

**The immediate effect of a 5-minute flexed posture on Lumbar Spine Reposition Sense.**

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## **ABSTRACT**

Proprioceptive control is considered important for maintenance of spinal stability and prevention of injury, and evidence exists to suggest that spinal proprioceptive structures, which are reflexive and viscoelastic, may be challenged by prolonged flexed postures. Alteration to lumbar spine position sense has been associated with low back pain patients; however, there has been little investigation into the effect different postural interventions may have on lumbar spine position sense in asymptomatic subjects. The aim of the current study was to investigate lumbar spine position sense after a 5-minute flexed posture in asymptomatic subjects. This dissertation is comprised of two main sections; a literature review followed by a manuscript for a research report that has been prepared in accordance with submission requirements for *Manual Therapy*. Following a familiarisation procedure, 30 asymptomatic subjects undertook two position sense tests to a neutral lumbar spine posture in the sagittal plane; one following a 5-minute fully flexed seated posture, one following a 5-minute sidelying posture, with a 15-minute interval in between tests. Absolute errors were calculated from data recorded by two orientation sensors, and compared between groups. Results showed a reduced lumbar spine reposition sense following 5-minutes in a flexed posture as compared with following 5-minutes in a sidelying position ( $p=0.042$ ), mean difference  $2.7^\circ$  (95% CI 0.10 to  $5.29^\circ$ ). The implications of this finding in relation to injury are discussed.

**Keywords:** *Low back pain; Proprioception; Position sense; Reposition sense.*



## **DECLARATION**

***Name of candidate:*** *Markos Pantelides*

This Research Project is submitted in partial fulfilment for the requirements for the Unitec degree of Masters of Osteopathy.

### ***Candidate's Declaration***

I confirm that:

- This Research Project represents my own work;
- The contribution of supervisors and others to this work was consistent with the Unitec Regulations and Policies;
- Research for this work has been conducted in accordance with the Unitec Research Ethics Committee Policy and Procedures, and has fulfilled any requirements set for this project by the Unitec Research Ethics Committee.

Research Ethics Committee Approval Number: 2008.831

Candidate Signature:

Date:

Student number: 1165871



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## **PREFACE**

This research project is divided into three sections. Section 1 consists of a Literature review that will firstly examine low back pain and the risk factors of sustained postures, then discuss position sense testing and literature supporting the methods used in the current study. Section 2 is a manuscript for a research report that has been formatted in accordance to *Manual Therapy* submission requirements. Note the literature review and manuscript use the *Manual Therapy* style of referencing as stipulated by the publisher. Section 3 of the dissertation is the appendices containing pictures of the experimental setup, documentation of ethics approval, tables not included in the other two sections, and the guide to authors for submission to *Manual Therapy*.

## TABLE OF CONTENTS

Abstract.....	2
Declaration.....	3
Acknowledgements.....	4
Preface.....	5
Table of Contents.....	6
List of Tables.....	7
<b>Section 1 – Literature Review.....</b>	<b>8</b>
Introduction.....	9
Low Back Pain Epidemiology.....	11
Aetiology of Low Back Pain.....	18
Altered Neuromuscular Control.....	22
Low Back Pain and Proprioception.....	25
Seated posture and Low Back Pain.....	35
Posture and Reposition Sense.....	37
Conclusion.....	39
References.....	40
<b>Section 2 – Manuscript.....</b>	<b>52</b>
Abstract.....	54
Introduction.....	55
Methods.....	60
Results.....	64
Discussion.....	65
Conclusion.....	72

References.....	73
<b>Section 3 – Appendices.....</b>	<b>86</b>
Appendix A – Table 1.....	87
Appendix B – Tables 2 and 3.....	89
Appendix C – Picture of experimental setup.....	90
Appendix D – Copy of Ethics approval.....	91
Appendix E – Manual therapy guidelines for authors.....	92

### LIST OF TABLES

Table 1 - Summary of studies on position sense & low back pain.....	87
Table 2 - Summary of descriptive data for reposition error tests 1 and 2.....	89
Table 3 - Summary of <i>t</i> -test results for reposition error tests 1 and 2.....	89

**Section 1:**

# **Literature Review**

## **INTRODUCTION**

Low back pain (LBP) is one of the most common medical problems of the middle-aged population, and from society's point of view, it is the most costly musculoskeletal disease of industrialized countries (Andersson, 1997; Van Tulder et al., 1995; Waddell, 1998). LBP causes a high level of morbidity by negatively affecting occupational and recreational activities, as well as activities of daily life.

Patients with LBP are known to have altered motor control (dysfunction) in the lumbopelvic region (Hodges, 2004) and, as methods of measuring proprioception in the region are devised, evidence is emerging that proprioception is also impaired (Brumagne et al., 2000; Brumagne et al., 2004; Leinonen et al., 2003; Mok et al., 2004; O'Sullivan et al., 2003; Parkhurst & Burnett, 1994). A loss of proprioception would contribute to neuromuscular dysfunction and likely poor segmental stability in LBP patients, which may lead to an increase in the risk of injury or further injury (Brumagne et al., 1999b).

Other research has shown that reflexive activity of proprioceptive structures, and viscoelastic properties of spinal tissues, are compromised by stretch or by flexed postures (Dolan et al., 1988; McGill & Brown, 1992), while sitting in a 'poor posture' has been found to be associated with LBP (Pearson's  $r = 0.57$ ,  $p < 0.001$ ) (Lee & Chiou, 1994). A review article by Lis et al. (2007) found that workers sitting for at least half a day or more who also adopted awkward

postures (defined as non-neutral i.e. bent forward or twisted trunk postures) were four times as likely as the general population to suffer from LBP. Studies investigating position sense on asymptomatic subjects, pre and post an intervention are few. It is generally believed that poor seated posture leads to LBP, and that LBP is related to decreased lumbar proprioception, but the relationship between sustained flexed posture and lumbar position sense has been scarcely studied and may be a useful way of investigating the aetiology of LBP.

The purpose of this review is to highlight current knowledge of LBP, sustained flexed posture, and lumbar spine position sense, with a focus on studies investigating lumbar spine position sense in symptomatic and asymptomatic subjects. It includes discussion on the prevalence, incidence, and aetiology of LBP; commonly adopted sustained postures; anatomical structures involved with proprioception; and position sense evaluation methods.

### **LITERATURE SEARCH**

A comprehensive literature search using electronic databases including Science Direct, Ebsco, Scopus, Academic Search Premier, the Cumulative Index to Nursing and Allied Health Literature, and the Medline database was undertaken to identify literature relating to LBP, lumbar proprioception, seated posture, position sense, reposition sense, and slouched posture. Additional studies were added by searching of the reference lists of original

investigations and review articles.

# LOW BACK PAIN EPIDEMIOLOGY

## Definition of LBP

Pain, in the sense of physical pain, is a typical sensory experience that may be described as the unpleasant awareness of a noxious stimulus or bodily harm. For scientific and clinical purposes, pain is defined by the International Association for the Study of Pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage"(Merskey & Bogduk, 1994). Pain is said to be subjective, because each individual learns the sensation of pain through their own experiences related to injuries in earlier life (Merskey & Bogduk, 1994).

LBP can be defined as pain perceived as arising anywhere within a region bounded superiorly by an imaginary transverse line through the tip of the last thoracic spinous process, inferiorly by an imaginary transverse line through the tip of the first sacral spinous process, and laterally by vertical lines tangential to the lateral borders of the lumbar erector spinae (Merskey & Bogduk, 1994) . It may further be described by the length of time symptoms persist: Acute LBP lasts less than six weeks, Subacute LBP lasts from six to 12 weeks, and Chronic LBP persists for more than 12 weeks (Bratton, 1999). LBP may also be classified according to aetiology. Mechanical or nonspecific LBP has no serious underlying pathology or nerve root compromise. Secondary LBP, occurring in fewer than 2% of patients, is associated with underlying pathology such as infection, prolapse, or tumour (Kuritzky et al., 2002). Clinically, it is generally

accepted that subjects with LBP, but no evidence of nerve root irritation or compression may also experience referred pain to the buttock, groin or thighs.

### Prevalence

A multitude of studies have investigated the prevalence of LBP in various populations, however, there exists a large variance between studies regarding the different time perspectives used, and the definitions and categorisation of back pain. Prevalence periods include point<sup>1</sup> (Pellis  et al., 2009), one-month (Papageorgiou et al., 1995), six-months (Cassidy et al., 1998), one-year (Leboeuf et al., 1996; Walker et al., 2004), and lifetime prevalence (Cassidy et al., 1998; Walker et al., 2004). Some of these studies have also examined the intensity of LBP and the degree of disability caused by LBP (Cassidy et al., 1998).

In a cross sectional, mailed survey of 1200 Saskatchewan adults, Cassidy et al. (1998) estimated that 28.4% (95% confidence interval (CI), 25.6% to 31.1%) of the Saskatchewan adult population were experiencing LBP at the time of the survey, and 84.1% (95% CI, 81.9% to 86.3%) had experienced it during their lifetime. Overall in the previous six months, 48.9% (95% CI, 45.9% to 52.0%) of the population had experienced low intensity/low-disability LBP, 12.3% (95% CI, 10.3% to 14.4%) had experienced high-intensity/low-disability LBP, and an

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<sup>1</sup> Point prevalence is a measure of the proportion of people in a population who have a condition at a particular time, such as a particular date.

additional 10.7% (95% CI, 8.8% to 12.5%) had experienced high-disability LBP. There was little variation in the estimates between age groups, but women experienced more high-disability back pain than men (14.9% cf. 7.3% for men). The authors concluded that low-intensity/low-disability LBP is a common problem in the general population.

In a prevalence and disability study of 2070 Australian adults, Walker et al. (2004), using similar categorization and prevalence points, reported findings similar to those of Cassidy et al. (1998). The sample point prevalence was estimated at 25.6% (95% CI, 23.6% to 27.5%), 12-month prevalence was 67.6% (95% CI, 65.5% to 69.7%), and lifetime prevalence was 79.2%, (95% CI, 77.3% to 81.0%). In the previous six-month period, 42.6% (95% CI, 40.4% to 44.8%) of the adult population had experienced low-intensity pain that was associated with low level disability. Another 10.9% (95% CI, 9.6% to 12.3%) had experienced high intensity-pain, but still low disability from this pain, however, 10.5% (95% CI, 9.2% to 11.9%) had experienced high-disability LBP. The Authors concluded that LBP is a common problem in the Australian adult population, and note that while most LBP is low-intensity and low-disability pain, over 10% had been highly disabled by LBP in the past six months.

