment approaches that focus on pain and reinforce the avoidance behaviour usually exacerbate these disorders and are contra-indicated.

3.2.1.1. Case study 1. A 28-year-old woman reported a 3 year history of disabling non-specific CLBP (central lower lumbar) that had developed following a lifting injury while working as a nurse. She was placed off work for three weeks and was told by her physiotherapist that she had injured her disc, should do 'McKenzie extension exercises', avoid flexion and maintain her lumbar lordosis at all times. She reported becoming disabled with pain and very fearful of bending her back which she avoided doing from that time.

Her treatment history consisted of McKenzie extension exercises, Pilates, stabilisation training (with a focus on pelvic floor, transverse abdominal wall and lumbar multifidus co-activation) and swimming. She had seen an orthopaedic surgeon, pain specialist, clinical psychologist, a number of physiotherapists and was taking
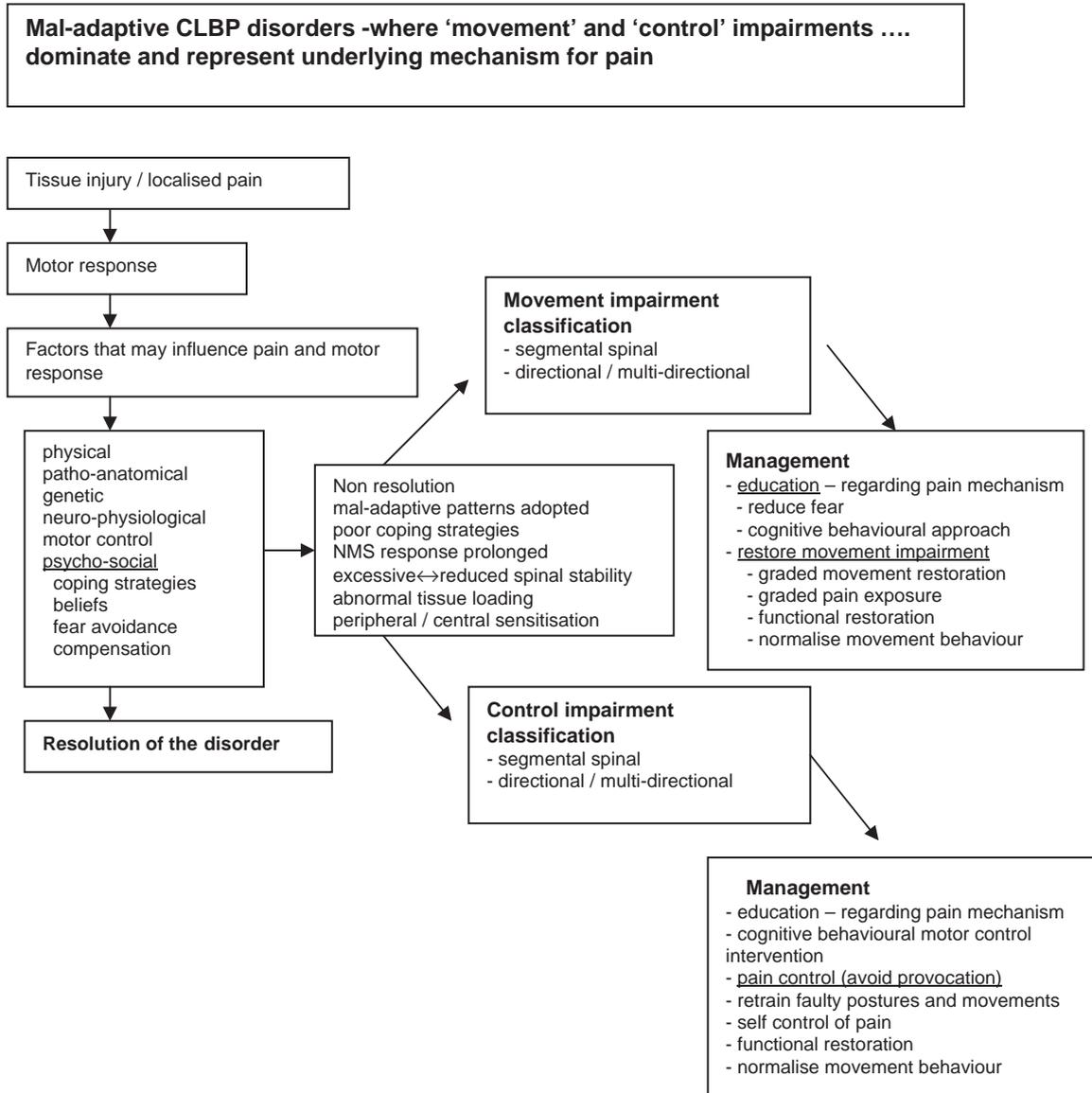
**Mal-adaptive CLBP disorders -where 'movement' and 'control' impairments ….
dominate and represent underlying mechanism for pain**

