Time-varying changes in the lumbar spine from exposure to sedentary tasks and their potential effects on injury mechanics and pain generation

by

Nadine Marie Dunk

A thesis
presented to the University of Waterloo
in fulfillment of the
thesis requirement for the degree of
Doctor of Philosophy
in
Kinesiology

Waterloo, Ontario, Canada, 2009

AUTHOR'S DECLARATION

I hereby declare that I am the sole author of this thesis. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners.
I understand that my thesis may be made electronically available to the public.

Nadine Marie Dunk

Abstract

General body discomfort increases over time during prolonged sitting and it is typically accepted that no single posture can be comfortably maintained for long periods. Despite this knowledge, workplace exposure to prolonged sitting is very common. Sedentary occupations that expose workers to prolonged sitting are associated with an increased risk of developing low back pain (LBP), disc degeneration and lumbar disc herniation. Given the prevalence of occupations with a large amount of seated work and the propensity for a dose-response relationship between sitting and LBP, refining our understanding of the biomechanics of the lumbar spine during sitting is important. Sitting imposes a flexed posture that, when held for a prolonged period of time, may cause detrimental effects on the tissues of the spine. While sitting is typically viewed as a sedentary and constrained task, several researchers have identified the importance of investigating movement during prolonged sitting. The studies in this thesis were designed to address the following two global questions: (1) How do the lumbar spine and pelvis move during sitting? (2) Can lumbar spine movement and postures explain LBP and injury associated with prolonged sitting?

The first study (Study 1) examined static X-ray images of the lower lumbo-sacral spine in a range of standing and seated postures to measure the intervertebral joint angles that contribute to spine flexion. The main finding was that the lower lumbo-sacral joints approach their total range of motion in seated postures. This suggests that there could be increased loading of the passive tissues surrounding the lower lumbo-sacral intervertebral joints, contributing to low back pain and/or injury from prolonged sitting. Study 2 compared external spine angles measured using accelerometers from L3 to the sacrum with corresponding angles measured from X-ray images. While the external and internal angles did not match, the accelerometers were sensitive to changes in seated lumbar posture and were consistent with measurements made using similar technology in other studies. This study also provided an in-depth analysis of the current methods for data treatment and how these methods affect the outcomes. A further study (Study 3) employed videofluoroscopy to

investigate the dynamic rotational kinematics of the intervertebral joints of the lumbo-sacral spine in a seated slouching motion in order to determine a sequence of vertebral motion. The pelvis did not initiate the slouching motion and a disordered sequence of vertebral rotation was observed at the initiation of the movement. Individuals performed the slouching movement using a number of different motion strategies that influenced the IVJ angles attained during the slouching motion. From the results of Study 1, it would appear as though the lowest lumbar intervertebral joint (L5/S1) contribute the most to lumbo-sacral flexion in upright sitting, as it is at approximately 60% of its end range in this posture. However, the results from Study 3 suggest that there is no consistent sequence of intervertebral joint rotation when flexing the spine from upright to slouched sitting. When moving from standing to sitting, lumbar spine flexion primarily occurs at the lowest joint (i.e. L5/S1); however, a disordered sequence of vertebral motion the different motion patterns observed may indicate that different joints approach their end range before the completion of the slouching movement.

In order to understand the biomechanical factors associated with sitting induced low back pain, Study 4 examined the postural responses and pain scores of low back pain sufferers compared with asymptomatic individuals during prolonged seated work. The distinguishing factor between these two groups was their respective time-varying seated lumbar spine movement patterns. Low back pain sufferers moved more than asymptomatic individuals did during 90 minutes of seated work and they reported increased low back pain over time. Frequent shifts in lumbar spine posture could be a mechanism for redistributing the load to different tissues of the spine, particularly if some tissues are more vulnerable than others. However, increased movement did not completely eliminate pain in individuals with pre-existing LBP. The LBP sufferers' seated spine movements increased in frequency and amplitude as time passed. It is likely that these movements became more difficult to properly control because LBP patients may lack proper lumbar spine postural control. The results of this study highlight the fact that short duration investigations of seated postures do not accurately represent the biological responses to prolonged exposure. Individuals with sitting-

induced low back pain and those without pain differ in how they move during seated work and this will have different impacts on the tissues of the lumbar spine.

A tissue-based rational for the detrimental effects on the spinal joint of prolonged sitting was examined in Study 5 using an in vitro spine model and simulated spine motion patterns documented in vivo from Study 4. The static protocol simulated 2 hours of sitting in one posture. The shift protocol simulated infrequent but large changes in posture, similar to the seated movements observed in a group of LBP sufferers. The fidget protocol replicated small, frequent movements about one posture, demonstrated by a group of asymptomatic individuals. Regardless of the amount of spine movement around one posture, all specimens lost a substantial amount of disc height. Furthermore, the passive range of motion of a joint changed substantially after 2 hours of simulated sitting. Specifically, there were step-like regions of reduced stiffness throughout the passive range of motion particularly around the adopted "seated flexion" angle. However, small movements around a posture (i.e. fidgeting) may mitigate the changes in the passive stiffness in around the seated flexion angle. The load transferred through the joint during the 2-hour test was varied either by changing postures (i.e. shifting) or by a potential creep mechanism (i.e. maintaining one static posture). Fidgeting appeared to reduce the variation of load carriage through the joint and may lead to a more uniform increase in stiffness across the entire passive range of motion. These changes in passive joint mechanics could have greater consequences for a low back pain population who may be more susceptible to abnormal muscular control and clinical instability. Nevertheless, the observed disc height loss and changes in joint mechanics may help explain the increased risk of developing disc herniation and degeneration if exposure to sitting is cumulative over many days, months and years.

In summary, this work has highlighted that seated postures place the joints of the lumbar spine towards their end range of motion, which is considered to be risky for pain/injury in a number of tissue sources. In-depth analyses of both internal and external measurements of spine postures identified different seated motion patterns and self-selected seated postures that may increase the risk for developing LBP. The model of seated

LBP/discomfort development used in this thesis provided evidence that large lumbar spine movements do not reduce pain in individuals with pre-existing LBP. Tissue-based evidence demonstrated that 2 hours of sitting substantially affects IVJ mechanics and may help explain the increased risk of developing disc herniation and degeneration if exposure to sitting is cumulative over many days, months and years. The information obtained from this thesis will help develop and refine interventions in the workplace to help reduce low back pain during seated work.

Acknowledgements

I would like to thank my supervisor, Dr. Jack Callaghan, without whom I would not have pursued graduate studies. I will take the independence and strong research skills that I have learned with me onto the next stages in my career. This experience has strengthened me not only academically, but personally as well.

Thank-you to the members of my committee, Dr. Jen Durkin, Dr. Stu McGill, Dr. Jeff Orchard and Dr. Greg Kawchuk, for taking the time to read my thesis and providing me with helpful suggestions to strengthen my work.

I would like to acknowledge the people who provided technical resources and advice:

Thank-you to the Callaghan Spine lab members, past and present, for all the advice, helping hands and programming code you have provided over the years.

The first three studies of my thesis would not have been possible without the generosity of Dr. Tom Jenkyn at the University of Western Ontario. My partner in crime, Angela Kedgely, was instrumental in welcoming me into the lab and making sure the study got up and running.

I would also like to recognize all of the time and effort Dr. Jeff Orchard put towards developing the image registration program. Jeff, you have gone above and beyond what is asked of a committee member. Your curiosity and interest in my work and science in general are truly inspiring.

I would also like to acknowledge how lucky I was to be in a department with such an incredible technical support team. In particular, Wendell Prime, Jeff Rice and Craig McDonald were always so helpful in troubleshooting technical problems.

I have the most incredible support network that has helped guide me through this process:

To my Waterloo support crew, past and present: in particular, Jennie, Diane, Jon, Tash, Janice, Jeannette, Ruth – I owe you my deepest gratitude for providing support, advice, humour and friendship over the years.

To my personal cheering section, the loudest section in the stands, including:

The “extended” Grahams – Thanks to Terry for all the guidance and advice you have given me; to Kirsten and Tim for being the best sister- and brother-in-law I’ve ever had!

Kiera, Uma – you are my voices of reason.

Mom, Dad, Michelle, Renee, Manny (aka the Battalion) – No words can describe how grateful, happy, comforted, encouraged, etc... I am because of all of you. My family is my life and you all make it so worthwhile.

Drew – I am overwhelmed by the person you are to me. We have been through so much together. So many times you have picked me up when I have faltered. Here’s to walking hand-in-hand towards the next big adventure.

Table of Contents

List of Figures

List of Tables

Chapter 1:
Description of Thesis

This thesis begins by providing a general background of the association between and general biomechanics of low back pain and prolonged sitting (Chapter 2). The global thesis questions and specific studies are also introduced in this chapter. Chapter 3 consists of the submitted manuscript (Clinical Biomechanics, in press) describing Study #1, which examined x-ray images of the lower lumbo-sacral spine in standing and seated postures. Study #2 (Chapter 4) describes the feasibility of using accelerometers to measure external lumbar spine and pelvic angles in sitting and includes a detailed description for the signal processing and analyses that are used in later studies (Studies #3 and 4; Chapter 5 and Chapter 6, respectively). Study #3 (Chapter 5) extends the static X-ray investigation of Study #1 and employed videofluoroscopy to investigate the dynamic rotational kinematics of the intervertebral joints of the lumbo-sacral spine in a seated slouching motion. Study #4 (Chapter 6) compared the postural responses and pain scores of a population of low back pain sufferers with an asymptomatic population during prolonged sitting in order to document the biomechanics and movement patterns associated with sitting induced low back pain. Part of this study was submitted as a manuscript to a special issue of *Work* and is in press. In Study #5 (Chapter 7), the spine movement patterns observed in the two *in vivo* populations from Study #4 were investigated using an *in vitro* spine model to quantify mechanical changes in the intervertebral joint associated with the development of low back pain. Chapter 8 addresses the findings of the five studies in relation to the global thesis questions and summarizes the contributions of the thesis.

There are seven appendices included in this work. Of note are Appendix D and Appendix G. Appendix D describes the steps used to verify the performance of the image registration program developed by Dr. Jeff Orchard used in Study #3 (Chapter 5) to track spine motion in the videofluoroscopic images. Appendix G outlines the issues encountered with EMG data collection and analyses during Study #4 (Chapter 6).

Chapter 2:
General Introduction

2.1 Sedentary Occupations and Low Back Pain

General body discomfort increases over time during prolonged sitting. Whether it is decreased blood flow to the lower limbs, pressure discomfort in the buttocks area, or discomfort in the upper or lower back, any prolonged posture can lead to static loading of muscles and passive tissues at and around joints (Magnusson and Pope, 1998). It is generally accepted, and a recognized ergonomic risk factor that no single posture can be comfortably maintained for long periods of time. Despite this knowledge, workplace exposure to prolonged sitting is very common. In industrialized countries, more than half of the workforce is employed in administration, services, credit, trade or insurance industries, which expose workers to sitting for much of the workday (Grieco, 1986). In Quebec, over 40% of the population reported that they sit while at work, with approximately 21% of those individuals indicating that they were not able to get up at will (Tissot et al., 2005). A survey of the Australian government workforce demonstrated that some workers, particularly in administrative and managerial positions, sit for over 75% of their workday (Table 2.1). A strong association was found between increased sitting time and less walking (Miller and Brown, 2004), suggesting that individuals whose daily work involves long hours of sitting are not able to, or chose not to move around.

Historically, it has been suggested that sedentary occupations that expose workers to prolonged sitting are associated with an increased risk of developing low back pain (LBP) (Magora, 1972; Frymoyer et al., 1980; Magnusson and Pope, 1998), disc degeneration (Videman et al., 1990) and lumbar disc herniation (Kelsey, 1975; Wilder et al., 1988). However, two recent meta-analyses of the epidemiological literature have suggested that the evidence does not support the associations between prolonged seated office work and LBP

(Hartvigsen et al., 2000; Lis et al., 2007). It is important to note that the majority of the reviewed studies were cross-sectional (i.e. not longitudinal by following LBP development over time), and therefore unable to establish a true cause-and-effect relationship. Any detrimental health effects of prolonged sitting might develop over long periods of time, thereby making it difficult to pinpoint the amount of critical exposure to sitting. Decisive evidence is difficult to establish by epidemiological methods as both seated postures and LBP are prevalent in the general population. Additionally, it is problematic to examine "non-specific" LBP as sitting can alleviate pain in some individuals while exacerbating pain in others. The fact remains that populations who have LBP that is induced and/or aggravated by prolonged sitting do exist (Womersley and May, 2006; Dankaerts et al., 2006b; O'Sullivan et al., 2006c; May and Donelson, 2008). Furthermore, subjective experimental findings demonstrate that low back discomfort increases with prolonged sitting, even in individuals with no prior history of LBP (Fenety et al., 2000; Fenety and Walker, 2002; Beach et al., 2005; Dunk and Callaghan, 2005; Gregory et al., 2006). Given the prevalence of occupations with a large amount of seated work and the propensity for a dose-response relationship between sitting and LBP, refining our understanding of the biomechanics of the lumbar spine during sitting is important.

Table 2.1: Self-reported hours of seated work, percent of workday spent sitting, total hours of sitting per weekday (including work and after work) and mean number of steps taken per weekday for workers in different occupation groups (modified from (Miller and Brown, 2004))

Occupation	Sitting at work		Total weekday sitting	Mean weekday steps
	Total hours	Percent of workday	Hours per day	(# steps per day)
Administrative (secretaries, administrative assistants)	5.7	75.7	10.3	7207
Professional/managerial (public health staff, med. officers, directors)	6.2	75	10.6	7883
Scientists (biologists, chemists, researchers)	4.9	63.2	9.4	10147
Technicians (lab technicians and assistants)	3.3	43	7.8	10731
Blue-collar workers (cleaners, maintenance staff)	1.6	22	6.5	11784

2.2 Seated Lumbar Spine Posture and Pain Generating Pathways

The link between low back pain and sitting has been attributed to the flexed curvature of the lumbar spine (Wilder and Pope, 1996). Keegan (1953) described the "normal" or "neutral" position of the lumbar spine as a position of balanced muscle relaxation between the posterior and anterior trunk-thigh muscles. This occurs when the hips are at an angle of 135° and the adjacent vertebral end-plates are parallel. Using lateral X-rays from four healthy adults (2 males, 2 females), he observed that upright standing increased the lumbar curve from the "neutral" position, and sitting considerably flattened the lumbar curve by decreasing the trunk-thigh angle. Andersson et al. (1979) observed from lateral radiographs that lumbar lordosis (the angle between the top of the first lumbar vertebra and the top of the sacrum) decreased by an average angle of 38° when moving from a standing to an unsupported sitting position. This decrease occurs mainly by rotation of the pelvis, which rotated 28° on average. The remaining 10° are mainly changes in the vertebral body angles of L4/5 and L5/S1, with small angular changes seen at the other lumbar levels. In general, the lumbar spine ranges between 40 and 80% of maximum standing flexion in various seated postures (Pearcy, 1993; Callaghan and McGill, 2001b; Callaghan and Dunk, 2002; Dunk et al., 2004; Gregory et al., 2006).

Sitting imposes a flexed posture that, when held for a prolonged period of time, may cause detrimental effects on the tissues of the spine. In order to understand how flexion could be detrimental to the spine, the biomechanical responses of the different spinal tissues of the spine to flexion will be described. A review of the innervations of the spinal tissues and potential pain generation pathways in response to the biomechanics will also be provided.

2.3 Pain generating pathways and the innervations of the spine

2.3.1 Innervations of the spinal tissues

The tissues of the lumbar spine are richly innervated by the central nervous system (Cavanaugh et al., 1996; Higuchi and Sato, 2002). Pain generators are usually free nerve endings of sensory fibres and are the smallest somatosensory nerve fibres found in the body (Cavanaugh, 1995; Cavanaugh et al., 1996; 1997). Pain fibres (or nociceptors) normally have high mechanical thresholds and fire when the stress applied to them is noxious (Cavanaugh et al., 1997).

Injury or damage to the structures of the lumbar spine is most likely caused by mechanical overload, either due to acute or repetitive loading scenarios. A cascade of biomechanical events occurs in response to the initial injury: mechanical overload leads to tissue damage, which in turn changes the biomechanics of the joint thus disturbing the normal mechanics/loading of other tissues in the area (McGill, 2002). In order for pain to be generated and sensed, nociceptors in the region of damage (usually pain-sensing free nerve endings) require activation that sends a message via the spinal cord and thalamus to the somatosensory cortex of the brain (Cavanaugh, 1995). Chemical mediators that are released during tissue damage and inflammation may lead to sensitization of pain fibres and can initiate the pain pathway (Cavanaugh, 1995). Lumbar spine tissue injury and inflammation can cause prolonged nociceptor excitation, or peripheral sensitization that can lead to central sensitization (in the spinal nerves and cord), which contributes to the persistence of pain (Cavanaugh et al., 1996). Furthermore, because of the chemical facilitation of pain, gross disruptions of the tissue are not necessary to produce pain; the inflammatory response due to microdamage may heighten the sensitivity of the neurons.

2.3.2 Intervertebral disc

The intervertebral disc (IVD) has a meager nerve supply that is generally limited to the outer layers of the annulus (Fagan et al., 2003). There is also a concentration of nerve fibres located in the central region of the endplate adjoining the nucleus (Fagan et al., 2003). The nerves of the outer annulus and central endplate may function as nociceptors (Cavanaugh et al., 1997; Fagan et al., 2003). Prolonged static spine flexion has been shown to cause stress peaks in the outer layers of the annulus that may activate annular nociceptors and elicit pain (Adams et al., 1996). Furthermore, moderate seated flexion causes the nucleus pulposus to migrate posteriorly, increasing the pressure in the disc and the risk for developing a disc herniation (Nachemson, 1966; Sato et al., 1999; Alexander et al., 2007). Clinical evidence also shows that prolonged sitting is problematic for individuals whose LBP is of a discogenic nature and that preserving lumbar lordosis is a successful way to alleviate this (Womersley and May, 2006; Dankaerts et al., 2006b; O'Sullivan et al., 2006c; McGill, 2007).

2.3.3 Facet Joints

The lumbar facet joint is a synovial joint that has the potential for overstress of the capsular ligament that may lead to degenerative changes in the joint (Cavanaugh, 1995). Excessive facet loads can stretch the joint capsule, which is much weaker in tension than the resistance offered by bony contact in compression (Yang and King, 1984). The facet is richly innervated and contains free nerve endings that respond to capsular stretch (Cavanaugh, 1995; Cavanaugh et al., 1996). Transient facet capsular strains of approximately 30% have elicited pain behaviour and neurophysiologic changes associated with chronic pain (Lee et al., 2004). Interestingly, these capsular strains are much less than the failure threshold, emphasizing that structural micro-damage is likely associated with pain development.

2.3.4 Nerve roots (dorsal root and dorsal root ganglion)

Posterior-lateral disc herniations can impinge on the lumbar dorsal root ganglion (DRG) and research has shown that moderate pressures applied to the DRG activates sensory fibres that

can continue long after the stimulus is removed (Cavanaugh, 1995; Cavanaugh et al., 1997). Cyclic loading, rapid loading and sustained loading all result in neural excitation and prolonged after-discharges of the sensory nerve fibres (Cavanaugh et al., 1997). The mechanical threshold for persistent pain to exist may fall within a nerve root strain value range of 17.4 to 22.2% (Winkelstein and DeLeo, 2004). Possible causes for compression of the nerve roots include IVD height loss and disc herniation (Nuckley et al., 2002), both of which are associated with biomechanical responses characteristic of prolonged sitting.

2.3.5 Fascia and ligaments

Several researchers have documented the sensory innervation of the lumbar spine ligaments (Figure 2.1) and lumbodorsal fascia (Figure 2.2) (Yahia et al., 1992; Rhalmi et al., 1993; Higuchi and Sato, 2002). These studies have also confirmed the presence of neurofilament immunoreactive nerve fibres, suggesting that irritation of the tissues could produce pain. High tensile forces are generated in the posterior ligaments of the spine for flexion angles greater than 75% RoM (Adams et al., 1994), which is within the flexion range of the seated lumbar spine (Pearcy, 1993; Callaghan and McGill, 2001b; Callaghan and Dunk, 2002; Gregory et al., 2006). Static loads applied to the lumbar ligaments and the development of cumulative creep can potentially cause neuromuscular disorders exhibiting symptoms such as decreased reflexive muscular activity and muscle spasms elicited by collagen fibre micro-damage and relayed reflexively by pain receptors (Solomonow, 2004).

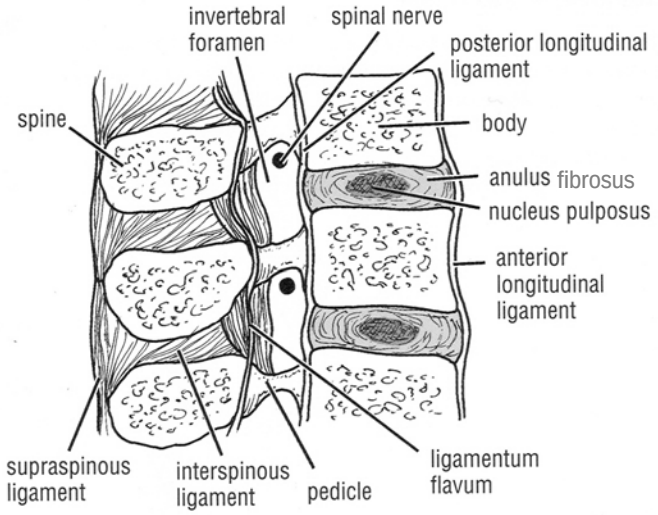


Figure 2.1: Sagittal section through three lumbar vertebrae, showing the ligaments and intervertebral discs. (from (Snell, 1995))

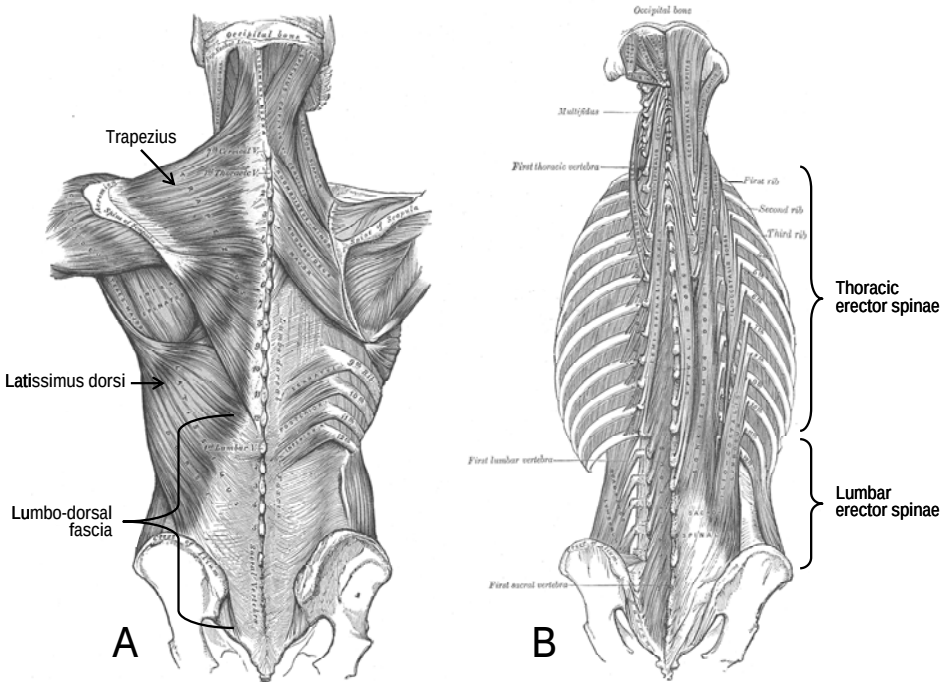


Figure 2.2: Superficial (A) and deep (B) muscles of the back. From (Gray, 2000) *Anatomy of the Human Body.* Philadelphia: Lea & Febiger, 1918; Bartleby.com, 2000. www.bartleby.com/107/ (accessed on October 20, 2008)

2.3.6 Muscles

Muscle contains a large number of nerve fibres that may have pain sensing functions (Cavanaugh, 1995). Physiological causes of fatigue and discomfort result from the accumulation of metabolites and the depletion of substrates (Sjogaard et al., 1988; Sjogaard, 1990). During muscle contractions, metabolic by-products including lactic acid, bradykinin, serotonin, potassium ions and prostaglandins are released from the muscle cells and can chemically induce pain (Mense, 1977; Kumazawa and Mizumura, 1977). Sustained contractions of the erector spinae muscles (Figure 2.2) will increase intramuscular pressure that in turn reduces blood flow causing ischemia and accelerating the onset of pain (Keyserling et al., 2005). However, the lack of blood flow may only become a muscle fatiguing factor at around 50 to 60% of maximum voluntary contraction (MVC) of the trunk extensor muscles (Yoshitake et al., 2001; Kell et al., 2004; Kramer et al., 2005). At lower levels of erector spinae muscle contraction, muscle oxygenation may be impaired at the beginning of a contraction (McGill et al., 2000) but may return to resting levels or even increase for longer duration contractions of around 5%MVC (Blangsted et al., 2005; Van Dieen et al., 2008). Back muscle activity as low as 2%MVC can cause fatigue-related changes in the EMG frequency content (e.g., a decrease in mean power frequency), with little to no associated decrease in muscle oxygenation (Van Dieen et al., 2008). Maintaining various seated postures requires low level activation of the ES muscles that rarely exceeds 10% MVC (Callaghan and McGill, 2001b; Callaghan and Dunk, 2002). Thus, muscle fatigue development during sustained low-level activity of the back muscles observed during prolonged sitting could result in discomfort or pain. Furthermore, mechanical loading of the muscles and resulting tension contributes to the sensations of pain and discomfort by stimulating mechanoreceptors located in the muscles and tendons (Keyserling et al., 2005). Thus, the muscles of the back could be a potential source for low back pain during sitting.

2.4 Sitting Biomechanics: What is Missing?

2.4.1 Intervertebral joint angles

Previous researchers have reported that the lumbar spine ranges between 40 and 80% of maximum standing flexion in various seated postures (Pearcy, 1993; Callaghan and McGill, 2001b; Callaghan and Dunk, 2002; Gregory et al., 2006). Considering this moderate amount of total lumbar flexion, it is unknown how each intervertebral joint (IVJ) of the lumbar spine contributes to total flexion. Although extensive research has produced kinematic data reporting the range of motion of the intervertebral joints in the lumbar spine, many of these studies have examined movements from upright standing postures (Pearcy and Whittle, 1982; Pearcy et al., 1984; Kanayama et al., 1996; Frobin et al., 1996; Okawa et al., 1998; Harada et al., 2000). While there is a small amount of data regarding seated IVJ rotations (Konig and Vitzthum, 2001; McGregor et al., 2001; Takayanagi et al., 2001; Makhsous et al., 2003; Snijders et al., 2004), much of this work has investigated static seated postures and these data have not been directly compared to IVJ rotations during standing flexion. One of the main differences between standing and sitting is the orientation of the pelvis and it has been suggested that the pelvis drives lumbar spine posture in sitting (Andersson et al., 1979; Gatton and Pearcy, 1999; Kasahara et al., 2008). There are likely differences in the IVJ rotations between standing and sitting flexion, and joint positioning will influence the distribution of forces to individual muscles and passive tissues. Two studies were designed as a part of this thesis to address the gaps identified above (Chapter 3 and Chapter 5) by determining how close the lumbar IVJs are to their end range of motion in sitting and whether seated flexion motions are driven by the pelvis.

2.4.2 Methods of documenting lumbar spine posture

A number of different techniques have been established for the external measurement of lumbar spine postures in sitting, including:

- Electromagnetic measurement device: Fastrak (O'Sullivan et al., 2002; Dankaerts et al., 2006b) or 3-space (Callaghan and McGill, 2001b; Gregory et al., 2006)
- Flexible electrogoniometer (Vergara and Page, 2000; Dolan and Green, 2006; Carcone and Keir, 2007; Kasahara et al., 2008)
- Accelerometers (Wong and Wong, 2008; Beach et al., 2008)
- Inclinometers (Adams et al., 1986; Dolan et al., 1988)
- Fins, with traditional motion tracking systems (Dunk and Callaghan, 2005)
- Skin markers (Vergara et al., 2006)

The use of a backrest in seated research restricts the function of many of the external measurement systems due to the lack of line of sight or movement of the device with respect to the skin. All external measurement instrumentation must be affixed to the skin overlying bony landmarks and is subject to errors due to skin movement, loss of contact with the skin and inaccurate anatomical landmarking. Therefore, a desirable external measurement device would be small enough to provide unimpeded use of a backrest and require simple and repeatable anatomical placement. One research group used small electronic inclinometers placed over the L1 spinous process and at the top of the sacrum to measure the lumbar spine angle in sitting (Adams et al., 1986). When compared to lumbar angles measured from X-ray images, they found that the inclinometers were susceptible to errors caused by "skin wrinkling". Differences between X-ray and inclinometer measurements of lumbar angle ranged from an under-prediction of 10° to an over-prediction of 12° (mean absolute error = 4.3°) (Adams et al., 1986). Furthermore, these instruments are limited by a significant amount of experimental error such as small ranges of measurement due to degradation of the signal as the inclination surpasses 60° in either direction (Otun and Anderson, 1988). Tri-axial accelerometers have shown promise for use in lumbar spine measurements in sitting (Wong and Wong, 2008). The output of the sensitive axis of the sensor is proportional to its angle with respect to the line of gravity without the limitations of inclinometers or uni-axial accelerometers (Hansson et al., 2001). As a part of this thesis, a study was conducted to

compare the use of tri-axial accelerometers for measuring lumbar spine angles with concurrent measurements made from X-rays (Chapter 4).

2.4.3 The time-varying responses during sitting

Advances in computer technology have allowed biomechanics researchers to more easily investigate the multidimensional complexities of human movement for prolonged periods of time. One of the challenges with research examining prolonged exposures is the biological variability inherent in the human response. Several researchers have identified the importance of investigating movement during sitting over long periods of time (Callaghan and McGill, 2001b; Vergara and Page, 2002). Frequent postural shifts while performing seated work may be an indication that a person is trying to alleviate musculoskeletal discomfort (Bhatnager et al., 1985; Liao and Drury, 2000). It is uncertain if these postural shifts also alleviate pain or injury generating scenarios. Jensen and Bendix (1992) suggested that there were three factors contributing to low back pain (LBP) in sitting: 1) insufficient nutrition to the intervertebral disc; 2) the stress-relaxation of spinal ligaments and; 3) muscular fatigue. Based on evidence from previous research, the benefits of lumbar spine movement include:

- Promotion of fluid flow into and out of disc to improve disc nutrition (Adams and Hutton, 1983; Adams and Hutton, 1986; McMillan et al., 1996a)
- Fluctuations in disc pressure (Wilke et al., 1999; Wilke et al., 2001) and the possible relief of potentially harmful stress concentrations in the disc (McNally and Adams, 1992; Adams et al., 1994; Adams et al., 1996)
- Spine creep recovery (McGill and Brown, 1992; Solomonow, 2004)
- Varied muscle activity (Mclean et al., 2000; Mclean et al., 2001; Callaghan and McGill, 2001b)
- Improved blood pumping to muscles (Sjogaard et al., 1988)

Until recently, most studies have examined a static snapshot of different sitting conditions to observe muscle activity, posture, and loading. A shift towards the examination of the time-varying responses of the lumbar spine is necessary in order to determine the mechanisms for pain generation and how certain variables can be manipulated to avoid or prolong the initiation of these mechanisms. Therefore, it is necessary to establish a more complete knowledge of how movement of the lumbar spine affects pain generating mechanisms in sitting. The final two studies of this work have addressed this problem (Chapter 6 and Chapter 7). First, sitting specific movement patterns of the lumbar spine were established for *in vivo* LBP and asymptomatic populations and a relationship between discomfort and movement variables was investigated. Finally, the time-varying effects of the documented movement patterns on the mechanical properties of the intervertebral joint, in particular the disc, were examined using an *in vitro* spine model. These studies provided insight into the potential pain generating mechanisms that can exist from exposure to prolonged sitting.

