3rd International conference on movement dysfunction 2009

The validity of O'Sullivan's classification system (CS) for a sub-group of NS-CLBP with motor control impairment (MCI): Overview of a series of studies and review of the literature

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A R T I C L E   I N F O

Article history:
Received 10 May 2010
Received in revised form 7 October 2010
Accepted 13 October 2010

Keywords:
Chronic low back pain
Motor control
Classification

A B S T R A C T

Chronic Low Back Pain (LBP) remains a common, recalcitrant and costly problem for the individual sufferer and for society. Effective treatments that reduce the social and economic burden have yet to be established for the majority of chronic LBP cases. Lack of evidence for specific interventions has been blamed on the heterogeneity of the chronic LBP population as well as a lack of a patient centred bio-psycho-social approach. This issue of heterogeneity has resulted in classification being considered the highest research priority in the area of chronic LBP. The potential for a 'wash-out effect' caused by the heterogeneity of the chronic LBP populations sampled for randomised controlled clinical trials (RCTs), has driven the need for classifying patients with nonspecific chronic LBP. A summary of a series of studies is outlined in this review paper. They represent a comprehensive investigation into the validity of O'Sullivan's proposed mechanism-based classification system (CS) for a sub-group of localized mechanically provoked nonspecific chronic LBP with motor control impairment (MCI). Further, the findings of these studies are discussed in relation to the relevant literature and the clinical implications arising are presented. Finally, the limitations of this research are outlined and recommendations for future research are made.

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1. O'Sullivan's classification system (CS) for a sub-group of nonspecific chronic LBP with motor control impairment (MCI)

Chronic lumbo-pelvic pain disorders are complex and multidimensional in nature. It is well acknowledged that these disorders can be associated with changes in physical, neurophysiology, lifestyle, altered motor control, psycho-social factors and in a small number of cases patho-anatomical factors (Waddell, 2004). There is a considerable debate as to the significance of these different factors and what is cause and effect. The majority of lumbo-pelvic pain disorders have no diagnosis leaving a management vacuum. From this perspective, classification of nonspecific chronic low back pain (LBP) disorders into sub-groups, based on the mechanism(s) underlying the disorder, is considered to ensure appropriate management.

In order to identify and sub-classify specific sub-groups of nonspecific chronic LBP patients within a mechanism-based CS, O'Sullivan proposed a division between disorders of the central nervous system (such as regional pain disorders, neuropathic pain and fibromyalgia), and localized pain disorders that have a peripheral pain pattern and mechanical pain behaviour (O'Sullivan, 2005). Similar divisions have previously been proposed by experts in the field (Bogduk and Merskey, 1994; Latremoliere and Woolf, 2009). It was further proposed that this second group can be classified into a large group of patients with localized nonspecific chronic LBP, with either movement impairments or control impairments that act as a peripheral driver of their disorder. Pain disorders associated with a 'movement impairment' classification are usually characterized by avoidant pain behaviour and are associated with a loss of normal physiological lumbo-pelvic mobility in the direction of pain provocation. Those with a 'MCI' classification, sub-group patients with localized nonspecific chronic LBP based on pain behaviours related to adopting provocative spinal postures and movement patterns (O'Sullivan, 2004, 2005), where there is no movement impairment in the direction of pain provocation. Five distinct (direction based)
patterns of MCI have been described in detail (O’Sullivan, 2004, 2005). These sub-groups of MCI consist of the; flexion pattern (FP), active extension pattern (AEP), passive extension pattern (sagittal plane), lateral shifting pattern (frontal plane) and a multi-directional pattern (combination of the others).