Tissue injury / localised pain

Motor response

Factors that may influence pain and motor response

physical
patho-anatomical
genetic
neuro-physiological
motor control
psycho-social
  coping strategies
  beliefs
  fear avoidance
  compensation

Resolution of the disorder

Non resolution
mal-adaptive patterns adopted
poor coping strategies
NMS response prolonged
excessive↔reduced spinal stability
abnormal tissue loading
peripheral / central sensitisation

Movement impairment classification
- segmental spinal
- directional / multi-directional

Management
- education – regarding pain mechanism
  - reduce fear
  - cognitive behavioural approach
- restore movement impairment
  - graded movement restoration
  - graded pain exposure
  - functional restoration
  - normalise movement behaviour

Control impairment classification
- segmental spinal
- directional / multi-directional

Management
- education – regarding pain mechanism
- cognitive behavioural motor control intervention
- pain control (avoid provocation)
- retrain faulty postures and movements
- self control of pain
- functional restoration
- normalise movement behaviour

Fig. 3. Mal-adaptive motor control impairment CLBP disorders.

**CLBP disorders associated
with altered motor control**

Adaptive / protective altered motor response to an underlying disorder
- inflammatory disorders
- centrally mediated pain
- sympathetically maintained pain
- neurogenic pain
- neuropathic pain

Altered motor response and centrally mediated pain secondary to dominant psychosocial factors

Mal-adaptive motor control patterns that drive the pain disorder
  - movement impairments
  - control impairments
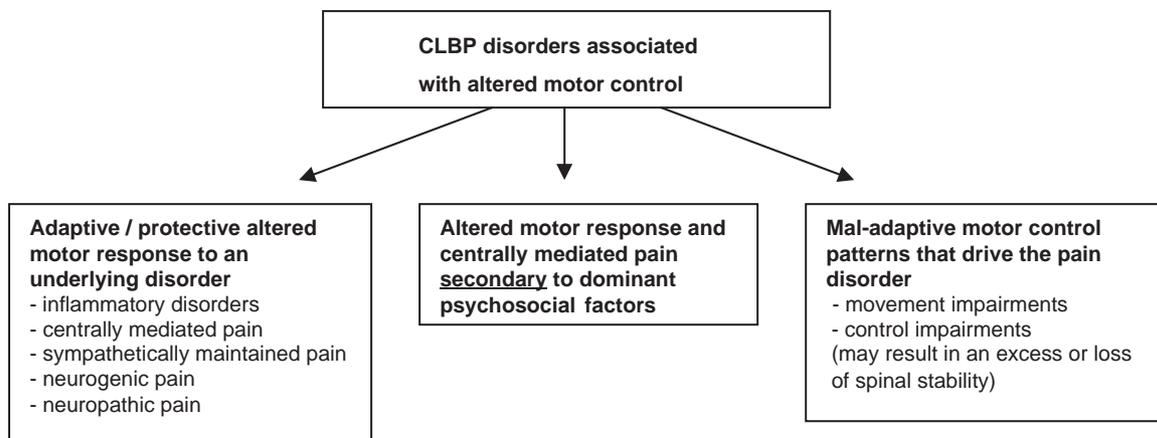(may result in an excess or loss of spinal stability)