2.5 Global Thesis Questions and Specific Study Purposes

Global Question #1: How do the lumbar spine and pelvis move during sitting?

Specific Questions:

a) Are the lower intervertebral joints (IVJ) close to their end range of motion in upright sitting?

b) Is lumbar spine flexion in sitting driven by the pelvis and does it demonstrate a "bottom-up" sequence of motion?

c) How well do external measurements of lumbar spine angles compare with internal measurements?

Three studies were included in this thesis to answer the above questions:

Study #1: This study used X-ray images of various standing and seated postures in order to quantify lumbo-sacral IVJ angles. Specifically, the goals were to determine if the lower

lumbar IVJs approach their end ranges of motion in seated postures and to investigate any gender differences.

Study #2: This study compared externally measured spine angles measured from L3 to the sacrum (L3/Sac) with corresponding angles measured from X-ray images. The goal was to determine the feasibility of using accelerometers to measure lumbo-sacral spine angles in standing and seated postures.

Study #3: Fluoroscopic video sequences were obtained during a seated slouching motion in order to examine the sequence of rotation of the pelvis and lower three lumbar vertebral bodies. The goal was to determine if seated spine motions were initiated by the pelvis, and if there were consistent motion patterns across all individuals.

Global Question #2: How can lumbar spine movements and postures explain LBP and injury associated with prolonged sitting?

Specific Questions:

a) What biomechanical variables are associated with increased LBP during prolonged sitting?

b) Is there a difference in the lumbar spine and whole body movement patterns of individuals with LBP compared with asymptomatic individuals during prolonged sitting?

c) Does one sitting movement pattern make the spinal joint more susceptible to injury?

Two studies were designed to examine these questions:

Study #4: The time-varying responses of individuals with LBP were compared with asymptomatic individuals during prolonged seated work. The goal was to determine the postural responses that are associated with increased LBP development. Various methods of

quantifying time-varying data were developed and utilized to distinguish between those who develop LBP and those who do not.

Study #5: The main goal was to examine the specific spine movement patterns previously documented in the LBP and asymptomatic populations using an *in vitro* model so that associated mechanical changes in the intervertebral joint could be quantified. This study was designed to provide a tissue-based rational for altered joint mechanics from prolonged sitting exposures and relate the associated changes to pain generating pathways.

Chapter 3:
Evidence of a pelvis driven flexion pattern: Are the joints of the lower lumbar spine fully flexed in seated postures?

Nadine M. Dunk, Angela E. Kedgely, Thomas R. Jenkyn, Jack P. Callaghan

Published in Clinical Biomechanics, 24(2), 164-168, 2009

3.1 Synopsis

Background: Seated postures are achieved with a moderate amount of lumbo-sacral flexion and sustained lumbo-sacral spine flexion has been associated with detrimental effects to the tissues surrounding a spinal joint. The purpose of this study was to determine if the lower intervertebral joints of the lumbo-sacral spine approach their end ranges of motion in seated postures.

Methods: Static sagittal digital X-ray images of the lumbo-sacral region from L3 to the top of the sacrum were obtained in five standing and seated postures from 27 participants. Vertebral body bony landmarks were manually digitized and intervertebral joint angles were calculated for the three lower lumbo-sacral joints.

Findings: In upright sitting, the L5/S1 intervertebral joint was flexed to more than 60% of its total RoM. Each of the lower three intervertebral joints approached their total flexion angles in the slouched sitting posture. These observations were the same regardless of gender. The results support the idea that lumbo-sacral flexion is driven by rotation of the pelvis and lower intervertebral joints in seated postures.

Interpretation: This is the first study to quantitatively show that the lower lumbo-sacral joints approach their total range of motion in seated postures. While not measured, the findings suggest that there could be increased loading of the passive tissues surrounding the lower lumbo-sacral intervertebral joints, contributing to low back pain and/or injury from prolonged sitting.

3.2 Introduction

The whole body postural demands of seated tasks require an upright torso while achieving moderate lumbo-sacral (LS) spine flexion (40% to 80% of maximum flexion (Pearcy, 1993; Callaghan and McGill, 2001b; Callaghan and Dunk, 2002; Gregory et al., 2006)). Sustained lumbar spine flexion can have severe detrimental effects on various structures of the spine and is implicated as a low back pain (LBP) generating mechanism. Prolonged sitting is associated with increased risk of lumbar disc degeneration (Videman et al., 1990) and herniation (Wilder et al., 1988). Given that seated postures are achieved with moderate LS spine flexion, one or more of the individual intervertebral joints (IVJ) could be approaching their flexion end range, making the joint more susceptible to pain/injury. Thus, we were driven to investigate the contribution of each individual IVJ to total LS spine flexion in seated postures.

In his study of lumbar spine X-rays in different standing and sitting positions, Keegan (1953) demonstrated that the LS spine was always more flexed in sitting than in standing, although no IVJ rotations were reported. Other researchers have demonstrated that the lumbar spine as well as each individual IVJ are more flexed in sitting than in standing (Andersson et al., 1979; Makhsous et al., 2003). Specifically, seated L1/2 rotation was identical to that during upright standing and the inferior IVJs (L3/4 to L5/S1) were progressively more flexed (Andersson et al., 1979; Makhsous et al., 2003). Given this evidence, the major factor in determining seated LS posture must be linked to the rotation of the pelvis. However, no study has compared seated IVJ angles to those in standing full flexion in order to determine

the range of motion (RoM) of each joint in sitting. Sustained end range of flexion can adversely affect the tissues surrounding a spinal joint (i.e. ligaments, intervertebral disc, etc.) (Wilder et al., 1988; McGill and Brown, 1992; Adams et al., 1994), which may lead to tissue injury and LBP in seated postures.

Gender-based differences in the LS spine and pelvic postures in sitting have been observed using external LS angle measurements (Dunk and Callaghan, 2005). This study found that females sat with more anterior pelvic rotation and less lumbar flexion than males. Females also exhibited greater lumbar spine flexibility than males (Van Herp et al., 2000), and tended to undergo greater changes in LS postures when moving from a standing to a sitting position (Dunk and Callaghan, 2005). Thus, the current study was also aimed at investigating whether individual LS IVJ angles could account for these gender differences observed in sitting.

3.2.1 Purpose

The overall purpose of this study was to determine if the lower IVJs of the LS spine approach their end ranges of motion in seated postures. Specific goals included determining the total range of motion (RoM) for each of the three lower IVJs and examining any gender differences in IVJ angles from X-ray images of the LS spine in various standing and seated postures.

3.2.2 Hypotheses

1) In seated postures, the L5/S1 joint will be closest to its end RoM followed by the L4/5 and L3/4 joints
2) Males will have greater flexion in all three IVJs in seated postures.

3.3 Materials and Methods

3.3.1 Participants

Twenty-seven participants, 13 males (mean age = 29.2 (SD 8.8) years; mean height = 1.78 (SD 0.05) m; mean mass = 78.5 (SD 8.0) kg) and 14 females (mean age = 26.7 (SD 6.5) years; mean height = 1.65 (SD 0.07) m; mean mass = 63.2 (SD 12.8) kg), were recruited. A justification for the chosen sample size can be found in 0. All participants were free of self-reported LBP for 12 months prior to testing and were screened to exclude any or all of the following: 1) potential or definitive pregnancy; 2) occupational exposure to radiation, and; 3) annual occurrence of two or more trunk and/or pelvis X-rays or other high-exposure diagnostic radiological procedures (e.g. CT, bone mineral density testing). All procedures were approved by the Universities of Waterloo and Western Ontario Offices of Research Ethics. Participants gave informed consent before testing began.

3.3.2 Instrumentation

The digital radiography setting of a video fluoroscope (9-inch image intensifier (II), 10 cm source-to-II distance; Siremobil Compact (L) Mobile X-ray Image Intensifier system, Siemens Medical Solutions USA Inc., Malvern, PA, USA) was used, with average technique factors of 2.4mA and 70 kVp. The effective dose was 0.21 mSv per image, which was less than that of a typical diagnostic lumbar radiograph (Wall and Hart, 1997). The video feed from the fluoroscope was captured directly to a computer hard drive using a digital video capture device and software (DVD Xpress DX2, ADS Technologies, Cerritos, CA, USA).

3.3.3 Data Collection and Analysis

One static X-ray image was obtained for standing, half flexion, full flexion, upright sitting and slouched sitting (Figure 3.1). Participants were instructed to maintain a lordotic spine in the upright standing and sitting postures. The slouched sitting posture was obtained by rounding the spine from upright sitting. Half flexion represented a standing posture with the

same trunk-thigh angle of 98.6° (SD 3.4°) in upright sitting as measured by a manual goniometer aligned with the long axis of the thigh and the line connecting the greater trochanter to the shoulder (at the greater tuberosity of the humerus). Participants were positioned with their right side touching the image intensifier and were instructed to place their hands on their shoulders for the upright standing and two seated postures to prevent the arms from obscuring the spine in the X-ray image. Once the posture was assumed, an experienced radiology technologist adjusted the location of the fluoroscope so that the top of the sacrum was visible in the lower part of the X-ray image.

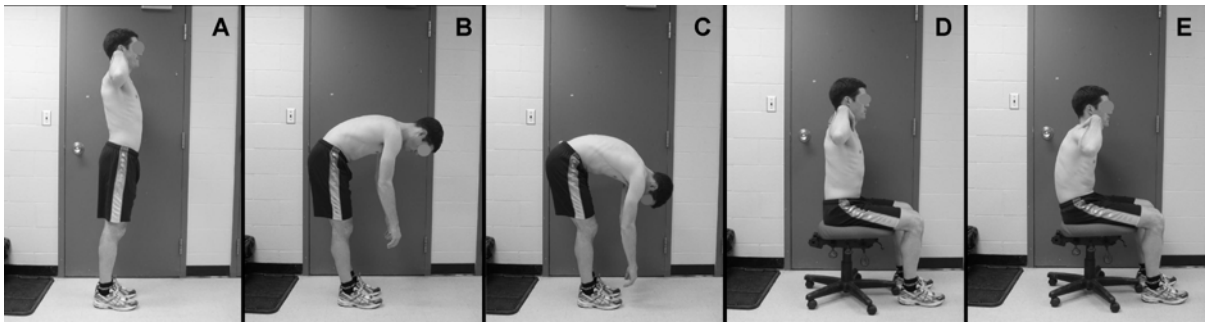


Figure 3.1: The five postures imaged in this study: A – upright standing, B – half flexion, C – full flexion, D – upright sitting, E – slouched sitting. Participants were instructed to place their hands on their shoulders in order to prevent the arms from obscuring the image of the spine.

The top two corners of the sacrum and four corners of the L3 to L5 vertebral bodies (VB) were manually digitized for each X-ray image using custom software (Visual Basic 6.0, Microsoft Corporation, Redmond, WA, USA) totalling 14 digitized landmarks. Each image was digitized 4 times by the same researcher to minimize random digitizing errors (Cholewicki et al., 1991). IVJ angles were calculated as the angle between sagittal "mid-plane" lines of adjacent VBs, defined as the line that intersected the mid-points between the two anterior and two posterior VB corners (Frobin et al., 1996). The upright standing IVJ angle was used as a baseline value and the IVJ angles in the 4 other postures were expressed

as a change from upright standing. The total RoM of each IVJ was defined as the greatest flexion value achieved by that IVJ in either the half or full standing flexion posture.

An error analysis was conducted as follows: the 5 images from a subset of 8 participants were digitized four times as described above. The average x and y co-ordinates (in pixels) for each of the 14 landmarks were calculated and the root mean square difference (RMSD) for each of the 4 digitized trials was determined. The IVJ angles for the four digitized trials were then calculated and the RMSD (in degrees) was determined. Error analysis revealed that the RMSD for digitizing was 0.8 (SD 0.4) pixels which corresponded to an angular error of 0.7° (SD 0.3°).

3.3.4 Statistical Analyses

Variables were initially analyzed using 2-way (gender*joint) repeated measures (joint) or 3-way (gender*posture*joint) repeated measures (joint, posture) analyses of variance (ANOVA). In most cases, gender was not a significant factor and thus 1-way (joint) or 2-way (posture*joint) ANOVAs were performed. Tukey's post-hoc multiple comparisons were used to examine significant effects and interactions.

3.4 Results

For up to 50% of the participants, the standing full flexion posture did not elicit the maximum observed RoM for a given joint (Table 3.1). Total standing RoM for L3/S1 was similar for both males and females (Table 3.1). The total RoM for each IVJ was not significantly different between genders, but the L4/5 had a higher RoM than both L3/4 *(P* = 0.003) and L5/S1 *(P* = 0.001) (Table 3.1). Females had significantly more total L3/S1 upright standing lordosis than males (Table 3.1). The L3/4 and L4/5 joints of the female participants exhibited greater lordosis than their male counterparts, but there was no significant gender difference between the L5/S1 lordosis.

Table 3.1: Mean (standard deviation) standing lordosis and total range of motion (RoM) for each of the three lower lumbo-sacral joints, including the RoM of all the joints spanning L3 and the sacrum (L3/S1). RoM values were calculated using the largest angle achieved by a particular joint in the two standing flexed postures. Negative values indicate extension or lordosis of the joint. The gender*joint interaction was significant for lordosis. For total RoM, only the joint main effect had significant differences and thus were collapsed across gender and compared between joints (see *Combined (n = 27)*). Significant differences ($P < 0.05$) are indicated by differing letters.

	Standing Lordosis			
	L3/4	**L4/5**	**L5/S1**	**L3/S1**
Female (n = 14)	-12.0° (1.6) d	-18.3° (4.4) f	-17.3° (5.6) f	-47.6° (7.1) h
Male (n = 13)	-8.5° (2.8) e	-14.9° (4.0) g	-18.8° (4.3) f	-42.2° (7.8) i
	Total RoM			
	L3/4	**L4/5**	**L5/S1**	**L3/S1**
	number of participants with max RoM from standing half flexion posture			
Female (n = 14)	*3*	*8*	*7*	*5*
Male (n = 13)	*4*	*6*	*3*	*3*
Combined (n = 27)	*12.6° (2.7)* *a*	*15.8° (3.4)* *b*	*12.2° (5.2)* *a*	*39.5° (8.1)* *c*

There were no significant gender effects or interactions for the subsequent variables and thus all reported data were pooled across gender. When the IVJ angles were expressed as a change from upright standing in the four other examined postures, there was a significant joint*posture interaction ($P < 0.0001$). The L5/S1 IVJ responded differently than the other two joints to the different postures (Figure 3.2). The L5/S1 IVJ rotated less than the L4/5 IVJ ($P < 0.005$) in all postures other than upright sitting. In the half flexion posture, the L5/S1 IVJ was less rotated ($P = 0.006$) than the L3/4 IVJ and more rotated ($P = 0.003$) in upright sitting (Figure 3.2). There was also a significant joint*posture interaction ($P <$

0.0001) when IVJ angles were expressed as a percentage of range of motion (% RoM) (Figure 3.3). In the half flexion posture, the L5/S1 joint was at a smaller % RoM ($P <$ 0.0001) than the other two IVJs which supports the well-established observation that full forward flexion is achieved by a top-down IVJ flexion pattern and terminated by forward pelvic rotation (Kanayama et al., 1996; Okawa et al., 1998; Harada et al., 2000). Upright sitting demonstrated a "bottom-up" flexion pattern whereby L5/S1 exceeded 60% of its total RoM followed by the L4/5 IVJ at around 50% RoM and L3/4 at 35% RoM (Figure 3.3). Each IVJ exceeded 80% RoM in the slouched sitting posture.

An interesting illustration of IVJ positioning was revealed when examining the percentage of contribution (%contribution) of each IVJ to the total amount of L3/S1 flexion. The L4/5 joint consistently contributed approximately 40% of total L3/S1 flexion in each of the 4 examined postures (Figure 3.4). In the two standing flexion postures, the %contribution of each joint did not change (Figure 3.4). Notably, in the upright sitting posture, L5/S1 contributed more to the total L3/S1 flexion than it did in any other posture ($P < 0.020$; Figure 3.4).

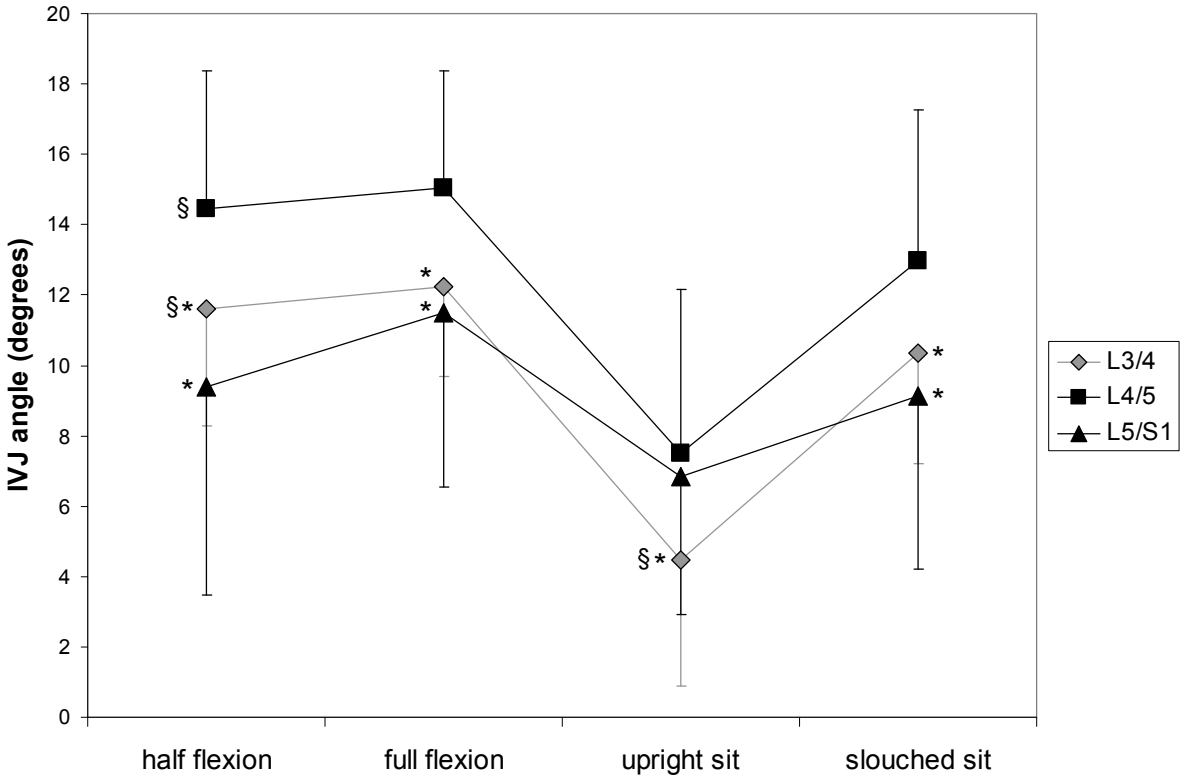


Figure 3.2: Mean angle of each intervertebral joint (IVJ) expressed as a change from upright standing in each of the adopted postures. The joint*posture interaction was significant ($P < 0.0001$). The L5/S1 IVJ responded differently than the other two joints in the different postures. In the upright sitting posture, the L5/S1 IVJ had a similar amount of flexion as the L4/5 joint. Within-posture IVJ angle significant differences ($P < 0.005$) are indicated by § (significant difference from L5/S1) and * (significant difference from L4/5).

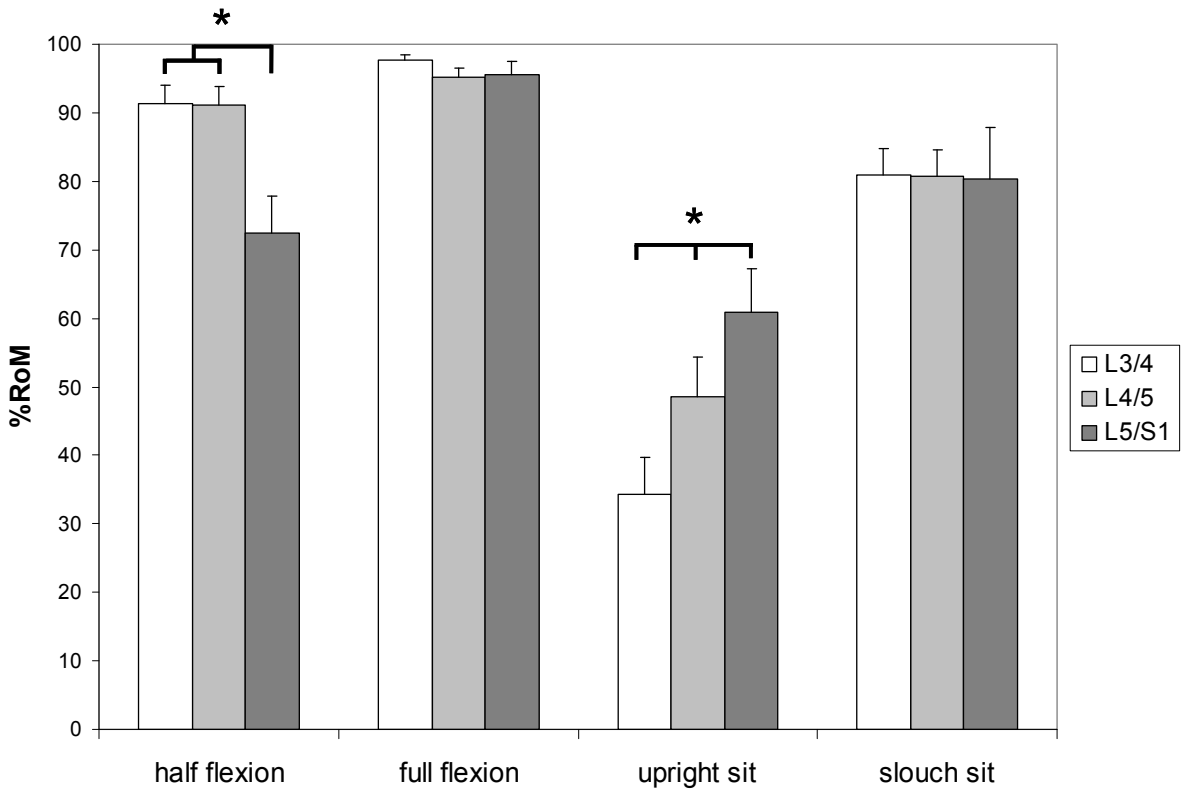


Figure 3.3: Mean intervertebral joint angle expressed as a percentage of total range of motion (%RoM) for each adopted posture. There was a significant joint*posture interaction ($P < 0.0001$). The asterisk (*) indicates significant differences between joint angles within a posture ($P < 0.002$).

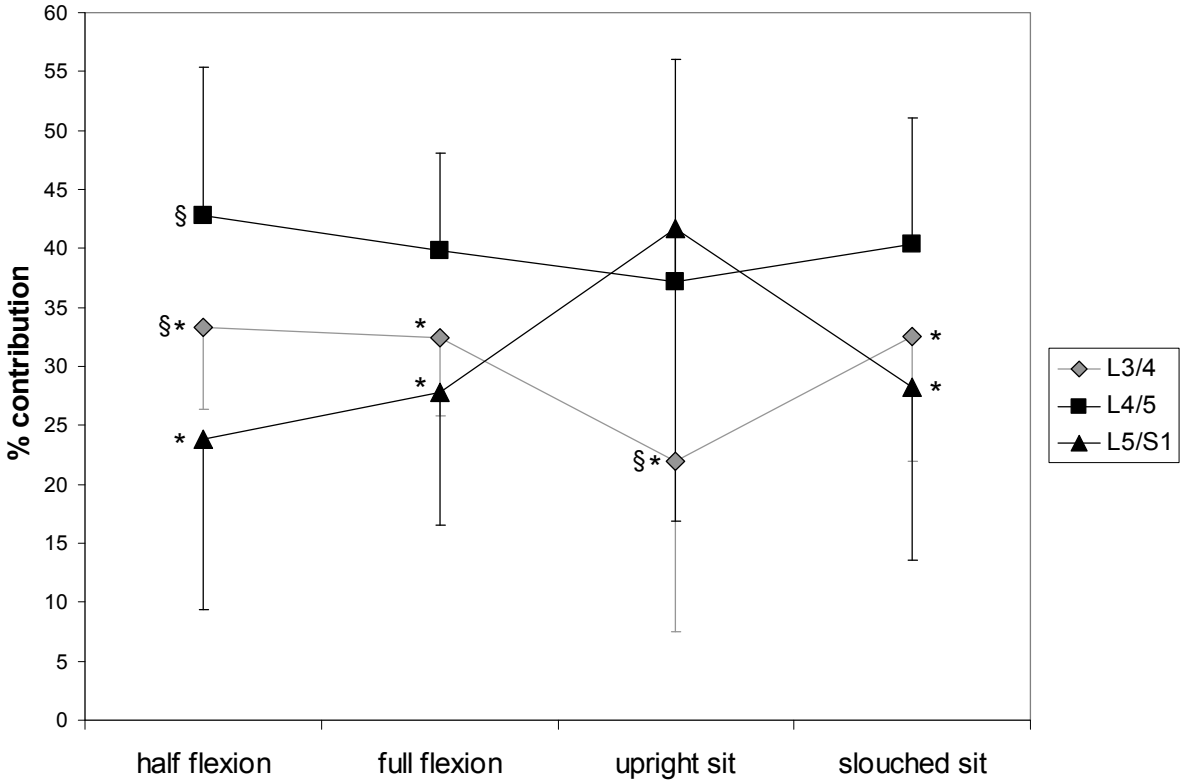


Figure 3.4: The contribution of each intervertebral joint to the L3/Sac flexion angle in each posture was expressed as a percentage (%contribution). There was a significant joint*posture interaction ($P < 0.0001$). The L5/S1 IVJ responded differently across the postures. It contributed the most to the total L3/Sac flexion in upright sitting. Within-posture IVJ angle significant differences ($P < 0.01$) are indicated by § (significant difference from L5/S1) and * (significant difference from L4/5).

3.5 Discussion

The lower LS spine was examined with static X-ray in various standing and sitting postures. The L4/5 IVJ had the highest RoM when compared to L3/4 and L5/S1. In upright sitting, L5/S1 was flexed to more than 60% of its total RoM and contributed to over 40% of the total L3/S1 flexion. In addition, each of the lower three IVJs approached their total standing

flexion angles in slouched sitting. These observations were the same regardless of gender. The results of this study support the idea that LS flexion is driven by rotation of the pelvis and lower IVJs in seated postures.

Sitting imposes a flexed posture that may have detrimental effects on the tissues of the spine. In the current study, the lower three IVJs approached their total standing flexion angles in slouched sitting. This suggests that there could be increased loading of the passive tissues surrounding the lower LS IVJs, leading to pain generation and potential injury. For flexion angles greater than 75% RoM, high tensile forces can be generated in the posterior ligaments of the spine (Adams et al., 1994). Furthermore, the erector spinae muscle activity is reduced by flexion-relaxation in slouched sitting, potentially redistributing the moment to the passive structures of the spine (Callaghan and Dunk, 2002). Higher intervertebral disc pressures *in vivo* have been demonstrated in sitting compared to standing (Andersson et al., 1975; Sato et al., 1999) and over 6 mm of posterior migration of the L4/5 and L5/S1 nucleus pulposus can occur in upright and flexed sitting (Alexander et al., 2007). The posterior annulus is at a higher risk for weakening and herniation because it is the narrowest part of the disc and least able to sustain large compressive loads (Adams et al., 1996). High annular stress peaks may also elicit pain from the innervated outer margins of the annulus, or from vertebral end plates (Adams et al., 1996). The current study's evidence that the lower lumbar IVJs approach their end RoM in slouched sitting lends insight into the relationship between prolonged sitting, LBP (Magnusson and Pope, 1998), disc herniation (Wilder et al., 1988) and disc degeneration (Videman et al., 1990).

This is the first study to compare the IVJ angles in sitting to total IVJ RoM. Thus, direct comparisons of our results to other studies are difficult due to the varying methods of measuring IVJ angles. The total IVJ RoM values observed in the current study are similar to values achieved from both static (Pearcy et al., 1984; Dvorak et al., 1991) and dynamic (Kanayama et al., 1996; Wong et al., 2004) RoM tests (L3/4: 10-15°; L4/5: 13-18°; L5/S1: 9-

17°). Sitting RoM is more difficult to quantify as there is large variability in the postures examined previously. Similar to the current study, others have demonstrated that the lumbar spine was more flexed in upright unsupported sitting than in standing (Andersson et al., 1979; Makhsous et al., 2003; Lin et al., 2006). This occurred mainly by rotation of the pelvis and the three lower lumbar IVJs, while there were few to no angular changes between L1/2 and L2/3. The values in the literature for upright sitting IVJ angles range from 4.9 to 8°, 6.7 to 10.2° and 4.6 to 7.0° for the L3/4, L4/5 and L5/S1 IVJ, respectively (Andersson et al., 1979; Makhsous et al., 2003; Lin et al., 2006), and agree very well with the values obtained in the current study (Figure 3.2). Furthermore, commonly adopted slouched postures increase the amount of flexion at all three of the lower lumbar joints, whether an individual has slouched from an upright sitting posture (the current study) or has slouched against a backrest (Lin et al., 2006).

Given the observation of gender differences in externally measured seated LS and pelvic postures (Dunk and Callaghan, 2005), it is intriguing that the lower 3 IVJs did not respond differently for the gender groups in the current study. It is possible that the amount of flexion observed in seated postures is distributed across the entire spinal column in a gender-specific manner. The upper lumbar IVJs were not imaged in this study due to the limited field of view of the image intensifier. Male L1 and L2 vertebral bodies tend to have more anterior wedging (i.e. anterior edge is shorter than posterior edge) than females (Cheng et al., 1998; Grados et al., 1999). This may allow for males to accommodate a more slumped lumbar posture in sitting (Dunk and Callaghan, 2005) that was not captured by the imaging method used in the current study. Furthermore, the ischial tuberosities of the male pelvis are more parallel in the anterior/posterior direction allowing males to roll the pelvis back and sit in a slouched position (Tague, 1992; Correia et al., 2005). Hamstring flexibility has also been shown to affect LS posture due to the muscles' direct attachment on the ischial tuberosities (Bridger et al., 1992). Given that females tend to have greater hamstring flexibility than males, it is possible that gender-specific seated postures are related to these inherent

differences in flexibility (Bridger et al., 1992). While it appears that IVJ RoMs may not account for the biological variation between genders (Dunk and Callaghan, 2005), further study of gender-specific pelvic and upper lumbar spine morphology, surrounding anatomy and how these affect global measurements of sitting posture is required to resolve these conflicting findings.