O’Sullivan’s CS is based on a process of diagnostics (Elvey and O’Sullivan, 2004) to make a clinical determination as to whether the patient has a classification of MCI (maladaptive movement behaviours) or whether the altered movement behaviour is simply a secondary effect of another process (adaptive movement behaviours). ‘Diagnostics’ places a strong emphasis on the correlation between the subjective history, radiology, pain behaviour, lifestyle factors, physical examination findings (examining the articular, neural and motor control systems) and screens for serious pathology (‘red flags’) and dominant psycho-social factors (including negative beliefs, coping, fear avoidance beliefs, stress, anxiety, depression, catastrophising etc.). As a result of this diagnostic process the relationship between the patient’s pain and the MCI is determined (Elvey and O’Sullivan, 2004; O’Sullivan, 2005). Where it is determined that the MCI is directly related to the provocation of the pain disorder, a classification of MCI with respect to the direction of pain provocation is made. Within this system there is the potential for peripheral and central pain drivers to co-exist, where a patient may have an MCI with associated cognitive factors such as fear avoidance behaviours and poor coping strategies reinforcing the disability associated with the disorder (O’Sullivan, 2005). Rather than a reductionist approach to decision making based on a series of prediction rules (Fritz et al., 2003), this approach provides a broad framework for the identification of underlying mechanisms and behaviours associated with a pain disorder in order to guide management.

2. A summary of the findings of a series of five studies on the validation of a proposed CS for nonspecific chronic LBP

Following the recommendations emerging from the literature to validate existing CS’s (Riddle, 1998; Petersen et al., 1999; Ford, 2002; McCarthy et al., 2004), this review paper provides an accumulation of evidence supporting the validity of a mechanism-based CS for a sub-group of nonspecific chronic LBP with a classification of MCI. A step-wise validation process for the classification of LBP, consisting of accumulating levels of evidence was undertaken. Different methods were developed for each of these stages and were then applied to the CS.

The following section summarizes the findings of five studies conducted by Wim Dankaerts as part of his doctoral research at Curtin University (referred to as Studies I–V).

3. Can clinicians agree on the classification of nonspecific chronic LBP patients with MCI? (Study I)

The results of Study I (Dankaerts et al., 2006c) revealed that there was almost perfect agreement (kappa-coefficient 0.96; % of-agreement 97%) between two ‘expert’ clinicians, in identifying and classifying patients with nonspecific chronic LBP into five specific sub-groups of MCI based on a comprehensive clinical examination. Study I also demonstrated substantial agreement (mean Kappa-coefficients 0.61 and mean % of-agreement 78%) across all five proposed MCI patterns based on combined subjective case reports and video observation of postures and movements between clinicians (physiotherapists and medical doctors) in two geographically separate regions. It was concluded that clinicians could agree upon the classification of nonspecific chronic LBP patients with MCI supporting its reliability. The fact that clinicians agree supports the clinical viability and face validity.

In support of this work a recent study by Fersum et al. (2009a) confirmed a moderate to substantial inter-tester reliability of the CS system for a wider range of patients within the nonspecific chronic LBP population, including patients with MCI. Both the studies (Dankaerts et al., 2006c; Fersum et al., 2009a) demonstrated that familiarity with the CS influenced the reliability results, demonstrating higher agreement among raters with more CS training (<100 h).

4. Can laboratory tests evaluating posture in sitting discriminate two sub-groups of nonspecific chronic LBP (AEP and FP) from no-LBP? Is classification into sub-groups important for clinical research in subjects who report aggravation of LBP in sitting? (Study II)

While there is consensus in the literature that sitting commonly exacerbates and perpetuates LBP, there is clearly a paucity of studies that had quantitatively examined different sitting postures among the LBP population (Dankaerts et al., 2006b). In Study II lumbo-sacral posture was measured during “usual” and “slumped” sitting in 33 nonspecific chronic LBP patients and 34 asymptomatic subjects using an electromagnetic measurement device. Before testing nonspecific chronic LBP patients were sub-classified by two blinded clinicians. Twenty patients were classified with a flexion MCI and 13 with an AEP MCI (see Table 1 for inclusion criteria, clinical features and exclusion criteria for FP and AEP classification).