Papageorgiou et al. (1995) conducted a larger scale study gaining 4501 responses from the patients of two family practices in a socio-demographically mixed suburban area of South Manchester. The study focussed only on the one-month prevalence of LBP, with no categorisation of LBP intensity or associated disability. The one-month period prevalence of LBP

was 39% (35% in males, 42% in females). The age distribution was unimodal, with peak prevalence in those aged 45 to 59 years. Responders to the first mailing had a small but non-significantly greater prevalence compared with those who responded to the second or third mailing. Non-responders had a subsequent consultation rate for LBP that was 22% lower than that for the survey responders. After considering potential differences in non-responders, the estimated one-month prevalence of LBP was between 35% and 37%, the authors noting that prevalence figures in survey responders may overestimate the true population prevalence by a modest amount.

While the literature on the epidemiology of LBP is accumulating, for the most part studies are restricted to countries with high per capita income, which comprise less than 15% of the world's population (Volinn, 1997). Interestingly, Volinn (1997) conducted a review of prevalence studies in low and middle income countries with the hypothesis that LBP rates are higher in low and middle-income countries than in high-income countries, not only because hard physical labour is more prevalent in low-income countries, but also because, unlike high-income countries, hard physical labour for older workers in low-income countries often is unavoidable. Point prevalence rates on the whole were higher among the general populations of selected high-income countries than among rural low-income populations; specifically, rates are two to four times higher among Swedish, German, and Belgium general populations than among Nigerian, southern Chinese, Indonesian, and Filipino farmers. Within low income countries, rates are higher among urban

populations than among rural populations. The considerably lower rates among populations of low-income farmers compared with rates of the affluent populations of selected northern European countries indicate that, contrary to the hypothesis proposed, hard physical labor itself is not necessarily related to LBP, but rather, that less physical and sedentary occupations may present a greater risk for back pain.

Another systematic review published in 2000, identified 30 methodologically acceptable studies for Meta-analysis. Of the studies analyzed there were notable differences in study design, patient age, mode of data collection, potential temporal effects, and prevalence results. Point prevalence ranged from 12% to 33%, one-year prevalence ranged from 22% to 65%, and lifetime prevalence ranged from 11% to 84% (Walker, 2000). This review clearly highlights that while there is a large variation between studies of different methodology and/or of different populations, that, even with respect to the most conservative estimates, LBP still presents a substantial problem to modern societies.

A comprehensive search was unable to locate any statistics with specific reference to the prevalence of pain only in the low back for a New Zealand population, however, a combined neck or back pain figure was available from the 2006/07 New Zealand Health Survey. The prevalence of neck or back pain lasting or expected to last for 6 months or more, from 755,100 participants was 24.2% (95% CI = 23.2% to 25.2%), males 23.1% (95% CI = 21.6% to 24.6%) and females 21.3% (95% CI = 20.3% to 22.4%) (New Zealand Ministry

of Health, 2008). Because the above statistics include both neck and back pain for the New Zealand population, it is difficult to make comparison with other international studies; however it indicates that a substantial number of New Zealanders too, may be affected by chronic LBP.

### Incidence

While the prevalence of LBP is highly investigated, the incidence<sup>2</sup> and natural course of LBP is an area relatively poorly researched. A study by Cassidy et al. (2005) investigated the incidence and course of LBP in the general Canadian population. The cumulative annual incidence was 18.6% (95% CI = 14.2% to 23.0%), most episodes of which were mild, with only 1.0% (95% CI = 0.0% to 2.2%) of the population developed intense, and 0.4% (95% CI = 0.0% to 1.0%) of the population developed disabling LBP. Resolution occurred in 26.8% of cases (95% CI = 23.7% to 30.0%), and 40.2% (95% CI = 36.7% to 43.8%) of episodes persisted. The severity of LBP increased for 14.2% (95% CI = 11.5% to 16.8%) and improved for 36.1% (95% CI = 29.7% to 42.2%). Of those that recovered, 28.7% (95% CI = 21.2% to 36.2%) had a recurrence within six months, and 82.4% of it was mild LBP. Younger subjects were found to be less likely to have persistent LBP (incidence rate ratio, 0.88; 95% CI = 0.80 to 0.97) and more likely to have resolution (incidence rate ratio, 1.26; 95% CI = 1.02 to 1.56). Overall, most new and recurrent LBP episodes were mild, but less than one third of cases resolved annually and more than 20% recurred within six

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<sup>2</sup> Incidence is a measure of the risk of developing some new condition within a specified period of time.

months. LBP episodes were found to be more recurrent and persistent in older adults (Cassidy et al., 2005).

A pathological cause cannot be identified for most new episodes of LBP presenting to the general practitioner, and one important potential influence on susceptibility is the patient's previous pain experience. Papageorgiou et al. (1996) investigated the incidence of LBP and the effect of previous pain experience on the incidence of LBP in 2715 adults in the South Manchester region, United Kingdom. The 12-month cumulative incidence of new consulting episodes was 3% in males and 5% in females; and for new non-consulting episodes 31% in males and 32% in females. Those with a history of previous LBP had twice the rate of new episodes, both consulting and non-consulting, compared to those with no LBP in the past. Neck pain or pain in other musculoskeletal sites at baseline also doubled the risk of a subsequent new episode of LBP. The results provided strong evidence that in those currently free of LBP, a previous history of the symptom substantially increases the risk of a further episode, with pain in other sites being an equally strong independent predictor of subsequent LBP (Papageorgiou et al., 1996).

Comparing the study of Papageorgiou et al. (1996) to that of Cassidy et al. (2005), we see large differences in the reporting of incidence rates. While this may be representative of the different populations studied the result may also reflect the lack of consensus in categorizing LBP severity between studies.

Using another methodology, George (2002) investigated the six-month incidence of 'clinically significant' LBP, using the Chronic Pain Questionnaire

(a 7-item scale that measures the intensity of chronic pain and associated disability). Of 848 respondents that reported no clinically significant LBP at baseline, a six-month cumulative incidence of 8% (95% CI = 6% to 10.4%) was found. In logistic regression models, marital status, rural residency, and history of back and neck pain were found to be associated with the onset of clinically significant LBP (George, 2002).

A recent Netherlands study investigated the incidence of occupational disability as a result of back pain from the period 1980-85 as compared to 1999-2000 (Steenstra et al., 2006). The authors found the incidence of occupational disability as a result of back pain decreased significantly by 37% (95% CI = 37% to 38%) in men and by 21% (95% CI = 20% to 24%) in women, after adjustment for age. The overall occupational disability as a result of all diagnoses decreased by 18% (95% CI = 18% to 19%) for men and increased by 34% (95% CI = 33% to 35%) for women. These figures indicate that occupational disability resulting from LBP is decreasing in total for both men and women, and LBP now accounts for a lower proportion of total occupational disability in both men and women. Despite the above findings it is surprising that the authors found the incidence of non-specific back pain and neck pain had increased by 196% (95% CI = 164% to 215%). The apparent incongruence of these results may possibly be explained by the emergence of new views on the management of back pain, in particular that continuation of work and activity is advisable in the management of back pain.

Looking at all the studies reviewed above we see that LBP severity is not uniformly categorized between studies, and also that there is a lack of consistency in chronicity categorization (i.e. acute, subacute, or chronic). Due to this, comparison of studies conducted on different sample populations is difficult, but still, it is clearly evident that LBP presents a problem of substantial proportions to the Western world. The most significantly affected age group appears to be 45-59 year olds, there is a slightly higher prevalence in women, and overall the numbers of people experiencing LBP seems to be increasing.

### **AETIOLOGY**

Due to the high prevalence, incidence, disability, and socioeconomic cost of LBP, investigation of the aetiology of LBP has been substantial. LBP may originate from many different conditions affecting the lumbar spine, or from many different structures within the lumbar spine. Conditions may include degenerative, infectious, inflammatory, neoplastic, neurological, and vascular, as well as congenital defect or traumatic injury. Non-specific chronic LBP (NSCLBP) is a description of back pain for which a cause cannot be definitively identified and a precise patho-anatomical diagnosis cannot be given (Slade & Keating, 2009). It accounts for approximately 85% of all LBP and does presume that specific pathologies, such as nerve root compression, infection or tumour have been ruled out by appropriate tests and imaging (Maher et al., 1999). Recent advances in our understanding of back pain

have shown that spinal pathology arises from interactions between genetic and environmental influences, and that subsequent pain and disability are strongly influenced by individual psycho-social factors, including personality (Dolan & Adams, 2007).

This literature review gives background to the Manuscript 'The immediate effect of a 5-minute flexed posture on Lumbar Spine Reposition Sense', which is a mechanically orientated study. As such, this review, although acknowledging the various pathological entities and psychosocial contributors to back pain, will focus on reviewing the mechanical and neuromuscular aetiology of NSCLBP.

Despite the wealth of research that exists however, and the growing knowledge of pathological conditions affecting the lower back, our understanding of NSCLBP is still relatively poor. In principle, any of the structures of the lumbar spine that receives an innervation could be a source of back pain. Accordingly, back pain could arise from any of the ligaments, muscles, fasciae, joints or discs of the lumbar spine (Adams et al., 2002). Early experimental studies in asymptomatic volunteers have shown that noxious stimulation of the back muscles (Kellgren, 1938), interspinous ligaments (Feinstein et al., 1954; Kellgren, 1939), dura matter (El Mahdi et al., 1981), zygapophysial joints (Fukui et al., 1997; Mooney & Roberston, 1976), and the sacroiliac joint (Fortin et al., 1994) can produce local and referred pain similar in quality and distribution to that seen in patients. These data corroborate that these structures can indeed be a source of back pain, but alone they do

not show causation in a particular patient (Adams et al., 2002). Regarding possible aetiologies, numerous structures have been investigated and discussed as being potentially causative of NSCLBP:

### Muscle

Muscular pain and spasm is a theory of back pain, which, having been previously popular, has now fallen mostly out of favour as a predominant diagnosis. Muscular pain, also known as myogenic pain, is thought to be associated with increased muscle tension resulting in a vicious tension-pain-cycle, leading to increased alertness and stress of the muscle (Kettenmann et al., 2007). Experimentally induced muscle pain can be produced by electrical stimulation, ischaemic contractions and algescic injection (Capra & Ro, 2004). However, in symptomatic patients, studies have failed to demonstrate electromyographic evidence or other features that independently correlate with pain from the allegedly affected muscle (Andersson & Bogduk, 1989). Interestingly, it has been found that muscle fibres themselves are not innervated by free nerve endings that code for nociception. Instead 'muscle nociceptors' are located on small arteries, arterioles and venules within the muscle tissue (Graven-Nielson & Mense, 2001). As such, muscle pain may be considered a misnomer and would be more appropriately labeled as vascular pain. While 'muscular pain' is a feasible diagnosis, it is not considered a major cause of non-specific chronic LBP.