The present study focused on a static analysis of a dynamic phenomenon. Due to the limited field of view of the fluoroscope, static postures were used to capture end RoMs. It is interesting to note that in the current study, up to 50% of the participants achieved a greater amount of IVJ flexion at a given joint in the standing half flexion posture. This suggests that bending down to touch one's toes may not be the best way to elicit maximum flexion in the lumbar spine. It also brings into question if the IVJs examined in this study ever reached their true maximum range of motion. As mentioned above, the observed IVJ RoM values (Table 3.1) do agree with previously reported active lumbar RoM (Pearcy et al., 1984; Dvorak et al., 1991; Kanayama et al., 1996) as well as *in vitro* passive flexion RoM values from human lumbar motion segments (mean RoM = 12.7° (SD 3.5)) (Adams et al., 1994). Thus, it is likely that the IVJs examined in the current study were very close to their end RoM. The current study's approach of examining static images could be missing valuable information about altered seated movement patterns. Dynamic mid-motion characteristics of standing lumbar IVJ RoMs have separated individuals with LBP from healthy controls (Okawa et al., 1998; Teyhen et al., 2007). It is possible to quantify rhythms, delays and alterations in movement patterns related to the patients' symptoms. For future sitting research, the quantification of movement patterns from dynamic imaging techniques could be useful in identifying individuals who develop LBP during prolonged sitting.

Lumbo-sacral vertebral imaging analyses are inherently fraught with errors and have been extensively studied by other research groups (Breen et al., 1989; Cholewicki et al., 1991;

Dvorak et al., 1991; Panjabi et al., 1992). Fluoroscopic images are known to have "pin cushion" distortion because of the curved image intensifier surface. While image distortion correction has reduced measurement error (Breen et al., 1989; Cholewicki et al., 1991), this type of error may be minimal with respect to other errors induced by the digitizing process and out-of-plane imaging (Brinckmann et al., 1994; Wearing et al., 2005). In the current study, care was taken to centrally locate the anatomical area of interest in the field of view where distortion is minimal. The digitized VB corners and the mid-plane IVJ angles were chosen because these parameters are minimally affected by radiographic distortion and off-centre positioning (Brinckmann et al., 1994; Frobin et al., 1996). Each image was also digitized four times in order to reduce random error associated with this process (Cholewicki et al., 1991).

3.6 Conclusions

In upright sitting, there is evidence of a "bottom-up" flexion pattern exhibited by the lower 3 LS IVJs. This is supported by observations that L5/S1 achieved more than 60% of its max RoM in this posture and the next two adjacent IVJs (L4/5 and L3/4) were at decreasing values of their total RoM. This is the first study to quantitatively show that the lower LS joints, in particular L5/S1, approach their total RoM in various seated postures. Upright sitting results in IVJ angles that are associated with less involvement of the passive tissues surrounding the joint. However, there could be increased loading of the passive tissues surrounding the lower LS IVJ in slouched sitting postures, leading to pain generation and potential injury from prolonged sitting.

Chapter 4:
The feasibility of using accelerometers to measure lumbo-sacral spine angles in standing and seated postures

4.1 Synopsis

Background: Accelerometers were used as a method of measuring external lumbar spine and pelvic tilt angles in two studies in this thesis. This chapter describes the collection equipment and analysis techniques in detail. Furthermore, externally measured lumbar spine angles were compared with corresponding angles measured from X-ray images.

Methods: Static sagittal digital X-ray images of the lumbo-sacral region from L3 to the top of the sacrum were obtained in five standing and seated postures. Accelerometers were placed over the L3 spinous process and sacrum in order to simultaneously measure the external L3/Sacral angle.

Findings: The accelerometers over-predicted the internal L3/Sacral intervertebral (IVJ) angles in standing postures by 11.7° (SD 4.3) and under-predicted them in sitting by approximately 7.4° (SD 3.0). However, the external measurement devices were sensitive to changes in angle, only when the postural change occurred between standing postures (e.g., half to full flexion), or between seated postures (e.g., upright to slouched sitting).

Interpretation: The main use of the accelerometers tested in the current study is to examine lumbar spine postures while sitting. The externally measured seated angles in this study were consistent with those measured with similar technology in other studies. Furthermore, accelerometers are sensitive to changes in seated lumbar spine posture where the motions may be small enough to limit erroneous results caused by skin motion and excessive pelvis or vertebral body rotation.

4.2 Introduction

Accelerometers were used as a method to measure external lumbar spine and pelvic tilt angles during prolonged sitting in Study #4 (Chapter 6) of this thesis. The collection equipment and techniques are presented in detail in this chapter along with an investigation to compare externally measured lumbar spine angles with those angles measured from X-ray images.

Accelerometers can be used as inclinometers in quasi-static and static situations (Willemsen et al., 1990; Hansson et al., 2001; Bernmark and Wiktorin, 2002; Wong and Wong, 2008; Beach et al., 2008). Given that accelerometers have a DC response, the output of the vertically aligned axis of the sensor is proportional to its angle with respect to the line of gravity. The small size of accelerometers (i.e. NexGen accelerometers are 29.5 x 11.6 x 15.3 mm) make them ideal for measuring lumbar spine and pelvic angles while sitting in a chair with a back rest (Figure 4.1).

Tri-axial accelerometers have been shown to be comparable to a skin-mounted marker motion analysis system in static conditions (RMS error < 1°) and less so in dynamic conditions (RMS error < 5°) (Wong and Wong, 2008). In dynamic seated conditions, lumbar and pelvic kinematics calculated from accelerometer outputs correlate well ($r > 0.9$) with lumbar spine and pelvic measurements made using a common fin marker method (Singer et al., 2007). Furthermore, angles measured from electronic inclinometers placed on the sacrum (at S1) and over the L1 spinous process have been shown to correlate well with X-ray angles (Adams et al., 1986). However, Adams' study, as well as others that have compared external and internal measurements of spine angles, are limited by small ranges of motion and constrained postures, which could limit confounding factors such as skin motion and external device movement during postural changes (Adams et al., 1986; Chen, 2000; Chen et al., 2004; Campbell-Kyureghyan et al., 2005; Vergara et al., 2006). Therefore, the purpose of

the current investigation was to determine the accuracy of external spine angles measured by tri-axial accelerometers in a wide range of spine postures, from upright standing to full flexion.

4.3 General Instrumentation

Tri-axial strain gauge accelerometers (S2-10G-MF, NexGen Ergonomics, Montreal, Quebec, Canada) were used in these studies. Accelerometer signals were digitized at a rate of 128 Hz using a 12-bit (NI 6024E, National Instruments Corporation, Austin, TX, USA) or 16-bit (Optotrak Data Acquisition Unit II, Northern Digital Inc., Waterloo, ON, Canada) analog-to-digital conversion system depending on the study. In the current investigation, the 12-bit system was used.

4.3.1 Accelerometer placement and collection procedures

Three accelerometers were placed over the spinous processes of the first and third lumbar vertebrae and over the sacrum at the level of S2. Each accelerometer was oriented so that the positive y-axis was aligned pointing down the vertical axis of the spine. The positive x-axis was aligned with the horizontal towards the left and the z-axis perpendicular to these pointing towards the posterior of the body (Figure 4.1). For angle normalization purposes, an upright standing trial was collected in order to determine the inclination of each accelerometer in an individual's neutral spine posture. Maximum lumbar spine flexion was also recorded, with the participant bending down to touch their toes and holding the posture for 5 seconds.

During data collection for all studies that used this methodology (Studies #2, 3 and 4), 3 accelerometers were used to calculate 3 different spine angles: lumbar spine (L1 & sacral accelerometers), L3/Sac (L3 & sacral accelerometers) and pelvis (sacral accelerometer). However, for the different comparisons/analyses presented throughout the thesis, the data presentation may not have included all three angle measurements. In this event, only 2

accelerometers were reported being used for simplicity (e.g. Study #4, Chapter 6 describes the lumbar spine and pelvic angles only, those reporting the use of only 2 accelerometers).

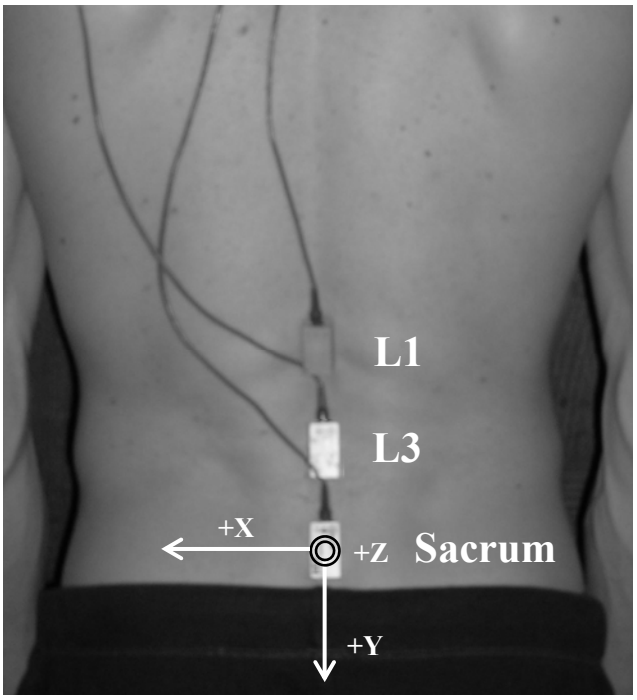


Figure 4.1: Three tri-axial accelerometers were placed over the spinous processes of the vertebrae at L1 and L3 as well as over the sacrum at the level of S2. The axes of each accelerometer were oriented such that positive Y pointed down the vertical axis of the spine, positive X pointed to the left along the horizontal and positive Z was perpendicular to X and Y, pointing posterior to the body (i.e. out of the page).

4.3.2 Calibration and angle calculation methodology

The output from an accelerometer is a sinusoidal function of the inclination, and the calibrations were performed at the maximum and minimum values of this function. Thus, the output from each axis was recorded at 0° (or +1g) and 180° (or -1g), providing a conversion factor from milli-volts to degrees.

Signal processing steps were as follows:

1) The raw data were originally filtered with a zero-lag 4th order low pass Butterworth filter with a cut-off frequency of 10 Hz to eliminate any high frequency artifacts from the data.

2) Voltage outputs were converted to degrees using the following equation:

$$Inclination.Angle = \arcsin\left[\frac{output(mV)}{conversion\_factor}\right] \quad \text{(Equation 4.1)}$$

3) The y-axis was used as the inclination axis and the z-axis was used in order to determine the correct orientation of the y-axis inclination (Figure 4.2). Because of the sinusoidal nature of the accelerometer output, two axes are necessary in order to determine the correct orientation. For example, the y-axis is pointed up (180°) and the z-axis is oriented to the left (90°) (Figure 4.2). It was assumed that changes in inclination occurred only in the sagittal plane, and any lateral bend or axial twist was ignored.

4) Lumbar spine angle was determined as the difference between the L1 and sacral accelerometer inclinations. The L3/Sac angle was calculated as the difference between the L3 and sacral accelerometers. Pelvic inclination was taken as the inclination angle of the sacral accelerometer.

5) Converted angle signals were then smoothed using a zero-lag 4th order low pass Butterworth filter with a cut-off frequency of 1 Hz to remove any inconsistencies in the signal that may have occurred during the conversion process.

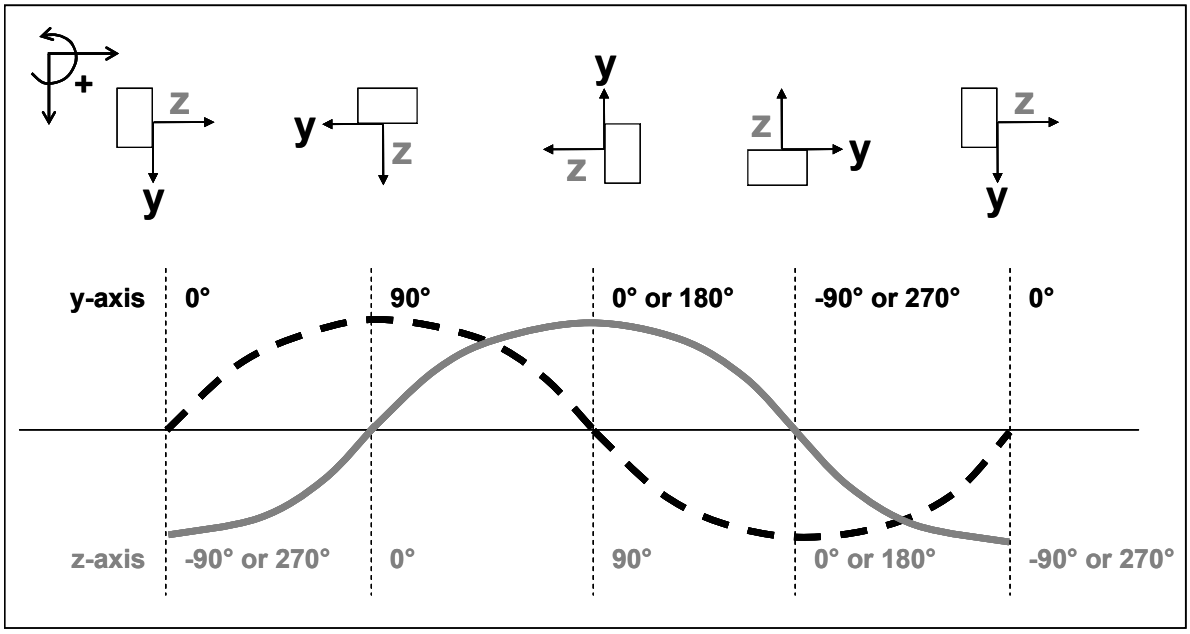


Figure 4.2: Method for determining the correct orientation of each accelerometer. The y-axis was aligned with the long axis of the spine and was used to determine the inclination angle. The z-axis was used to determine the correct orientation of the y-axis.

4.3.3 Instrumentation considerations

There are a number of potential concerns associated with the use of accelerometers as inclinometers, namely issues related to electro-mechanical drifts and cross-talk (due to misalignment between the spinal column and the sensitive axis of the accelerometers). To address these issues, care was taken to ensure proper alignment and securing of the sensors, and pilot tests were conducted to ensure that accelerometer offsets (zero bias) and sensitivities (mV per degree) were not drifting over the course of the testing sessions.

4.4 Methods: Internal vs. external measures

4.4.1 Participants

The data presented in this study were collected from the same participant pool as the study presented in Study #1 (Chapter 3). However, 1 female and 1 male participant were excluded because of missing accelerometer data. Therefore, twenty-three participants, 11 males (mean age = 29.6 (SD 9.1) years; mean height = 1.79 (SD 0.05) m; mean mass = 78.8 (SD 8.3) kg; mean BMI = 24.6 (SD 2.9) kg/m^2) and 12 females (mean age = 27.2 (SD 7.2) years; mean height = 1.64 (SD 0.06) m; mean mass = 59.1 (SD 7.1) kg; mean BMI = 21.9 (SD 2.4)g/m^2), were recruited. The males had significantly higher BMI than the females ($P = 0.02$). A justification for the chosen sample size can be found in 0.

4.4.2 Instrumentation

External lumbar spine and pelvic angles were measured using accelerometers placed over the spinous processes of the L1 and L3 and over the sacrum at the level of S2. The accelerometer signals were digitized at a rate of 128 Hz using a 12-bit analog-to-digital conversion system (NI 6024E, National Instruments Corporation, Austin, TX, USA). Accelerometer data were collected for 5 seconds while sagittal digital X-ray images of the lumbo-sacral region were concurrently obtained (see Study #1 (Chapter 3, Section 3.3.2) for full description of digital X-ray equipment).

4.4.3 Data Collection and Analysis

One static X-ray image and accompanying accelerometer data were obtained for standing, half flexion, full flexion, upright sitting and slouched sitting (see Chapter 3, Section 3.3.3 for full description of each posture). The internal L3/Sac IVJ angle was calculated as described in Chapter 3, Section 3.3.3. Briefly, the top two corners of the sacrum and the four corners of the L3 vertebral body (VB) were manually digitized in each X-ray image. The internal L3/Sac angle was calculated as the angle between sagittal "mid-plane" lines of the L3 VB

and the top of the sacrum. Furthermore, the internal sacral angle was calculated as the angle between the top of the sacrum and the horizontal. Accelerometer outputs were converted to degrees (as described above in Section 4.3.2) and the total lumbo-sacral (LS) spine angle, as well as the L3/Sacral (L3/Sac) angle were calculated. The inclination of the sacral sensor was considered to be representative of anterior/posterior pelvic tilt. The average of the 5-second trial was computed to represent the externally measured angles of the given posture.

For this investigation, the external (EXT) L3/Sac angles measured by accelerometers were compared to the internal (INT) L3/Sac angles from the X-rays and the lumbo-sacral (L1/Sac) angle was calculated for the sole purpose of comparing the current study's results with previous results from the literature (presented in the Discussion, Section 4.6.1). For the measurement comparison study, L3/Sac angles were expressed in two different ways: the first, termed "absolute angles" (ABS), were the angles derived directly from the calculations (i.e. INT angle between L3 mid-plane and top of the sacrum from x-ray or the difference between the EXT inclination angle of the L3 and pelvic accelerometers). The second method for expressing angles was termed "change from upright standing" (CHANGE), indicating that the INT or EXT L3/Sac angle measured in upright standing was considered "zero" and was subtracted from the angle measured in each of the other postures. Furthermore, the absolute difference between the two angle measurement methods (INT or EXT) was calculated within each posture for each method of angle expression (ABS or CHANGE) to examine the effect of data treatment on angle measurements (i.e. |EXT angle – INT angle| within a given posture, for a given angle calculation method (ABS or CHANGE)). One final variable was calculated in order to assess how each measurement method (EXT or INT) tracked changes in posture. This was done by computing the difference between every combination of the L3/Sac angle in each of the 5 postures assumed, leading to 2 (EXT or INT) x 10 (number of comparisons between the 5 postures).

Please note that the INT sacral angle measured from x-rays could only be expressed as a "change from upright standing". This is because of the portable nature of the fluoroscope, and its C-arm design that allowed the x-ray source and image intensifier unit to be positioned in different orientations. Thus, the orientation of the actual x-ray image was performed manually and not necessarily consistent between participants. However, since the C-arm orientation did not change between postures within a given participant, relative changes from one posture to the next could be examined. Therefore, for the purpose of comparison, the INT sacral angles and the orientation of the sacral accelerometer (EXT sacral angle) were expressed as a "change from upright standing".

4.4.4 Statistical Analyses

EXT (accelerometer) and INT (X-ray) L3/Sac and sacral angles were compared across gender and postures using a 3-way mixed general linear model (GLM) with repeated measures on two factors (measure, posture). This statistical analysis was performed separately on the ABS and CHANGE angle datasets. EXT L3/Sac and sacral angles were compared across gender and postures using a 2-way mixed GLM with one repeated factor (posture). Three-way repeated measures GLMs were also used to analyze the absolute differences between the two angle measurement methods (INT or EXT) (factors: gender, posture, calculation method; repeated measures: posture, calculation method) and compare the measurement method posture tracking (factors: gender, 2 posture comparison, measure; repeated measures: 2 posture comparison, measure). Pairwise comparisons were examined using Tukey's post-hoc test and differences were considered significant when $P < 0.05$.

4.5 Results

In upright standing, the mean external L3/Sac lordosis angle was 19.5° (SD 15.2) and was lower than the internal L3/Sac lordosis measured from X-ray (45.0° (SD 8.4); $P < 0.0001$). The gender main effect was not significant, nor was the gender*measure interaction. When comparing the absolute internal and external L3/Sac angles across the 4 other postures, there was a significant measure*posture interaction (($P < 0.0001$): the angles measured by accelerometers were significantly different from those measured from x-rays in all postures (Figure 4.3). In fact, the accelerometer absolute angles indicated that the L3/Sac spine was kyphosed in the two standing flexion and slouched sitting postures while some lordosis was maintained in the upright sitting posture. On the other hand, the L3/Sac spine angle measured from x-ray images indicated that there was always some lordosis maintained in every posture.

When the internal and external L3/Sac angles were expressed as a change from upright standing, the significant measure*posture interaction ($P < 0.0001$) was maintained: however, the accelerometers significantly under-predicted the internal L3/Sac angle in both standing postures ($P = 0.0004$ for standing half flexion; $P = 0.001$ for standing full flexion) and tended to over-predict the angle in both sitting postures ($P = 0.057$ for upright sitting; $P = 0.003$ for slouched sitting; Figure 4.4). This phenomenon was the same regardless of gender (i.e. the gender effect and interactions were not significant). However, when the external L3/Sac angles were compared alone across postures and expressed as a change from upright standing, there was a trend towards a gender effect ($P = 0.062$) where the male external L3/Sac angles were significantly higher than female angles (Figure 4.5). This gender effect did not exist with the internally measured L3/Sac angle ($P = 0.84$).

The angles measured by accelerometers did not match with the internal angles from X-rays, regardless of the method of expressing the angles (i.e. absolute vs. change from upright standing). However, it appears as though expressing the angles as a change from upright standing (or removing the standing bias in each posture) reduced the difference between the two methods (significant posture*calculation method ($P = 0.003$)) (Figure 4.6). Specifically, within the "absolute angle" calculation method, the angle differences seen in the standing postures were greater than those seen in the sitting posture ($P < 0.0001$). Within the "change from upright" calculation method, the difference between measurements was reduced and not significant between all postures (Figure 4.6).

The top of the sacrum and the sacral accelerometer inclination were compared when the angles were expressed as a change from upright standing (absolute angles were not compared for reasons explained in Section 4.4.3). There was a significant gender effect ($P = 0.01$) where females always had more anterior rotation (16.9° (SD 35.9)) than males (7.9° (SD 34.9)), regardless of posture. This gender effect also existed when the internal and external sacral angles were analyzed separately (internal sacral angle $P = 0.03$; external sacral angle $P = 0.01$). Comparing the internal and external angles, the measure*posture interaction was significant ($P = 0.0004$): the sacral accelerometer under-estimated the amount of sacral rotation in only the full standing flexion and seated slouched postures (Figure 4.7). However, the differences between the two measurements of sacral rotation were 4.6° and 6.0° for the full flexion and slouched sitting postures, respectively. Otherwise, it was observed that the sacral accelerometer tracked sacral rotations quite well in the half flexion and upright sitting postures, where pelvic rotation was not at its extreme. In order to examine the gender effect more closely, both the external and internal sacral angle changes were analyzed separately. The external sacral angle results are reported here: there were significant effects of both gender ($P = 0.01$) and posture ($P < 0.0001$). Specifically for the gender effect, the female sacrum had more anterior rotation in the 2 standing flexion postures

and less posterior rotation in the 2 seated postures. In other words, the female sacrum always had more anterior rotation than the male sacrum.

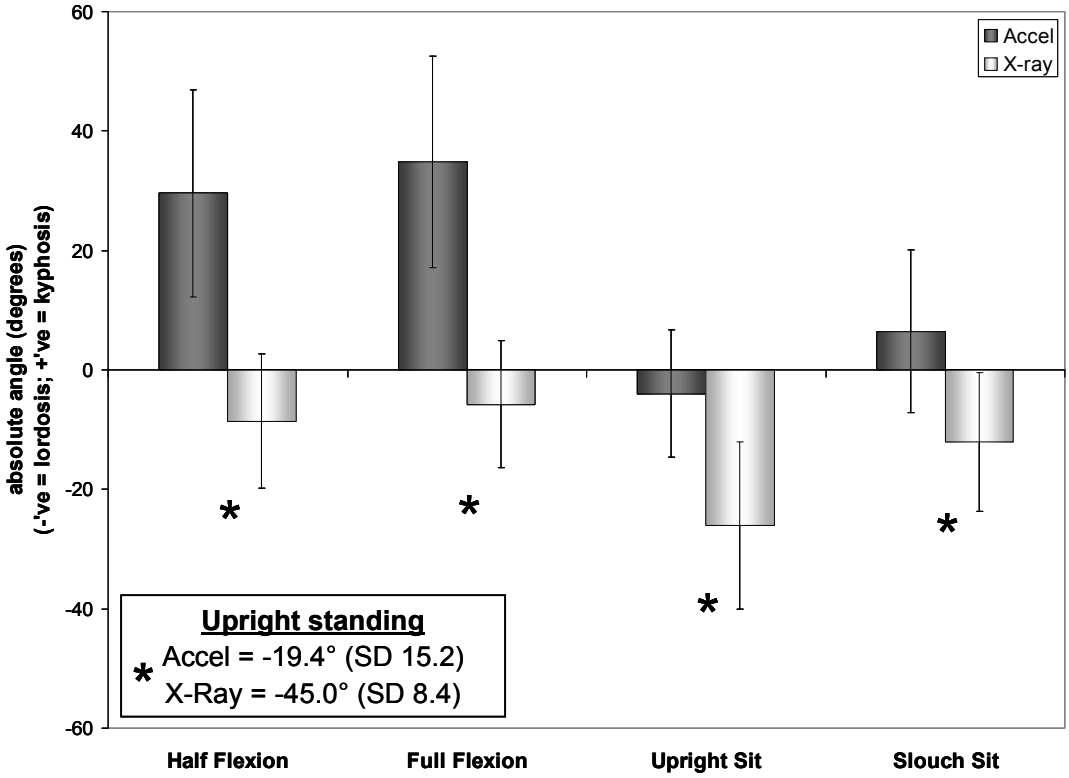


Figure 4.3: L3/Sacral ABSOLUTE angles measured from X-ray images and by accelerometers acting as inclinometers. Negative values indicate lordosis; positive values indicate kyphosis. There was a significant measure*posture interaction (($P < 0.0001$): the angles measured by accelerometers were significantly different from those measured from x-rays in all postures (* indicates $P < 0.0001$).

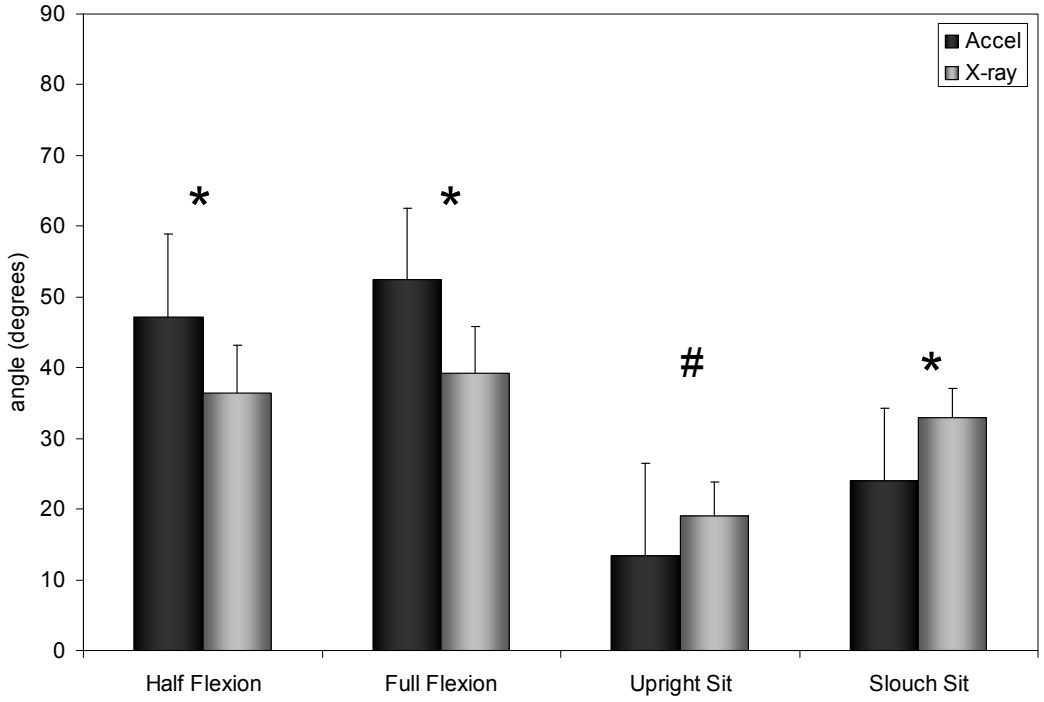


Figure 4.4: L3/sacral (L3/Sac) angle expressed as a CHANGE FROM UPRIGHT, measured from X-ray images and by accelerometers acting as inclinometers. The internal (X-ray) angles were significantly different from the external (accelerometer) angles (* indicates $P < 0.005$; # indicates $P = 0.057$).

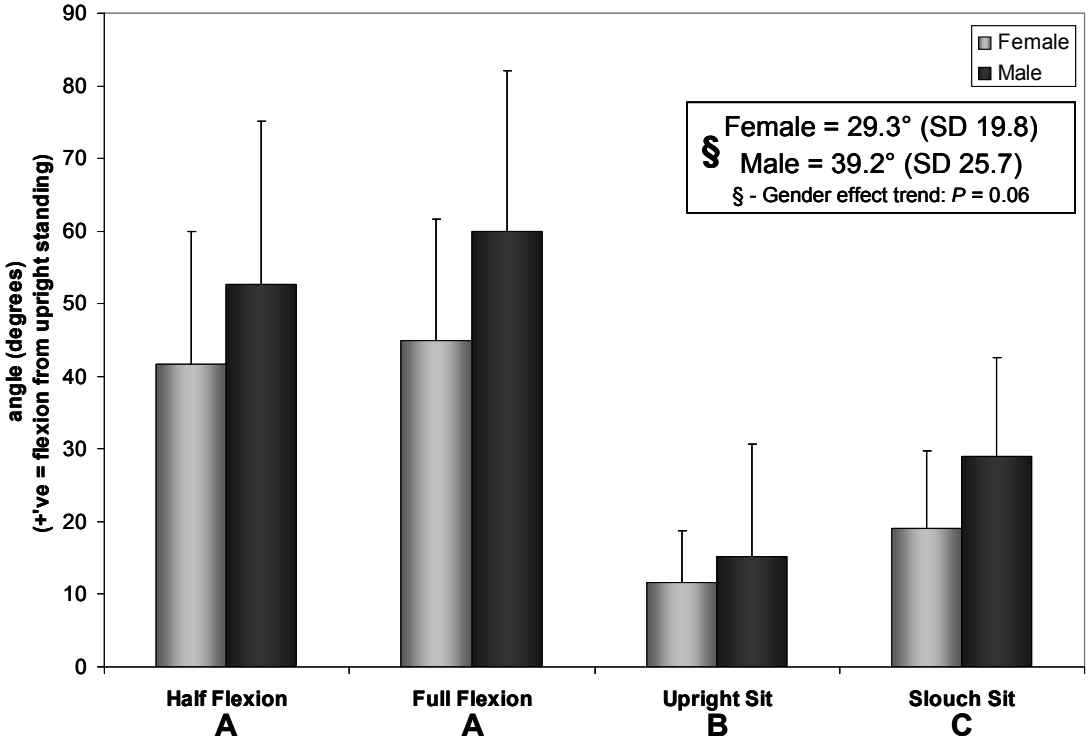


Figure 4.5: L3/Sacral (L3/Sac) angle measured externally by accelerometers, expressed as a change from upright standing. The main effect posture ($P < 0.0001$) was significant: different letters indicate which postures were significantly different (e.g. half flexion was not significantly different from full flexion, but different from upright and slouched sitting). There was also a gender effect trend ($P = 0.062$); however the interaction between these two variables was not signficant. In general, male L3/Sac angles were higher than female angles. The two standing postures were not significantly different from each other; the two sitting postures were different from each other and from both of the standing postures.