The results of Study II (Dankaerts et al., 2006b) showed that, when nonspecific chronic LBP patients were pooled into one group no differences were detected in lumbo-sacral posture during usual sitting when compared to controls. In contrast, analyses based on sub-classification revealed that patients classified with an AEP sat with a more lordotic posture at the symptomatic lower lumbar spine, whereas patients with an FP sat with a more kyphotic posture, when compared to healthy controls. Further, both groups of nonspecific chronic LBP patients had less ability to change their lumbo-sacral posture when asked to slump from usual sitting when compared to controls.

The irony with these patients is that they adopt the very postures that they report they are sensitized to, they have difficulty varying from these positions and appear to have little awareness that they do this. This would appear to be a maladaptive behaviour.

It was concluded that discriminative differences in lumbo-sacral sitting posture existed in these sub-groups that supported the proposed CS. The heterogeneity of the nonspecific chronic LBP subjects’ posture when pooled highlights the importance of subclassification in clinical research and provided evidence for construct validity of the CS.

5. Can laboratory tests evaluating trunk muscle activity in sitting discriminate two clinical sub-groups of nonspecific chronic LBP with a classification of MCI (AEP and FP) from no-LBP? Is classification of sub-groups important for clinical research into muscle activation patterns in subjects who report aggravation of LBP in sitting? (Study III)

The results of Study III (Dankaerts et al., 2006a) demonstrated that no differences in trunk muscle activity were observed between healthy controls and a pooled nonspecific chronic LBP group in usual sitting. However, when the CS was applied differences were identified. When compared to controls the AEP group presented with high levels of co-contraction of superficial fibres of lumbar multifidi (+12%), ilio-costalis lumbarorum pars thoracis (+36%) and transverse fibres of internal oblique (+43%). The FP group had less activation of these muscles than the AEP groups and showed a trend towards lower activation when compared to the controls.
Inclusion criteria for nonspecific chronic LBP with motor control impairment of flexion pattern or active extension pattern classification.

- History of chronic (>3 months) nonspecific (no radiologic diagnosis) LBP with at least moderate disability (Revised Oswestry score >15%)
- Pain localized to the lower lumbar spine (L4–L5 or L5–S1) region with minimal radiation
- Absence of red flags
- Absence of dominant yellow flags
- Clear mechanical basis of disorder: specific postural and functional movements that aggravate and ease symptoms; relief of symptoms when reducing the strain to the symptomatic spinal segment in the provocation direction
- Associated impairments in the control of the motion segment(s) in the provocative movement direction(s)
- Absence of impaired movement of the symptomatic segment in the painful direction of movement or loading (based on clinical joint motion palpation examination)
- Clinical diagnosis of a flexion pattern or active extension pattern disorder, both clinicians (independently) agreed upon the diagnosis
  
  Key clinical features flexion pattern
  - Aggravation of symptoms with movements and postures involving flexion of the lower lumbar spine
  - Loss of segmental lordosis at asymptomatic level, difficulty assuming and/or maintaining neutral lordotic postures with a tendency to drop into hyper-flexion
  - Pain relief with spinal extension
  
  Key clinical features active extension pattern
  - Aggravation of symptoms with movements and postures involving extension of the lower lumbar spine (commonly reported as a provocative activity is forward bending and sitting, with the key feature here being the tendency to hold the lumbar spine into segmental hyper-extension)
  - Excess of segmental lordosis at symptomatic level with posture and movements
  - Difficulty assuming and/or maintaining neutral lordotic postures with a tendency to position themselves into hyper-extension
  - Pain relief with spinal flexion

Exclusion criteria for nonspecific chronic LBP with motor control impairment of flexion pattern or active extension pattern classification.