### Ligament

Ligament sprain is an attractive explanation for acute LBP following exertion or effort, however no valid and reliable tests exist for diagnosis of a specific ligament (Adams et al., 2002). A couple of studies have used selective injection of the interspinous ligament with local anesthetic as a means of diagnosis. These studies report a 14% (Steindler & Luck, 1938) and a 10% prevalence (Wilk, 1995) of interspinous ligament pain, but this diagnostic technique has not been rigorously evaluated and neither of these studies incorporated controls for false positive responses.

### Zygapophyseal joints

A structure that has received reasonable attention as a source of LBP is the lumbar zygapophyseal joints (Schwarzer et al., 1994a; Schwarzer et al., 1994b; Schwarzer et al., 1995b; Schwarzer et al., 1995c; Yang & King, 1984).

Controlled studies using diagnostic blocks of these joints have shown that, in an elderly population, the prevalence of zygapophyseal joint pain is about 40% (Schwarzer et al., 1995b), while in younger injured workers, the prevalence is around 10-15% (Schwarzer et al., 1994a). Another study by Schwarzer et al. (1995) examined the computed tomography findings in patients with zygapophyseal joint pain, finding no demonstrable features that relate to the joint being painful. However, earlier biomechanical and post mortem studies exist that indicate the underlying pathology may be a disruption of the joint capsule (Yang et al., 1984), and/or microscopic impaction fractures (Farfan et al., 1970).

### Intervertebral Disc

Another key area of focus has been on intervertebral discs as a source of LBP. Discogenic pain means pain arising as a result of stimulation of nociceptive nerve endings in the intervertebral disc, and is distinct from disc prolapse, which involves displacement of a mixture of nuclear and annular material beyond the normal perimeter of the disc (Adams et al., 2002). Internal Disc Disruption has been identified as the cardinal pathological basis for lumbar discogenic pain, which is characterised by disruption of the internal architecture of the disc in the form of radial fissures extending from the nucleus to the outer annulus with the outer perimeter remaining essentially intact (Bogduk, 1991). The further the extent of the radial fissure, the more likely the disc is to be painful (Vanharanta et al., 1987). Schwarzer et al. (1995), using two diagnostic criteria; a positive response to controlled disc stimulation, coupled with demonstration of radial fissures on computed tomography-discography, found the prevalence of intervertebral disc degeneration to be at least 40% in patients with chronic LBP.

### Sacroiliac joint

The sacroiliac joint forms a major entity considered to cause chronic LBP. The gold standard for diagnosing this condition is considered to be intra-articular sacroiliac joint blocks (Adams et al., 2002). Using such blocks, two controlled studies have shown the prevalence of sacroiliac joint pain amongst people with chronic LBP below L5/S1 to be 30% (Schwarzer et al., 1995a), and 18.5% (Maigne et al., 1996).

A more recent article however, casts doubt on intra-articular sacroiliac joint blocks as a gold standard for diagnosis (Berthelot et al., 2006). This research states that the effects of two consecutive blocks are identical in only 60% of cases, and the anaesthetic diffuses out of the joint in 61% of cases. Diagnosis is confused because anaesthetic comes into contact with the sheaths of the adjacent nerve trunks or roots. Despite this challenge to the conventional diagnostic gold standard, the literature overall validates the sacroiliac joint as being a causative structure in a significant number of chronic LBP sufferers.

### **ALTERED NEUROMUSCULAR CONTROL**

The structures mentioned above have been implicated as being the actual tissues causing NSCLBP symptoms. Focussing our aetiological lens further out however, we may also identify theories of underlying factors or mechanisms that attempt to explain the aetiology of NSCLBP; that is, the theories of dysfunction or factors that lead to tissue dysfunction and pain.

One of these theories is that altered neuromuscular control may represent a causative factor in patients with recurrent LBP (Barker et al., 2004; Hides et al., 2008; Hodges & Richardson, 1996; Kiesel et al., 2007; Macdonald et al., 2009; Richardson et al., 2002; Tsao et al., 2008). A recent study investigated whether the control of the short and long fibres of the deep back muscles was different in people with recurrent unilateral LBP (who were symptom free during testing) as compared with a group of healthy volunteers.

Intramuscular and surface electrodes recorded the electromyographic (EMG) activity of the short and long fibres of the lumbar multifidus and the deltoid muscle, during a postural perturbation associated with a rapid arm movement. Short fibre EMG onset occurred later in participants with recurrent unilateral LBP than in healthy participants ( $p=0.022$ ). The short fibres were active earlier than long fibres on both sides in the healthy participants ( $p<0.001$ ) and on the non-painful side in the LBP group ( $p=0.045$ ), but not on the previously painful side in the LBP group (Macdonald et al., 2009). Deep back muscle activity is critical for normal spinal control, and the authors' findings that the activity of deep back muscles is different in people with a recurrent unilateral LBP, despite the resolution of symptoms may provide evidence of a candidate mechanism for recurrent episodes.

The role of the transversus abdominus muscle has also been extensively researched in relation to LBP, with ultrasound studies having shown altered recruitment of transversus abdominus in LBP patients (Ferreira et al., 2004; Hides et al., 2008; Kiesel et al., 2007). Other studies have identified that decreased Transversus Abdominus contraction leads to altered lumbopelvic stability (Hodges et al., 1996; Richardson et al., 2002). The subsequently altered lumbopelvic mechanics in patients with decreased transversus abdominus tone is widely considered to play a possible role in the aetiology of LBP.

A very recent study by Tsao et. al. (2008) investigated the possible reorganization of the motor cortex in association with postural control deficits

in patients with recurrent LBP, as compared with asymptomatic participants. By assessing the activity of Transversus abdominus during postural control, as well as cortical representation of trunk muscles, Tsao et al. (2008) found evidence that individuals' with recurrent LBP exhibit reorganization of trunk muscle representation at the motor cortex. Tsao et al. (2008) suggest this reorganization is associated with deficits in postural control.

The Psoas muscle, although receiving less attention than the muscles discussed above has also been the focus of investigation. In a study investigating 50 patients with unilateral LBP of 12 weeks or more, the authors reported a statistically significant decrease ( $p < 0.001$ ) in the cross sectional area of psoas on the symptomatic side (Barker et al., 2004). The magnitude of the decrease however was not reported. These studies, and the other muscular based studies discussed above, represent a small fraction of the research into altered muscular function/neuromuscular control and LBP. Although only briefly reviewed here, altered muscular function and/or neuromuscular control mechanisms have been a strong focus of research, and their dysfunction likely represents an important relationship to LBP.

## **LOW BACK PAIN AND PROPRIOCEPTION**

*Proprioception* is described as ascending information from the afferent receptors towards the central nervous system contributing to the neuromuscular control of movement (Lephart & Fu, 2000). It includes the sensation of joint movement and joint position (Marks, 1998; Swinkels & Dolan, 1998), allowing the body to maintain stability and orientation during both static and dynamic activities (Laskowski et al., 1997). Although proprioception may be considered a subset of neuromuscular control, in respect to the research project for which this literature review gives background, proprioceptive control is a highly relevant topic. What follows is an in-depth review of its relationship to LBP.

### Anatomical structures involved in proprioception

The afferent aspect of lumbar proprioception is derived principally from mechanoreceptors in the lumbar and adjacent regions. Mechanoreceptors are abundant in human lumbar facet joints (Ozaktay et al., 1991), in discs (Roberts et al., 1995), as well as in skeletal muscle, ligament and skin (Gandevia et al., 1992). Receptors found in the various tissues have been described as playing different roles depending on the range at a given joint (Swinkels & Dolan, 1998). Joint receptor afferents are believed to be activated towards the end of the range of motion, whereas muscle spindles are assumed to provide afferent input throughout the physiological range (Burgess et al., 1991).

## Measurement of Proprioception/Position sense

There are several methods to investigate the conscious aspects of proprioception. Either by awareness of the joint position of a body segment in passive movement, or by active movement instigated by the participant. The ability to perceive movement or orientation of a body segment in space, either actively or passively, is known as position sense (Newcomer et al., 2000a). Studies investigating proprioception have predominantly used position sense or reposition error (RE) as a measure of proprioceptive ability (Asell et al., 2006; Brumagne et al., 1999a; Brumagne et al., 1999b; Descarreaux et al., 2005; Dolan & Green, 2006; Gill & Callaghan, 1998; Iwasa et al., 2005; Newcomer et al., 2000a; O'Sullivan et al., 2003), while other measures such as passive motion threshold (Silfies et al., 2007), and force plate analysis (Nies & Sinnott, 1991) have also been employed.

Movement detection is markedly improved by muscular contraction and it has been suggested that it is more functionally relevant to assess proprioceptive deficits during the active implementation of normal movements, compared with assessing passive joint position sense (Brumagne et al., 1999b; Gill & Callaghan, 1998; Swinkels & Dolan, 2000). Any proprioceptive discrepancies that appear in the mid-range of active movement probably characterize irregular afferent information derived from muscle receptors. These muscles are the primary providers of proprioceptive information regarding joint position and movement (Brumagne et al., 1999b; Gandevia et al., 1992).

The reliability of active repositioning error was examined via a test-retest design in a study of 20 volunteers, with the authors concluding that i) healthy volunteers can in fact reposition their spine with considerable accuracy, and ii) that this ability does not change significantly on a day to day basis (Swinkels & Dolan, 1998). Another experimental protocol utilizing a 3-Space Fastrak to determine the error, within and between days, of 10 healthy subjects in reproducing a neutral lumbopelvic (T10-S2) position, found no statistically significant difference in asymptomatic subjects (mean diff. between days = 0°) (Maffey-Ward et al., 1996). These results infer that any substantial within-subject alteration to position sense, as a result of an intervention, can likely be attributed to that intervention rather than to natural variation.