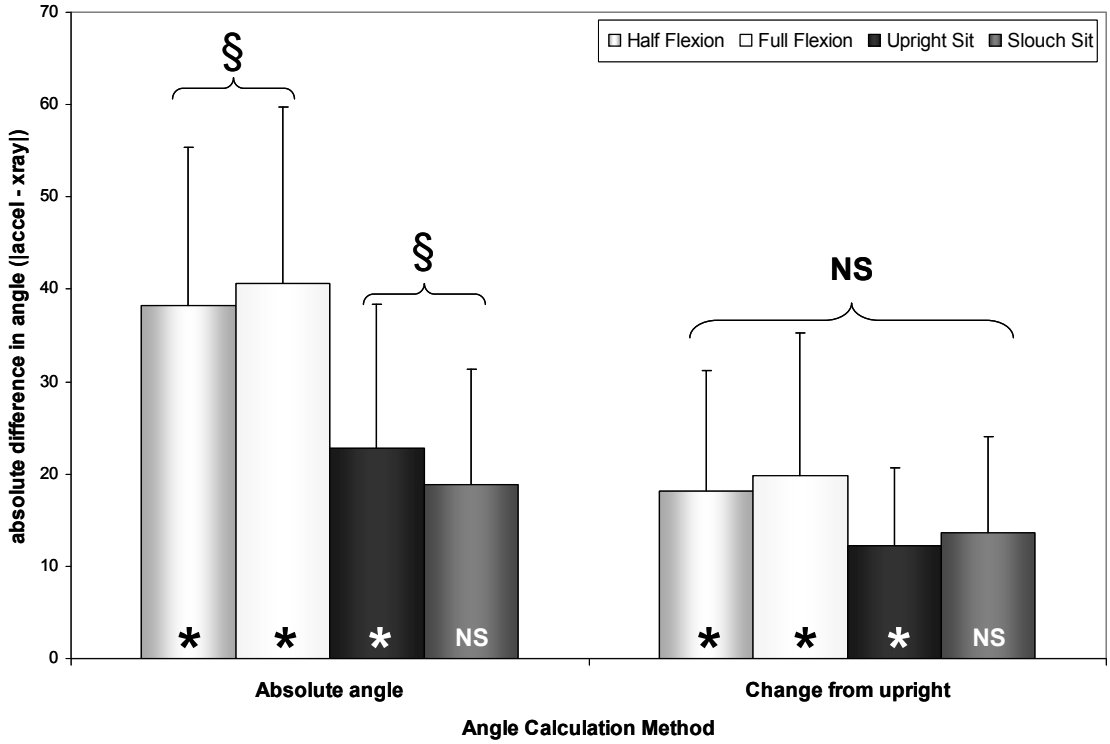


Figure 4.6: Absolute difference in angle between the external (EXT) and internal (INT) L3/Sacral angles (|accel – xray|) for each of the four flexion postures. There was a significant posture*calculation method (*P* = 0.003): expressing angles as a change from upright significantly reduced the difference between the accelerometer and X-ray angles (* indicates *P* < 0.003). Furthermore, within the "absolute angle" calculation method, the angle differences seen in the standing postures were greater than those seen in the sitting posture (§ indicates *P* < 0.0001). Within the "change from upright" calculation method, the difference between measurements was reduced and not significant between all postures.

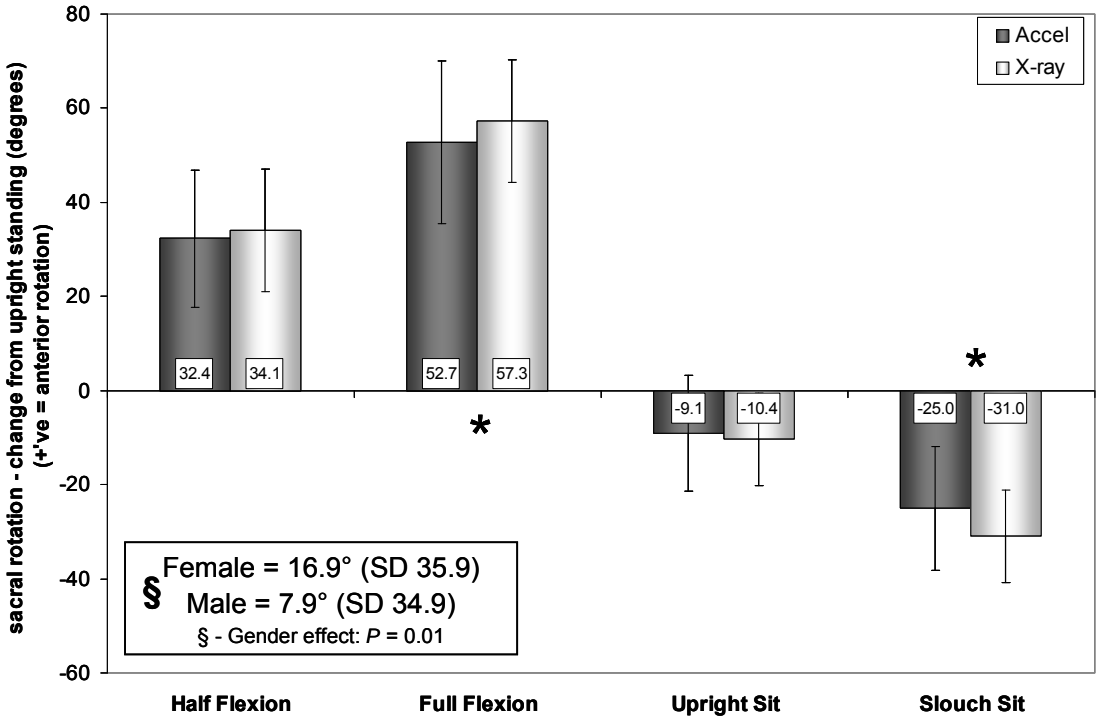


Figure 4.7: Sacral angle, expressed as a change from upright standing, measured externally by accelerometers and internally as the top of the sacrum. There was a significant main effect of gender ($P = 0.01$) where the female sacrum always had more anterior rotation than males. The measure*posture interaction was also significant ($P = 0.0004$): the sacral accelerometer angle was less than the internal angle of the top of the sacrum in full standing flexion and slouched sitting (* indicates $P < 0.05$).

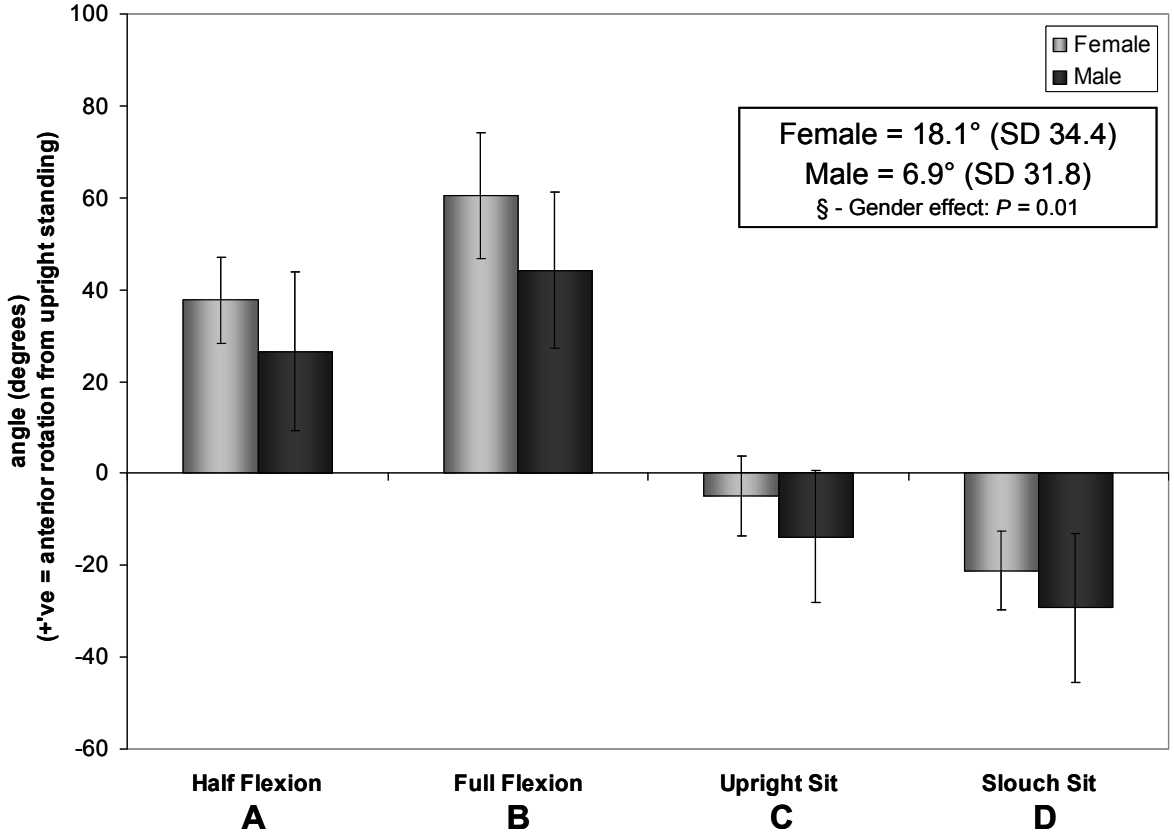


Figure 4.8: Sacral angle measured externally by accelerometers, expressed as a change from upright standing. The main effects of posture ($P < 0.0001$) and gender ($P = 0.01$) were significant while the interaction between these two variables was not. Posture differences are indicated by differing letters (i.e. all postures were significantly different from each other). In general, the female pelvis had more anterior rotation in the 2 standing flexion postures and less posterior rotation in the 2 seated postures.

In order to get an idea of how each measurement method tracked changes in posture, the change in L3/Sac angle for a given method (external or internal) was compared between postures. Although there were differences between genders in the external angles, the gender effect was not significant for the changes in angle between postures as measured by accelerometers ($P = 0.2$). However, there was a significant posture*measure interaction ($P < 0.0001$); Table 4.1 displays the angle differences collapsed across gender for the

combinations of posture comparisons. The external L3/Sac measurement method generally registered larger changes in angle than the internal measurement method except when comparing the change in angle from upright sitting to slouched sitting (Table 4.1). Also of note is that angle changes were not significantly different between measurement methods in either standing (half to full flexion) or sitting (upright to slouched sitting) postural changes, and when changing from upright standing to upright sitting.

Table 4.1: The values in each cell represent a mean difference (SD in brackets) in L3/Sac between postures, measured by accelerometers (Ext) or from X-ray images (Int). For example in row 4 column 7, the upright sitting external L3/Sac angle was 10.4° less than the external angle in slouched sitting. Positive value means that the "column" posture was less than the "row" posture. Significant differences between the external and internal angles are indicated by an asterisk (*, $P < 0.05$)

	Half flexion		**Full flexion**		**Upright sit**		**Slouch sit**	
	Ext	**Int**	**Ext**	**Int**	**Ext**	**Int**	**Ext**	**Int**
Upright Stand	*-49.1° (24.8)	*-36.4° (9.5)	*-54.3° (22.1)	*-39.2° (8.8)	*-15.5° (14.1)*	*-19.0° (10.0)*	*-25.9° (15.3)	*-33.0° (10.3)
Half flexion			*-5.2° (20.9)*	*-2.8° (5.1)*	*33.6° (19.4)	*17.4° (13.6)	*23.2° (22.7)	*3.4° (8.2)
Full flexion					*38.8° (16.4)	*20.3° (12.1)	*28.4° (16.7)	*6.3° (6.1)
Upright sit							*-10.4° (11.0)*	*-14.0° (12.3)*

There were 9 participants where the L3 accelerometer was visible in their upright standing and upright sitting X-ray images. The top and bottom corners of the accelerometer that lay against the skin surface were digitized along with the four corners of the L3 vertebral body (Figure 4.9). The L3 VB mid-plane line was calculated with same method employed in Chapter 3. The angle between the L3 mid-plane line and the accelerometer line was calculated using the dot-product (Figure 4.9). On average, the angle between the L3 VB and corresponding accelerometer was 95.7° (SD 6.2) for upright standing and 91.6° (SD 10.9) for upright sitting, indicating that the accelerometer had rotated with respect to the L3 vertebral body. Calculations for the linear translation of the accelerometer (to investigate skin motion) were difficult to separate from rotational motions. The 2 images (upright standing, upright

sitting) of these 9 participants were visually inspected to determine how the accelerometer moved with respect to the L3 spinous process and VB. No consistent pattern of skin movement could be identified when moving from an upright standing to upright sitting posture. However, for 4 out of these 9 participants, the L3 accelerometer was also visible in the full flexion posture and it was consistently more superior with respect to its position in either the upright standing or sitting postures (Figure 4.10).

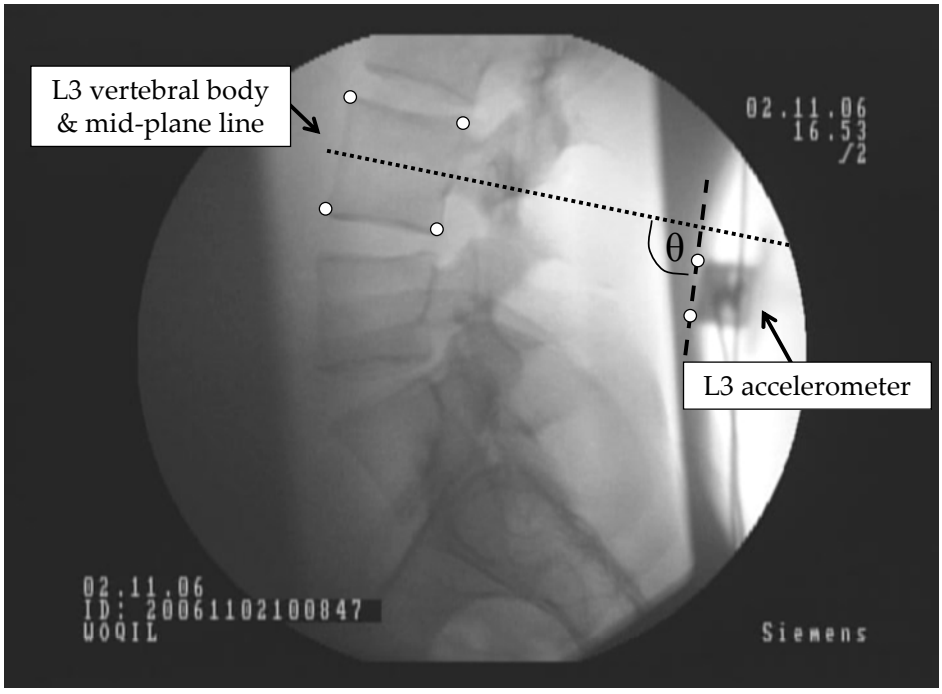


Figure 4.9: A sample X-ray image of upright standing with the L3 accelerometer visible against the skin. The two corners of the accelerometer that lay against the skin were digitized along with the 4 corners of the L3 vertebral body (VB). The VB mid-plane line was calculated, and the angle between this line and the accelerometer line was determined. In this case, the angle (θ) is about 95°.

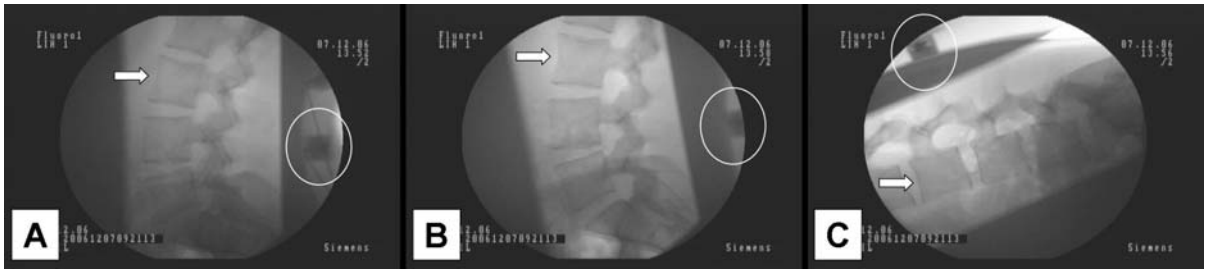


Figure 4.10: Series of 3 X-rays from one participant showing movement of the L3 accelerometer (white circle) with respect to the L3 vertebral body (white arrow). In upright standing (A) and upright sitting (B), the accelerometer is positioned in between the L3 and L4 spinous processes. It moves more superior with respect to the L3 vertebral body in the fully flexed standing posture (C).

4.6 Discussion

The main finding from this investigation was that externally measured lower lumbar spine angles did not match with internally measured ones. External L3/Sac angles measured by accelerometers over-predicted the internal L3/Sac angle in standing and under-predicted in sitting. However, removing the upright standing "bias" and expressing angles as a change from upright appeared to reduce the difference between the two measurements. Furthermore, the sacral accelerometer tracked changes in internal sacral orientation quite well, except towards the extreme ends of the pelvic range of motion. A gender trend was noted in the external L3/Sac angles that was not seen in the internal angles; males tended to demonstrate higher external L3/Sac flexion angles than females in the 4 standing and sitting postures. However, this gender effect may be partially driven by the significant gender effect observed in sacral orientation; in the 2 standing flexion postures, the female sacrum went through more anterior rotation than males, and less posterior rotation in the 2 seated postures. However, regardless of gender, both the internal and external measurement methods tracked ***changes*** in L3/Sac posture in a similar manner, but only within standing or seated postural changes. Thus, when using accelerometers to measure external angles, the response is a function of both gender and posture. While accelerometers fixed to the skin over a lumbar spinous process and on top of the sacrum do not accurately represent the internal rotations of the

vertebrae, these devices have the ability to track changes in the external shape of the spine particularly within seated postures.

4.6.1 Do externally measured angles match with previous (or concurrent) results?

The lumbar spine from L1 to the sacrum (L1/Sacral) and pelvic angles measured in this study were compared with other studies that used similar measurement methods: one study's results were obtained using the same external devices in a different population at different time points (see Chapter 6) and the other study used uniaxial accelerometers in a different sitting investigation (Beach et al., 2008). The current study's results are consistent with lumbar and pelvis RoM results obtained from these two comparison studies (Table 4.2). However, the sitting postures in the current study differ from the two comparison studies in several ways: 1) the participants sat on a stool without a back rest in the current study, while the other two studies used standard office chairs with a back rest; 2) the participants were instructed to achieve specific postures in the current study, where the naturally selected sitting postures were documented on the other two studies. Thus, direct comparisons are not possible; however, by comparing the seated lumbar spine and pelvic angle results, certain hypotheses can be made about directed versus self-selected seating postures while sitting in a chair with a back rest. First of all, slouched sitting can happen one of two ways – through rounding of the spine, while maintaining the upper body centre of mass over the ischial tuberosities as seen in the current study (Callaghan and Dunk, 2002), or by rounding the spine and leaning against a back rest with the upper body CoM behind the ischial tuberosities (Snijders et al., 2004). The current study's ***slouched sitting*** lumbar spine angles are similar to the seated angles in the two other studies, suggesting that the lumbar spine achieved a slouched posture when leaning against a back rest. However, Beach et al. (2008) demonstrated gender differences in pelvic posture when participants sat in a chair with a back rest (Table 4.2). In fact, the female pelvic rotation in Beach's study was almost identical to the upright sitting pelvic rotation angle in the current study. This suggests that when there is a back rest present, some females may achieve a slouched lumbar spine posture without the

concurrent posterior pelvic rotation. These results were not replicated in the study presented in Chapter 6 of this thesis. Nevertheless, these comparisons suggest that the external devices used to measure lumbar spine and pelvic postures in this study are consistent across different populations and time points, and compare well to similar devices used by other experimenters.

Table 4.2: Comparison of the external lumbar (L1/Sacral) spine angles measured by accelerometers in the current study and other studies using similar technology.

	Lumbar RoM	**Pelvis RoM**	**Seated Lumbar angle (upright)**	**Seated Pelvic angle (upright)**
This study **Females** **Males**	 45.5° (16.1) 61.0° (21.3)	 59.0° (14.2) 43.4° (16.5)	***upright** 11.3° (6.9) 15.6° (14.9) ***slouched** 18.6° (10.5) 28.3° (13.3)	***upright** -5.6° (8.8) -14.5° (13.9) ***slouched** -22.7° (9.9) -29.4° (15.5)
Study #4 (Chapter 6) **Females (healthy)** **Males (healthy)**	 50.5° (11.4) 67.7° (16.2)	 61.3° (11.1) 48.0° (11.5)	***with backrest** 26.0° (9.6) 36.2° (9.5)	***with backrest** -29.7° (10.9) -36.2° (5.6)
Beach et al. (2008) (unpublished data) **Females** **Males**	 48.5° (15.3) 48.9° (14.8)		***with backrest** 31.9° (10.6) 37.4° (10.9)	***with backrest** -5.6° (12.6) -24.9° (4.4)

4.6.2 Why do accelerometers over-predict in standing and under-predict in sitting?

In the current study, the external L3/Sac angle was measured using the inclinations of accelerometers placed over the spinous process of L3 and on the sacrum at S2 (Figure 4.11). The X-ray L3/Sac angle was calculated as the angle between the L3 mid-plane line and the top of the sacrum (Figure 4.11). In theory, these two measurements are equivalent but in reality, this is not necessarily the case. First of all, there is a large difference between the lordosis measured in upright standing by each technique (external angle = 19.5° (SD 15.2); internal angle = 45.0° (SD 8.4)). As can be seen in the schematic drawing in Figure 4.11, there are likely differences between the initial alignment of both the L3 and sacral

accelerometers with their respective tracking bones. The L3 accelerometer was, on average, 5° more vertically oriented than the L3 vertebra, based on the accelerometer orientation measurements obtained directly from the X-ray image of 9 participants (see Figure 4.9). This error would lead to an initial under-estimation of the lordosis of the L3 vertebra. Because of the variable fluoroscope image orientation limitation (mentioned in Section 4.4.3), the absolute angles of the sacral accelerometer could not be compared to the sacral angle measured from X-rays. However, the posterior surface where the sacral accelerometer was mounted and the top of the sacrum measured in the X-ray image, were most likely not perpendicular to each other. Thus, it is hypothesized that the systematic differences observed between the initial internal and external sacral and L3 orientations likely contributes substantially to the difference observed between internally and externally measured upright standing lordosis.

Despite the initial sacral orientation error, it was observed that the difference between the angle of the top of the sacrum and the sacral accelerometer remained constant throughout a large range of pelvic motion (see Figure 4.7). In other words, the sacral accelerometer tracked changes in sacral (or pelvic) rotation quite well, except in the postures where pelvic motion was more extreme (i.e. full flexion, slouched sitting) and skin motion would likely come into effect. Thus, it is likely that the over-prediction in standing and under-prediction in sitting is due to errors associated with the L3 accelerometer. Inclination measurements require the assumption that the sensor lies against a flat surface and is rigidly fixed to it. Neither is the case with the spine; the spinous processes are not flat and they lie under soft tissue that will move independent of the bone underneath. There is variability in the placement of the accelerometers over the spinous processes and sacrum and this will affect how they move over the skin. This was demonstrated by the lack of a consistent skin motion pattern in the 9 participants where the L3 accelerometer was visible in the upright standing and seated images. Since obtaining a clear image of the necessary vertebrae was the primary purpose of the data collection, the X-ray imaging needed to be optimized by collimating the

X-ray beam and this sacrificed the ability to examine motion of the external devices on the skin of all participants. However, visual inspection of the full standing flexion images where the L3 accelerometer was visible revealed that superior movement of the L3 accelerometer with respect to the L3 vertebra might partially explain by the accelerometers over-predicted the internal L3/Sac angles. Skin motion could also be the culprit for the under-predicted external angles in seated postures: because it is not rigidly fixed to the spinous process, the accelerometer placed over L3 did not follow the internal rotation of the VB and instead measured the inclination of the skin surface. In fact, the L3/4 IVJ changes from lordosis to kyphosis in the half and full flexion postures (see Appendix B, Figure B1). The L3/4 IVJ was still lordotic in upright sitting, but approached kyphosis in slouched sitting (Figure B1). In the current study, the over-prediction of the internal L3/Sac angle by the accelerometers was not significant in upright sitting, and this was the only posture where the L3/4 IVJ remained lordotic. It is possible that the angular motion at L3/4, coupled with the apparent superior shift of the L3 accelerometer attached to the skin over the spine in standing flexion, could explain the errors seen in the external L3/Sac measurements in the various postures.

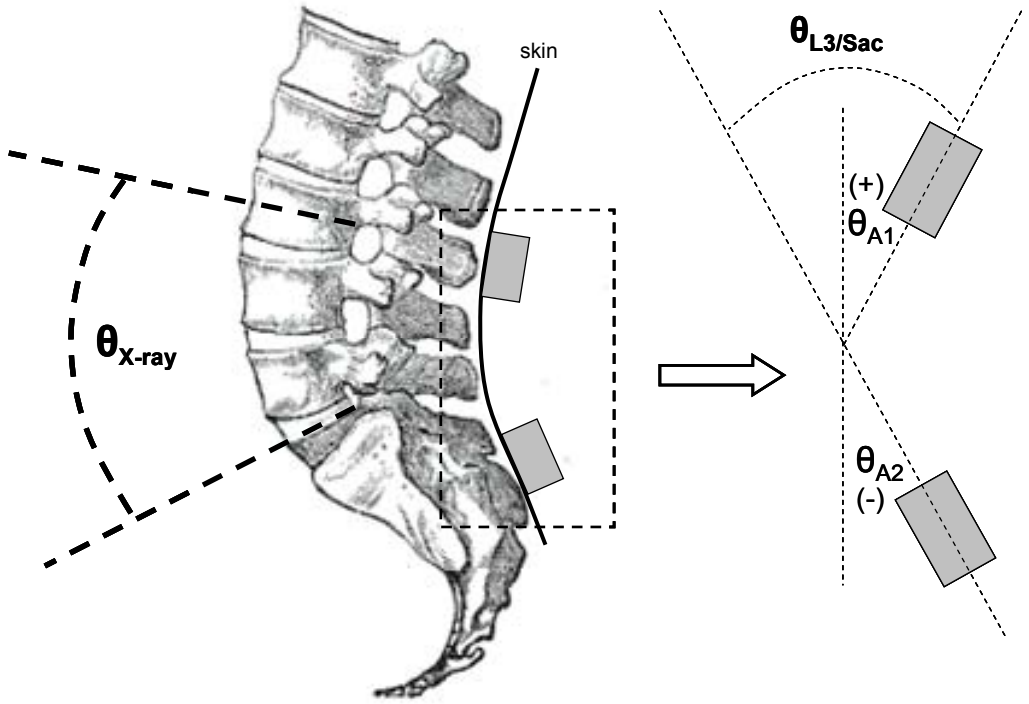


Figure 4.11: Schematic diagram of the lumbar spine and sacrum in relation to the externally placed accelerometers (grey boxes). The thick dashed lines on the spine indicate the lines of the L3 mid-plane and the top of the sacrum used to calculate the L3/Sac angle ($\theta_{X\text{-}ray}$). The external L3/Sac angle ($\theta_{L3/Sac}$) is calculated as the difference between the inclinations of the top (A1) and bottom (A2) accelerometers ($\theta_{A2} - \theta_{A1}$).

4.6.3 Why are there gender differences in externally measured angles?

Gender differences in mass distribution should be mentioned in the discussion of external measures of lumbar spine and pelvic angles. In the current study, male BMI was significantly higher than female BMI. It would have been of greater interest to know the distribution of mass (or subcutaneous fat) over the entire body, or in particular, around the pelvic/buttocks region as this could affect the movement of the externally placed accelerometer over the sacrum. Given the absences of these measures, one can only speculate about the potential effects of mass distribution on external measurements. Males tend to have a larger distribution of muscle mass in their upper body, and their higher BMI in

this study could be indicative of larger muscle mass in general. Conversely, lower BMI in females could indicate that the participants in this study were very lean with less muscle mass than their male counterparts. However, all participants tested in the current study had BMIs that were considered to be in the "normal weight" category. It is also unlikely that mass distribution would have an effect on the external measurements as there is no muscle mass over the spinous processes or posterior sacrum. These landmarks are easy to palpate in healthy, lean individuals.

In order to further explain the differences between internal and external angles measured in standing and sitting flexion, one must consider the gender differences in sacral orientation. In the 2 standing flexion postures, the female sacrum went through more anterior rotation than males, and less posterior rotation in the 2 seated postures. The externally measured L3/Sac angles in the current study also follow the trend of males having more lumbo-sacral spine flexion in both standing and sitting. However, this trend was not seen in the internal angles. The main difference between standing and sitting is the orientation of the pelvis and the angle of the hip joint (Gatton and Pearcy, 1999; Kasahara et al., 2008). It has been suggested that gender differences in hamstring flexibility plays a role in pelvic rotation (Bridger et al., 1992). As can be seen in the results from the accelerometer placed on the sacrum, pelvic orientation differs both across gender and standing or sitting posture (Figure 4.8). For males, standing flexion must be accomplished more by the lumbar spine than females because the pelvis does not rotate as much but the lumbar spine angle is greater in standing flexion. Female standing forward flexion is accomplished by more pelvic rotation. In fact, a closer look at the external L3/Sac angles revealed that 6 out of 13 females had less flexion in the "full flexion" posture than in the "half flexion" posture. Three out of 12 male participants demonstrated this phenomenon. This suggests that females tend to execute standing forward flexion by rotation at the hips (and thus the pelvis) rather than flexing the lumbar spine. A separate analysis of the same data set also demonstrated the same response in the individual IVJs of several individuals, mostly female; the lower lumbar IVJs reached a

higher amount of flexion in the half flexion posture (see Chapter 3). It is also hypothesized that the amount of internal IVJ flexion may be distributed across the entire spinal column in a gender-specific manner. The upper lumbar IVJs were not imaged in this study due to the limited field of view of the image intensifier. Male L1 and L2 vertebral bodies tend to have more anterior wedging (i.e. anterior edge is shorter than posterior edge) than females (Cheng et al., 1998; Grados et al., 1999). This may allow for males to accommodate more lumbar spine flexion in standing and a more slumped lumbar posture in sitting (Dunk and Callaghan, 2005) that was not captured by the imaging method used in the current study. Several studies have documented that the male pelvis contributes more to lumbo-sacral spine flexion in sitting which agree with the results of the current study (Dunk and Callaghan, 2005; Beach et al., 2008). The ischial tuberosities of the male pelvis are more parallel in the anterior/posterior direction allowing males to roll the pelvis back and sit in a slouched position (Tague, 1992; Correia et al., 2005). Given that females tend to have greater hamstring flexibility than males, it is possible that gender-specific seated pelvic postures are related to these inherent differences in flexibility (Bridger et al., 1992). Further study of gender-specific pelvic and upper lumbar spine morphology, surrounding anatomy and how these affect global measurements of sitting posture is required to resolve the conflicting findings between internal and external angles. Nevertheless, it is reasonable to assume that the external measurements are dependent on the ability of the pelvis to rotate and internal measurements of L3/Sac angle are not, and this is likely the reason that gender differences are observed in external, but not internal, measures of L3/Sac posture.