- Not fulfilling inclusion criteria: low (<15) Revised Oswestry score, signs of neurologic involvement (radicular pain), non-mechanical pain, more generalized pain, evidence of specific diagnosis, e.g., spondylolisthesis, inflammatory disease, no agreement upon the motor control impairment between the two independent clinicians
- Presence of red flags
- Presence of dominant yellow flags
- Previous spine surgery, pregnant at the time of the study or 6 months postpartum, recently undergone a period of motor control rehabilitation

6. Can a statistical classification model using laboratory tests (sEMG and lumbo-sacral kinematics) accurately discriminate between two groups of nonspecific chronic LBP patients (AEP and FP) and a group of no-LBP controls? Are the clinically reported sub-group characteristics ‘real phenomena’? (Study IV)

The laboratory tests analysed in Study IV (Dankaerts et al., 2009) involved commonly reported aggravating spinal postures and movements that form part of the basis of the clinical classification process. The results of Study IV demonstrated that a Statistical Classification Model identified five lumbo-sacral kinematics (lower lumbar spine kinematics in sitting and forward bending) and two trunk muscle activation (lack of flexion relaxation of the superficial fibers of lumbar multifidus in slump sitting and end-range of forward bending) variables that were able to correctly classify 96.4% of cases and to discriminate the three groups (no-LBP, AEP and FP). These findings suggest that the sub-group characteristics’ reported clinically are ‘real phenomena’ supporting that the lumbar kinematics of functional postures and movement, and the inability of the superficial lumbar multifidus to relax, were key predictors of nonspecific chronic LBP.

This study provided evidence of construct validity in support of the CS, and highlights the heterogeneity of the nonspecific chronic LBP population with regards to motor control of the lumbar spine and the potential importance of classification in the identification of sub-groups. This finding supports the validity of the clinical process of diagnostics, where a diagnosis is determined as a result of a complete physical evaluation involving a series of tests while closely correlating these findings to the individual’s complaints.

7. Can a CS and laboratory tests (trunk muscle activation and lumbo-sacral kinematics) be applied in outcome studies investigating the predictive validity of a CS for a sub-group of nonspecific chronic LBP with a classification of MCI? (Study V)

The results of this case study (Study V) (Dankaerts et al., 2007) of a patient with nonspecific chronic LBP, classified as having a multidirectional MCI (O’Sullivan, 2004, 2005) demonstrated normalised motor control following a targeted motor learning intervention specific to the patient’s classification. The improvement in motor control was associated with reductions in pain, disability and fear related to movement. This was obtained at 3 months, and maintained at 6 and 12-months follow-up. Laboratory-based tests supported the classification (pre-intervention) and showed normalisation of motor control at 6 months (including normalisation of flexion relaxation response to forward bending), providing preliminary evidence that the MCI was the mechanism underlying the LBP disorder.

It was concluded that the use of this CS and laboratory tests (trunk muscle activation and lumbo-sacral kinematics) could be applied in outcome studies investigating the predictive validity of a CS for a sub-group of nonspecific chronic LBP and MCI.

In summary, the five studies conducted provide an accumulation of evidence towards the validation of a proposed mechanism-based CS for nonspecific chronic LBP patients with a classification of MCI. The following section discusses the relevance of these findings with respect to the specific aims of the studies and the background.
literature relevant to the validation of a CS for patients with nonspecific chronic LBP and MCI.

8. What drives the altered motor control?

The present series of studies demonstrate that motor control patterns exhibited by patients with AEP and FP are different from each other and from those in healthy subjects. These motor patterns observed in the LBP subjects appear maladaptive and potentially provocative. However there is no clear answer to the question of causality. In other words, it is not known what leads to these very different motor control patterns.

In light of this, it is important to highlight some of the more recent work by our team. Astfalck et al. (2010a,b) showed that similar postural characteristics, but not the EMG patterns, are present in adolescents with nonspecific chronic LBP suggesting that they are present early in development of the disorder. The fact that the EMG changes were not present in these adolescents suggests they are present early in development of the disorder. The fact that the EMG changes were not present in these adolescents suggests that the motor changes may evolve with the disorder and may be secondary, whereas the kinematic patterns present early and may in themselves be provocative.

In support of this view a study investigating sagittal standing posture by Smith et al. (2008) in 14 year olds highlighted that some postural sub-groups carry an increased risk for LBP. It was suggested that some of these postural changes may be influenced by developmental and/or genetic factors, or as a response to patterns of loading such as increased BMI, and carry a greater risk for LBP.