While many investigations have used lumbar spine position sense as a measure of proprioception, there has been little consistency in the testing methods or testing positions used (Preuss et al., 2003). Seventy asymptomatic males were tested 3-dimensionally for lumbar spine repositioning accuracy. The researchers investigated three test positions (standing, sitting and four point kneeling), under two conditions (eyes open and blindfolded), using the neutral spine posture as the initial reference position. Both the accuracy and precision of lumbar spine repositioning was found to be significantly affected by test position. Repositioning errors (reflective of accuracy) were significantly larger in four point kneeling than in both sitting and standing, and significantly larger in sitting than in standing, under both eyes-open and blindfolded

conditions (Preuss et al., 2003). These results indicate that test position does have a substantial effect on the acuity of lumbar spine position sense, and while whichever method used in a particular study does allow comparison between groups, test position should be considered when examining the current literature on lumbar spine proprioception.

## **Studies utilizing lumbar spine position sense**

It has been shown in peripheral joints that stress or injury to a joint caused by instability may result from an individual's inability to control joint position accurately (Forwell & Carhahan, 1996; Smith & Brunolli, 1989), and strong evidence exists that repositioning acuity of the cervical spine is disturbed in chronic neck pain (Kristajansson et al., 2003; Loudon et al., 1997; Revel et al., 1991). It has been suggested, that in the lumbar spine, proprioceptive deficit may lead to altered coordination and delayed neuromuscular protective reflexes, resulting in muscular contraction which is too late to protect a joint from excessive movement (O'Sullivan et al., 2003). Altered proprioception is also theorised to lead to compromised segmental spinal stability, abnormal loading transmitted across joint surfaces, and possibly lead to pain and articular damage (Cholewicki & McGill, 1996; Radebold et al., 2001). Clinically, patients with LBP frequently demonstrate difficulty adopting or maintaining a neutral or midrange position of the lumbar spine (Lam et al., 1999), an observation that may indicate a proprioceptive deficit in LBP populations.

In the past decade numerous investigations have been conducted examining the relationship between proprioception and LBP, using position sense or repositioning error as a measure of proprioceptive ability. Various experimental protocols, LBP definitions, sample populations, and measurement tools have been employed, leading to a variety of findings. The mixed findings have made the relationship between LBP and altered lumbar spine proprioception a controversial one. Table 1 in Appendix A summarises the investigations of LBP and position sense. These studies are discussed in greater depth below.

#### *Studies reporting altered position sense in LBP patients*

Gill and Callaghan (1998) were the first researchers to investigate differences in lumbar spine position sense between asymptomatic (n=20) and LBP patients (n=20). They used a two group experimental design, and a device involving chest and pelvic harnesses, which may have provided large amounts of sensory input. Reposition sense was tested in two positions finding a statistically significant difference in both four point kneeling (2.43°, p<0.05) and in standing (2.25°, p<0.05). The target position used in this study was one of approximately 20° flexion.

Newcomer et al (2000a,b) conducted two studies very similar to each other comparing 20 LBP patients with 20 healthy subjects in the standing position. In the first study (Newcomer et al., 2000a) the authors observed no trunk repositioning differences between groups. In the second study (Newcomer et al., 2000b) the subjects stood with their legs and pelvis stabilised to limit

proprioceptive input from the lower limb, and had the unusual finding that LBP patients displayed 1.75° greater RE in flexion ( $p < 0.036$ ) and 1.5° less RE in extension ( $p < 0.015$ ). The authors speculate that the decreased RE found in extension could possibly be caused by increased activation of mechanoreceptors in facet joints of LBP patients.

Brumagne et. al. (2000) reported that Lumbosacral position sense was decreased in a group of LBP subjects as compared with that of an asymptomatic control group (Absolute Error difference=2.7°,  $P < 0.0001$ ). This study used a sitting pelvic tilting test with a piezoelectric accelerometer placed over the sacrum, with S2 being the only reference point. While this method may be useful in the measurement of sacral position and pelvic tilting, without any direct measure of lumbar spine position, the results cannot be considered synonymous with lumbar spine position sense. The sample of 23 patients was broadly defined in terms of LBP characteristics and lacked homogeneity.

O'Sullivan et al (2003) used a seated neutral target posture, using a clearly defined subgroup of 15 LBP patients who were diagnosed as having lumbar segmental instability, and compared their lumbar RE to 15 asymptomatic controls. This subgroup of LBP patients were found to exhibit 2.48cm greater RE than control subjects ( $p < 0.02$ ).

A study published by Koumantakis et al. (2002), compared 62 LBP subjects to 18 asymptomatic volunteers, measuring absolute error and variable error, and applying clinical applicability measures to determine the value of the

findings. Clinical applicability measures were limited, with right sided rotation absolute error the best with 83.3% specificity and 54.8% sensitivity. No location of pain for LBP patients was described, but all participants except for one were right handed. Simple comparisons of mean error values between the 2 groups with independent-samples *t*-tests showed significant differences of 0.645° variable error in flexion ( $p=0.01$ ), left rotation absolute error 1.27° ( $p=0.003$ ), and right rotation absolute error 1.44° ( $p=0.002$ ). All of these errors were greater in the LBP group. Although the size of these effects is not particularly large, they cannot be disregarded and do point towards some altered proprioceptive ability in LBP patients.

Descarreaux et al. (2005) assessed active standing position sense, using target positions of 15, 30, and 60° of flexion and 15° extension. Although they found no difference between groups in absolute RE, LBP subjects demonstrated longer movement time and smaller peak velocities and symmetry ratios than normal subjects. The authors conclude that given sufficient time, LBP patients could reposition their spines as accurately as control subjects. These findings however, of reduced rapidity of proprioceptive response, do indicate altered proprioceptive function in LBP patients, and may also have relevance to the interpretation of other studies. For example; in the studies by Newcomer et al. (2000a, b) in which the time taken to move to target positions was standardized to 5 seconds, it can be speculated that the standardization may have resulted in the authors finding larger differences in RE, than other studies not incorporating a standardized

timeframe. Given that standardizing or limiting the timeframe for movement is more likely to highlight differences in absolute RE between groups; it could also be argued that the enforced slow speed in assuming target positions used in Newcomer's studies (i.e. 5-seconds), may have decreased the difference in RE between groups, as compared to if a faster speed of target adoption was enforced.

As mentioned, it is hypothesized that lumbar spine proprioceptive deficit may lead to altered coordination and delayed neuromuscular protective reflexes, resulting in muscular contraction which is too late to protect a joint from excessive movement (O'Sullivan et al., 2003). That said, decreased timeliness of position sense, as well as absolute errors, may well predispose to injury, and should both be considered relevant variables in the measurement of position sense.

#### *Studies failing to report altered position sense in LBP patients*

A relatively poor single group study was conducted by Lam et al. (1999), in which RE was examined seated in 20 LBP subjects, moving from flexion to a target position of a neutral upright posture. Key flaws of this study relate to the sample population, which was a convenience sample drawn from the authors acquaintances and students from the University of Queensland, with no homogeneity of back pain characteristics. The authors, comparing their findings to an earlier study of 10 asymptomatic volunteers conducted by Maffey and Ward (1996), concluded that no proprioceptive deficit existed in LBP populations. Although the instrumentation and protocol used was

matched as closely to that of the earlier study as possible, the sample populations were only matched approximately in terms of age (Lam et al. = mean age 29yrs, Maffey & Ward = mean age 23yrs) and sex and the sample size of the comparison study by Maffey & Ward was only 10 participants.

A relatively large, single-blinded, controlled, multi-group comparative study by Asell et al. (2006), attempted to resolve the debate over whether lumbar repositioning acuity is reduced in patients with chronic LBP. Ninety-two chronic LBP patients were divided into distinct diagnostic subgroups and compared to 31 asymptomatic participants. Using a seated target position of 1/3 maximal extension to compare RE, the researchers reported no statistically significant difference between LBP patients and asymptomatic controls (constant error,  $p=.90$ ). RE was greater in the LBP group but the exact difference was not reported by the authors. The researchers incorporated a number of components to minimize methodological flaws of other studies. They used a large sample of patients with well-defined chronic LBP, blinded the experimenter to the patients back pain status, and restricted food intake for two hours prior to the experiment to minimize any abdominal cutaneous feedback. The authors acknowledge it may have been a methodological flaw to assess patients in only one target position, which restricts being able to generalize the findings to general reposition sense ability. While some other experiments have also used only one target position to measure RE, and thus limiting generalisability, the selection of an extension target position is unusual given the earlier research by Newcomer et al (2000b), which found extension

to be one position in which LBP patients RE's conflicted with the general findings.

The most recent, and largest study of reposition errors in LBP patients was conducted on 292 college athletes and included a follow up of three years (Silfies et al., 2007). Sixty of the participants (21%) had a history of some LBP in the previous 5 years, of which many (72%) had sustained only a single LBP episode. The average amount of time post injury was 23 months. Using a specially built apparatus which fixed the torso, and via a motorized swivel stool with a clutch, the authors measured active and passive position sense, as well as motion perception threshold in the transverse plane. The authors reported small, insignificant differences between groups in active RE, passive RE, and motion perception threshold. There was no reported difference in RE between participants who did or did not sustain an injury in the three year follow up period. Given that a number of studies have found a relationship between impaired lumbar reposition sense and LBP patients (Brumagne et al., 1999a; Gill & Callaghan, 1998; Newcomer et al., 2000b; O'Sullivan et al., 2003), the value of a prospective cohort study to try and determine a causal link between impaired lumbar proprioception and LBP is a valuable exercise. The value of this quite large study however seems undermined by a number of factors. Firstly, in trying to compare LBP history patients with asymptomatic patients the sample selection of patients who predominantly experienced only one episode of LBP an average of 23 months ago does not represent a true LBP population for comparison. In numerous other studies these patients

would have been categorized in the asymptomatic group (Descarreaux et al., 2005; Newcomer et al., 2000a; Newcomer et al., 2000b). Secondly, the specifically designed measurement instrument is previously untested. It is the current authors opinion that by fixing the torso and attempting to recreate target positions by twisting the stool below; i) may introduce confounding factors relating to hip and pelvis movement, and ii) the action in no way represents normal movement patterns in order to approximate the findings to normal proprioceptive ability. The decision to assess rotational RE and motion perception threshold seems an unusual choice given that rotational REs are relatively small compared to other planes of movement. Only one previous study has reported a deficit in rotational position sense in LBP populations (Koumantakis et al., 2002). The most significant differences have been found in sagittal plane repositioning tasks (Brumagne et al., 1999a; Descarreaux et al., 2005; Gill & Callaghan, 1998; Newcomer et al., 2000b; O'Sullivan et al., 2003). In Silfies et al. (2007)'s study of fit, active, college athletes, the entire sample population exhibited an average active RE of only 1.6° (SD 0.7). It is possible that relative differences in REs between participants may still be within a range that does not increase risk of injury and no definitive conclusions regarding the cause effect relationship is possible. Very subtle differences in RE were not shown to increase the risk of back pain in this particular population. It is still not clear what role more significant deficits in reposition sense might play, in other, perhaps more sedentary sample populations.