4.6.4 Integration of results with the literature

Other research groups have examined the relationship between internal and external spine angles. Some research suggests that skin motion may not play a large factor when relating small, flat skin markers to bony landmarks on vertebral bodies (Vergara et al., 2006; Morl and Blickhan, 2006). However, these studies have been limited by the small range of motion

between imaged postures. One research group used stick markers placed over the spinous processes of the lumbar vertebrae and pelvis and found that the external marker orientation was a much better predictor of the upper lumbar vertebrae then the pelvis or total LS angle (Chen and Lee, 1997). In a follow-up study, Chen (2000) used stick markers attached to the skin the spinous processes of L1, L3, L5 and S1 in order to develop models for predicting the inclinations of the vertebrae. Trunk angle was also measured as the line between the shoulder and the greater trochanter. Radiographic measurements were made in various amount of trunk flexion, but the lumbar spine lordosis was maintained throughout all the measurements (i.e. participants bent at the hips in order to achieve the desired trunk angle). The inclination of the L1, L2 and L3 VBs had very strong linear relationships to trunk posture, while the internal inclinations of L4, L5 and S1 demonstrated strong exponential relationships. The postures examined in Chen's studies were very highly controlled and may not be representative of the biological variation of VB motion during spine flexion (Chen and Lee, 1997; Chen, 2000). Another research group was able to accurately predict the position and orientation of each vertebral segment ranging from T12 to S1 along with the lumbar spine lordosis angle in a neutral posture using data from a back goniometer (the Lumbar Motion Monitor, Biomec Inc., Cleveland, OH, USA) (Campbell-Kyureghyan et al., 2005). They also established a relationship between the external goniometer angle change during 30° of trunk flexion and the neutral internal lordosis of the spine. Their model could predict the external shape of the spine, but not the internal VB rotations. All of the studies that have attempted to represent the internal angles of the lumbar spine have been limited by lack of generalizability because of constrained postures. Adams et al. (1986) measured lumbar angles with inclinometers and compared them with angles measure from X-rays and showed a mean absolute difference of 4.3° (SD 4.2) between internal and external measurements, where the inclinometers generally under-predicted the internal spine angle. The X-ray/inclinometer comparisons were only in seated postures, which agree with the sitting results in this study. However, the current study showed a mean absolute difference between seated internal and external angles of 13.0° (SD 8.4). Part of the difference between the

Adams study and the current one is that the sacral accelerometer was placed lower on the sacrum which could help explain the larger mean absolute difference in the current study.

4.6.5 Methodological Considerations: Lumbar Normalization Procedures

Several pieces of evidence from the current study and Study #1 (Chapter 3) point to the fact that standing forward flexion may not be the best way to elicit maximum lumbar RoM. First, up to 50% of the participants in Study #1 (Chapter 3) did not achieve the greatest flexion angle at a given IVJ in standing full flexion; instead, a standing "half" flexion characterized by a rounded spine elicited greater IVJ flexion angles. Second, when accelerometers are used as inclinometers to measure lumbar spine and pelvic postures, the response of this instrumentation appears to be a function of the inclination of the pelvis. In other words, using a standing full flexion posture to represent maximum lumbar flexion may actually inflate this value because of the larger anterior rotation of the pelvis. It is necessary to be aware of this issue since normalizing seated spine angles to full standing flexion will likely affect the estimate of %RoM in seated postures. In other words, if accelerometers over-predict lumbar spine RoM in full standing flexion, then normalizing seated angles will lead to a conservative estimate, or reduce the sensitivity of a change in spine angle detected in setting. Thus, any changes detected in sitting may, in reality, be larger when considering changes in angle at the IVJ level.

Several recommendations are made regarding lumbar spine normalization procedures:

1) Future research is needed in order to determine the most appropriate means of eliciting full lumbar flexion. Suggested methods include: a) seated full flexion (with the legs flat on the floor) in order to ensure that the pelvis is fully rotated in the posterior direction (Adams et al., 1986); b) passive RoM measured on a frictionless table that keeps the pelvis and legs in a fixed position while allowing the upper body to move through its full range of flexion (Parkinson et al., 2004; Beach et al., 2005).

This method has been used to quantify passive stiffness of the lumbar spine as well as eliminates gravity, and the effect of the hamstrings on lumbar/pelvic rotation.

2) When conducting seated research, seated full flexion may be a more appropriate means to elicit full flexion especially when using external measurement devices that are sensitive to gravity.

3) When using electronic measurement devices such as accelerometers, it is suggested that devices be located over the L1 and L5 spinous processes and a separate sensor be placed over the sacrum. This would enable two separate measurements – lumbar spine angle and pelvic inclination. In the set-up that was used in the studies described in this thesis, lumbar spine angle was not independent of pelvic inclination, thus leading to the over-estimation issues discussed above.

4.7 Conclusions

Based on the results of this study, it is reasonable to state that externally placed devices to measure lumbar spine angles do not accurately represent the internal rotations of the spine. The accelerometers used in this study over-predicted the L3/Sac IVJ angles in standing postures and under-predicted them in sitting. However, the external device was sensitive to changes in angle, provided the postural change occurs between standing postures (e.g., half to full flexion), or between seated postures (e.g., upright to slouched sitting). The main use of the accelerometers tested in the current study is to examine lumbar spine posture while sitting. The externally measured seated angles in this study are consistent with those measured with similar technology in other studies. Furthermore, accelerometers are sensitive to changes in seated lumbar spine posture where the motions may be small enough to limit erroneous results caused by skin motion and excessive pelvis or VB rotation. However, methods of normalizing angle data lead to a necessity for interpreting results with caution, and provide direction for future work to improve data normalization methods.

Chapter 5:
Individual differences in lumbar vertebral body and pelvic movement patterns during a seated flexion motion

5.1 Synopsis

Background: In standing, there is a "normal" dominant sequence of rotation of vertebral bodies where the forward flexion motion is initiated by the L1 vertebra, proceeds in a "top-down" manner and finishes with pelvic rotation. However, it is unknown whether it is the rotation of the pelvis or the muscles of the lumbar spine that drive the slouching motion. This would be important to know because identifying different movement strategies may be useful in assessing normal and abnormal or pathological spine motion in sitting. Thus, the aim of the current study was to investigate the dynamic rotational kinematics of the IVJs of the LS spine and pelvis in a seated slouching motion in order to determine if a dominant sequence of vertebral motion exists. Gender-specific kinematics were also investigated.

Methods: Dynamic sagittal fluoroscopic videos of the lower lumbar-sacral region were obtained from 19 participants (9 males, 10 females) while they flexed their spines from an upright to a slouched seated posture. Pelvic motion was simultaneously measured using an accelerometer placed on the sacrum. Vertebral body motion was tracked in the fluoroscopic video sequence using a frame-by-frame image registration method.

Findings: There was no consistent order of vertebral body movement initiation and none of the participants initiated the movement with their pelvis. Three vertebral body motion patterns were identified. Group 1 (16% of total participants) completed the motion with all vertebral bodies rotating "en bloc" and had very little L3/4 and L4/5 IVJ flexion (0.3° (SD 2.6) and 1.3° (SD 2.5), respectively). Group 2 participants (32%) started the motion with all vertebral bodies rotating at the same rate, but diverging towards the end of the motion. This motion pattern was also characterized by small IVJ angles (L3/4 = 3.1° (SD 1.3); L4/5 = 1.6°

(SD 2.1)). The vertebral bodies of Group 3 participants (52%) diverged in their rotation angles from the onset of motion and the IVJ angles were high (L3/4 = 7.2° (SD 4.2); L4/5 = 7.8° (SD 4.0)). Gender differences were noted in pelvic kinematics where females had more posterior pelvic rotation throughout the slouching motion.

Interpretation: Although it was initially hypothesized that the slouched sitting motion would be initiated by the pelvis, this study demonstrated that this is not the case. Joint positioning will alter the load on the passive tissues surrounding the joint, the force-generating capacity of individual muscles and the demands on the neuromuscular control system. A disordered sequence of vertebral motion the different motion patterns observed may indicate that different joints approach their end range before the completion of the slouching movement.

5.2 Introduction

Certain lumbar spine and pelvic movement patterns may predispose individuals to development of low back pain (LBP) or can be used to identify "normal" and "pathological" patterns. In standing, there is a "normal" dominant sequence of rotation of vertebral bodies where the forward flexion motion is initiated by the L1 vertebra, proceeds in a "top-down" manner and finishes with pelvic rotation (Kanayama et al., 1996; Okawa et al., 1998; Harada et al., 2000). In a seated posture, the pelvis acts as the base of support for the spine and its motion is constrained by its association with the seat pan (Andersson et al., 1979; Pope et al., 2002; Makhsous et al., 2003; Snijders et al., 2004; Lin et al., 2006). Upper body weight is carried mainly by the ischial tuberosities and it is suggested that these bony structures act as a fulcrum for movement of the upper body centre of mass (Makhsous et al., 2003; Snijders et al., 2004; Dunk and Callaghan, 2005; Lin et al., 2006). The transition from an upright sitting posture to a slouched posture involves transition from one stable position to another and anything in between could be considered unstable (Snijders et al., 2004). Previous research suggests that the pelvis may contribute the most to lumbar spine flexion in slouched sitting motions (Snijders et al., 2004; Kasahara et al., 2008). Furthermore, the lower three lumbar

IVJs approach their maximum RoM in slouched sitting (see Study #1, Chapter 3). However, it is unknown whether it is the rotation of the pelvis or the muscles of the lumbar spine that drive the slouching motion. This would be important to know because identifying different movement strategies may be useful in assessing normal and abnormal or pathological spine motion in sitting. Thus, the aim of the current study was to investigate the dynamic rotational kinematics of the IVJs of the LS spine and pelvis in a seated slouching motion in order to determine if a dominant sequence of vertebral motion exists.

Several studies have used videofluoroscopy to determine the sequence of rotation of the individual joints during standing lumbar flexion and extension. Kanayama et al. (1996) determined that lower lumbar and lumbosacral flexion (L3 to S1) was initiated at the L3/4 segment and proceeded in a stepwise or sequential manner with intersegmental angular motion lags. A later study from the same lab demonstrated similar results, also noting that the angular velocity at the onset of motion increased as the segmental level descended (Harada et al., 2000). Similarly in another study, a sequential flexion pattern starting with L2 was observed in 46% of a healthy population, with 31% showing a simultaneous motion pattern and 23% showing a disordered pattern that was thought to be abnormal (Okawa et al., 1998). Wong et al. (2004) assessed spinal motion for common magnitudes of motion (analyses of IVJ rotations were performed between 40° of flexion and 10° of extension, in 10° intervals, with the pelvis free). They noted that at most of the common assessment points of RoM, the IVJ rotation angle decreased from L1/L2 to L5/S1, suggesting that standing lumbar flexion is initiated from the upper segments to lower segments.

In upright sitting, there is evidence of a "bottom-up" flexion pattern exhibited by the lower 3 lumbar IVJs (see Study #1, Chapter 3). In this previous study, L5/S1 achieved more than 60% of its total RoM in an upright sitting posture and the next two adjacent IVJs (L4/5 and L3/4) were at decreasing values of their total RoM. However, this previous study focused on

a static analysis of a dynamic phenomenon and could be missing valuable information about altered movement patterns. Traditional variables attained from static radiographs, including global and segmental ranges of motion, may not differ between asymptomatic and LBP groups (Dvorak et al., 1991; Teyhen et al., 2007). It has been demonstrated that standing dynamic mid-motion characteristics of lumbar IVJ RoMs are necessary to separate individuals with LBP from healthy controls (Kanayama et al., 1996; Okawa et al., 1998; Harada et al., 2000; Teyhen et al., 2007). Previous research has quantified rhythms, delays and alterations in standing movement patterns of LBP patients using videofluoroscopy. For example, patients with degenerative spondylolisthesis have spine flexion/extension motions patterns characterized by disordered initiation of vertebral body flexion and prolonged deflection of the affected joint in extension (Okawa et al., 1998). Furthermore, differences in the rates of angular and linear IVJ displacements during the onset of flexion and extension were used to distinguish between a LBP and an asymptomatic group (Teyhen et al., 2007). Therefore, videofluoroscopy overcomes the limitations of static radiographs by capturing relevant information only observed during movement. Specifically, the quantification of seated spine movement patterns from dynamic imaging techniques could be useful in distinguishing between those who develop LBP and those who do not during prolonged sitting.

5.2.1 Purpose

The purpose of the current study was to assess the vertebral rotations of the lower 3 lumbar spine segments throughout a seated slouching motion using videofluoroscopy. The main goal was to examine the initiation of the slouching motion and determine the sequence of rotation of each of the lower three lumbar vertebrae. Differences between males and females were also examined since gender-specific pelvis postures have been observed in sitting (Dunk and Callaghan, 2005).

5.2.2 Hypotheses

1) In the seated slouching motion, the pelvis and lower lumbar spine will exhibit a bottom-up sequence of rotation: the motion will be initiated by the pelvis followed by the lumbar vertebrae from L5 to L3.
2) The L4/5 IVJ will achieve a greater amount of flexion than the L3/4 IVJ at the end of the slouching motion.

5.3 Methods

5.3.1 Overview of Experiment

Dynamic sagittal fluoroscopic videos of the lumbar-sacral region ranging from the top of the sacrum to the top of the third lumbar vertebra were obtained while participants moved their spine from an upright to a slouched seated posture.

5.3.2 Participants

A total of twenty-two participants, 9 males (mean age = 29.3 (SD 8.8) years; mean height = 1.78 (SD 0.05) m; mean mass = 78.5 (SD 8.0) kg) and 10 females (mean age = 27.4 (SD 7.5) years; mean height = 1.65 (SD 0.06) m; mean mass = 59.8 (SD 7.1) kg), were recruited from a university population. A justification for the chosen sample size can be found in Appendix A. All participants were free of low back pain for 12 months prior to the testing period and were screened to exclude any or all of the following: 1) individuals who were or thought they might be pregnant; 2) individuals who were exposed to radiation as a part of their job, and; 3) individuals who had had two or more X-rays of the trunk and/or pelvis or other high-exposure radiological procedures (i.e. CT, bone mineral density testing or radiotherapy) due to injury or illness. The study protocol received approval from the University of Waterloo and the University of Western Ontario Offices of Research and participants gave informed consent before testing began.

5.3.3 Instrumentation

The video fluoroscope (Siremobil Compact (L) Mobile X-ray Image Intensifier system, Siemens Medical Solutions USA Inc., Malvern, PA) was equipped with a 9-inch image intensifier and a source to image intensifier distance of 100cm. The average X-ray technique factors were 3.4 mA and 107 kVp and the total imaging time did not exceed 20 seconds. The total effective dose was less than 6 mSv, which was less than that of a typical CT examination (FDA - http://www.fda.gov/cdrh/ct/risks.html). The FDA also estimates that 10 mSv of X-ray exposure increases the risk of fatal cancer by 0.05%, which is considered insignificant. The video feed from the fluoroscope was captured at 30 Hz directly to a computer hard drive using a digital video capture device and software (DVD Xpress DX2, ADS Technologies, Cerritos, CA). External spine and pelvic angles were measured using accelerometers placed over the spinous process of the L3 and over the sacrum at the level of S2. The accelerometer signals were digitized at a rate of 128 Hz using a 12-bit (NI 6024E, National Instruments Corporation, Austin, TX, USA) analog-to-digital conversion system and synchronized in time with the fluoroscopy video using an electromechanical contact switch placed on the fluoroscope control foot pedal. Accelerometer outputs were converted to degrees (as described in Chapter 4, Section 4.3.2) and the L3/Sac angle was calculated as the difference in inclinations between the L3 and sacral sensors. The inclination of the sacral sensor was considered to be representative of anterior/posterior pelvic tilt.

5.3.4 Data Collection and Analysis

The participants were positioned with their right side as close to the image intensifier as possible and an experienced radiology technologist adjusted the location of the fluoroscope with respect to each participant so that the top of the sacrum would be visible in the lower part of the image. During each trial, participants were asked to place their hands on their shoulders in order to prevent the arms from obscuring the spine in the X-ray image. Spinal motion was described as a rounding of the lumbar spine to a “slouched” seated position. The lower spine was imaged for approximately 5 seconds while the participant started in an

upright seated position, achieved the slouched posture and returned to upright. In some cases, the end range of the slouching motion was limited in order to keep the lower 3 lumbar vertebrae within the fluoroscope field of view. Since all individuals participating in the current study also had static X-ray images of their upright and slouched sitting postures (as presented in Study #2, Chapter 4), it was possible to examine the effect of interfering with the slouching movement by comparing the static postures with the dynamic ones. To do this, the external L3/Sac and pelvic angles obtained in the initial upright sitting posture and the maximum slouched posture in the dynamic trial were determined and compared with the corresponding external angles in the static upright and slouched postures. Preliminary statistical analyses were performed using paired t-tests where the "dynamic" posture was compared with the corresponding "static" posture. Results revealed that there were no significant differences between any static/dynamic posture pairs (Table 5.1).

Table 5.1: External L3/Sac and pelvic angles, measured by accelerometers, obtained during static upright and slouched sitting, compared with upright and slouched sitting postures achieved during the dynamic slouching trials. Angles are expressed as a change from upright standing.

	Upright		Slouched	
	Static	**Dynamic**	**Static**	**Dynamic**
External L3/Sac	12.6° (9.6)	13.9° (10.1)	23.5° (10.4)	22.6° (10.3)
	$P = 0.407$		$P = 0.863$	
External pelvis	-10.1° (10.6)	-11.0° (10.2)	-26.0° (12.7)	-22.5° (8.7)
	$P = 0.126$		$P = 0.149$	

Vertebral body motion was tracked in the fluoroscopic video sequence using a frame-by-frame image registration (or rigid body alignment) method developed by Dr. Jeff Orchard (School of Computer Science, University of Waterloo). One motion trial was analyzed per participant because quality of the fluoroscopic video sequence (e.g., the spine remained within the field of view, frame-to-frame contrast homogeneity) was usually limited to one

good trial. Due to equipment limitations (i.e. the small field of view of the fluoroscope), it was very difficult to obtain a trial where the relevant vertebrae were in view at both the beginning and end of the motion, which is necessary for the registration method where a template image is used in the tracking process (explained in more detail in the Steps outlined below). Therefore, only the flexion (or slouching) phase of the movement was analysed. The end of the flexion phase was identified as the frame before the spine motion reversed directions and the fluoroscopic video sequence was clipped to remove frames after this point. The registration algorithm is an intensity-based least-squares optimization; the method rotates and translates a "floating" image with respect to a "fixed" template image in order to solve the intensity-based problem by seeking the motion parameters that minimize the sum of squared residuals (or pixel-by-pixel difference) between the two images (Orchard, 2003). Without loss of generality, the first image is assumed to be fixed and used as the template for registration. This method takes care of fine-tuning adjustments efficiently, and works well if the images to be registered are almost registered to begin with.

Steps of the registration method (Figure 5.1):

1) One or multiple region(s) of interest (ROI) were specified by the user – areas of the vertebrae that were well-defined (e.g., vertebral body, the pedicle that attaches the posterior elements to the vertebral body, the most ventral part of the spinous process) and remained within the field of view throughout the video sequence were included in the ROI.
2) The ROI was implemented using a weighting image that excluded the influence of pixels outside the ROI by assigning them a weight of zero. Thus, in the weighted least-squares cost function only pixels inside the ROI were used in the registration.
3) Images were enhanced using a Difference-of-Gaussians filter in order to increase the visibility of edges and other details present.
4) The filtered floating image was registered to the filtered template (fixed) image using the following steps:

a. The rigid-body transformations were linearly approximated using the partial derivatives of each pixel with respect to each of the motion parameters (x translation, y translation and rotation about the z-axis).
b. The optimal motion parameters were determined by minimizing the sum of squared residuals (or pixel-by-pixel difference) between the fixed image and the floating image. The resulting rotation and translations that were applied to the image to align it with the fixed image were based only on the user-defined ROI.
c. In order to increase the signal-to-noise ratio, the last 3 registered frames were averaged and used as a template to which the next frame was registered. Averaging the frames tended to improve the quality of the template image by canceling out the noise, since noise should not be correlated from frame to frame.

5) The motion parameters were fine-tuned by creating an average template using all of the registered images and then registering each image to this average template.
6) Image registration (steps 1 – 6) was repeated for the L3, L4 and L5 VBs.

The performance of the image registration process could be verified by applying motion parameters to the initial video sequence. This process essentially changed the frame of reference from the fluoroscope field of view to the VB being tracked. This resulted in a corrected video where the tracked VB remained fixed and all other image details rotated around it. The VB could be visually assessed to ensure that it remained stationary, thus verifying the tracking performance of the program.

To further verify the performance of the registration program, two different analyses were performed. A phantom spine model was imaged and the outputs from the registration program were compared with two other methods of quantifying angles (manual digitization, and direct measurement of the phantom angular displacement with an electrogoniometer).

Furthermore, 7 different trials were downsampled and hand-digitized and the VB angular motions were compared with the registration program outputs. Theses processes are outlined in Appendix D. Briefly, the phantom model was rotated through the fluoroscope field of view and "vertebral body" angular displacements were determined using manually digitized landmark co-ordinates, the output of the electrogoniometer and the rotations obtained from the image registration program. The registration method was highly linearly correlated with the two other measurements ($r > 0.990$) and had mean absolute errors (calculated across the entire motion trial) of less than 2°. Additionally, the mean absolute difference between the hand-digitized trials and the registration output was less than 2° (range = 0.38 – 2.11°) for all VBs and the correlations were greater than 0.9 for all trials. *In vitro* studies of spine joint motion could be used to put the mean absolute difference in context: assuming that one VB remains relatively fixed while the adjacent VB moves with respect to the fixed VB, values for total VB RoM in the literature range from 13° to 19° (Adams et al., 1994; Wilke et al., 1997). In the worst case scenario, a mean absolute difference of 2.11° could represent 16% error in VB motion tracking (% error = (2.11°/13°)*100). In most cases, the registration program over-predicted the rotation of the VB but the high correlations indicate that although the absolute angle values differed between the two tracking methods, they both followed the movement of the VB in the same way. (See Appendix D for complete details).

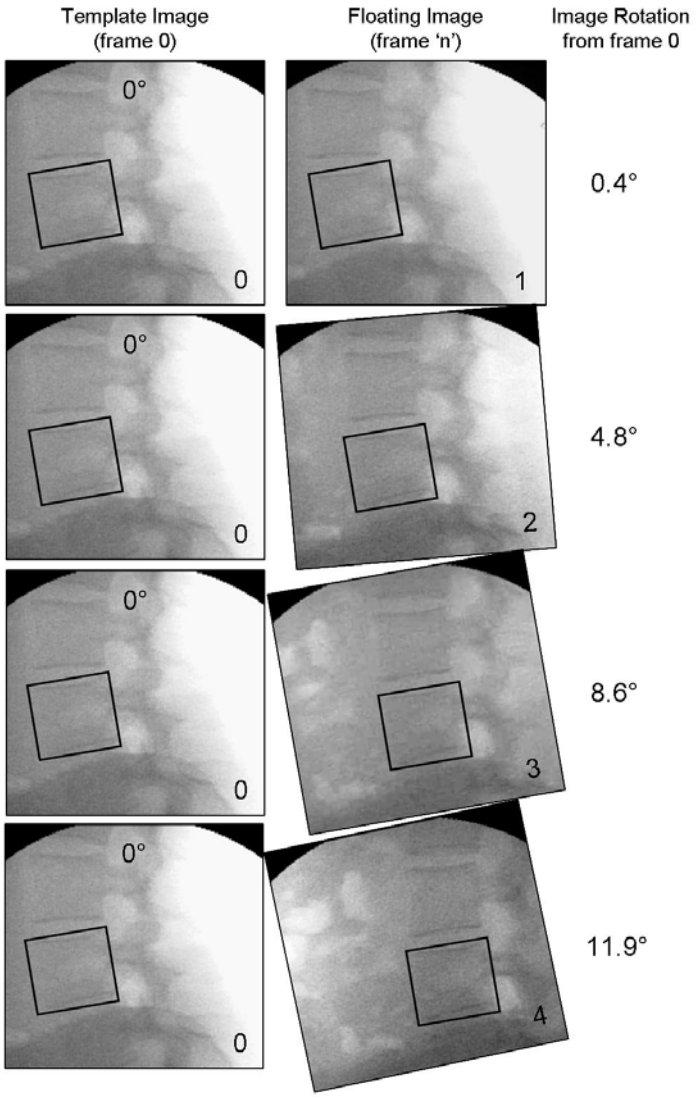


Figure 5.1: Representative fluoroscopic image sequence demonstrating the image registration process (downsampled from 30Hz to 2Hz). The region of interest (ROI) around the L4 vertebral body is defined in the template image (black square in all images). The floating image is registered to the template image by applying the least-squares cost function only to pixels inside the ROI. The resulting rotations are listed in the right-hand column.

5.3.5 Data reduction and presentation

Since the registration program could not reliably track the sacrum, the time series of pelvic inclination measured by the accelerometer was used in the analyses. As was demonstrated in a previous study in this thesis, the sacral accelerometer tracked changes in sacral orientation quite well (see Chapter 4, Figure 4.7). The initiation of movement was determined using a method similar to the method used in determining the initiation of gait (Winter et al., 1974): the first second of the trial consisted of "quiet sitting" in the upright posture. The mean and standard deviation (SD) of this first second was computed and a segment (VB or pelvis) was considered to be in motion once the angular displacement exceeded ±2SD. Simultaneous initiation of movement was considered to occur if a segment started to move within one frame (or 0.03s) of the initiation of another segment. The frame in which the first segment initiated movement was labeled as T_0 for that trial and the corresponding angle values for the pelvis and 3 lower VBs were expressed as a change from this time point. In other words, the angle at T_0 was subtracted from the rest of the time series. The angular velocities of L3, L4, and L5 and pelvis were calculated using three-point differentiation. The rotational motion profiles for the 3 VBs and pelvic inclination profile were resampled using linear interpolation onto a common time axis so that the time scale reflected 0 to 100% of the cycle. This was done so that the variability in timing between subjects could be eliminated. The motion profiles were examined in order to group the participants based on the patterns of segment motions. Three motion profile groups were identified and will be presented and further discussed in the Results section.

The angle between the L3 and L4 VBs, or the L3/4 intervertebral joint (IVJ) angle, was calculated as the difference between the L3 and L4 VB rotations. The L4/5 IVJ angle was calculated in a similar manner, using the L4 and L5 rotations. The external L3/Sac angle (measured by accelerometers) was used as a measure of the "global" spine range of motion (RoM) and the L3/Sac time series was normalized with respect to the local maximum relative

angle achieved during the slouching motion. The L3/4, L4/5 and pelvic angles were plotted with respect to the normalized L3/Sac RoM and the angles at 10% increments of the L3/Sac RoM were obtained (Figure 5.5 in the Results section). The maximum IVJ angles as well as the maximum externally measured L3/Sac and pelvic angles were identified and mean values were determined for each of the three motion profile groups.

5.3.6 Statistical Analyses

One-way analyses of variance (ANOVA) were used to examine significant differences between motion profile groups for the maximum IVJ angles and externally measured L3/Sac and pelvic angles. In order to determine if knowing the final relative external L3/Sac angle would be indicative of the VB motion patterns, all L3/Sac values were ranked and a Kruskal-Wallis one-way ANOVA was performed. Time-varying gender differences were examined using a 2-way ANOVA (gender, time) with one repeated measure (time). Pairwise comparisons were examined using Tukey's post-hoc test and the alpha-level was set at 0.05.

5.4 Results

5.4.1 Motion profile groups – Initiation of movement

There was no consistent order of segment movement initiation (Table 5.2). The pelvis was the last segment to move for 13 participants and this case included all of the male participants (Table 5.2). Only one participant (F7) initiated the slouching movement with their pelvis. Two participants (M5, M9) exhibited a top-down sequence of rotation ending with the pelvis, two more (F8, M4) demonstrated a bottom-up sequence starting with L5 and all three VBs started to move simultaneously for 1 participant (M1) of the participants (Table 5.2). Eight out of 19 participants (5 males, 3 females) had a disordered sequence of VB movement initiation ending with the pelvis, while five participants (all female) had a completely disordered sequence.

Three VB motion pattern groups were identified based on how the three VBs moved with respect to each other:

Group 1: "En bloc" – all VBs moved at the same rate throughout the slouching motion (Figure 5.2).

Group 2: "Divergent at end" – all VBs start rotating at same rate and diverge towards the end of the motion (Figure 5.3).

Group 3: "Divergent from start" – all VBs rotate at different rates throughout the slouching motion (Figure 5.4).

The most commonly observed VB motion pattern was Group 3, where all VBs rotated at different rates, demonstrated by 10 out of 19 (53%) participants (Table 5.2).

Table 5.2: Order of segment movement initiation and vertebral body (VB) motion pattern group for all participants. When two or three segments are listed in one cell, this means that movement was initiated simultaneously (within 0.3 seconds).

Participant	Order of movement initiation			
	1	**2**	**3**	**4**
VB motion pattern Group 1: En Block				
F5	L3	L5	L4	pelvis
F6	L5	L4	pelvis	L3
F8	L5	L4	L3	pelvis
VB motion pattern Group 2: Divergent @ end				
F3	L3	pelvis	L4	L5
F7	pelvis/L4	pelvis/L4	L5/L3	L5/L3
M2	L4	L5	L3/pelvis	L3/pelvis
M3	L4	L5/L3	L5/L3	pelvis
M5	L3/L4	L3/L4	L5	pelvis
M6	L3/L5	L3/L5	L4	pelvis
VB motion pattern Group 3: Divergent @ start				
F1	L5	pelvis	L3	L4
F2	L5	pelvis/L3	pelvis/L3	L4
F4	L4	L5	L3	pelvis
F9	L3	L4	pelvis	L5
F10	L5	L3	L4/pelvis	L4/pelvis
M1	L3/L4/L5	L3/L4/L5	L3/L4/L5	pelvis
M4	L5	L4	L3	pelvis
M7	L4	L5	L3	pelvis
M8	L4	L5	L3	pelvis
M9	L3	L4/L5	L4/L5	pelvis

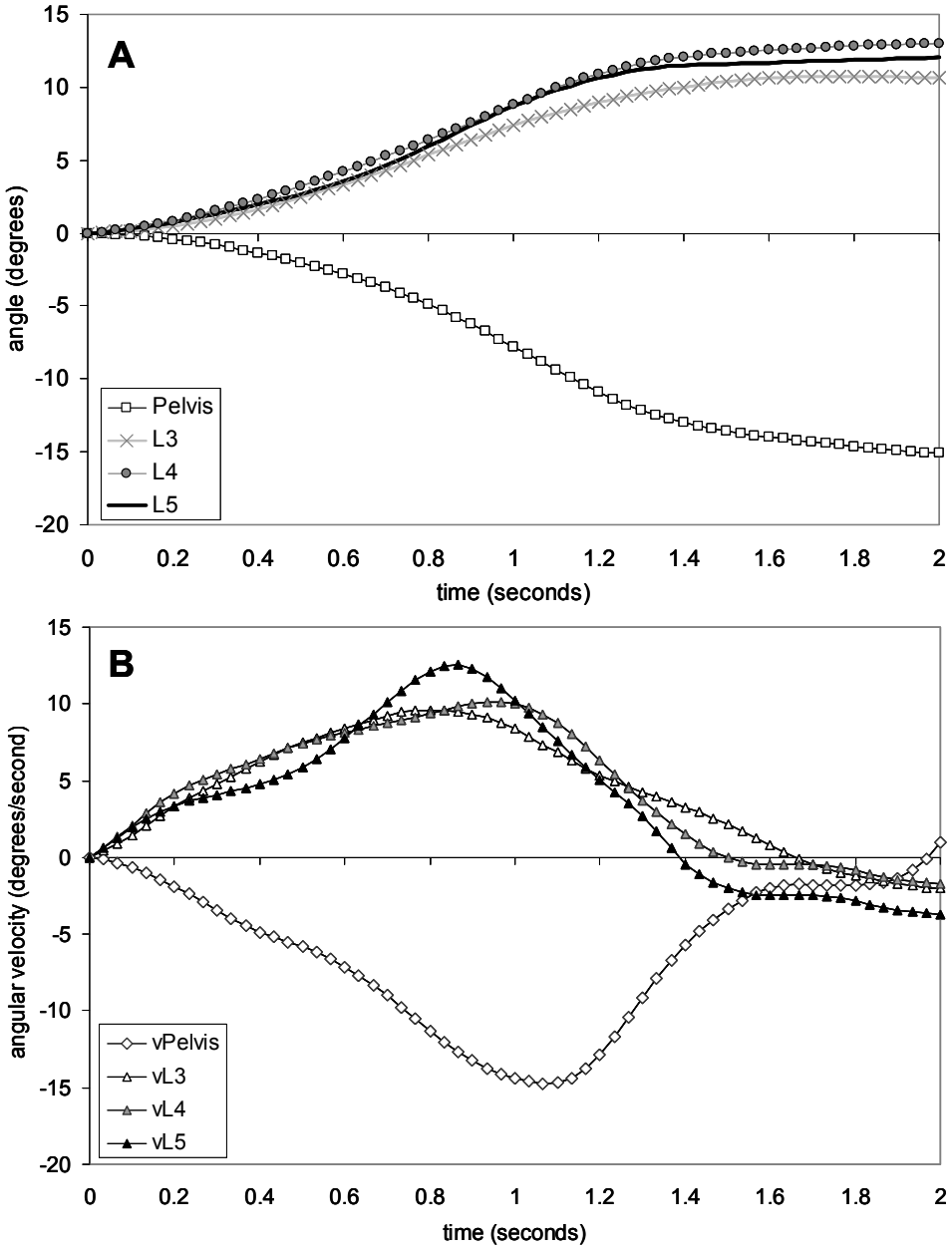


Figure 5.2: Example of Group 1 - the "en bloc" motion pattern. (A) Angular rotation and (B) angular velocity of the pelvis and three vertebral bodies (L3, L4, L5). All three vertebral bodies rotated at approximately the same rate.