Longitudinal prospective studies are necessary to further unravel the question of causality, but to date none have been published. Several recent extensive literature reviews have discussed models explaining the mechanism underlying pain and dysfunction in the low back (for example Hodges and Moseley, 2003; van Dieen et al., 2003; Moseley and Hodges, 2004). A detailed review of all the potential reasons that underlie these findings is beyond the scope of this review paper, rather the purpose of this section is to discuss the importance of these studies in light of some of the proposed mechanisms.

Why would patients develop behaviours of self-provocation?

While it is not yet fully understood, it is hypothesised that complex changes in the central nervous system result in altered patterns of sensorimotor integration (Moseley, 2003). As a consequence, the chronic LBP patient is left with disrupted feedback sense and altered body schema. This hypothesis fits with findings of impaired repositioning sense (O’Sullivan et al., 2003), impaired two point discrimination (Moseley, 2008; Luomajoki and Moseley, 2009), and cortical changes in chronic LBP patients (Wand et al., 2011). Other factors such as stress, anxiety, patients beliefs and clinicians advise to hold erect postures and co-contract the spine with an inherent underlying maladaptive movement behaviour. Studies II-IV demonstrated that the direct experience of pain and disability or (iii) driven by levels of fear avoidance beliefs. Studies II-IV demonstrated that the patients consistently postured themselves in the direction of reported pain provocation.

These results support the concept that these patients present with an inherent underlying maladaptive movement behaviour that acts as a potential ongoing peripheral nociceptive drive rather than as a strategy to avoid pain. This kind of provocative pain phenomena has been clearly reported by Sullivan et al. (2009) who reported that repeated lifting tasks resulted in a summation of pain for those with nonspecific chronic LBP. It is our hypothesis that one of the mechanisms for this pain summation is the presence of maladaptive movement behaviours, previously described by Szeto et al. (2005) in neck and arm pain subjects during a typing task.

It is further hypothesised that a multitude of factors could conceivably change the spinal motor control leading to this MCI such as, physical factors (mechanism of injury, previous injury, where initial adaptive behaviours to pain in the acute phase of a disorder remain and become maladaptive in the chronic phase of the disorder), altered cortical organisation resulting in decreased body awareness and proprioception, cognitive factors such as beliefs about body schema, psychological factors (personality trait) and changes in neuroplasticity secondary to a chronic pain disorder and possible (gender related) genetic factors as well as factors such as the beliefs and training methods of
physiotherapists that have tended to reinforce training of muscles such as lumbar multifidus.

10. Clinical implications

The findings of this series of studies confirm the presence of distinct patterns of MCI in the AEP and the FP groups. These results advance the clinical understanding of nonspecific chronic LBP patients and have potential implications for the classification and therapeutic management of nonspecific chronic LBP subjects.

Contemporary literature advocates a process of diagnostics, where factors across multiple domains that are involved in these disorders are identified within a bio-psycho-social framework. This is as a result of a complete subjective and physical evaluation that identified the patient’s experience of pain, its behaviour, functional impairments, beliefs, coping, pain-related fear and distress. The examination involves analysis of the functional impairments reported by the patient to determine if the movement and pain behaviours are adaptive (protective) or maladaptive (provocative) (Elvey and O’Sullivan, 2004; O’Sullivan, 2004, 2005).

Consistent with this body of clinical literature, Study IV demonstrated that not one single factor but rather a series of factors (involving static posture, dynamic movement including both kinematics and an inability to relax spinal muscles) discriminated between the three groups examined in this study. An important clinical implication of Study IV is therefore that to clinically sub-classify nonspecific chronic LBP patients with MCI, clinicians should not simply rely on one test, but rather consistency across a series of functional tests that are linked to the patient’s pain behaviour and functional impairments.

Study I demonstrated that clinicians agree on the classification of nonspecific chronic LBP and MCI using this reasoning process. While the above described inter-examiner reliability study suggests that substantial specialised training for clinicians is required, the feasibility to implement the CS into clinical practice seems satisfactory.

