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Lumbar kinematic variability during gait in chronic low back pain and associations with pain, disability and isolated lumbar extension strength

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ABSTRACT

Background: Chronic low back pain is a multifactorial condition with many dysfunctions including gait variability. The lumbar spine and its musculature are involved during gait and in chronic low back pain the lumbar extensors are often deconditioned. It was therefore of interest to examine relationships between lumbar kinematic variability during gait, with pain, disability and isolated lumbar extension strength in participants with chronic low back pain.

Methods: Twenty four participants with chronic low back pain were assessed for lumbar kinematics during gait, isolated lumbar extension strength, pain, and disability. Angular displacement and kinematic waveform pattern and offset variability were examined.

Findings: Angular displacement and kinematic waveform pattern and offset variability differed across movement planes; displacement was highest and similar in frontal and transverse planes, and pattern variability and offset variability higher in the sagittal plane compared to frontal and transverse planes which were similar. Spearman's correlations showed significant correlations between transverse plane pattern variability and isolated lumbar extension strength ($r = -.411$) and disability ($r = .401$). However, pain was not correlated with pattern variability in any plane. The r^2 values suggested 80.5% to 86.3% of variance was accounted for by other variables.

Interpretation: Considering the lumbar extensors role in gait, the relationship between both isolated lumbar extension strength and disability with transverse plane pattern variability suggests that gait variability may result in consequence of lumbar extensor deconditioning or disability accompanying chronic low back pain. However, further study should examine the temporality of these relationships and other variables might account for the unexplained variance.

1. Introduction

Chronic low back pain (CLBP) is a highly prevalent musculoskeletal disorder (National Institute for Health and Clinical Excellence, 2009; Waddell and Burton, 2000; Walker, 2000; World Health Organisation, 1998) with costs amounting to billions worldwide (Ekman et al., 2005; Freburger et al., 2009; Guo et al., 1999; Katz, 2006; Maniadakis and Gray, 2000; Ricci et al., 2006; Stewart et al., 2003; van Tulder et al., 1995; Waddell et al., 2002). Despite its prevalence, in as much as 85% of LBP cases no specific patho-anatomical diagnosis can be found (White and Gordon, 1982). However, more recently it is acknowledged as a multifactorial condition with a variety of associated dysfunctions (National Research Council, 1998; National Research Council and The Institute of Medicine, 2001). One of the multifactorial dysfunctions reported is gait variability (Roffey et al., 2010; Vogt et al., 2001; Waddell et al., 1997). It has been suggested that deficiencies in motor control during gait may produce excessive stresses to the lumbar spine, which may contribute to development of CLBP (Vogt et al., 2001). However, a recent review has suggested that there is evidence against walking itself being causally associated with CLBP (Roffey et al., 2010).

Healthy participants demonstrate relatively low stride-to-stride variability in lumbar kinematic patterns during both level and incline gaits (Vogt and Banzer, 1999). However, greater stride-to-stride variability at the lumbar spine in all movement planes (Vogt et al., 2001), greater frontal plane coordination variability of the pelvis and trunk (Lamoth et al., 2006a; Seay et al., 2011a) and more rigid transverse plane coordination variability of the pelvis and trunk (Lamoth et al., 2002, 2006a; van Der Hoorn et al., 2012) are reported in participants with CLBP compared with healthy controls. It also appears that pain per se may not be responsible for these gait differences. Lumbar spine kinematics during gait appear to be complex and developed over time, as patterns are evident before pain is experienced (Anders et al., 2005) and both induced pain and fear of pain produce little change in muscle activity in CLBP patients (Lamoth et al., 2004). Indeed recently studies have shown that even

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those with a previous history of CLBP who are currently asymptomatic demonstrate abnormal gait patterns (Crosbie et al., 2012; Seay et al., 2011a). Thus pain per se may not be the factor responsible. There is contrasting evidence reporting no residual effect upon gait from an episode of low back pain in nurses returning to work with very low pain levels (Rowe and White, 1997); however this study lacked a directly comparable control group.

Evidence instead suggests that the lumbar extensor musculature might play a role in gait variability in CLBP (Arendt-Nielsen et al., 1996; Hanada et al., 2011; Lamoth et al., 2004, 2006a, 2006b; van Der Hulst et al., 2010a, 2010b, 2010c; Vogt et al., 2003). It appears that the kinematic patterns seen in participants with CLBP are combined with poorer erector spinae activity adaptability to unexpected perturbations (Lamoth et al., 2004), or walking velocity changes (Lamoth et al., 2002). In fact, the findings of numerous studies are suggestive of muscular dysfunction of the lumbar extensors during gait in those with CLBP compared with asymptomatic controls (Arendt-Nielsen et al., 1996; Lamoth et al., 2004, 2006a, 2006b; Vogt et al., 2003). Hanada et al. (2011) also report that where asymptomatic controls significantly activated their rectus abdominus and internal obliques more, symptomatic participants had significantly greater activation of the lumbar extensors. More recent work shows evidence of greater lumbar extensor activity in participants with CLBP compared with controls (van Der Hulst et al., 2010a), at a range walking velocities (van Der Hulst et al., 2010b), and that neither disability nor fear of movement is associated with this greater activity (van Der Hulst et al., 2010a). However, different coping strategies may be associated with either greater activity (catastrophizing) or greater relaxation during double support (distraction) suggesting some influence of cognitive control (van Der Hulst et al., 2010c).