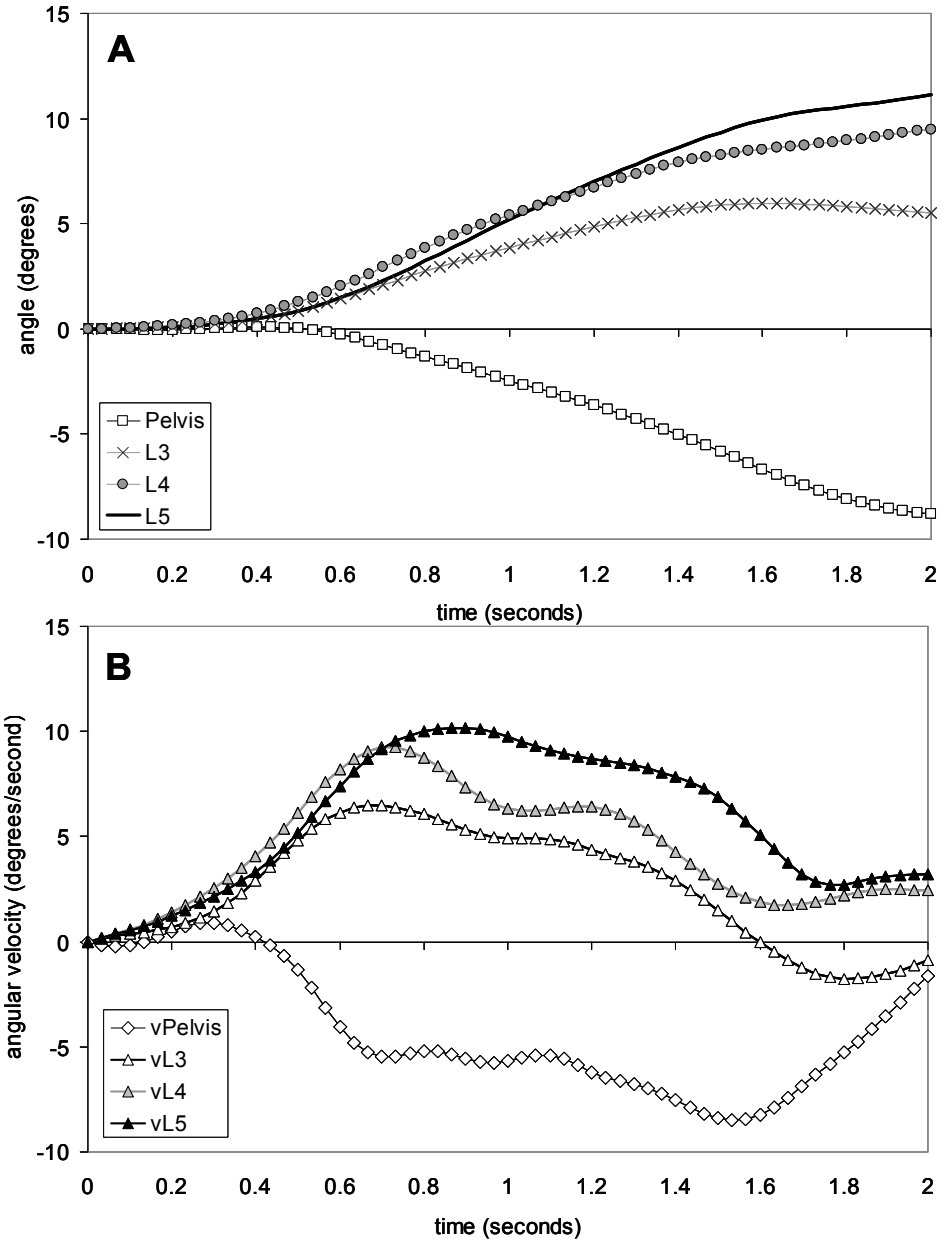


Figure 5.3: Example of Group 2 - the "divergent at end" motion pattern. (A) Angular rotation and (B) angular velocity of the pelvis and three vertebral bodies (L3, L4, L5). All three vertebral bodies start rotating at approximately the same rate and then diverge towards the end of the motion.

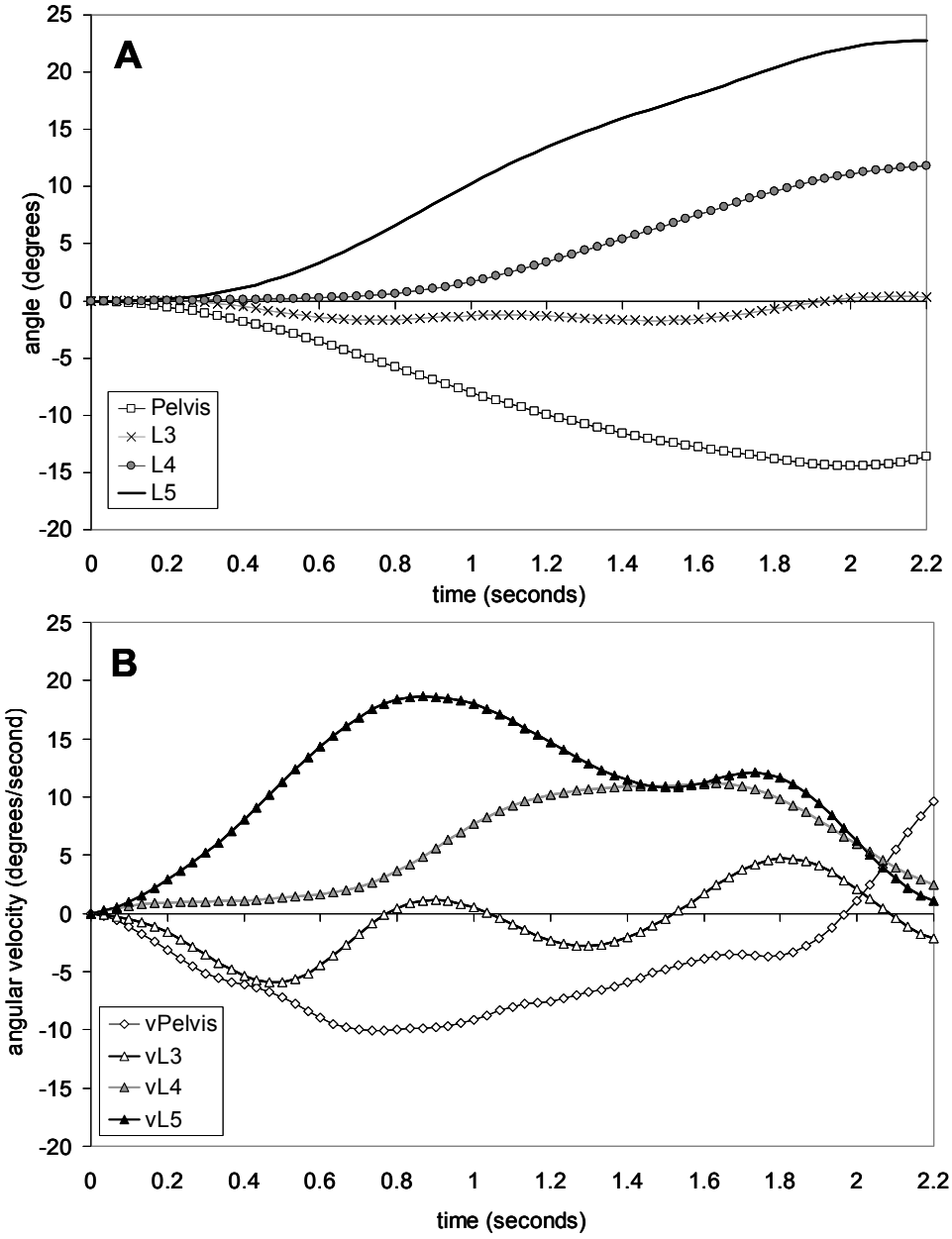


Figure 5.4: Example of Group 3 - the "divergent from start" motion pattern. (A) Angular rotation and (B) angular velocity of the pelvis and three vertebral bodies (L3, L4, L5). All three vertebral bodies rotate at different rates from the initiation of the motion

5.4.2 Intervertebral joint kinematics

The participants in motion profile Group 3 (VBs divergent from start) demonstrated the largest L3/4 and L4/5 angular changes throughout the slouching motion (Table 5.3). This group also exhibited the largest relative L3/Sac angle as measured externally by accelerometers (L3/Sac angle = 11.2° (SD 7.1); Table 5.3). The L3/Sac relative angle for Group 2 (VBs divergent at end) was greater than Group 1 (VBs moved "en bloc") but this difference was not significant (Group 2 L3/Sac angle = 7.5° (SD 5.0); Group 1 L3/Sac angle = 4.0° (SD 3.1)). During the slouching motion, the L3/4 and L4/5 IVJ angles did not change much for Group 1 and the L3/Sac angle appeared to be achieved mostly by the rotation of the pelvis (Figure 5.5A). Group 2 demonstrated increasing L3/4 and L4/5 IVJ angles over the L3/Sac RoM; however, the IVJ angles were, on average, less than 2° and did not start to increase until after 30% of the L3/Sac RoM (Figure 5.5B). In contrast, the L3/4 and L4/5 IVJ angles of Group 3 increased from the beginning of the L3/Sac RoM and continued to increase until 100% of L3/Sac RoM (Figure 5.5C). It is interesting to note that for all three groups, the slope of the L4/5 IVJ angle line (or rate of attainment) was less than L3/4 towards the end of the motion (between 90 and 100%) and the slope became negative for Group 1 and 2 (Figure 5.5). This supports the idea that the L4/5 IVJ approached a local maximum of relative rotation towards the end of the motion. In contrast, the L3/4 IVJ angle was still increasing towards the end of the motion.

The results of the Kruskal-Wallis ANOVA revealed that the mean rankings for the three groups were not significantly different (K-W = 3.928, df = 2, $P = 0.14$): 5.3 (SD 4.0) for Group 1, 8.7 (SD 6.3) for Group 2 and 12.2 (SD 4.8) for Group 3. However, assuming even group sizes, any difference between mean ranks was considered significant if it was larger than 3.25. Therefore, there was a trend in the ranking indicating that knowing the final relative external L3/Sac angle could be a useful way to indicate the VB motion pattern. The results of the current study are inconclusive because of the small sample sizes.

Table 5.3: Mean (and standard deviation) maximum angles reached by the L3/4 and L4/5 intervertebral joints, and maximum externally measured L3/Sac and pelvic angle. The asterisk (*) indicates that the Group 3 value is significantly different from both Groups 1 and 2 ($P < 0.05$). The number sign (#) indicates that Group 3 is significantly different from Group 1 only ($P < 0.05$).

	Maximum angle reached (degrees)		Max ext L3/Sac angle (degrees)	Max ext pelvic angle (degrees)
	L3/4	L4/5		
Group 1: Close to parallel (n = 3)	0.3 (2.6)	1.3 (2.5)	4.0 (3.1)	11.8 (5.3)
Group 2: Divergent at end (n = 6)	3.1 (1.3)	1.6 (2.1)	7.5 (5.0)	13.6 (5.9)
Group 3: Divergent at start (n = 10)	7.2 (4.2) *	7.8 (4.0) *	11.2 (7.1) #	10.2 (6.2)

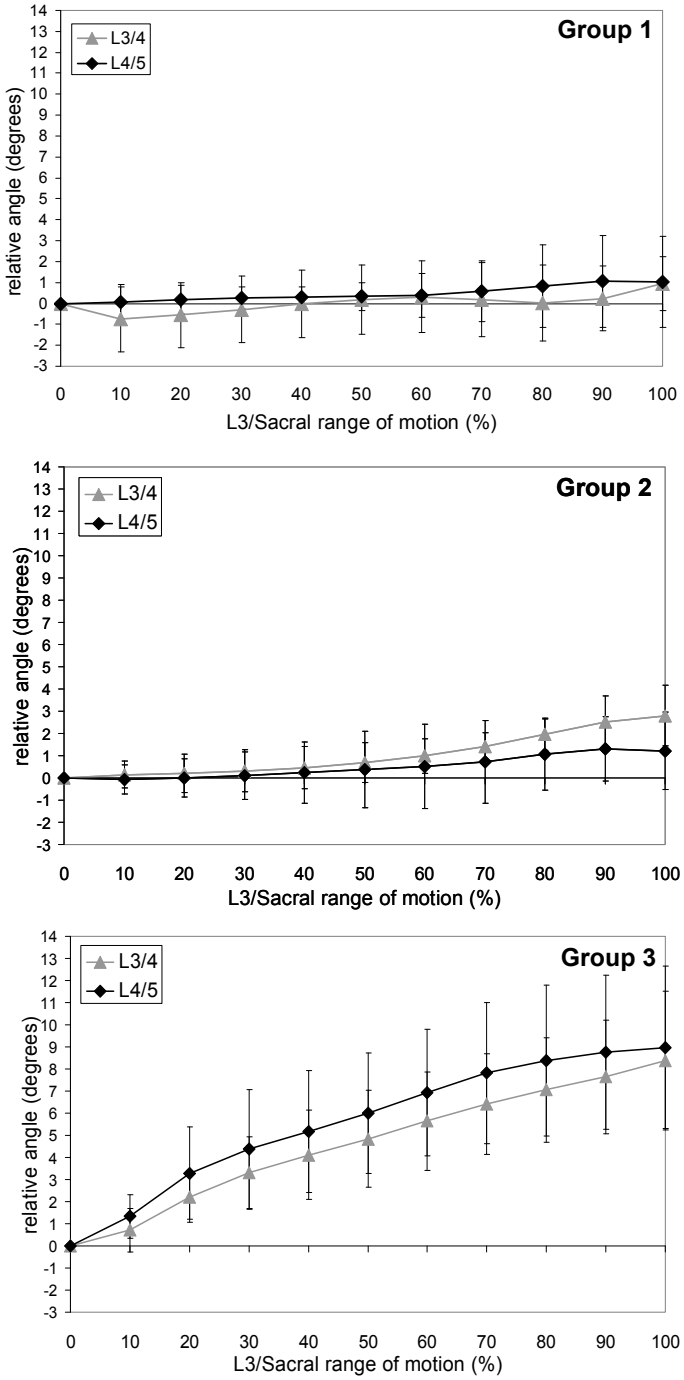


Figure 5.5: Relation between the relative L3/4 and L4/5 intervertebral joint and total L3/Sacral range of motion during the slouching movement. Each curve represents the mean values for the different vertebral body (VB) motion profile groups: Group 1 (en bloc), Group 2 (divergent at end), Group 3 (divergent at start).

5.4.3 Gender differences summary

The "en bloc" motion profile (Group 1) was comprised of all female participants. Males and females were evenly distributed between the other two motion profile groups (Table 5.2). The pelvis was the last segment to move for all of the male participants while only 40% of the female participants completed the slouching motion with their pelvis (Table 5.2). Pelvic kinematics throughout the slouching motion differed significantly when grouped by gender; there was a significant gender*time interaction ($P = 0.02$). Females rotated their pelvis at a greater rate throughout the entire slouching motion (Figure 5.6) and they had more relative posterior pelvic rotation than males when the slouching motion was complete (maximum pelvic relative rotation angle: females = 13.5° (SD 5.1); males = 8.8° (SD 5.2), $P < 0.0001$). Females also tended to start the motion with more anterior rotation of the pelvis than males but the difference was not significant (upright sitting pelvic angle from vertical: females = 10.0° (SD 7.3); males = 4.7° (SD 9.7)).

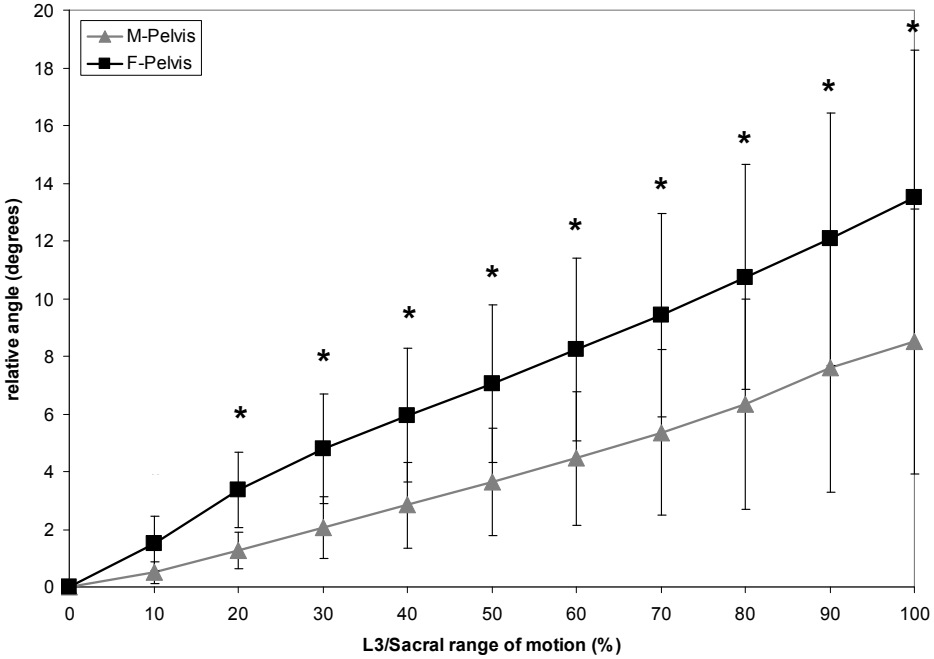


Figure 5.6: Relation between relative posterior pelvic rotation and total L3/Sac range of motion during the slouching motion. Significant differences between genders are indicated by an asterisk (*, $P < 0.01$).

5.5 Discussion

The results of this study indicate that individuals perform a slouched seated flexion movement using a number of different motion strategies. Three vertebral body motion patterns were identified based on how the VBs moved with respect to each other and these motion patterns affected the IVJ angles attained during the slouching motion. Although it was initially hypothesized that the slouched sitting motion would be initiated by the pelvis, this study demonstrated that this may not be the case. This suggests that individuals use their back muscles to initiate this motion. Interestingly, females had greater posterior pelvic rotation than males throughout the entire slouching motion, indicating that females have a greater capacity to rotate the pelvis in seated postures. The lowest lumbar IVJ (i.e., L5/S1) contributes the most to lumbo-sacral flexion in upright sitting, as it is at approximately 60% of its end range in this posture (Study #1, Chapter 3). Greater pelvic rotation with less lumbar flexion suggests that the majority of flexion could occur at the L5/S1 IVJ, concentrating the stress at this joint throughout the motion. A disordered sequence of VB motion initiation was also observed in all the participants in the current study, suggesting that different joints can approach their end range before the completion of the slouching movement. Joint positioning will alter the load on the passive tissues surrounding the joint, the force-generating capacity of individual muscles and the demands on the neuromuscular control system. Given that clinicians use hip flexion to rotate the pelvis and reduce L5/S1 flexion, the next step to investigate this issue would be to see if training individuals to initiate slouching with their pelvis makes the sequence of rotation ordered.

Only the flexion phase of the slouching motion was examined due to equipment limitations (i.e. the small field of view of the fluoroscope). The return to upright sitting phase is of equal interest because if the pelvis and VB movement patterns are different, it would be reasonable to suggest that the two movements may require different neuromuscular control patterns. Different movement patterns between standing flexion and extension have been observed in

both global lumbar spine and pelvic co-ordination (Nelson et al., 1995; Gatton and Pearcy, 1999; Lee and Wong, 2002; Pal et al., 2007) and intervertebral joint motions (Kanayama et al., 1996; Okawa et al., 1998; Harada et al., 2000). Both the pelvis and spine contribute to standing flexion tasks but the spine may have greater contribution to the early stages of the movement (Nelson et al., 1995; Gatton and Pearcy, 1999; Lee and Wong, 2002; Pal et al., 2007). Furthermore, a top-down sequential rotation of the lumbar VBs appears to be the most common pattern observed among individuals with no history of back pain (Kanayama et al., 1996; Okawa et al., 1998; Harada et al., 2000). Previous research suggest that the pelvis may contribute the most to lumbar spine flexion motion in slouched sitting motions (Snijders et al., 2004; Kasahara et al., 2008). Furthermore, the lower three lumbar IVJs approach their maximum RoM in slouched sitting (Study #1, Chapter 3). However, the results of the current study demonstrate that there is no clear "normal" pattern for how the lumbar VB movements are coordinated with pelvic motion in the initiation of a seated slouching movement. In other words, seated slouching is not initiated by a bottom-up sequence of IVJ joint rotation suggesting that individuals perform this motion by primarily using their back muscles.

In the current study, the pattern of VB movement influences the lower lumbar IVJ kinematics. When the three lower lumbar VBs initially rotated at similar rates (as was the case for Groups 1 and 2 identified in this study) the resulting L3/4 and L4/5 IVJs were less than 2° on average (Figure 5.5A, B). Conversely, when all three VBs initially diverged from the beginning of the slouching motion (i.e. Group 3), the L3/4 and L4/5 IVJs achieved much larger flexion angles. It has been suggested that in standing flexion, a disordered VB motion pattern (Okawa et al., 1998; Takayanagi et al., 2001) and differential rates of IVJ angular displacement (Teyhen et al., 2007) are characteristic of individuals with LBP. The results of the current study suggest that it is normal for some individuals to have disordered VB motion patterns in seated flexion. This suggests that there is a greater demand on the neuromuscular control system to avoid abnormal movement or buckling. Two motor patterns could cause

similar motion patterns but with very different consequences in terms of joint loading and spinal stability (McGill et al., 2003). For example, it has been demonstrated that the thoracic erector spinae muscles function either independently or synergistically with the lumbar erector spinae and internal oblique muscles to control lumbar flexion in sitting (O'Sullivan et al., 2006a). In some individuals, the extensor moment in slouched sitting is supported by activity in the thoracic ES when the lumbar ES activity decreases (O'Sullivan et al., 2006a). In other individuals, the thoracic ES activity decreases while lumbar activity remains the same (Callaghan and Dunk, 2002). Segmental deep multifidus muscles are differentially activated during trunk movements and most likely contribute to intersegmental control (Moseley et al., 2002). Quadratus lumborum activity does not change from an upright to a slouched sitting posture (McGill et al., 1996a) but does appear to have more activity in sitting than in standing (Andersson et al., 1996). Psoas activity has been shown to decrease in slumped sitting when compared to upright sitting, but has little to no activity in upright standing (Andersson et al., 1995). These results suggest that QL and psoas have postural functions in sitting, perhaps because of the rotation of the pelvis and increased lumbar flexion. Gatton and Pearcy (1999) noted that individuals performed a specific task twice (i.e. stand-to-sit motion) using different lumbar vertebral motion sequences each time. This suggests that there is not one "optimal" muscle recruitment strategy to perform a specific task, even in a constrained task such as slouched sitting. Still, one motion pattern may be more protective of spine health than another.

Only one motion trial was analyzed per participant. This was due to the fact that the participant's movement and the quality of the fluoroscopic video sequence were usually limited to one or possibly two trials. Issues that reduced the quality of the fluoroscopic video sequences included: the initial starting position of the spine was outside the field of view, not allowing for the initial determination of the ROI to be tracked, and; other objects (such as air pockets in the intestines) or large image contrast differences between the initial and subsequent frames, which would cause the registration program to fail. If multiple motion

trials were analyzed, it is possible that a participant could achieve the slouching motion with a different segmental motion pattern. This phenomenon was documented by Gatton and Pearcy (1999), who reported that individuals performed specific tasks twice using different spine motion sequences each time. If this were the case in the current study, however, it would add evidence to the idea that there is not one "optimal" movement pattern to perform a specific task.

The slouching motion needed to be restricted for some participants in order to keep the spine within the fluoroscope field of view. Thus, some participants were unable to go to an "end-point". These participants tended to be the ones who were classified in Group 3 (divergent from start) and achieved the largest external L3/Sac rotation. For half the participants dispersed throughout all three groups, the L4/5 IVJ angle reached a maximum angle towards the end of the slouching motion while the L3/4 IVJ was still flexing. This suggests that the L4/5 IVJ could be close to its end range of motion before the slouching motion is finished, as reported in a previous study (Study #1, Chapter 3). When given verbal and visual cues, Claus et al. (2009) observed that individuals were able to imitate seated postures with the same curvature direction in the thoraco-lumbar and lumbar regions, specifically, slouched, flat-back and long lordosis sitting. In the current study, the same verbal instructions about the adopted seated postures were given to all participants. The participants otherwise adopted their most natural posture. The purpose of this study was to examine the natural motion patterns of individuals. Before an intervention can be tried, it was necessary to know the natural motion patterns and this study laid the groundwork for understanding how a slouching motion is achieved in a normal, asymptomatic population. Nevertheless, the slouching motion described may not be representative of how individuals sit in everyday tasks. Slouched sitting can happen one of two ways – through rounding of the spine, while maintaining the upper body centre of mass over the ischial tuberosities (Callaghan and Dunk, 2002), or by rounding the spine and leaning against a back rest with the upper body CoM behind the ischial tuberosities (Snijders et al., 2004). When the spine is not supported during

the slouching motion, such as in the current study, the upper lumbar spine may make reciprocal movements in the opposite direction to prevent the whole trunk from falling forwards or backwards. In fact, some participants in the current study demonstrated an L3 VB rotation in the opposite direction to the overall rotation during slouching (Table 5.2). If a back rest were present, the VB kinematics could be very different than those documented in the current study and thus have different implications on spine tissue loading.

It remains unknown whether certain seated lumbar spine and pelvic movement patterns might predispose individuals to the development of low back pain during sitting. Simultaneous VB and pelvic motion during slouching, as demonstrated by some individuals in this study, may potentially help decrease the risk of back injury by reducing the degree of IVJ flexion during slouching. Less lumbar spine flexion may reduce the strain on the iliolumbar ligaments (Snijders et al., 2004) or reduce the likelihood of flexion relaxation, or reduced activity, in the superficial erector spinae muscles that has the potential to place large loads on the ligaments and disc of the lumbar IVJs (Dolan et al., 1988; Callaghan and Dunk, 2002; O'Sullivan et al., 2006a). On the other hand, larger IVJ rotations could lead to greater variation in muscle activity and provide periods of rest to the superficial erector spinae muscles. While the results of the current study provide some insight into the lower lumbar IVJ kinematics of asymptomatic individuals, future work could be aimed at investigating individuals with sitting-induced LBP. It is possible that delays and alterations in lumbar and pelvis movement patterns are related to a LBP patient's symptoms (Teyhen et al., 2007). In fact, abnormal movement of a lumbar IVJ, resulting in pain, was documented during lifting using fluoroscopy (Cholewicki and McGill, 1992). The quantification of seated movement patterns from dynamic imaging techniques could be useful in distinguishing between those who develop LBP and those who do not during prolonged sitting.

Gender differences were observed in the current study in the pelvic kinematics. Females had greater posterior pelvic rotation than males throughout the entire slouching motion,

indicating that females have a greater capacity to rotate the pelvis in seated postures. In the initial upright sitting posture, the male pelvis was more posteriorly inclined than the females. Males tend to have less hamstring flexibility than females (Bridger et al., 1992); since the muscles attach directly to the ischial tuberosities of the pelvis, decreased flexibility could be responsible for a more posteriorly inclined pelvis in sitting. The upper lumbar IVJs were not imaged in this study due to the limited field of view of the image intensifier. Given that the pelvis was the last segment to move for males in the current study, it is likely that males initiate a slouching motion with flexion of the upper lumbar IVJs rather than pelvic rotation. Interestingly, the lower 3 lumbar IVJs do not respond differently for the gender groups when examining static X-rays of seated postures (Study #1, Chapter 3). However, dynamic gender-specific lumbar-pelvic coordination patterns could mean that males and females are prone to injury or pain in different regions of the spinal column. More posterior pelvic rotation to accomplish the slouching motion in females suggests that the majority of flexion could occur at the L5/S1 IVJ, concentrating the stress at this joint throughout the motions.

The image registration method eliminated the onerous task of manually digitizing vertebral body landmarks frame by frame through a video sequence. One of the benefits of using the image registration method is that the video could be analyzed at 30Hz, rather than needing to be down-sampled to reduce the analysis time for manual digitization. Furthermore, the intensity-based registration approach can offer higher accuracy for tracking objects because it takes into account all available information from the user-defined region of interest, rather than relying on the user's visual location and selection of VB landmarks (see Appendix D). The image registration was still susceptible to noise from contrast non-homogeneity in the image, especially during periods where there was little motion of the bony structures. This made it difficult to determine an initiation of VB rotation directly from the image registration output. However, hand-digitizing of videofluoroscopy sequences is also susceptible to errors from poor image quality and should not be considered the "gold standard" in terms of accurate representation of VB angular displacement. While trials can be digitized multiple

times in order to reduce random error associated with this process (Cholewicki et al., 1991), this method is still extremely laborious, time-consuming and potentially harmful for the individual performing the hand-digitizing. The automated registration performance used in this study could be verified by applying the motion parameters determined by the image registration program the initial video sequence and visualizing the stationarity of the tracked VB. Both the hand-digitizing method and the automated registration program are susceptible to errors; however, because of the steps taken to verify the registration program output, it is reasonable to assume that the registration program's performance is adequate. Several future changes may further improve the performance of the registration program including using a fluoroscope with a larger field of view and further development of image tracking methods that are robust to changes in image contrast, soft tissue scatter, motion of other internal anatomical structures and partial occlusion of the vertebral bodies (Wong et al., 2009).

5.6 Conclusions

The main finding of this study was that there was no dominant sequence of rotation of the pelvis and lower VB of the lumbar spine during a slouched sitting motion. Many mathematical models allocate the amount of total lumbar spine flexion to each IVJ based on proportions, that is to say a percentage of total flexion is allotted to each IVJ (Chaffin, 1969; McGill and Norman, 1986; Marras and Granata, 1997). The results of the current study do not support this assumption. The sequence of individual vertebral joint motion is an important assumption within dynamic and quasi-static models, since joint positioning will influence the mechanical advantage of individual muscles and passive tissues. The relationship between IVJ motions and total lumbar spine motion during sitting must be defined in order to better understand how the tissues of the spine are affected during seated motions.