The presentation of these patients was commonly related to increased co-contraction of the trunk stabilising muscles during pain provocative tasks, and an inability to relax muscles such as Superficial Lumbar Multifidus during forward bending with associated differences in spinal posture and movement patterning. These findings coupled with other recent research highlighting that absence of timing delays in the transverse abdominis in nonspecific chronic LBP subjects (Gubler et al., 2010), question current clinical beliefs regarding spinal instability and deficits in the spines stabilising muscles as an underlying basis for these disorders. It may in fact be that the increased motor activity of the spines stabilising muscles in some cases results in increased and abnormal loading forces across pain sensitive structures. This may be a reason why clinical trials that target specific training of spinal stabilising muscles for nonspecific chronic LBP are no more effective than other interventions which have small effect sizes (e.g. spinal manipulative therapy) (Ferreira et al., 2006). It was recently demonstrated (Costa et al., 2009) in a randomised placebo-controlled trial that for people with chronic LBP motor control exercise produced short-term improvements in global impression of recovery and activity, but not pain. This also highlights that generic approaches to managing nonspecific chronic LBP using stabilising training do not address the individual motor control deficits identified in patients with the disorder. Acknowledging this factor requires a change in the belief systems of physiotherapists and other health professionals, with a focus less on muscle and more on behaviours and control of movement.

When developing treatment and management for chronic LBP patients it would appear essential to have an understanding of the mechanism underlying the disorder. A behavioural intervention that focuses on cognitive and behavioural change (Cognitive Functional Therapy, CFT) (O’Sullivan, 2004) directed to changing the maladaptive patterns of control and associated cognitive behaviours seems a logical clinical approach when considering this data. This approach involves changing patients beliefs, confronting their fears, providing them awareness of pain mechanisms, enhancing mindfulness of the control of their body during pain provocative functional tasks, training them to reduce excessive trunk muscle activity and change behaviours related to pain provocative postures and movements. This approach has the possibility to reduce the central wind up of pain and disability and unload sensitised spinal structures peripherally (O’Sullivan, 2004). The capacity of this approach to reduce pain, disability and fear as well as normalise abnormal motor control patterns was demonstrated in case Study V (Dankaerts et al., 2007). Preliminary results of recent research investigating the efficacy of CFT in a randomised controlled clinical trial (RCT) supports its efficacy for nonspecific chronic LBP (Fersum et al., 2009b). We await the publication of the full results of this trial.

11. Limitations of present research

Despite its’ strengths, a number of limitations need to be highlighted. The application of strict inclusion/exclusion criteria (inherent to sub-classification) limits the clinical generalisability of the results of Study II – IV to patients with an AEP and FP classification. The fact no other clinical sub-groups were evaluated was based on logistics and convenience of sampling. It is acknowledged that the nonspecific chronic LBP group consists of other distinct sub-groups than the two sub-groups with clinical signs of MCI.

It is further acknowledged that the results of the EMG studies conducted (Study III and IV) are limited to the superficial muscle sites under examination. Therefore future work should focus on the involvement of deeper muscles deemed important in LBP, such as quadratus lumbarorum, transversus abdominis, deep lumbar multifidus and psoas (Ingber, 1989; De Franca and Levine, 1991; Hodges and Richardson, 1996; McGill et al., 1996; Andersson et al., 2002; Barker et al., 2004).

Finally, it could be argued that a potential limitation of Study I and IV was the use of ‘expert’ clinician’s classification as ‘gold standard’ as it is prone to bias. However, in the absence of a true criterion standard for MCI diagnosis, this method reflects current clinical practice (Gracovetsky et al., 1995).

12. Recommendations for further research

Further clinical research in the form of RCTs is required, comparing CFT to other ‘approaches’. This is the logical and essential next step in the multi-step validation process outlined in this review to further test the predictive validity of the proposed CS.

Further investigation (including brain research) is required into the underlying basis of the development of these patterns and why some patients appear to develop provocative movement behaviours that maintain pain cycles.

References