### *Summary of studies investigating LBP and position sense*

In reviewing the research examining lumbar position sense in LBP patients, there is no clear consensus to the findings. Numerous studies do exist however, to suggest some form of altered position sense in LBP patients. What is apparent is that numerous different instruments and methodologies have been used, and considerable variations in results have emerged. In what is a relatively new field of research it is still difficult to elucidate how subtle differences in methodology such as test position and protocol may affect results, or how sample selection and categorization of LBP subcategories may alter position sense findings. Although not clear cut, evidence does exist to suggest altered position sense in LBP patients, and review of the literature suggests that these deficits may be more pronounced in sagittal plane movements, especially neutral or flexed postures, and that position sense deficits may be more prominent in certain subgroups of LBP patients (e.g. those with lumbar segmental instability). Also, proprioceptive deficits may be seen in variables such as movement time rather than purely in the measurement of RE.

## **SEATED POSTURE AND LOW BACK PAIN**

Sitting has been a complex topic for LBP researchers. Nachemson and Elfstrom (1970) found that body position affects the magnitude of the loads on the lumbar spine, and that these loads increase markedly when sitting is compared with upright standing and well-supported reclining. Other studies have identified a creep response of the lumbar spine to prolonged flexed postures; finding a flexion moment of 20-minutes duration to increase peak flexion by 5.5°, it taking approximately 20 minutes for spinal structures to return to baseline stiffness (McGill & Brown, 1992). It has also been found that reflex muscular responses are diminished following sustained flexion postures ( $P < 0.029$ ) (Rogers & Granata, 2006) and following application of a stretch to spinal ligaments (Solomonow et al., 2001). As mentioned earlier the ligaments, muscles, discs and facet joints all contribute to proprioceptive spinal control so any alteration to these structures could feasibly affect proprioceptive ability and reflex activity.

The neutral zone is a phenomenon described as 'a few degrees of spinal movement that is controlled by proprioceptive neuromuscular reflexes' (Panjabi, 1992). The neutral zone is clinically important in spinal stability and change to the neutral zone may expose the spine to potential injury (Panjabi, 1992). Maintaining a slouched posture may compromise the integrity of the joints and decrease the proprioceptive response, resulting in an increase of the neutral zone (Campbell, 2001; Panjabi, 1992).

For many years, prolonged seated posture has generally been accepted as a substantial risk factor for the development of LBP (Kelsey & White, 1980; Magora, 1972). More recently, a review article on the association between sitting and LBP has challenged that belief (Chen et al., 2009). Chen et al. (2009) reviewed studies published between 1998 and 2008 identifying 15 high quality studies to be included in the review, of which only one high-quality cohort study reported a positive association between LBP and sitting (OR 6.2, 95% CI 2.2 to 17.3) (Sjolie, 2004). All other studies reported no significant associations, and hence, the authors concluded there was limited evidence to demonstrate that prolonged sitting is a risk factor for developing LBP. An earlier study however, conducted in 1994 and therefore not included in Chen et al. (2009)'s review, found sitting in a poor posture to be significantly correlated with LBP (Pearson's  $r = 0.57$ ,  $p < 0.001$ ) (Lee et al., 1994). Another review article was conducted by Lis et al. (2007) that examined occupational sitting and LBP but with co-exposure factors such as whole body vibration and/or the adoption of awkward postures. This review identified four studies (Bovenzi & Betta, 1994a; Bovenzi & Zadini, 1994b; Bridger, Groom, Jones, Pethybride, & Pullinger, 2002; Massaccesi et al., 2003) finding that occupations requiring prolonged sitting (of half a day or more) when combined with whole body vibration or the adoption of awkward postures, did present a significantly increased risk of LBP (OR=4).

Some studies have reported odds ratios as low as 0.7 for occupations in which the major physical requirement is sitting, indicating that these people

actually have a lower risk of LBP than in other occupations (Levangie, 1999; Vingard et al., 2000). Although the rate of LBP among occupations requiring extended periods of sitting may not be as high when compared with more strenuous occupations, Lee et al. (2001) reported that this group had the highest hospitalisation rate for LBP. This may indicate that when low back injuries occur in these occupations, they tend to be more severe. Following from this, one might speculate that long term seated postures may not directly injure the spine but rather may increase injury risk via some other mechanism, perhaps by altering neuromuscular control and proprioceptive sensibility.

Although the balance of the literature does not support the association between LBP and sustained seated postures, some studies have shown an association (OR = 6.2, Pearson's  $r = 0.57$ ) and it appears that prolonged seated postures when combined with whole body vibration or the adoption of awkward postures, increases the incidence of LBP (OR = 4).

## **POSTURE AND REPOSITION SENSE**

The body of research reviewed above has shown, although not conclusively, that there is an association between altered position sense in the lumbar spine and LBP patients, and also that there may be some association between flexed seated postures and LBP. Given the known effect of sustained flexed postures on spinal structures, including structures responsible

for spinal proprioceptive control, the question naturally begs as to what effect flexed postures might have on spinal proprioceptive control and position sense.

To the best of the authors knowledge, following an in-depth literature search, only one study exists which examines the effect of sustained flexed posture on lumbar spine position sense. Using a repeated measures design, Dolan and Green (2006) examined the immediate effects on lumbar spine reposition sense following a 5-minute 'slouched posture' in 32 participants. Slouched posture was defined as "a relaxed sitting posture with a flexed lumbar spine". Using a flexible electrogoniometer, the authors examined participants seated ability to reposition to a neutral posture in the sagittal plane.

Prior to each test, ten practice repetitions of repositioning to 'upright posture' were performed with the researcher providing manual facilitation and verbal feedback. For each reposition test the subject was asked either to 'slouch and return immediately to an upright posture' (3s, test 1) or requested to 'slouch and return to your upright posture when instructed (300s, test 2). All participants completed both the 'slouch' test and the 'non-slouch' test in a randomly selected order with a rest period of 15 minutes between tests to allow for tissue recovery (McGill & Brown, 1992). The reposition sense was compared between the two tests. Results indicated that a slouched posture of 5-minutes duration increased mean RE by  $3.92^{\circ}$  (95% CI  $2.35^{\circ}$  to  $5.48^{\circ}$ ), as compared to 3-seconds in a slouched posture.

Although there is no research describing the effects of training-performance delay on reposition tasks or short term motor memory, it seems intuitive that the ability to accurately reposition to a specific learnt position may diminish over time if there is no practice or reinforcement of the position. The inherent flaw of Dolan and Greens methodology, is that in one test participants tried to assume the target upright position 3-seconds after they had practiced achieving that position, while in the other test participants tried to assume the target upright position 300-seconds after they had practiced achieving the position. If the ability to reposition to a specific learnt position does diminish over time, which seems a reasonable possibility, then the difference in RE found by Dolan and Green (2006) may overestimate the true effect of 'slouched posture' on position sense, by incorporating possible time-dependant effects on attention and short-term position memory. In order to isolate the neurophysiological effects of the slouched posture on position sense, the gap in time between learning the position and performance of the reposition task needs to be standardized between the two tests.

## **CONCLUSION**

It has been well established that LBP is a common complaint in society today; however, the aetiology appears difficult to attribute to a precise origin, with many lumbar spine structures and conditions capable of causing pain. Risk factors for LBP are also many and varied, broadly including individual, physical and sociodemographic factors.

One way to study the aetiology of LBP is to investigate proprioceptive information and the control of lumbar spine position and movement. Lumbar proprioceptive disturbances have been investigated using position sense, predominantly on subjects with chronic LBP, with numerous studies finding proprioceptive differences between symptomatic subjects and controls. Despite these studies, the role of proprioception in LBP is poorly understood and there has been little investigation into lumbar spine position sense in asymptomatic subjects who have undertaken an intervention. Investigating position sense in asymptomatic subjects may inform questions regarding the aetiology of LBP. The body of literature has pointed towards investigating the effect of sustained flexed postures on lumbar spine reposition sense; however only one study of this nature has been conducted, which failed to control for possible confounding factors relating to the effect of training-performance delay on repositioning error. Consequently, the aim of the current study was to evaluate lumbar spine reposition sense before and after a 5-minute static flexed posture in asymptomatic subjects, controlling for the effect time dependant variables may have on reposition sense.

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**Section 2:**

# **Manuscript**

# **The immediate effect of a 5-minute flexed posture on Lumbar Spine Reposition Sense.**

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## ABSTRACT

Proprioceptive control is considered important for maintenance of spinal stability and prevention of injury. Evidence exists to suggest that spinal proprioceptive structures, which are reflexive and viscoelastic, may be challenged by prolonged flexed postures. Alteration to lumbar spine position sense has been associated with low back pain patients; however, there has been little investigation into the effect different postural interventions may have on lumbar spine position sense in asymptomatic subjects. The aim of the current study was to investigate lumbar spine position sense after a 5-minute flexed posture in asymptomatic subjects. Following a familiarisation procedure, 30 asymptomatic subjects undertook two position sense tests to a neutral lumbar spine posture in the sagittal plane; one following a 5-minute flexed seated posture, one following a 5-minute sidelying posture, with a 15-minute interval in between. Absolute errors were calculated from data recorded by the two orientation sensors, and compared between groups. Results showed a significantly reduced lumbar spine reposition sense following 5-minutes in a flexed posture as compared with following 5-minutes in a sidelying position ( $p=0.042$ ), mean difference  $2.7^\circ$ . Based on this sample there is evidence that a flexed posture of 5-minutes duration would increase reposition error by more than  $0.10^\circ$  and less than  $5.29^\circ$  ( $n=30$ , 95% CI).

**Keywords:** *Low back pain; Proprioception; Position sense; Reposition sense.*

## **INTRODUCTION**

Low back pain (LBP) is a major health and socioeconomic problem in Western countries (Andersson, 1997), having important consequences not only for patients but also for their families, employers and society in general (Waddell, 1998). Many people will experience one or more episodes of LBP in their lifetime. A summary by Andersson (1997) of prevalence data from numerous studies shows the lifetime prevalence of LBP to range between 49 to 70%, while point prevalence ranges between 12 to 30%. LBP is associated with high economic costs of health care utilisation, work absenteeism and disablement, with one study from the Netherlands estimating the total direct and indirect costs to be as much as 1.7% of the gross national product (Van Tulder et al., 1995).