Chapter 6:
Time-varying postural responses during prolonged sitting differentiate low back pain developers from matched asymptomatic controls

6.1 Synopsis

Background: Little is known about how lumbar spine movement influences mechanical changes and the potential injurious effects of prolonged flexion associated with seated postures. The purpose of this study was to examine the postural responses and pain scores of low back pain sufferers compared with asymptomatic individuals during prolonged sitting in order to understand the biomechanical factors that may be associated with sitting induced low back pain.

Method: Sixteen participants with sitting-aggravated low back pain were age- and gender-matched with 16 asymptomatic participants. Tri-axial accelerometers were used to monitor lumbar spine angles during 90 minutes of seated computer work. Lumbar spine postures were examined using a movement pattern analysis of two types of postural adjustments, termed shifts (step-like adjustments larger than 5°) and fidgets (small change and return to approximately the same position).

Findings: The LBP group reported large significant increases ($P < 0.0001$) in low back pain while asymptomatic individuals reported little to no pain. On average, every participant fidgeted every 40 to 50 seconds. However, only the LBP sufferers demonstrated a significant increase ($P = 0.04$) in the number of shifts over 90 minutes of seated work; the LBP group shifted every 4 minutes in the last 30 minutes of sitting compared to every 10 minutes for the asymptomatic group. LBP sufferers also demonstrated larger amplitudes of shifts and fidgets when compared to the asymptomatic group.

Implications: Frequent shifts in lumbar spine posture could be a mechanism for redistributing the load to different tissues of the spine, particularly if some tissues are more vulnerable than others. However, increased movement did not completely eliminate pain in individuals with pre-existing LBP. The seated spine movements in LBP sufferers may be more detrimental to the spine and counteract the beneficial effects of movement. Future work to understand the biomechanical effects of proactively inducing slow and controlled movement may help to explain the paradox of the relationship between movement and pain.

6.2 Introduction

The relationship between workplace exposure to prolonged sitting and low back pain (LBP) is controversial; systematic reviews of the literature fail to show convincing evidence that LBP is strongly linked to prolonged sitting (Hartvigsen et al., 2000; Lis et al., 2007). However, the fact remains that populations who have LBP that is induced and/or aggravated by prolonged sitting do indeed exist (Womersley and May, 2006; Dankaerts et al., 2006b; O'Sullivan et al., 2006c). Furthermore, subjective experimental findings demonstrate that low back discomfort increases with prolonged seated exposures, even in individuals with no prior history of LBP (Fenety et al., 2000; Beach et al., 2005; Dunk and Callaghan, 2005; Gregory et al., 2006). A move towards the examination of time-varying responses of the lumbar spine is necessary in order to determine the mechanisms for sitting-induced LBP in situations relevant to workplace exposures. Little is known about how movement in the lumbar spine influences the mechanical changes and potential injurious effects of the prolonged flexion associated with seated postures and whether movements are preventative of or in response to discomfort/pain. Thus we were driven to document the seated movement patterns of a group of individuals with a history of LBP induced or aggravated by prolonged sitting and compare them to an asymptomatic group.

Several research attempts have increased the understanding of the biomechanical differences between individuals with LBP and asymptomatic individuals and how these factors affect seated postural and muscular responses. A common clinical observation is that individuals with non-specific LBP cannot touch the ground with their fingers during a standing toe-touch test (Halbertsma et al., 2001). This may not be due to limited lumbar spine range of motion (RoM) (Ng et al., 2002). Rather, altered hip and/or pelvic motion may cause altered lumbar spine kinematics (Shum et al., 2005; Shum et al., 2007) and pelvic movement may be directly affected by limited hamstring extensibility (Halbertsma et al., 2001). Given that hamstring flexibility has an effect on lumbo-pelvic posture in sitting (Bridger et al., 1992; Beach et al., 2008), it is reasonable to postulate that decreased hamstring flexibility may alter the seated lumbo-pelvic posture in LBP individuals. Previous research shows that LBP status can affect lumbar spine and pelvic postures in sitting. Individuals with flexion-provoked pain sit closer to lumbar spine flexion end range with greater posterior pelvic tilt (O'Sullivan et al., 2006c) and this results in a reduced "usable" lumbar range of movement in sitting (Dankaerts et al., 2006b). Furthermore, LBP patients with skeletal asymmetries compensate with altered motion in both the thoracic and lumbar regions during seated movements (Al Eisa et al., 2006). Several studies demonstrate impaired trunk postural control in individuals with LBP that could be caused by poor proprioception in the lumbar spine (Radebold et al., 2001; O'Sullivan et al., 2003). In fact, LBP patients appear to lack the ability to produce fine postural adjustments in both flexion/extension and lateral bending and they have difficulty repositioning their lumbar spine into a neutral posture (Radebold et al., 2001; O'Sullivan et al., 2003). In seated postures, LBP patients demonstrate higher back muscle activation (Dankaerts et al., 2006a) and delayed muscle responses to sudden loading (Radebold et al., 2001). The increased muscle activity is consistent with a protective reflex at the end range of lumbar spine flexion (Solomonow et al., 2003b). It is thought that these altered muscle co-ordination patterns are aimed at protecting the lumbar spine in order to avoid or reduce pain. However, if this guarding behaviour persists, it may exacerbate the pain or lead to new complaints (Main and Watson, 1999). Co-contraction will increase

forces acting on the spine and hyperactivity in the muscles could lead to spasm and more pain and limit the functional abilities of LBP patients (Van Dieen et al., 2003b).

Most studies have examined a static snapshot of different sitting conditions to observe representative muscle activity, posture, and loading. Seated office work can involve a wide range of postures of up to 50% of lumbar spine range of motion (RoM) with frequent movements (Van Dieen et al., 2001a; Callaghan and McGill, 2001b). Thus, a shift towards the examination of the time-varying responses of the lumbar spine is necessary in order to determine the mechanisms for LBP generation and how certain variables can be manipulated to avoid or prolong the initiation of these mechanisms. Jensen and Bendix (1992) suggested that there are three factors contributing to LBP in sitting: 1) insufficient nutrition to the intervertebral disc; 2) the stress-relaxation of spinal ligaments and; 3) muscular fatigue. Therefore, it is necessary to establish a more complete knowledge of how movement of the lumbar spine affects pain generating mechanisms in sitting. Sitting-specific movement patterns of the lumbar spine need to be established in order to attempt to understand the relationship between lumbar spine movement and LBP.

One of the difficulties with prolonged examinations is finding ways to quantify time-varying data. In other words, large amounts of data need to be reduced down to meaningful numbers that can be compared between or within individuals. Specifically, for this study, how could movement patterns of the lumbo-sacral spine be quantified? Several different approaches have been attempted to document postural changes during prolonged sitting. Several researchers have counted the number of times that whole body posture changes (Bhatnager et al., 1985; Jurgens, 1989; Liao and Drury, 2000) while others have employed more complex methods of documenting movement. Fenety and colleagues (2000; 2002) assumed an association between general discomfort and movement of the seat pan centre of pressure (CoP). Callaghan and McGill (2001b) used the amplitude probability distribution function

(APDF) to quantify the variation in lumbar spine posture. Vergara and Page (2000; 2002) used a flexible goniometer placed over the lumbar spine to measure curvature and the signal was digitally filtered to discriminate between two different types of movement: global postural changes (macro-movements) and small "fidgeting" changes (micro-movements). In a related technical approach, Duarte and Zatsiorsky (1999) developed algorithms to detect different migration patterns of the centre of pressure under the feet during prolonged standing. The method assumed that CoP migration is not random and contains specific and consistent patterns of movement. A similar approach was developed for the current study in order to quantify lumbar spine movement and is discussed in Section 6.3.

6.2.1 Purpose

In order to understand the relationship between lumbar spine movement and LBP, it is necessary to first examine the sitting behaviour of individuals with sitting-induced LBP. Additionally, gender has been identified as a factor that affects lumbar spine and pelvic posture in sitting (Dunk and Callaghan, 2005; Gregory et al., 2006; Beach et al., 2008) and is important to consider when examining sitting behaviour. Thus, the purpose of this study was to examine any association between postural responses and pain development in a LBP population and compare it to an asymptomatic control population in order to understand the biomechanical factors associated with LBP during sitting. Specific goals included: 1) investigating the potential implications of gender, altered flexibility, spine range of motion, muscular activation patterns on sitting-related LBP; 2) examining the postural responses and pain scores of LBP and asymptomatic individuals to 90 minutes of seated computer work; 3) developing different methods of quantifying lumbar spine movement patterns and to determine if it is possible to distinguish between LBP and asymptomatic groups based on postural responses.

6.2.2 Hypotheses

Specific hypotheses were:

1) Males and females with LBP will have less LS RoM and hamstring flexibility than the age- and gender-matched asymptomatic individuals.
2) LS RoM will be affected after 90 minutes of seated computer work: males will have reduced LS RoM while females will not show a distinct response.
3) The LBP group will sit with more LS flexion and greater posterior pelvic tilt than the asymptomatic group throughout the 90 minutes of sitting, because previous research has suggested that this is the case the short-term sitting behaviour of LBP individuals (O'Sullivan et al., 2006c). Furthermore, both groups will demonstrate systematic increases in LS flexion and posterior pelvic tilt over 90 minutes.
4) The lumbar spine movement patterns will differ between the LBP and asymptomatic groups: LBP individuals will move less, demonstrating guarding behaviour.

6.3 Quantifying patterns of lumbo-sacral spine movement in prolonged sitting

When lumbar spine postures were displayed as a time-series, two patterns of movement were discernible (Figure 6.1 and Figure 6.2):

1) Shifts: step-like angular change in lumbar angle
2) Fidgets: small rapid changes of the lumbar angle about the same average position

Two separate algorithms were developed in order to detect these two movement patterns in time-varying lumbar angle.

6.3.1 Shift Algorithm

To detect shifts in the lumbar spine angle time series (Figure 6.1):

1) The average values ($\bar{x}_{W1i}$ and $\bar{x}_{W2i}$) for two consecutive moving windows, W_1 and W_2 each of 15 seconds in length, centered around a point (x_{1i} and x_{2i}) and separated by the 3 second period W_s, were computed. $\bar{x}_{W1i}$ and $\bar{x}_{W2i}$ were calculated and compared on a point by point basis (where *i* corresponds to the centre point of the moving window).

2) Any two consecutive windows being offset by a change greater than five degrees (Equation 6.1) were classified as a shift:

$$\left|\bar{x}_{W1i} - \bar{x}_{W2i}\right| \geq 5° \qquad \text{(Equation 6.1)}$$

3) The shift duration was determined as the length of time Equation 6.1 was consecutively satisfied, as long as the shift duration was greater than W_s.

4) The shift amplitude was calculated as:

$$Amplitude_{shift} = \left(\sum_{i=m}^{n}\left|\bar{x}_{W1i} - \bar{x}_{W2i}\right|\right)\Big/ n \qquad \text{(Equation 6.2)}$$

Where: n = the total number of consecutive points satisfying Equation 6.1 for a given shift
i = frame number where Equation 6.1 is satisfied
m = the last consecutive frame satisfying Equation 6.1 for a given shift

Parameter values:

The identification of the shift pattern depends on the following parameters: a) the shift amplitude threshold value of 5°; b) the length of time of windows W_1 and W_2; and c) the

constant value of W_s, the period separating the two consecutive windows. The length of W_1 and W_2 was chosen to be 15 seconds. These values were used by Duarte & Zatsiorky (1999) when developing their algorithm and they demonstrated that the size of the windows had little effect on the shift outcome. The period separating the two windows (W_s) was set at 3 seconds. An informal sensitivity analysis was performed where the shift duration was varied from 1 second up to 5 seconds on 4 different datasets. Changing the distance between the two moving windows only affected shift amplitude, not shift frequency. Furthermore, the dynamic slouching motion performed in Study #3 (Chapter 5) was achieved in approximately 3 seconds. Thus, it was determined that 3 seconds was an appropriate time to complete a large change in spine posture while sitting. According to the work done by Duarte and Zatsiorsky (1999), the most important parameter is the shift threshold. A shift amplitude threshold of 5° was chosen for the following reasons:

- It has been used as a threshold by other researchers to define large lumbar spine movements (Vergara and Page, 2000; Vergara and Page, 2002).
- Previous research has shown that flexion at the L4/5 joint contributes approximately 40% to the total flexion angle in sitting (see Chapter 3). Based on this partitioning, a 5° change in total lumbar spine angle will effect a 2° change at the L4/5 IVJ. While it is not fully understood how much of an angular change will affect the IVJ, 2° increment changes of a spinal segment have been shown to affect mean IVD pressure (McNally and Adams, 1992).

6.3.2 Fidget Algorithm

To detect a fidget in the LS angle time series (Figure 6.2):

1) The average and standard deviation values ($\bar{x}_{Wi}$ and SD_{Wi}) of a moving window (W), centered on a point (x_i) were computed. The length of W was 60 seconds.

2) The fidget amplitude threshold value was set as $3*SD_{Wi}$ and the fidget duration, W_F, was selected as less than 3 seconds in order to distinguish it from a shift (Equation 6.3).

$$\left|\bar{x}_W - x_i\right| > 3SD_W \qquad \text{(Equation 6.3)}$$

3) The fidget duration, W_F was determined as the length of time Equation 6.3 was consecutively satisfied, as long as the time did not exceed 3 seconds.

4) The fidget amplitude was calculated as:

$$Amplitude_{fidget} = \left(\sum_{i=m}^{n}\left|\bar{x}_{Wi} - x_i\right|\right)\Big/ n \qquad \text{(Equation 6.4)}$$

Where: n = the total number of consecutive points satisfying Equation 6.3 for a given fidget

i = frame number where Equation 6.3 is satisfied

m = the last consecutive frame satisfying Equation 6.3 for a given fidget

Parameter values:

The identification of the fidget pattern depends on the following parameters: a) the fidget amplitude threshold value of $3*SD_{Wi}$; b) the length of time of the window, W; and c) the constant value of W_F, the width or length of time for the fidget. The length of W was chosen to be 60 seconds and the fidget width (W_F) was set to less than 3 seconds. The fidget threshold value of $3*SD_W$ was chosen because upon visual inspection, it was the threshold value that returned the most realistic answers based on the lumbar spine time series.

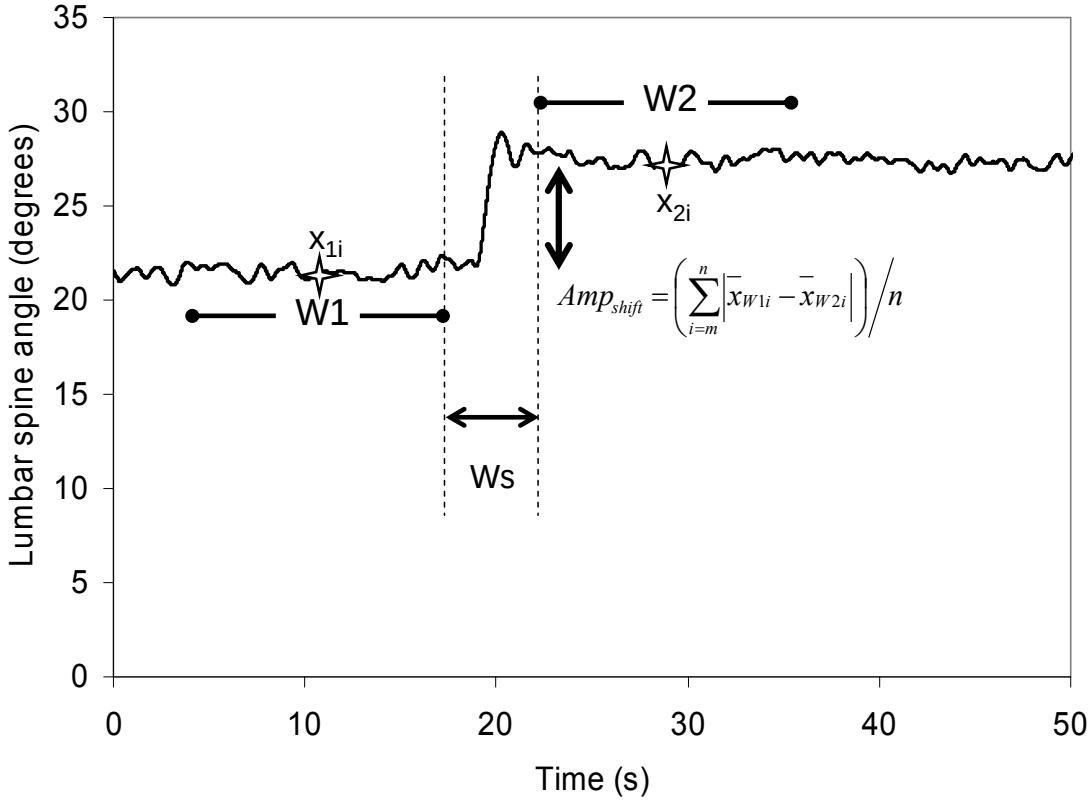


Figure 6.1: An example of the shift pattern and the parameters used in the recognition algorithm. The average values were computed on a point-by-point basis for two 15-second moving windows (W_1 and W_2), each centered on a point (x_{1i} and x_{2i}) and separated by W_s (3 seconds). Any two consecutive windows being offset by a change greater than five degrees were classified as a shift. Amp_{shift} indicates the calculation for the shift amplitude.

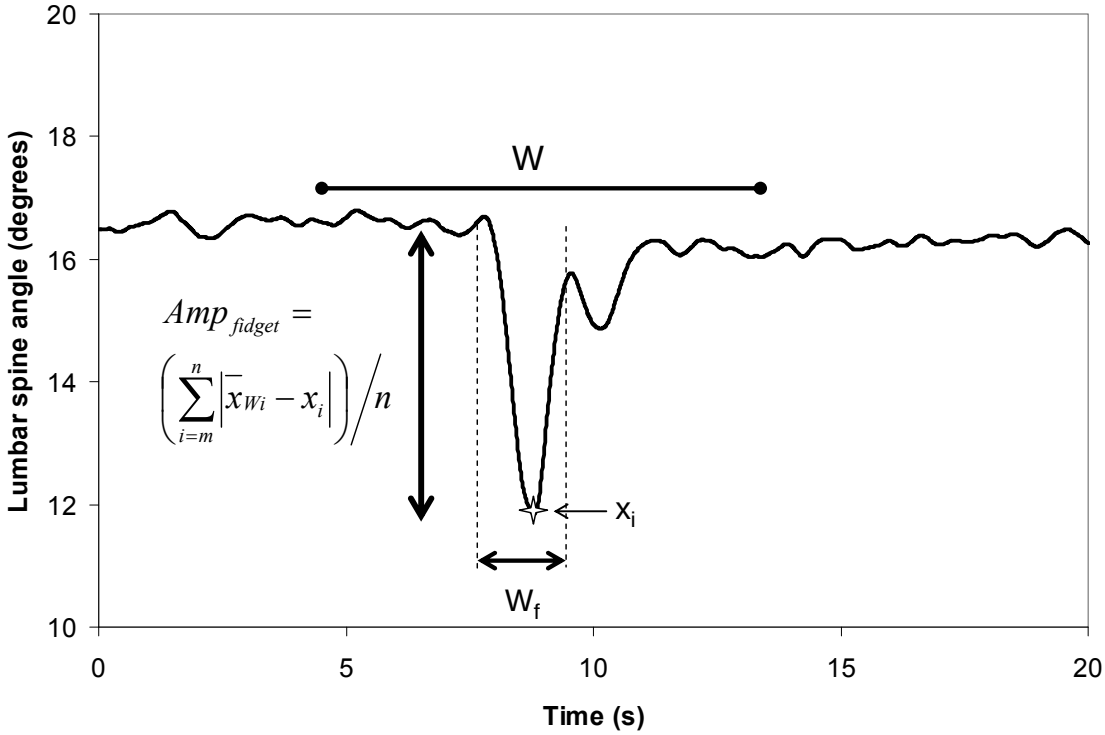


Figure 6.2: An example of the fidget pattern and the parameters used in the recognition algorithm. The average and standard deviation were computed on a point-by-point basis for a moving window (W) of length W (60 seconds) and centered on a point (x_i). Amp_{fidget} indicates the calculation for the fidget amplitude.

6.4 Methods

6.4.1 Overview of Experiment

Individuals with sitting-induced LBP and asymptomatic individuals were asked to perform 90 minutes of seated computer work while upper body kinematics, lumbar spine and pelvic posture, seat pan pressure, and pain scores were monitored. Lumbar spine range of motion (RoM), hamstring flexibility and erector spinae (ES) muscle fatigue were tested before and after the 90 minutes of seated work.

6.4.2 Participants

A total of thirty-two participants were recruited from the community at large. Eight asymptomatic males and eight asymptomatic females with no history of LBP for 12 months prior to the testing period were age- and gender-matched to eight males and eight females with sitting-induced LBP (Table 6.1). It was decided to test eight participants per group because results from a previous study with very similar methodology observed significant differences between gender groups with the same number of participants per group (Dunk and Callaghan, 2005).

Inclusion criteria for asymptomatic participants:

- No activity-limiting back pain over the 12 months prior to the testing period

Inclusion criteria for LBP participants:

- Non-specific back pain with insidious onset for longer than 2 months (less than 2 years)
- Self-reported lumbar pain and discomfort elicited by prolonged sitting

- Minimal pain at the time of testing (i.e. at the start of the test, participants were not having an episode of LBP that would prevent them from sitting for 90 minutes)

Exclusion criteria for LBP participants:

- Neurological deficits and/or lower extremity impairments (i.e. numbness, weakness, decreased reflex)
- Medically diagnosed stenosis, spondylolisthesis or recent fractures
- Severe structural deformities (kyphosis or scoliosis)
- Previous surgical intervention

The pre-test conditioning of the participants was controlled with as much rigor that human testing allows. All participants were asked to refrain from vigorous activity for 24 hours prior to the testing session. Participants also went through the same 60 to 90 minute experimental set-up routine, whereby they were asked to stand while instrumentation was attached to their body, or they were performing the same calibration tasks (e.g. full flexion, the RVE task, described in Section 6.4.4). In studies where prior activity history is an important factor to control (i.e. for studies examining height loss using stadiometry), the standard pre-test conditioning time ranged from 30 to 60 minutes of one common activity (e.g. lying down, upright standing or slow walking). Therefore, it was reasonable to assume that the experimental set-up time period in the current study was an acceptable pre-conditioning activity. All participants were either students or members of the workforce. Full schedules made it very difficult to standardize the time of day at which testing occurred. It is acknowledged that this may be a confounding factor since responses seen in the morning may differ after a full day of work. However, the time of day of the testing session was a random factor with participants from both groups being tested at all times of the day. Therefore, any differences seen between the two groups (LBP vs. asymptomatic) are likely not attributed to the time of day.

All participants (both asymptomatic and LBP) completed the Oswestry Disability Index 2.0 (Fairbank and Pynsent, 2000) (Appendix E) in order to ascertain how LBP affected their ability to manage in everyday life (Table 6.1). An ODI score of 0 to 20% indicates minimal disability and 21 to 40% indicates moderate disability (Fairbank and Pynsent, 2000). The study protocol received approval from the University of Waterloo Office of Research and participants gave informed consent before testing began.

Table 6.1: Participant characteristics and Oswestry Disability Index (ODI) scores.

	Age (years)	**Height (m)**	**Mass (kg)**	**BMI (kg/m^2)**	**ODI Score**
Female					
Asymptomatic (n = 8)	25.9 (4.6)	1.67 (0.06)	64.4 (13.3)	23.0 (4.7)	1.1 (2.0)
LBP (n = 8)	27.0 (5.3)	1.70 (0.06)	70.7 (13.3)	24.6 (5.3)	17.5 (9.2)
	n/s	n/s	n/s	n/s	$P = 0.002$
Male					
Asymptomatic (n = 8)	24.0 (3.8)	1.80 (0.10)	74.0 (11.5)	23.3 (3.3)	0.3 (0.8)
LBP (n = 8)	22.8 (3.2)	1.80 (0.10)	81.3 (17.3)	24.8 (3.4)	14.2 (5.2)
	n/s	n/s	n/s	n/s	$P = 0.0001$

6.4.3 Instrumentation

Whole body kinematics were recorded using an optoelectronic motion analysis system (Optotrak Certus, Northern Digital Inc., Waterloo, ON). Markers were placed on the chair being tested (on the side of the seat pan) and over the following anatomical landmarks on both sides of the participant's body: right/left hand at the base of the third metacarpal, right/left wrist, right/left elbow, right/left shoulder, right/left ear canal, C7/T1, right/left greater trochanter, right/left knee and right/left ankle. Lumbar spine and pelvic tilt angles were obtained using accelerometers (S2-10G-MF, NexGen Ergonomics, Montreal, Quebec, Canada) placed over the L1 spinous process and the sacrum at the level of S2. Accelerometers have been used as inclinometers to measure the spine in quasi-static and static situations (Hansson et al., 2001; Bernmark and Wiktorin, 2002; Wong and Wong, 2008). Muscle activity of the upper and lower erector spinae (ES) was monitored using

electromyography (EMG). Blue Sensor bi-polar Ag-AgCl electrodes (Ambu A/S, Denmark, intra-electrode distance of 2.5 cm) were applied to the skin in pairs bilaterally over thoracic ES muscle belly, approximately 5 cm lateral to the T9 spinous process, and lumbar ES muscle belly, approximately 3 cm lateral to the L3 spinous process. The EMG signals were differentially amplified (common-mode rejection ratio 115 dB, input impedance 10 GΩ) (model AMT-8, Bortec, Calgary, AB, Canada), and bandpass filtered (10–1000 Hz) prior to digital conversion. Accelerometer and EMG signals were A/D converted at rates of 128 Hz and 2048 Hz, respectively, with a 16-bit A/D system (Optotrak Data Acquisition Unit II, Northern Digital Inc., Waterloo, ON, Canada). Seat pan interface pressure was measured using a pressure mapping device (X2 Seating System, XSensor Technology Corporation, Calgary, AB) and continuously sampled at 4Hz. The seat pressure signal collections were initiated by a pulse sent from the Optotrak Data Acquisition Unit in order to synchronize the data in time.

6.4.4 Data Collection protocol

The testing session involved 90 minutes of seated computer work, performed at a workstation that was adjusted for each individual user so that the elbows were at 90° with relaxed shoulders when each participant was typing on the keyboard. The chair was a standard office chair (Jendra #3614, Borgo Contract Seating, Etobicoke, Ontario, Canada) and was adjusted such that the initial seated position allowed the knees to be at 90° when the feet were in full contact with the floor. The seat pan to back rest angle was fixed at an angle of 105° and the seat pan was locked in a position parallel to the floor. Participants were instructed to sit how they would normally and move around in the chair at will without crossing their legs. During the 90 minute sitting period, participants performed 15 minute intervals of controlled simulated office work consisting of a mousing task, a typing task and task involving a combination of the two. Standardized tasks were used because the type of computer tasks could have an effect on postural responses while sitting (Van Dieen et al., 2001a; Dunk and Callaghan, 2005; Gregory et al., 2006). The three tasks were presented in a random order

during the first 45 minutes to ensure that any observed differences were not attributable to the order of task performed and then repeated in the same order. It is acknowledged that the randomization of the tasks was not complete; however, this was done to ensure that a full data set was obtained should a participant not be able to complete the 90 minute sitting period. Optotrak, accelerometer, EMG and seat pressure data were continuously collected throughout each 15 minute interval.

Participants rated their perceived low back pain on a 10 cm visual analogue scale (VAS) during the 90 minute sitting period (Appendix F). VASs are generally accepted by the International Association for the Study of Pain as a valid scale for pain and discomfort assessments (Borg, 1998) and have the sensitivity to distinguish between diseased and non-diseased participants (Randolph, 2000). Participants were asked to place a mark along the 10 cm line where they felt their current level pain was on a scale of "no discomfort" to "worst discomfort imaginable". VAS scores were obtained for three regions of the low back: the central area over the lumbar spine, and the right and left sides lateral to the spine. Measures were taken at the start and after each of the six 15 minute intervals, yielding a total of seven VAS scores per participant.

In order to normalize the EMG data, participants performed a sub-maximal reference voluntary exertion (RVE) of the ES muscles prior to data collection. Briefly, participants were asked to hold a 10 kg mass with their arms straight and bend at the hips so that the trunk was at an angle of 60° to the vertical (Figure 6.3). Participants were instructed to maintain a lordotic (i.e. neutral) spine, squeezing their shoulder blades together and looking straight ahead while data were collected for 10 seconds. An RVE was obtained instead of a maximum voluntary contraction so that all participants would have reliable EMG reference levels, as it has been shown that individuals with LBP may not be able to perform a true MVC (Marras and Davis, 2001; Dankaerts et al., 2004). A resting EMG level was also

recorded with the participant lying prone. The RVE test was also performed immediately before and after the 90 minute sitting period in order to assess the presence of ES muscle fatigue.

Further tests were performed before and after the 90 minute sitting period to assess lumbar spine RoM and hamstring flexibility. For lumbar spine RoM, participants were asked to bend over and touch their toes while focusing on maximally flexing their lumbar spine and data were collected for 5 seconds while the position was held. Right and left hamstring flexibility was determined with the following test, which was recommended by a Canadian Registered Physiotherapist: the hip was flexed and the thigh was held to the chest by grasping the posterior aspect of the thigh; in this position, the knee joint was maximally extended while maintaining contact of the thigh to the chest (Davis et al., 2008). This position was held for 5 seconds while data were collected and repeated on both the right and left side of the body.

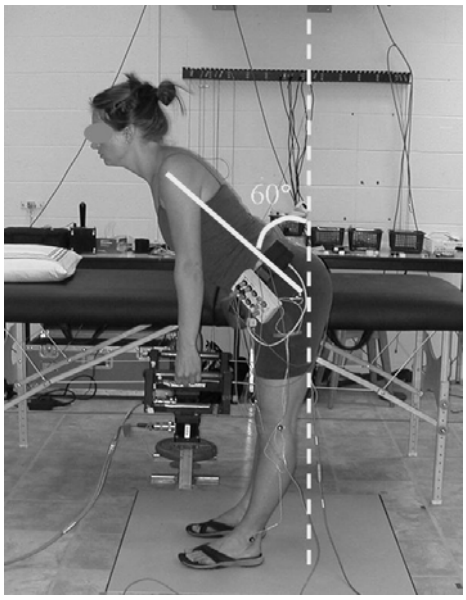


Figure 6.3: Erector spinae muscle submaximal reference voluntary exertion (RVE) posture. Participants held a 10 kg mass in their hand while bending the trunk to 60° from the vertical.

Summary of the data collection protocol:

1) Pre-sitting hamstring flexibility tests with the following markers: right and left hip, right and left knees and right and left ankles
2) EMG electrode placement and normalization procedures (RVE, rest trial)
3) Accelerometer and remaining marker placement
4) Pre-sitting RVE for ES fatigue baseline, upright standing trial, maximum flexion trial
5) 90 minute sitting period with VAS scores obtained throughout
6) Post-sitting RVE for ES fatigue detection, maximum flexion trial
7) Post-sitting flexibility tests with necessary markers (all instrumentation remained on the participant, but only the markers described in Step 1 were activated during these tests)

6.4.5 Data reduction and processing

Hamstring flexibility was calculated as the 2D angle between the shank (the line between the ankle and the knee) and the thigh (the line between the knee and the hip). Larger angles corresponded to increased flexibility (Figure 6.4). Lumbar spine RoM was calculated as the change in angle from upright standing to the fully flexed posture in the toe touch RoM test.