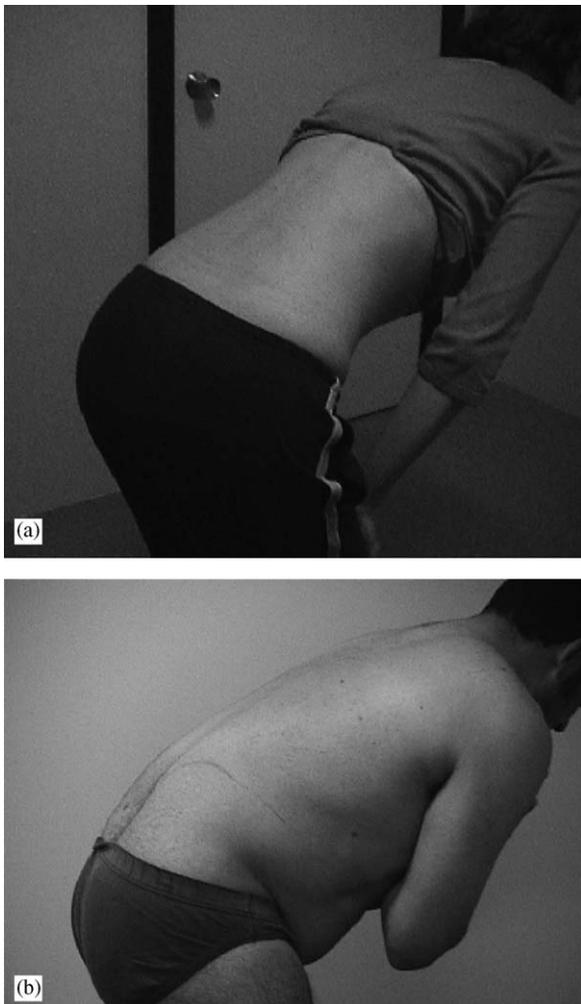Fig. 4. Altered motor responses in the presence of CLBP (3 groups).

Fig. 5. (a) Patient with classification of movement impairment into flexion (note the pain provocation into flexion *is* associated with an impairment of lumbar spine flexion). (b) Patient with classification of control impairment into flexion (note the pain provocation into flexion is *not* associated with an impairment of lumbar spinal flexion).

anti-depressants, strong analgesic and muscle relaxant medication.

She was only able to work 2 days per week doing light duties because of her CLBP disorder.

She reported that her symptoms were exacerbated by all flexion postures and movements such as slump sitting, bending, dressing and lifting activities. Extension related spinal movements such as standing and walking were pain free. She gained relief from her pain with heat and rest.

She reported high levels of anxiety relating to pain, disability and an inability to work full time. She constantly worried about her back pain and believed that she would not get better as she had a disc injury that had not resolved. She coped with her back pain by avoiding provoking it and restricting her activities involving spinal flexion. Her pain intensity level was

8/10, her disability index (Oswestry disability index) was 40% and she had high levels of kinesiophobia (Tampa scale of Kinesiaphobia).

Investigations:	X-rays/MRI Lumbar spine—NAD

Physical examination

Observation	• she sat and walked with a rigid erect thoraco-lumbar spine posture
	• she sat forward on the chair with a lordotic spinal posture
	• she maintained thoracolumbar lordosis and avoided flexion when moving from sitting to standing and while undressing
AROM	Flexion—hip flexion 50°, no thoraco-lumbar flexion with use of hands to support her and assist her return to upright (Fig. 5a) Extension—30° no pain Side bending—full ROM and pain free Repeated flexion increased guarding and report of pain
Motion palpation	L5/S1—hypo-mobile in flexion
Provocation palpation of L4 and L5 centrally—reproduced pain (highly sensitized)	
SIJ	NAD
Neural provocation tests	NAD

Motor control

1. Functional movement tests—stated under observation
2. Specific movement testing—attempts to posteriorly rotate pelvis in sitting, supine and four point kneeling were associated with pain and muscle guarding.
3. Specific muscle testing—able to isolate co-activation of the transverse abdominal wall and lower lumbar multifidus in neutral lordosis (difficulty observed relaxing them).

Diagnosis	non-specific CLBP
Classification	*Movement impairment disorder–flexion pattern L5/S1*

The disorder *diagnosis* of non-specific CLBP was based upon the non-resolution of a *flexion back sprain* and the absence of a specific diagnosis.

The disorder *classification* of this patient was a *movement impairment disorder* (into flexion with localized pain at L5/S1).