Human gait is normally quite robust in the face of muscular weakness of the lower limbs (van Der Krogt et al., 2012). The lumbar spine, however, may play a primary role in human bipedal gait (Gracovetsky, 1985). It is possible that the greater activation of the lumbar extensors, and altered lumbar spine kinematics during gait in participants with CLBP, are a manifestation of the lumbar extensor deconditioning (i.e. reduced lumbar extensor strength/endurance, atrophy, and excessive fatigability) commonly associated with CLBP (Steele et al., 2014). Greater activation in the face of fatigue due to deconditioning could be a compensatory attempt to maintain control of the lumbar spine during gait. Hart et al. (2009) demonstrate that inducing fatigue in the lumbar extensors impacts lumbar kinematics during running gait of healthy participants and participants with CLBP. Arjunan et al. (2010) also show significantly greater lumbar extensor activity during running gait in participants with CLBP. Indeed, prospective evidence has demonstrated that reduced lumbar extensor strength/endurance, atrophy, and excessive fatigability increase risk of low back injury and LBP in asymptomatic persons (Steele et al., 2014). Thus it may be responsible for the development of the gait variability associated with CLBP also.

Considering this it was therefore of interest in the present study to examine the relationships between lumbar kinematic variability during gait, with pain, disability and isolated lumbar extension (ILEX) strength. Previous research has focused upon trunk/pelvis co-ordination (Lamoth et al., 2002, 2006a, 2006b, 2008; Seay et al., 2011a, 2011b; Selles et al., 2001). Those interested in stride-to-stride variability of the lumbar spine with respect to the pelvis instead have utilised Winter's coefficient of variation (CV) (Winter, 1983) to examine the consistency of movement patterns using the ensemble average of the raw waveforms of repeated trials (Vogt and Banzer, 1999; Vogt et al., 2001). However, a new method of differentiating between pattern and offset variability has been recently suggested (O'Dwyer et al., 2009). A large mean offset value effectively deflates the value calculated for variability using the CV (O'Dwyer et al., 2009). Because of this O'Dwyer et al. (2009) have suggested the use of methods to differentiate the offset from calculation of the variability in the waveform pattern; the latter they suggest being far more representative of movement replication whereas the offset incorporates a greater degree of other variance sources (i.e. marker error). Thus this study in particular aimed to examine variation in lumbar kinematic pattern variability in relation to pain, disability and isolated lumbar extension (ILEX) strength.

2. Methods

2.1. Study design

The study was part of a wider investigation examining ILEX in participants with CLBP published in part elsewhere (Steele et al., 2013a). Gait data were also collected as part of this wider investigation. The present manuscript presents the cross-sectional data from the combined sample of the study collected at baseline.

2.2. Participants

Thirty eight participants (males n = 21, females n = 17) were initially identified and recruited into the wider investigation by posters, group email and word of mouth from a University and the surrounding locality. Direct referral was also provided from a local private chiropractor through posters in their practice. Inclusion criteria were as follows; participants suffered from non-specific low back pain having lasted longer than 12 weeks (Frymoyer, 1988) and had no medical condition for which resistance training would be contraindicated. Exclusion criteria were as follows; participants must have no medical condition for which movement therapy would be contraindicated. These included: acute (not re-occurring) low back injury occurring within the last 12 weeks, pregnancy, evidence of sciatic nerve root compression (sciatica), leg pain radiating to below the knee, paraesthesia (tingling or numbness), current tension sign, lower limb motor deficit, current disc herniation, previous vertebral fractures or other major structural abnormalities. All participants were cleared prior to involvement in the study by either their General Practitioner or the Chiropractor in the research group and provided written informed consent. The study was approved by the NHS National Research Ethics Service, Southampton & South West Hampshire Research Ethics Committee B (REC Reference: 11/H0504/9).

2.3. Equipment

Participants' stature was measured using a stadiometer (Holtan ltd, Crymych, Dyfed), body mass measured using scales (SECA, Germany) and Body Mass Index (BMI) calculated. Isometric ILEX strength testing and ROM were performed using the (MedX, Ocala, Florida; Fig. 1). Fig. 1 shows the restraint system. The has been shown to be reliable in assessing isometric strength at repeated angles in asymptomatic (Graves et al., 1990) and symptomatic participants (Robinson et al., 1992), and valid in measurement through removal of gravitational effects (Pollock et al., 1991) and pelvic movement (Inanami, 1991). Pain was measured using a 100 mm point visual analogue scale (VAS) (Ogon et al., 1996), and disability measured using the revised Oswestry disability index (ODI) (Fairbank et al., 1980). Gait kinematic variables were captured at 500 Hz using a 10 MX T20 camera three dimensional motion capture system (Vicon, Oxford) and analysed using both Vicon Nexus software version 1.4.116 (Vicon, Oxford), MATLAB version R2012a (MathWorks, Cambridge) and Microsoft Excel version 2010 (Microsoft, Reading).

2.4. Participant testing

For baseline testing participants visited the lab on three occasions. Participants were required to complete the VAS and the ODI on their first visit to the laboratory. The first two visits also involved testing of isometric ILEX strength. This was tested on separate days (at least 72 h apart in order to avoid the effects of residual fatigue or soreness).