Although LBP is complex and multi-factorial, one way to study the aetiology of LBP is to investigate sensorimotor proprioceptive information and the control of lumbar spine movement and posture. There are several methods to investigate proprioception: by awareness of the joint position of a body segment in passive movement; estimating the relative position or amplitude of a static limb; and by active movement instigated by the participant (Marks, 1998). Movement detection is markedly improved by muscular contraction and it has been suggested that assessment of proprioceptive deficits during the active implementation of normal movements are more functionally and clinically relevant (Brumagne et al., 1999a; Gill & Callaghan, 1998; Swinkels & Dolan, 2000). Proprioceptive discrepancies that occur in the

mid-range of active movement probably characterise irregular afferent information derived from muscle receptors, and it is the muscle spindles within these muscles that are the primary providers of proprioceptive information regarding joint position and movement (Boyd-Clark et al., 2002; Brumagne et al., 1999b; Gandevia et al., 1992b; Kulkarni et al., 2001).

Numerous studies have investigated the presence of proprioceptive deficits in LBP populations, however the relationship between proprioception and LBP remains contentious. Several studies have found decreased position sense in LBP patients, the deficit ranging between 1.5° to 2.7° greater reposition error (RE) than control subjects (Brumagne et al., 2000; Descarreaux et al., 2005; Gill & Callaghan, 1998; Newcomer et al., 2000a; Newcomer et al., 2000b; O'Sullivan et al., 2003). Other studies have found no significant correlations between altered position sense and LBP patients (Asell et al., 2006; Lam et al., 1999; Silfies et al., 2007). O'Sullivan et al. (2003) specifically investigated LBP patients with a clinical diagnosis of lumbar segmental instability, finding them to exhibit 2.48 cm greater RE than control subjects ( $p < 0.02$ ). O'Sullivan et al. (2003) suggest that proprioceptive deficits may be more pronounced in certain groups of LBP patients and the mixed results from other studies could be the result of poor categorisation of LBP patients, and lack of clear sub groupings of diagnostic characteristics.

Panjabi (2006) describes a 'neutral zone'; approximately 1-3° of segmental intervertebral motion where little resistance is offered by the passive spinal column. Panjabi (2006) identifies control of intersegmental motion around the

neutral zone as a major parameter of spinal instability involved in the mechanism of clinical instability. It is hypothesised that maintaining a slouched posture, may compromise the integrity of the joints and decrease the proprioceptive response resulting in an increase of the neutral zone (Panjabi, 1992). These few degrees of mid-range spinal movement, controlled by proprioceptive neuromuscular reflexes, are considered clinically important in spinal stability, with change to the neutral zone exposing the spine to potential injury (Panjabi, 1992).

Adoption of a flexed spine posture caused by computer and desk work, as well as by driving, has become an integral part of many working environments (Callaghan & Dunk, 2002; Porter & Gyi, 2002). For many years prolonged seated posture has generally been accepted as a substantial risk factor for the development of LBP (Kelsey et al., 1980; Magora, 1972). More recently however, a review article on the association between sitting and LBP has challenged that belief. Chen et al. (2009) reviewed studies published between 1998 and 2008 identifying 15 high quality studies, of which only one high-quality cohort study reported a positive association between LBP and sitting (Sjolie, 2004), and all other studies reported no significant associations. An earlier study however, of 3159 Taiwanese nurses, found sitting in a 'poor' posture did increase the risk of experiencing LBP (adjusted odds ratio =2.13, 95% CI 1.46-3.11, P<0.01) (Lee et al., 1994). Another review article examining occupational sitting and LBP (Lis et al., 2007) identified four high quality studies (Bovenzi et al., 1994a; Bovenzi et al., 1994b; Bridger et al., 2002;

Massaccesi et al., 2003) which found prolonged sitting (of half a day or more) when combined with co-exposure factors (such as whole body vibration or the adoption of awkward postures) did present a significantly increased risk of LBP (OR=4).

Sustained flexion has been shown to be associated with slow deformation creep and may change the physico-chemical properties of spinal structures (McGill & Brown, 1992; Panjabi, 2006). It has also been found that reflex muscular responses are diminished following sustained flexion postures ( $P < 0.029$ ) (Rogers et al., 2006), and following application of stretch to spinal ligaments (Solomonow et al., 2001).

Given the known physical effects of sustained flexed postures on spinal structures (including proprioceptive structures), the possible association between seated postures and LBP populations, and the association between decreased position sense and LBP populations; the question naturally begs as to what effect flexed postures might have on spinal proprioceptive control and position sense.

To the best of this authors knowledge, following an in-depth literature search, only one study exists which investigates the effect of sustained flexed posture on lumbar spine position sense. Dolan and Green (2006) using a repeated measures design, examined the immediate effects on lumbar spine reposition sense following a 5-minute 'slouched posture' as compared to a 3-second slouched posture, in 12 male and 20 female subjects. They defined a slouched posture as 'a relaxed sitting posture with a flexed lumbar spine',

and using a flexible electrogoniometer, examined subjects' ability to reposition to a neutral posture in sitting. The researchers found evidence that a slouched posture of 5-minutes duration would increase mean RE by 3.92°, as compared to 3-seconds in a slouched posture (95% CI, 2.35° to 5.48°).

Dolan and Green (2006)'s research provides preliminary evidence that spinal control is altered immediately following a sustained slouched posture. Based on their research design however, it is impossible to conclude if the reduced position sense is due to the biomechanical and physiological effects of the 5-minute slouched posture, or possibly due to the 4-minute 57 second difference in delay between learning the target position and completing the reposition task between groups. Although no research describing the effects of training-performance delay on reposition tasks or short term motor memory exist, it seems intuitive that the ability to accurately reposition to a specified target may diminish over time if there is no practice or reinforcement of that target. This time-dependant decrease in reposition accuracy may be independent of the posture adopted during that time.

Consequently, the aim of the current study is to determine if spinal proprioceptive control and position sense are in fact altered immediately following a flexed posture, using a research design that controls for time-dependant loss of position sense that may occur independently to the intervention posture.

## **METHODS**

### Design

A repeated measures design was used to determine the effect of a sustained flexed posture on reposition sense of the lumbar spine. All subjects completed two repositioning tasks in a randomly allocated order; repositioning after 300 seconds in a flexed posture; and repositioning after 300 seconds sidelying. Subjects were blind to the results and the data was not displayed during data collection. The outcome measure was RE (in degrees) as a measure of proprioception.

### Inclusion and exclusion criteria

A questionnaire was completed by subjects to identify relevant medical history and confirm inclusion and exclusion criteria. Inclusion criteria were: i) Age range of 18-40; this age group helped to ensure that subjects were neurologically, muscularly, and skeletally mature, yet minimised the effects of age-related changes to the biochemistry and structure of the lumbar tissues (Maffey-Ward et al., 1996), ii) Subjects were required to have a score of zero in the McGill short form Pain Questionnaire, have no current LBP, and have experienced no LBP or received any treatment for LBP in the last year. Exclusion criteria were: i) any previous back surgery; and ii) existence of any known pathologies of the spine (Brumagne et al., 1999; O'Sullivan et al., 2003). All subjects were required to give written informed consent.

### Sample

A convenience sample of asymptomatic subjects was recruited based upon an *a priori* power analysis (Erdfelder et al., 1996). Assuming  $\alpha=0.05$ , minimum power of 0.9, and minimum clinically important effect size of 0.6; a sample of 32 subjects was required. Subjects were recruited from the Unitec undergraduate osteopathy programme.

### Measurement

The same investigator prepared and tested each subject. Lumbar spine position was measured using two 3DM-GX1 Gyro Enhanced Orientation Sensors, (MicroStrain, Inc. Williston, VT) interfaced with a notebook computer running custom designed data acquisition and display software (LabView, National Instruments Corp. Austin, TX).

Subjects wore only shorts and undergarments to reduce sensory cues from clothing (O'Sullivan et al., 2003), and were seated on the end of a plinth, arms crossed, facing a blank wall 1m away. Subjects were seated so that the edge of the plinth made light contact with the popliteal fossa and the height of the plinth was adjusted so that joint angles were observed to be the same for all participants. The two orientation sensors were attached to T12 and S1 with a custom designed adhesive tape and Velcro application system that gave secure attachment but minimised cutaneous cues from the device. The orientation sensors remained attached throughout the entire experimental protocol to prevent introduction of errors related to positioning of the sensors.

### Protocol

Prior to each trial, five practice repetitions of repositioning to arbitrary targets in the sagittal plane were performed. This familiarization protocol used target positions distinct from the neutral 'upright' posture. Following the familiarisation protocol all participants completed both the 'flexed posture' trial and the 'sidelying' trial in a randomly selected order with a rest period of 15-minutes between tests to allow for tissue recovery. McGill & Brown (1992) demonstrated in 20 female subjects that it takes approximately 25-minutes for baseline stiffness of spinal structures to return following a 20-minute flexion moment. As such, a rest period three times the duration of the flexion moment (i.e. 15-minutes) was considered sufficient for washout of tissue effects in the current study.

For the flexed posture trial the subject was moved into the target upright posture, aligned by the researcher as the anterior and posterior superior iliac spines being level in the horizontal plane (Maffrey-Ward, Jull, & Wellington, 1996). The subjects were asked to hold the position for approximately 5-seconds while focusing on and remembering the position of their lower back. Subjects were then asked to relax into full forward flexion, and on request (after 300s) to sit up and move into full extension, move into full forward flexion again, then sit up and try to adopt the target upright posture. In the 'sidelying' trial, subjects were shown the target upright posture and asked to remember it, and then lay down on their left side in a relaxed recumbent position for 300s. They were then asked to sit up and position themselves in the same position on the plinth, then extend fully back, flex fully forward, and

then sit up and try to achieve the target upright posture. For both trials subjects were left alone in the room during the 5-minute intervention posture to avoid any interaction with the investigator which may influence position sense memory. During the 15-minute rest period all participants lay in a comfortable left sidelying position to control against individual postural effects on structures of the lumbar spine.