The mechanism underlying the pain is a movement impairment with a loss of normal physiological movement into flexion, with associated muscle guarding and fear of forward bending. This movement impairment and associated fear was initiated in the acute phase and was reinforced by her beliefs that pain associated with flexion of her spine was damaging for her. This patient avoided bending due to the knowledge that flexion will provoke pain and the belief (reinforced by treatment providers) that this movement causes 'further damage' and that by not moving into this painful direction will prevent damage. The basis of this pain disorder is linked to both dominant peripheral and secondary central pain mechanisms.

Management of this patient was directed at both the dominant peripheral and secondary central mechanisms of the pain disorder over a 12 week period. Management first focussed on educating the patient regarding the basis and mechanism of her disorder. It was critical to change the patient's beliefs, so that she understood that to relax the spinal muscles and restore normal movement in the direction of her pain was essential for resolution of the pain disorder. The patient was assured that her movement-provoked pain into flexion was not dangerous or damaging.

The restoration of normal tissue compliance and reduction of muscle guarding was facilitated by 'passive' treatment techniques directed to restore flexion mobility to the lower lumbar spine (L5/S1 flexion articular mobilisation techniques and soft tissue inhibitory techniques directed to her back extensor and psoas muscles). This was combined with graded active movement into the restored range. This involved the patient initially being taught to posteriorly tilt her pelvis in a relaxed manner without trunk muscle guarding and breath holding (initially in supine and four point kneeling progressed to sitting and standing). She was instructed to cease cognitively contracting her spinal 'stabilising muscles' but rather to relax her upright postures so to reduce her thoraco-lumbar hyper-lordosis to a neutral spine posture. Finally the patient was trained to flex her spine in upright postures (sitting and standing) in a normal physiological manner without guarding. As the movement impairment was restored, the pain, disability and fear of bending also reduced. At this stage the patient reported that she had the capacity to control her pain. This new control was then introduced into previously provocative functional tasks such as dressing and housework. She reported that she could work longer and increase her general activity levels. She was encouraged to carry out regular cardio-vascular exercise and join a yoga class to maintain her spine mobility in a relaxed manner. The resolution of her CLBP disorder supported the classification and management approach taken.

3.3.1. Control impairment classification

CLBP disorders classified as 'control impairment' appear to be most common in clinical practice. These disorders are associated with impairment or deficits in the control of the symptomatic spinal segment in the primary direction of pain. In these disorders there is no movement impairment in the direction of pain (Figs. 3 and 5b). Pain in these disorders is associated with a loss of functional control around the neutral zone of the spinal motion segment due to specific motor control deficits (and muscle guarding in some situations) of the spinal stabilising muscles. This is manifest during dynamic and/or static tasks as

1. 'through range movement pain' due to non-physiological motion of the spinal segment observed during dynamic tasks,
2. 'loading pain' due to non-physiological loading of the spinal segment (not end range) observed during static loading tasks and
3. 'end of range pain' or 'overstrain' due to repetitive strain of the spinal motion segment at the end of range observed during static and dynamic functional tasks.

The irony with these patients is that they adopt postures and movement patterns that maximally stress their pain sensitive tissue (Burnett et al., 2004; O'Sullivan et al., 2004; Dankaerts et al., 2005b), and yet they have no awareness that they do this. One reason for this may relate to the fact that their pain is often of a gradual onset and therefore they lack a withdrawal reflex motor response, coupled with a lack of proprioceptive awareness of the lumbo-pelvic region (Fig. 2) (O'Sullivan et al., 2003; Burnett et al., 2004). This control deficit is clearly *mal-adaptive* and represents a powerful mechanism for ongoing pain (which is both peripherally and centrally mediated) and disability. These patients present with movement based fear that is real, as their movement strategies are highly provocative of their pain disorder, resulting in failure to respond to general exercise and conditioning interventions. These disorders frequently present in a directional manner (flexion, extension (passive or active) and lateral shift control impairment) as well as combinations of these directions (multi-directional control impairment). These disorders may be associated with deficits in the spinal stabilising muscles (i.e. flexion pattern) or excessive muscle activity resulting in increased spinal loading (i.e. active extension pattern). These directional patterns are described in detail elsewhere (O'Sullivan, 2000, 2004). Clinical instability of the lumbar spine represents a sub-group of these disorders (O'Sullivan, 2000, 2004).

Management of this sub-group is based on a cognitive behavioural motor learning intervention model. This intervention is based on the premise that mal-adaptive

motor control behaviour provides an ongoing mechanism for tissue strain and peripheral nociceptive drive. The aim of the intervention is to desensitize the nervous system by educating the patient to control their pain provocative postures and movement patterns so as to *avoid* repetitive strain on the painful tissue, reduce the peripheral nociceptive drive and in turn enhance function. This is not simply an exercise program rather it follows a motor learning intervention model with the aim of changing movement behaviour via physical as well as cognitive learning processes. As the motor control is enhanced, the repeated stress on the symptomatic tissue reduces, resulting in less peripheral nociceptive drive into the nervous system, allowing the pain disorder to resolve. This provides the patient with the capacity to manage their disorder in an effective manner, which reduces their fear of activity and increase their levels of function. This intervention directly impacts on both the dominant peripheral nociceptive as well as the secondary central drives for the pain disorder.

The role of manual therapy treatment in control impairment disorders is limited only to the restoration of articular movement away from the direction of pain provocation and only if this movement is impaired and inhibiting the muscle synergies controlling this movement. These techniques are never used in isolation, but rather they facilitate movement so as to enhance the restoration of motor control to dynamically unload the pain sensitive tissue. For example in a flexion pattern control impairment disorder, if a loss of segmental spinal extension prohibits restoring control over the lower lumbar lordosis, then manual therapy treatment may be used to facilitate extension. This is immediately followed by training active control over this movement so as to reduce the flexion load of the motion segment. The specifics of this intervention have been reported in detail previously (O'Sullivan, 2000, 2004).

3.3.1.1. Case study 2. A 42-year-old male reports a 2 year history of non-specific CLBP. He first developed central LBP while lifting (with a flexed lumbar spine) a 30 kg bag of fertilizer while working as a labourer. His back pain disorder did not resolve and he had not been able to return to work.

His previous treatment consisted of physiotherapy, Pilates, gym based exercise programs, psychological intervention and medication (strong analgesics and anti-depressants).

He reported that his back pain was provoked by static flexed spinal postures (sitting, driving, semi-inclined bending) and activities (such as lifting, sit—stand, dressing). He reported that he avoided all such activities as they exacerbated his pain and it took days then to settle. He reported relief with extension or lordotic postures.

He reported feeling depressed due to the nature of his disability, his loss of independence and his alienation with his health providers, work and family and was tearful when describing this. He was also limited in his ability to socialize with his friends. He had been told there was nothing structurally wrong with his back and that he would have to learn to live with his problem and he believed that his condition was unlikely to improve. His pain intensity level was 7/10, his disability index (Oswestry disability index) was 42% and he had high levels of kinesiophobia (Tampa scale).

Physical examination

Observation	• he sat down to undress, and used his hands to assist transferring from sitting to standing
AROM	Flexion—no lower lumbar movement impairment (full low lumbar ROM) into flexion with report of LBP mid range (Fig. 5b) Extension—30° no pain Right and left side bending—full ROM Repeated and sustained spinal flexion increased his LBP
PPIVM	L5/S1—hyper-mobile in flexion

Provocation palpation of L5/S1 central—painful with reproduction of back pain

Neural provocation tests	NAD

Motor control:

1. Functional movement tests—forward bending, reaching, lifting, sit to stand and squatting were associated with increased flexion at the lower lumbar spine, a loss of anterior pelvic rotation and lordosis in the upper lumbar and thoracic spine (Fig. 4b). The use of the arms was observed to support the trunk with these activities.
2. Specific movement tests—Attempts to initiate anterior pelvic tilt and extend the lower lumbar spine in standing, sitting and supine were associated with upper lumbar and thoracic spine extension
3. Specific muscle testing—Inability to isolate the activation of the pelvic floor, transverse abdominal muscles and lumbar multifidus with posterior pelvic rotation and flexion of the lower lumbar spine, with bracing of the upper abdominal wall.

Investigations	X-rays/MRI lumbar spine—degenerative disc disease L5/S1 (mild)
Diagnosis	non-specific CLBP
Classification	*control impairment disorder—flexion pattern at L5/S1*

The diagnosis of non-specific CLBP was based on the non-resolution of a *flexion back sprain* beyond normal healing time and the lack of a specific diagnosis.