### Data Extraction

Raw data was extracted as a discreet excel data file for each trial. Raw data generated for each sensor (T12 and S1) was combined to calculate RE in degrees. The value of the caudal sensor angle was subtracted from the value of the cephalad sensor angle to determine the target angular position of the lumbar spine, and a constant was added to normalise the starting position to equal zero. This constant was added to the relative sensor angle at conclusion of the reposition task to determine the RE in degrees. Negative values represented adoption of a posture more flexed than the target, while positive values represented a posture more extended than the target.

### Data Analysis

All statistical analysis was performed using SPSS, v 17 (SPSS Inc, Chicago, IL). Normality of raw data was determined with the Shapiro-Wilk statistic, inspection of normal Q-Q plots, and by calculating skewness and kurtosis. Effects of trial order were explored and because no substantial order effect was found, data for all 'flexed posture' trials were pooled and data for all

'sidelying' trials were pooled. Descriptive data including means and standard deviations were calculated. A two-tailed dependent t-test was used to investigate both procedures to determine whether absolute RE varied following the sustained flexed posture, as compared to the sidelying posture. Confidence limits and probabilities for effect size were calculated from p values as described by Hopkins (2002). Effect sizes were interpreted according to the descriptors described by Cohen (1988). It was assumed that each subject would display a unique 'neutral target posture' representing biomechanical and postural characteristics of the participant. Raw data plots were generated to see if any correlations existed between these individual postural characteristics and reposition accuracy. All data are presented as mean  $\pm$  SD.

## **RESULTS**

Of the 36 subjects recruited, 5 were used for development and refinement of the experimental protocol. Of the remaining 31, one subject was identified as an outlier and removed from analysis because their RE following the flexed procedure was greater than three SD from the mean (Hopkins et al., 2009). The resultant sample consisted of 13 males and 17 females with a mean age of  $24.9 \pm 6.35$  yrs. Differences in RE were observed between the two interventions ( $p=0.042$ ,  $d=0.49$ ). Using Cohen's qualitative descriptors, the magnitude of this effect is moderate to large (Cohen, 1988). The results of the individual RE tests are summarised in Table 2, Appendix B. Table 3 in Appendix B summarises the *t*-test results of the two RE tests. Following the 5-minute flexed posture RE was found to be  $2.7^\circ$  less accurate than following 5-minutes in a left sidelying position (95% CI,  $0.10^\circ$  to  $5.29^\circ$ ). Visual inspection of raw data plots revealed no association between individual postural characteristics (i.e. displaying a relatively flexed or extended 'neutral' position) and either relative or absolute RE's.

## **DISCUSSION**

The results of this study indicate that lumbar spine position sense immediately following a 5-minute flexed posture, is different to lumbar spine reposition sense immediately following 5-minutes of sidelying. The subjects in this sample showed moderately reduced lumbar spine repositioning accuracy following 5-minutes in a flexed posture as compared with the sidelying posture. In this sample, there is evidence that a flexed posture of 5-minutes duration increased RE by between  $0.10^{\circ}$  and  $5.29^{\circ}$  ( $n=30$ , CI 95%).

The effect of prolonged flexed posture on reposition sense has scarcely been studied. Only one other similar study exists in which a 5-minute slouched seated posture was compared with a 3-second slouched seated posture, finding a significant reduction in accuracy following the 5-minute intervention (Dolan & Green, 2006). Dolan and Green (2006)'s study appeared to provide evidence that a 300-second slouched posture would increase RE by between  $2.35^{\circ}$  and  $5.48^{\circ}$  ( $n=32$ , CI 95%), however, the two groups in the study waited vastly different time periods between learning the target posture and executing the reposition task. It is not possible to conclude that the decrease in position sense was an effect of the slouched posture and not just a result of the 300 second delay. Put simply, it is possible their results partly represented the effect of just 'forgetting' rather than purely the effect of altered proprioceptive control. The current study controlled for time-dependant factors extraneous to the flexed posture intervention which may impact upon reposition sense. As such, the results of this study provide evidence that the

decrease in position sense following time spent in flexed postures represents a posturally mediated alteration in proprioceptive ability and spinal control mechanisms. Previous research indicates that this reduced position sense accuracy may be due to viscoelastic effects on soft tissues (Adams & Dolan, 1996; McGill & Brown, 1992), and alterations to proprioceptive neuromuscular reflexes (Callaghan & Dunk, 2002; Solomonow et al., 2003). It was not the goal of the current study, however, to elucidate the mechanism but simply to gain evidence either in support of, or against the presence of an effect.

We found results very similar to those of Dolan and Green (2006). It was hypothesised that the inclusion of a control procedure that used the same time period between learning the target position and executing the repositioning task would result in a smaller difference in error between the two tests as compared with the study of Dolan and Green (2006). This effect may have been offset because the intervention posture used in the current study was somewhat more challenging, involving greater total lumbar flexion than occurs during slouched sitting (Dolan et al., 1988) and thus potentially increasing the difference in RE between groups. These two distinct differences in design make direct comparison between the two studies difficult. While no definitive conclusions can yet be made regarding the effect of slouched posture on position sense, we now have evidence that position sense in the lumbar spine is decreased immediately following a 5-minute flexion moment.

The use of a sidelying control intervention could be seen as a possible weakness of this study. It is feasible that the extra step of moving subjects into a sidelying position and repositioning them back to sitting for the repositioning task may have resulted in a slightly altered set up, potentially introducing a degree of measurement error that might overestimate RE in the sidelying group. Any increase in RE this may have caused would serve to underestimate the true difference in RE between interventions, making the reported effect size relatively conservative. Alternately it could be argued that the sidelying intervention may reduce muscle fatigue and therefore may confound results as position sense has been shown to decrease following fatigue of lumbar postural muscles (Taimela et al., 1999). In spite of this possibility, relaxed flexed postures have been shown to exhibit a flexion-relaxation phenomenon (Callaghan & Dunk, 2002) where a relative myoelectric 'quietness' occurs in the lumbar erector spinae muscles, and as such, any fatigue effects caused by either of the two interventions are considered minimal.

Arguably, another methodological shortcoming of this study may be that repositioning errors were only assessed for one position in only one trial. This method was employed because the current research sought to examine the immediate change to position sense and by performing multiple positions and trials, results might have been confounded by washout of tissue effects. It has also been suggested by previous researchers in this area (Koumantakis et al., 2002) that average errors would tend to be larger with a greater number

of repetitions. Multiple trials in the absence of feedback may confuse subjects and accentuate their RE rather than provide a more accurate measure of RE.

The subjects' affective disposition, mood and motivation at the time of the tests may have some influence on the results, for example if subjects were tired, excited or distracted this may compromise their concentration and be reflected in the experimental data. Also because all subjects were from the same tertiary institution, studying the same health science degree, it is possible they may have had pre-existing ideas and beliefs as to how an intervention may influence movement patterns. While this effect is acknowledged, it is not considered substantial, and the researcher ensured subjects remained naïve to the hypothesis in order to minimise expectation bias. While there are positive aspects to the use of a highly homogenous sample population, it must be noted that the similar subjects are unlikely to represent the diversity of the population (Alreck & Settle, 1995) and therefore the extent to which these findings may be generalised is limited.

It is questionable whether the mean increase of 2.7° RE following the flexed posture would be sufficient to compromise spinal stability, or challenge Panjabi's neutral zone which has been identified as being between approximately 5-15° in a normal upright lumbar spine. While an increase in error of 2.7° may be considered relatively small, nonetheless it still presents an increased challenge to spinal stability. Creep response to prolonged flexion is thought to principally affect passive tissues, most likely compromising spinal

stability at end range movement (McGill & Brown, 1992). Gandevia et al. (1999) suggest that precise muscle spindle input of the paraspinal muscles is essential for accurate positioning of the pelvis and lumbosacral spine in a sitting posture. We now have a strong indication that muscle spindle activity is also affected by flexed postures, because muscle spindles are the primary control mechanism of the mid range active movement investigated in this study (Boyd-Clark et al., 2002; Brumagne et al., 1999b; Gandevia et al., 1992; Kulkarni et al., 2001). Risk of injury may be increased during a period of impaired proprioception, and as such the loss of spinal control associated with flexed postures found in this study, combined with the increase in passive tissue laxity associated with flexed postures (McGill & Brown, 1992) may represent an increased risk for the onset of traumatically induced LBP.

As mentioned, the intervention posture used in the current study was a more challenging posture than that of normal slouched sitting and as such findings cannot be generalised to commonly adopted, slouched seated postures. It is probable however, that even in less challenging postures, that compromise of spinal stability may increase with more prolonged loading; or with repetitive flexion postures which may occur during daily activities (Dolan & Green, 2006). Research into creep deformation of the lumbar spine resulting from prolonged full flexion, indicates that tissue deformation in humans continues to occur for at least 20 minutes after adopting a flexed posture (McGill & Brown, 1992). Using a feline model, Jackson et al. (2001) found a 20min stretch to in vivo spines inhibited multifidus muscle activity for 7-hours,

and repetitive flexion periods of 10-minutes have been shown to have a cumulative effect on muscle activity and soft tissues, also taking over 7-hours to recover (Solomonow et al., 2003).

Traumatic injury immediately following prolonged flexion, while feasible, is unlikely to represent a substantial cause of LBP. In peripheral joints however, it has been proposed that proprioceptive deficits may lead to abnormal loading across joint surfaces (Forwell et al., 1996), and thus contribute to degenerative disease (Gross, 1987). Furthermore, it has been reported that even very small errors in position sense may result in spinal tissue overload and injury (Cholewicki & McGill, 1996). The current researchers speculate that cumulative effects of flexed postures may result in chronically altered spinal control mechanisms. Altered position sense may have the effect of altering postural habits and the sensation of normal position over time, and thus may present a source of nociception due to mechanical loading of innervated structures, altered muscle function, or structural change, secondary to the altered posture. The time course and/or cumulative effects of flexed postures on position sense need to be further explored in order to establish any aetiological link between altered proprioception and altered joint mechanics which may be associated with LBP.

While the current study investigated the effect of flexed posture on the ability to adopt a specified target posture, another avenue for research may be to investigate how sustained flexed postures affect an individual's own sense of

'neutral' posture, thereby examining the effect flexed postures may have in changing postural habits.

Numerous studies have associated position sense deficits with LBP populations, but no clear understanding of the cause-effect relationship exists. It is possible that decreased position sense may contribute to LBP, or possibly decreased position sense may be an epiphenomenon of the back pain itself. Only one prospective study has examined the causality of this relationship. In 232 asymptomatic college athletes, no difference in RE was found between those who did or did not sustain a low back injury during the 3-year follow up period (RE deficit between groups=0.1°, p=0.63) (Silfies et al., 2007). Despite these findings, issues relating to sample population and the measures used in the study make any firm conclusions unfounded and it would be premature to conclude that position sense deficit does not contribute to LBP. Very subtle differences in RE appeared not to increase the risk of back pain in the population Silfies et al. (2007) studied, but it is still not clear what role more substantial deficits might play in other populations.