The classification of this patient as *control impairment disorder* (flexion pattern) is based on the underlying mechanism of this pain disorder being directly linked to an ongoing flexion strain of the L5/S1 motion segment secondary to a loss of functional control of the segment into flexion. The patients' sense of alienation, frustration, anger and depression further confounds his situation resulting in increased central drive of his pain.

Management of this patient was directed on a cognitive behavioural motor learning frame-work (O'Sullivan, 2004). The patient was first educated that subsequent to his initial back sprain he had adopted a mal-adaptive motor control pattern that exposed the symptomatic segment to abnormal and repetitive strain into flexion, which in turn maintained his pain. This was further reinforced by his anxiety levels related to work and home, lack of control over his pain disorder and inactivity.

Management focused on a motor control intervention to reduce the flexion strain at L5/S1 in a functionally specific manner with relaxation of the thoraco-lumbar spine and enhancing control of segmental lordosis at L5/S1. Initally he was taught to dis-associate lumbo-pelvic lordosis from thoracic in supine, sitting and standing. This was in order to develop proprioceptive awareness and control of this region and so reduce the flexion strain at L5/S1. Once this was achieved he was then taught to co-activate his lower lumbar multifidus with his transverse abdominal wall (in a neutral lordosis), with relaxation of his thoracic erector spinae and upper abdominal muscles (with normal respiration) in these postures. At this stage previously aggravating postures and movements into forward bending were targeted and retrained so that the patient could perform them (controlling the L5/S1 within a neutral lordosis), in a pain-free manner thereby enhancing his functional capacity. This in turn reduced his fear of movement and activity. His exercise program was then progressed into a gym setting where he was taught to integrate his lumbo-pelvic control into a graded cardio-vascular exercise program as well as training strength and endurance with loaded tasks such as squats, lunges and resistance lifting tasks. As the patient's functional mobility increased and pain reduced his coping strategies improved and he was capable of a graduated return to work. The resolution of the disorder supports the classification that the control impairment into flexion represented the dominant underlying mechanism driving the disorder.

4. Validity of the classification system

There is a growing concensus within the literature that current diagnostic and classification approaches for CLBP are limited, and a mechanism based classification of CLBP disorders from a biopsychosocial perspective is required (McCarthy et al., 2004). Although considerable research has documented the biopsychosocial nature of CLBP, further research is required to test the validity of this approach in management of CLBP disorders to determine whether it predicts and indeed improves patient outcomes.

There is growing evidence to support the validity of the 'control impairment' classification system as a subgroup with CLBP. Recent research has shown that physiotherapists trained in the classification system can reliably identify five different subgroups with a classification of control impairment (Dankaerts et al., 2005a, b). Laboratory evidence for the presence of specific motor control and postural deficits have been documented in a series of studies conducted on patients with CLBP with a classification of 'control impairments' (O'Sullivan et al., 1997a–c, 2003; Burnett et al., 2004; O'Sullivan et al., 2004; Dankaerts et al., 2005b).

Motor learning interventions have been shown efficacious in patient groups with a classification of control impairment, with documented reductions in pain and disability (O'Sullivan et al., 1997a–c, 1998, 2001; Dankaerts et al., 2004).

5. Summary

CLBP disorders must be considered within a biopsychosocial framework. The presence and dominance of the potential pathoanatomical, physical, neurophysiological, psychological and social factors that may impact on these disorders is different for each individual with CLBP. This highlights the enormous complexity and individual nature of the problem. It is critical that classification of CLBP pain disorders be based on the mechanism (s) underlying and driving the disorder. It is proposed that motor control impairments may be adaptive or mal-adaptive in nature. The treatment of the signs and symptoms of a pain disorder cannot be justified without an understanding of its underlying mechanism as there are sub-groups of patients for whom physiotherapy treatment is not indicated. It is proposed that there is a large sub-group of CLBP disorders where mal-adaptive movement and control impairments dominate the disorder, resulting in either excessive or impaired dynamic spinal stability and loading. This in turn becomes a mechanism for ongoing pain. Physiotherapy interventions that are classification based and specifically directed to the underlying driving mechanism, have the potential to alter these disorders and impact on both the primary physical and secondary cognitive drivers of pain. This approach is not limited only to the lumbo-pelvic region but can be applied to all regions of the musculoskeletal system. The evidence to

date supports these proposals although further research is required to further develop and validate this approach.

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