The findings of the current study show that even in the absence of pain, certain postures have the effect of decreasing position sense. While we cannot determine that the decreased proprioception in LBP patients is not a result of the LBP itself, understanding the spectrum of mechanisms which may contribute to altered proprioception gives us valuable background knowledge with which to better interpret findings related to the complex enigma that LBP and altered sensorimotor control presents.

Manual therapies advocate postural awareness and re-education, and for a modern lifestyle where there is a tendency towards flexed postures, understanding the effect that flexed postures may have on the ability to maintain postural control may be important for prevention of LBP and injury. This research may have implications in patient education for patients who spend time in flexed positions as well as doing lifting, such as recreational gardeners or perhaps warehouse workers. It may be prudent to advise such people not to attempt awkward, or heavy lifting activities immediately after prolonged flexion activities.

O'Sullivan et al. (2003) investigated a defined LBP population and found that proprioceptive deficits were more pronounced in LBP patients diagnosed as having lumbar segmental instability. The relationship between segmental instability, reduced proprioception, and flexed posture also requires more research, however, the results of the current study provide evidence that postures causing creep deformation of lumbar structures also cause decreased position sense, even in pain free subjects. The combination of both increased passive tissue laxity and decreased proprioceptive control may play a role in the aetiology of lumbar segmental instability.

Although the current study did not measure the time variability of repositioning tasks, future research into the rapidity of proprioceptive ability may also be valuable. It has been found that LBP subjects take longer to perform reposition tasks than healthy controls (Descarreaux et al., 2005) and it is hypothesised this decreased speediness of lumbar sensorimotor control

mechanisms might be a candidate mechanism in injury susceptibility. While we know the rate at which lumbar structures return to baseline stiffness following creep deformation; it may also be valuable to know the rate at which proprioceptive deficits return to baseline levels in order to appropriately advise occupational groups.

## **CONCLUSION**

Lumbar spine position sense immediately following a 5-minute flexed posture is significantly different to lumbar spine reposition sense immediately following 5-minutes of sidelying. More research is required to establish the duration of altered position sense following flexed postures, and to explore if this effect represents a candidate mechanism for LBP and injury.

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## **Section 3:**

# **Appendices**

**Appendix A:** Table 1. Summary table of studies investigating LBP and position sense

Study	Year	Design	Symptomatic/ Asymptomatic	Test Position/s and Instrumentation	Outcome measures	Conclusion/ Results
Gill, K. P. Callaghan, M. J.	1998	Two group experimental design. Elbow position sense measured to compare short term motor memory between groups.	20 CLBP patients vs. 20 asymptomatic controls	Target position of 20° lumbar flexion, measured in standing and four point kneeling (FPK). Measured using a Lumbar motion monitor, consisting of exoskeleton that attaches between pelvic and chest harnesses.	Active RE in degrees	No difference in short term motor memory between groups.  LBP patients exhibited 2.25 degrees greater RE in standing ( $p < 0.05$ ), and 2.43 degrees greater RE in FPK ( $p < 0.05$ ).
Lam, S. S. Jull, G. Treleaven, J.	1999	Single-group post-test only design, using a sample of convenience	20 LBP patients.	Seated, moving to a neutral target position from a flexed position. Measured in the sagittal, coronal and transverse planes using a 3-space Fastrak system.	RE in degrees.	No control group. Authors compared results to earlier study by Maffey-Ward et al. (1996) of 10 asympt. participants using same methods. Found no significant differences in RE, only that LBP patients overshoot target position in 79% of cases c.f. 50% in asymptomatic participants.
Brumagne, S. Cordo, P. Lysens, R. Verscheuren, S. Swinnen, S.	2000	Two group experimental design with repeated measures.	23 LBP patients Vs. 21 asymptomatic controls	Seated. 6 sacral tilt angles measured by a piezoelectric accelerometer attached at S2.	Active RE in degrees.	AE difference between groups = 2.7 degrees, $P < 0.0001$ , i.e. repositioning accuracy was significantly lower in patient group than in healthy individuals.
Newcomer, K. Laskowski, E. R. Yu, B. Larson, D. R. An, K. N.	2000	Two group experimental design.	20 CLBP patients vs. 20 asymptomatic controls	Standing. 50% of total range of motion in flexion, extension, bilateral sidebending and rotation. Measured with a 3space tracker.	Active RE in degrees.	No significant difference in RE between LBP patients and control groups.
Newcomer, K. L. Laskowski, E. R. Yu, B. Johnson, J.	2000	Two group experimental design.	20 CLBP patients vs. 20 asymptomatic controls	Standing, legs and pelvis immobilized. 3 target positions, approx 30%, 60%, and 90% of total range of motion in	Active RE in degrees.	LBP patients displayed 1.75 degrees greater RE in flexion ( $p < 0.036$ ) & 1.5 degrees less RE in extension ( $p < 0.015$ ). No difference noted in

C. An, K. N.				flexion, extension and bilateral sidebending. Measured with a 3space tracker.		left and right sidebending.
Koumantakis, G. A. Winstanley, J. Oldham, J. A.	2002	Two group repeated measures design.	62 LBP patients (tested on 2 occasions) vs. 18 asymptomatic controls (tested on 3 occasions).	Standing, 5 target positions - 15° Flexion, R & L rotation 15°, R & L sidebending 15°. Measured using a triaxial spinal electrogoniometer attached to thorax and pelvis.	2 different error indices of active reposition error – Absolute Error (AE) and Variable Error (VE).	Clinical applicability measures were limited, with right sided rotation AE the best with 83.3% specificity and 54.8% sensitivity. However, although not reported, AE in LBP patients was larger in flexion, R rotation, and L rotation by 93%, 68%, and 60%, respectively.
O'Sullivan, P. B. Burnett, A. Floyd, A. N. Gadsdon, K. Logiudice, J. Miller, D. Quirke, H.	2003	Two group experimental design.	15 Chronic LBP patients with a clinical diagnosis of lumbar spine instability vs. 15 asymptomatic controls	Seated, neutral target position. Measured in the sagittal plane using a 3-Space Fastrak.	Active RE in centimetres translation	LBP patients exhibited 2.48 centimetres greater RE than control subjects (p<0.02)
Descarreaux, M. Blouin, J. S. Teasdale, N.	2005	Two group experimental design with repeated measures.	16 Chronic LBP (CLBP) patients vs. 15 asymptomatic controls	Neutral standing posture, pelvis and legs immobilized. Targets 15, 30, & 60 degrees of flexion and 15 degrees extension. Recorded using unspecified 'rehabilitation device' produced by Loredan Biomedical.	Movement time, movement time variability, peak velocity, and absolute RE in degrees.	Found no difference between groups in Absolute RE, but LBP subjects demonstrated longer movement time and smaller peak velocities and symmetry ratios than normal subjects.
Asell, M. Sjolander, P. Kerschbaum, H. Djupsjobacka, M.	2006	Single-blinded, controlled, multi-group comparative study.	92 CLBP patients divided into subgroups based on LBP characteristics, and 31 age and sex matched healthy controls.	Seated. Target position of 1/3 extension of the lumbar spine, measured using 3space Fastrak.	Absolute RE, and Variable RE in degrees.	No differences in RE between the subjects with CLBP or the subgroups of patients and the control group. A weak correlation was found between RE's, and self-reported disability, self-efficacy, and pain.
Silfies, S. P. Cholewicki, J. Reeves, N. P. Greene, H. S.	2007	Prospective cohort study with 3 year follow-up.	292 athletes, 60 of which (21%) had history of LBP within last 5 years – not current LBP!	Seated, neutral target position (0 degrees rotation). Measured using specifically built apparatus, body secured to backrest by a four point harness.	Using degrees of rotation, measured active and passive RE, as well as Motion Perception Threshold.	No significant differences in trunk RE between athletes with and without a history of Low Back Injury (p=0.25), or between those who did or did not sustain a low back injury during the follow up period (p=0.63).

					Also measured recurrence of LBP/injury in follow up.	
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## Appendix B:

Table 2.

Summary of descriptive data for reposition error tests 1 and 2.

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	<b>Mean RE (deg)</b>	<b>S.D. (deg)</b>	<b>Min.</b>	<b>Max.</b>	<b>Standard error</b>	<b>95% confidence interval (deg)</b>
<b>Test 1 (sidelying)</b>	5.82	4.52	0.10	18.1	0.82	4.14 to 7.51
<b>Test 2 (flexed)</b>	8.52	6.06	0.20	20.8	1.11	6.26 to 10.78

---

Table 3.

Summary of *t*-test results for reposition error tests 1 and 2.

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	<b>Mean diff.</b>	<b>S.D. (deg)</b>	<b><i>t</i></b>	<b>df</b>	<b><i>P</i>-value</b>	<b>95% confidence interval of difference (deg)</b>
<b>RE1-RE2</b>	-2.69°	6.95	-2.13	29	0.042	-5.29 to -0.10

---

**Appendix C:** Experimental setup in the evaluation of lumbar spine position sense using the electrogoniometers.



**Appendix D:** Confirmation letter of ethical approval for this study.



Markos Pantelides  
72 Vermont St  
Ponsonby,  
Auckland

3 June 2008

Dear Markos

Your file number for this application: 2008.831

Your application for ethics approval has been reviewed by the Unitec Research Ethics Committee (UREC) and has been **approved** for the following period:

Start date: 30 May 2008

Finish date: 30 May 2009

Please note that:

1. the above dates must be referred to on the information AND consent forms given to all participants
2. you must inform UREC, in advance, of any ethically-relevant deviation in the project. This may require additional approval.

You may now commence your research according to the protocols approved by UREC. We wish you every success with your project.

Yours sincerely

A handwritten signature in black ink, appearing to read 'Deborah Rolland', written over a horizontal line.

Deborah Rolland  
Deputy Chair, UREC

## **Appendix E:** Guidelines for submission to Manual Therapy.

### **Guide for Authors**

The journal editors, Ann Moore and Gwen Jull, welcome the submission of papers for publication.

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Masterclass 4000 words

Letters to the Editors 500 words

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The **title page** should give the following information:

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- name and address of the department or institution to which the work should be attributed
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### **Text**

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