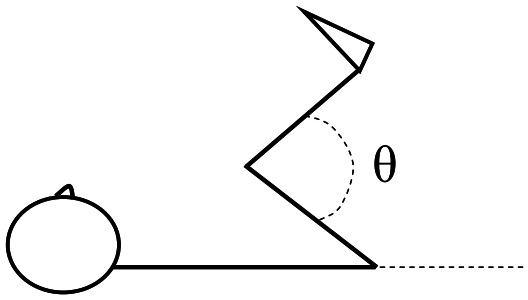


Figure 6.4: Knee extension angle was used as a measure of hamstring flexibility. The angle (θ) between the line of the shank (from ankle to knee) and the line of the thigh (from knee to hip) was calculated. Larger angles correspond to increased flexibility.

ES muscle fatigue was assessed by examining the changes in the frequency content and amplitude of the EMG signal obtained during the pre- and post-sitting RVE. The power spectrum of the EMG signal was calculated and the mean power frequency (MPF) was used to characterize the frequency content. The change in EMG amplitude was expressed as a percent (%) change of the post-test amplitude from the pre-sitting amplitude.

Due to noise contamination of the EMG signals during the 90 minute sitting period, the data were not reliable. The explanation of the issues and associated steps to attempt to remedy the situation can be found in Appendix G.

Given that the outputs from the accelerometers are a sinusoidal function of the inclination of the sensor relative to gravity, each axis was recorded at 0° (or +1g) and 180° (or -1g) in order to provide a conversion from voltage outputs to degrees. Each accelerometer was oriented so that the positive y-axis was aligned pointing down the vertical axis of the spine. The y-axis was used as the inclination axis and the corresponding orthogonal z-axis was used in order to determine the correct orientation of the y-axis inclination. Because of the sinusoidal nature of the accelerometer output, two axes are necessary in order to determine the correct orientation of the sensor. For example, when the z-axis was oriented to the left (90°), the y-axis was pointed up (180°). The lumbar spine angle was calculated as the difference between the y-axis inclinations of the L1 and sacral sensors, and expressed as a change from upright standing. Converted angles were smoothed using a zero-lag 4^{th} order low pass Butterworth filter with a cut-off frequency of 1 Hz to remove any higher frequency accelerations.

The mean lumbar spine angles from the pre- and post-sitting RoM trials were calculated and compared to determine any effects of prolonged sitting on lumbar spine RoM. The pre-sitting lumbar spine RoM value was used to normalize all lumbar spine angles throughout the

90 minute sitting period. Time-varying lumbar spine and pelvic angles were determined and the following methods were used to quantify the signals:

1) All variables were initially averaged over the 15 minute intervals to examine gross changes over time during the 90 minute sitting period.
2) The minimum and maximum lumbar spine angles during each 15 minute time block were determined and the range of lumbar spine movement within each block was calculated.
3) The amplitude probability distribution function (APDF) was used to examine the range and time distribution of lumbar spine postures adopted during the 90 minute sitting period (Callaghan and McGill, 2001b). Briefly, the normalized time-varying lumbar spine angles were binned in 1% increments to generate an APDF for each individual.
4) Shift and fidget patterns were detected using the movement pattern algorithms described in Section 6.3.

Three-dimensional upper body centre of mass (CoM) was calculated from the marker data using anthropometric properties summarized by Winter (1990) with modifications for the trunk (Pearsall et al., 1996). The vertical trunk axis was represented as the line connecting the mid-point between the two hip markers and the C7 marker. Sagittal and medial/lateral trunk inclinations were calculated as the 2D angle of the vertical trunk axis to the horizontal in the respective planes.

The seat pressure profile was used to obtain the location of the centre of pressure (CoP) and peak pressure (PP) over time for each 15 minute interval. The area of peak pressure under the ischial tuberosities (IT) was calculated by locating the cells containing the peak pressure to the left and right side of the midpoint of the pressure area on the mat; any adjacent cells

whose pressure was within 10% of the maximum value were included in the peak pressure area. Left and right peak pressure areas (PP area) were expressed as a percentage of the total seat pressure area and averaged to obtain an overall mean value for each trial within each subject. Time-varying CoP signals were used to describe whole body movement patterns. CoP range of movement within each 15 minute time block was calculated as the difference between the maximum and minimum CoP locations in both the A/P and M/L directions. Similarly, CoP total distance traveled was calculated as the sum of the changes in location between successive time points in the signal.

A calibration procedure was performed in order to locate the pressure mat on the chair in the global co-ordinate system of the motion analysis system. Briefly, points located on the pressure mat at the front edge and right edge of the chair were digitized with a 4 marker rigid body digitizing probe (i.e. global x, y and z co-ordinates were determined) and these points were located with respect to a marker fixed on the seat pan. Since the distance between the two digitized points and the seat pan marker remained fixed, the front edge of the chair (FEC) and right edge of the chair (REC) could be tracked regardless of the movement of the chair in space. Using the dimensions of the pressure mat cells, the location of the CoP could also be determined with respect to the FEC and REC points, thereby allowing the pressure system measures to be related to the anatomical kinematic data and located on the chair. CoP was expressed with respect to the front edge of the chair (FEC), the right edge of the chair (REC) and the upper body CoM. In order to analyze where the individual was sitting on the chair, the hip joint and PP location were expressed with respect to the FEC and REC. All variables were averaged over the 15 minute intervals to examine gross changes over time during the 90 minute sitting period.

6.4.6 Statistical Analyses

Hamstring flexibility, MPF and EMG amplitude variables from the pre- and post-sitting tests were compared using 4-way analyses of variance (ANOVA) (gender, LBP status, leg or muscle, time) with repeated measures on two factors (leg/muscle, time). Lumbar spine pre/post variables were compared using 3-way ANOVAs (gender, LBP status, time) with repeated measure on one factor (time). Tukey's post-hoc multiple comparisons were used to examine significant main effects and interactions. The level of significance was set at $P < 0.05$.

Time-varying variables were analyzed using either 3-way (1 repeated measure) or 2-way ANOVAs. Gender and LBP status main effects were consistent factors in both ANOVA designs. For the 3-way ANOVA, the repeated factor was time (to evaluate changes over 90 minutes), task (to verify the task randomization process), or direction of movement (for variables collapse over 90 minutes). Tukey's post-hoc multiple comparisons were used to examine significant main effects and interactions. The level of significance was set at $P < 0.05$.

Time-varying measures included:

a) Lumbar spine and pelvic angles, shifts and fidgets

b) Body positioning on chair:

- PP vs. upper body CoM, FEC
- Hip vs. FEC
- Upper body CoM vs. FEC, REC, CoP(M/L), CoP(A/P)
- PP area
- Trunk inclination in A/P and M/L directions

c) CoP measurements

- CoP range of movement in A/P and M/L directions
- CoP total distance traveled in A/P and M/L directions

d) VAS scores

- VAS over 90 minutes of sitting
- VAS baseline scores by low back region

6.5 Results

PRE/POST-SITTING TEST RESULTS

The following three sections summarize the results from the tests that were performed before and after exposure to 90 minutes of sitting in order to ascertain the effect of prolonged sitting on various muscular and postural responses.

6.5.1 Erector Spinae muscle fatigue tests

There was a significant LBP*muscle interaction ($P = 0.05$) where the lower ES muscles had significantly higher MPF for the LBP group when compared to the asymptomatic group. The upper ES muscle MPFs were similar between the two groups, and there were no significant effects of gender or time. EMG amplitude dropped by approximately 8% (significant time effect, $P = 0.003$) after 90 minutes of sitting for all participants.

6.5.2 Hamstring Flexibility

Hamstring flexibility was not affected by 90 minutes of sitting for either the LBP group or the asymptomatic group. However, regardless of LBP status females demonstrated significantly greater hamstring flexibility than males (female knee angle = 140.3° (SD 12.4); male knee angle = 128.6° (SD 9.1); $P = 0.003$).

6.5.3 Lumbar spine range of motion

Lumbar spine RoM was significantly different ($P = 0.04$) between the LBP group that had an average RoM of 47.4° (SD 14.0) and the asymptomatic group with an average of 58.0° (SD 15.1). There was no significant difference between the groups in pre and post sitting lumbar spine RoM; however in both groups, participants lost an average of 2.4° (SD 4.1) of lumbar spine RoM after 90 minutes of sitting ($P = 0.003$).

TIME-VARYING RESPONSES DURING 90 MINUTES OF SEATED WORK

All variables were examined for task and time effects, and only significant time effects were noted. Therefore, task effect results were not reported.

6.5.4 VAS scores

Baseline VAS scores for the three low back regions were not significantly different and were pooled to give an overall low back pain score when comparing the LBP and asymptomatic groups. The difference between baseline VAS scores was significant between the groups ($P = 0.01$); the asymptomatic group's baseline score was 0.1 mm (SD 0.1) and the LBP baseline score was 9.6 mm (SD 3.8). There was a significant LBP status*time interaction ($P < 0.0001$) for low back VAS scores in all three regions; scores increased significantly over time for the LBP group, while remaining relatively close to zero for the asymptomatic group (Figure 6.5).

6.5.5 Body Positioning on Chair during 90 minutes of sitting

Neither time nor task had significant effects on any of the body position variables measures, thus the results reported are mean values over the entire 90 minute sitting period. There was a trend ($P = 0.08$) towards female PP being closer to the FEC than males (Distance of PP to FEC for females = 28.8 cm (SD 2.5), for males = 30.2 cm (SD 1.9)). This was mainly due to the fact that females with LBP sat with their PP closest to the FEC (27.8 cm (SD 2.5)).

Every other group sat with their PP approximately 30.1 cm (SD 2.0) away from the FEC. Similarly, there was also a trend ($P = 0.08$) towards females sitting with their hips closer to the FEC than males but the difference was only about 1cm (Distance to FEC for females = 23.3 cm (SD 1.7), for males = 24.3 cm (SD 1.2)). Individuals with LBP tended ($P = 0.07$) to sit with their upper body CoM closer to the FEC than the asymptomatic group (posterior distance of CoM from FEC asymptomatic = 33.1 cm (SD 4.1); LBP = 30.5 cm (SD 4.3). A similar but non-significant trend ($P = 0.37$) was that the LBP group sat with their upper body CoM closer to but still posterior to their PP than asymptomatic individuals (LBP PP vs. CoM = 1.5 cm (SD 3.5); asymptomatic PP vs. CoM = 2.9 cm (SD 5.0)). Similar results were observed for upper body CoM vs. CoP (CoM distance posterior to CoP for asymptomatic group = 9.1 cm (SD 4.6); LBP group = 6.7 cm (SD 3.7)).

There were no LBP status, gender or time effects found for trunk inclination; participants had a trunk inclination of 105.2° (SD 7.5) to the horizontal, indicating that the adopted seating posture involved reclining against the back rest. Furthermore, all participants tended to lean their trunk towards their left side by 1.5° (SD 1.8). This evidence was supported by the upper body CoM being 2.8 cm (SD 0.9) to the left of the CoP for all participants. In addition, the peak pressure area under the left ischial tuberosity was significantly greater than that of the right side (LPPArea = 8.9 cm^2 (SD 2.7), RPPArea = 6.8 cm^2 (SD 1.8); $P = 0.002$).

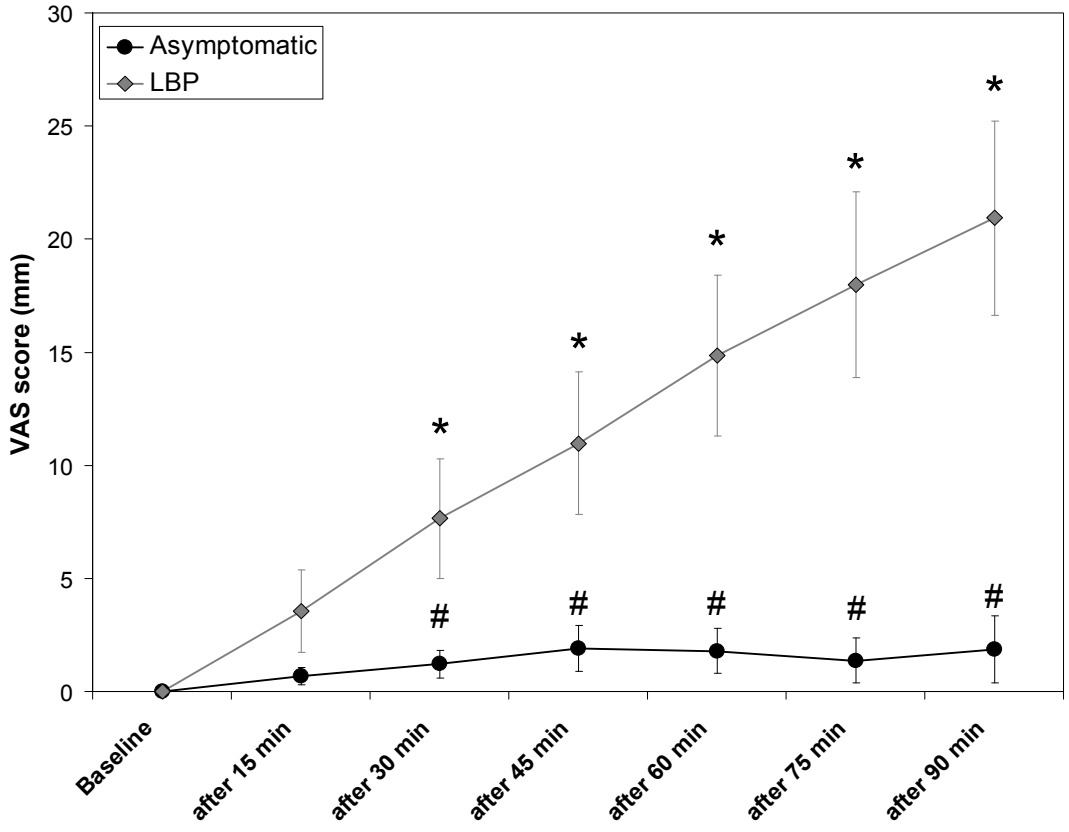


Figure 6.5: Average (+/- 1 SD) Visual Analogue Scale (VAS) scores for the low back over 90 minutes of sitting for the low back pain (n = 16) and asymptomatic (n =16) groups. There was a significant LBP*time interaction ($P < 0.0001$). The asterisk (*) indicates that the LBP VAS score is significantly different from baseline. The number sign (#) indicates that the LBP VAS score is significantly different from the asymptomatic group.

6.5.6 Lumbar spine and Pelvic posture during 90 minutes of sitting

Lumbar spine posture, when normalized to %RoM, was variable for all groups over the 90 minutes (Figure 6.6). The gender*LBP*time interaction was significant ($P = 0.0005$), but the only significant pairwise comparisons occurred in Time 1 (Figure 6.6). The female LBP group ($P = 0.0002$) and the asymptomatic male group ($P = 0.03$) were significantly different from the male LBP group. There was also a significant time effect ($P = 0.003$) indicating

that lumbar spine angle varied over time. While there were no significant pairwise comparisons, there was a general trend towards less lumbar spine flexion over time. Similarly for pelvic rotation, the pelvis became less posteriorly rotated over time (significant time effect, $P = 0.009$) (Figure 6.6). Also, females sat with less posterior pelvic rotation than men throughout the entire 90 minutes (females = 0.4° (SD 10.2), males = -6.0° (SD 7.8); $P = 0.045$).

The variability in lumbar spine posture can be attributed to the amount of movement throughout each 15 minute block. For instance, Figure 6.7 demonstrates that asymptomatic individuals used up to 30% of their lumbar spine RoM during each 15 minute time block. Individuals with LBP initially used up to 50% of their lumbar spine RoM and this value increased over time to almost 80% RoM used. Asymptomatic individuals used 26.5% (SD 9.8) of their RoM on average while LBP individuals used an average of 61.4% (SD 27.3), which were significantly different ($P < 0.0001$). Thus, mean values of lumbar spine posture over 15 minutes did not accurately represent the time-varying postural changes. There were two patterns of seated lumbar spine postures adopted by the asymptomatic and the LBP groups, as demonstrated by the APDF (Figure 6.8). Asymptomatic individuals generally adopted a more "static" sitting strategy where they used a smaller range of their lumbar spine RoM over the 90 minute sitting period. LBP individuals generally adopted a wide range of postures over a larger RoM (Figure 6.8).

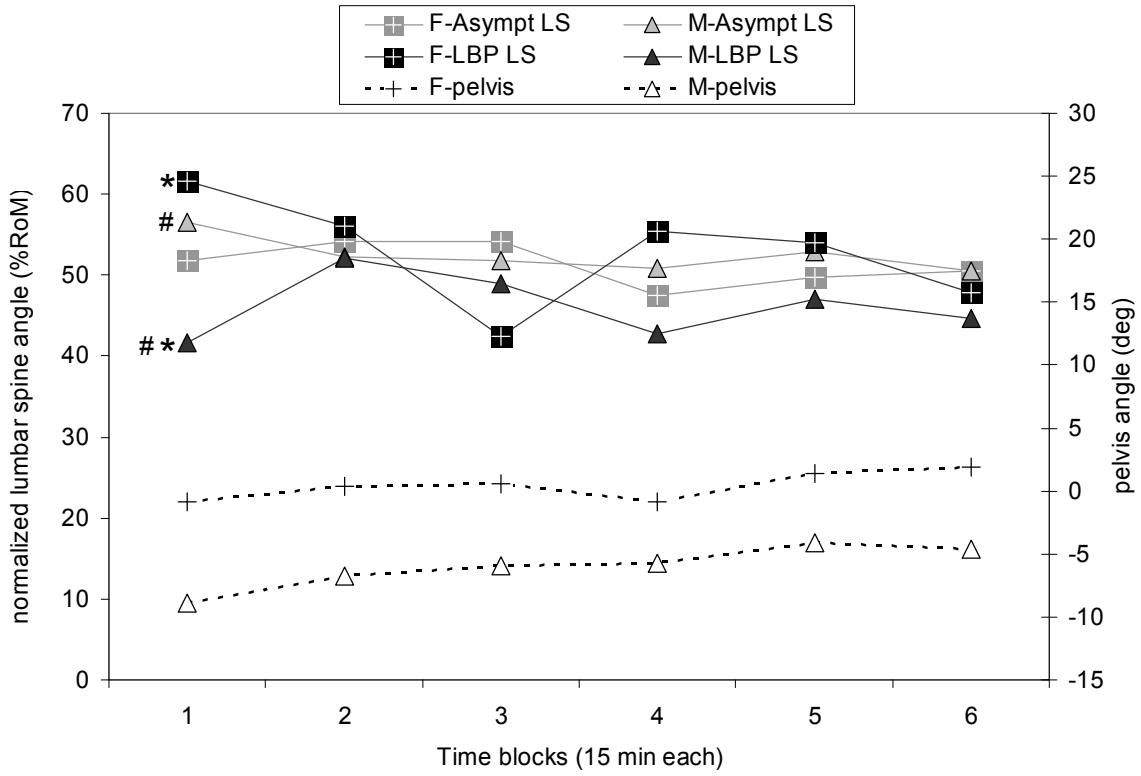


Figure 6.6: Normalized lumbar spine (LS) and pelvic posture (angle with respect to vertical) over the 90 minute sitting period. Each data point represents an average over 15 minutes. There was a significant gender*LBP*time interaction (*P* = 0.0005) for the lumbar spine angle, but the only significant pairwise comparisons occurred in Time 1. The female low back pain (F-LBP) group (* - *P* = 0.0002) and the asymptomatic male (M-Asympt) group (# - *P* = 0.03) were significantly different from the male LBP group. For the pelvis, the male pelvis was more posteriorly rotated (*P* = 0.04) than the female pelvis. Both genders demonstrated a shift towards more anterior pelvic rotation over time (*P* = 0.009). Negative pelvic angle indicates posterior rotation with respect to vertical.

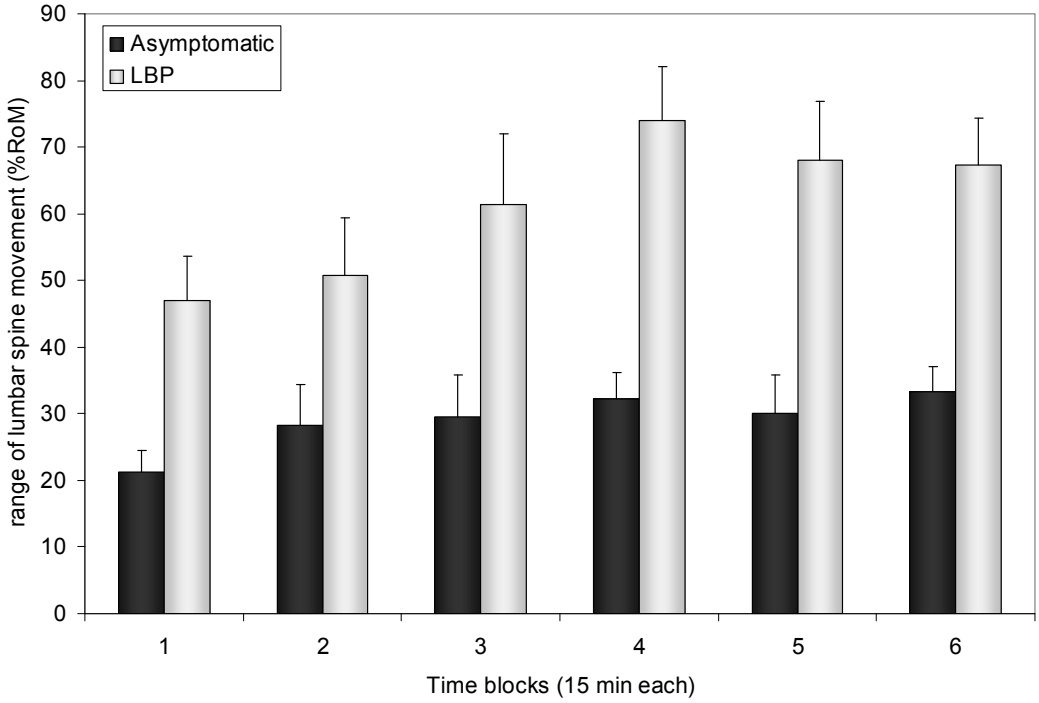


Figure 6.7: Range of lumbar spine movement for the asymptomatic and LBP groups. The range was calculated as the largest minus the smallest normalized spine angle measured in each 15 minute time block. The LBP group had a larger range of movement than the asymptomatic group (P = 0.0002) and all individuals used a larger range of lumbar spine RoM over time (P = 0.0008).

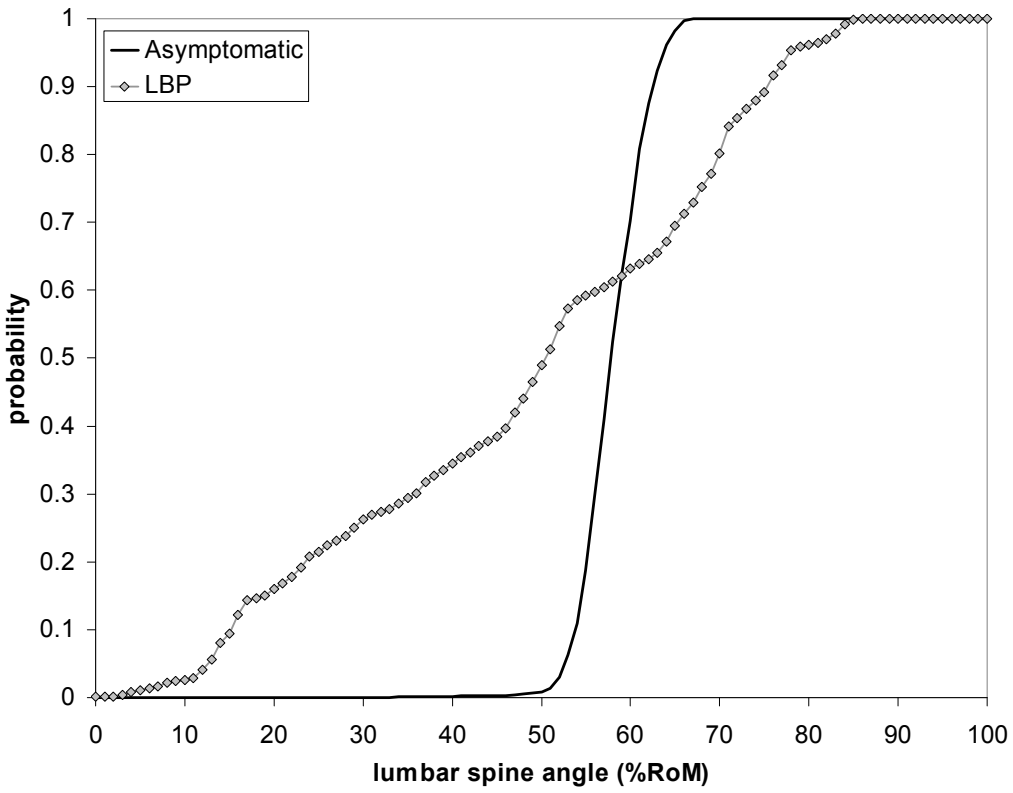


Figure 6.8: Amplitude probability distribution functions of lumbar spine angles for one asymptomatic and one LBP participant for the duration of the sitting period.

Over the entire sitting period, LBP sufferers shifted once every 7.2±4.7 minutes, whereas healthy participants shifted every 12.6±4.6 minutes and the difference in shift frequency was statistically significant ($P = 0.003$; Table 6.2). Every participant demonstrated some level of a fidget movement pattern, where a fidget occurred on average every 40 to 50 seconds (Table 6.2). However, the amplitudes of both the shift and fidget movement patterns of the LBP group were 1.5 and 2.5 times larger than the asymptomatic group (Table 6.2). There was also a significant LBP*Time interaction ($P = 0.04$) whereby the LBP group shifted more times than the asymptomatic group in the last three 15 minute time blocks (Figure 6.9).

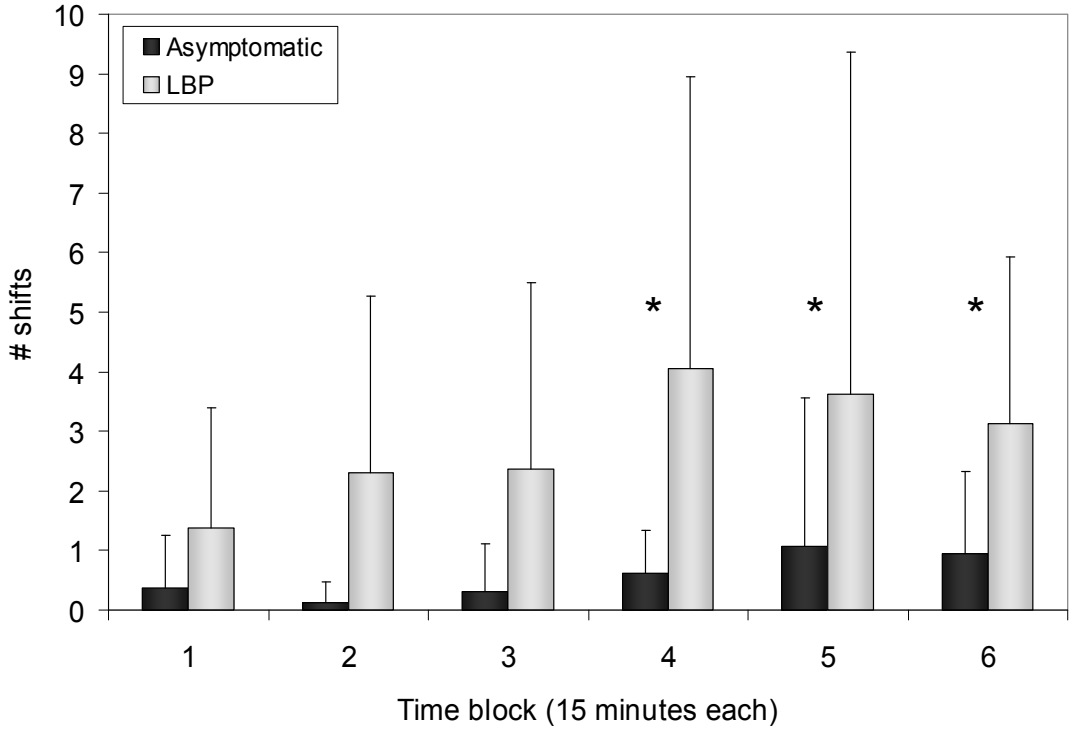


Figure 6.9: The average (+1SD) number of shifts during 90 minutes of sitting for each 15 minute time block for the two test groups (low back pain and asymptomatic). There was a significant LBP*time interaction ($P = 0.04$) whereby LBP group had significantly more shifts in the last 3 time blocks than the asymptomatic group. The asterisk (*) indicates a significant difference between LBP and asymptomatic groups ($P < 0.05$).

Table 6.2: Lumbar spine shift and fidget analysis. Shifts are defined as a step-like change in lumbar spine angle. Fidgets are defined as a change and return of lumbar spine angle to approximately the same position. Significant differences between the low back pain (LBP) and asymptomatic groups are indicated by asterisks (*).

	Asymptomatic	**LBP**	***P*-value**
Shift amplitude (normalized to %RoM)	11.2% (5.2) *	17.3% (6.7) *	*P*=0.01
Shift frequency (# minutes between shifts)	12.6 min (4.6) *	7.2 min (4.7) *	*P*=0.003
Fidget amplitude (normalized to %RoM)	4.4% (3.6) *	10.9% (8.0) *	*P*=0.001
Fidget frequency (# seconds between fidgets)	47.0 s (9.2)	42.1 s (9.9)	n/s

6.5.7 Centre of Pressure measurements

CoP range of movement and the total excursion of the CoP in the A/P and M/L directions were significantly different between the LBP and asymptomatic groups (Table 6.3). This means that the LBP group CoP had a larger total excursion (P = 0.0007) and range of CoP movement (P = 0.0006) than the asymptomatic group. The CoP range and excursion in the A/P and M/L directions were also significantly different; the CoP had a larger range (P = 0.02) and more total excursion (P = 0.003) in the M/L direction. There was a significant LBP*time interaction for total CoP excursion in both directions (P < 0.01; Figure 6.10). In other words, the CoP traveled increasing distances in both the A/P and M/L directions for the LBP group when compared to the asymptomatic group in the second half of the 90 minute sitting period (Figure 6.10) which agrees well with the increasing shifts seen in the LBP group over the same period.

Table 6.3: Mean centre of pressure (CoP) variables with one standard deviation in brackets. The main effects of low back pain status (LBP) and direction (anterior/posterior (A/P) vs. medial/lateral (M/L)) were significant for both the CoP range and distance traveled. Significant LBP effects are indicated in the rows labeled "LBP vs. Asympt" and significant direction effects are indicated in the row labeled "A/P vs. M/L".

	A/P vs. M/L	**Asymptomatic**	**LBP**
CoP Range (cm)	*** *P* = 0.02**		
A/P direction	***2.9 (1.3)* ***	2.1 (0.7)	3.8 (1.3)
M/L direction	***3.8 (3.2)* ***	2.2 (1.7)	5.4 (3.5)
LBP vs. Asympt **# *P* = 0.0006**		***2.2 (1.3)* #**	***4.6 (2.7)* #**
CoP distance traveled (cm)	**§ *P* = 0.003**		
A/P direction	***353.1 (166.2)* §**	264.4 (82.1)	441.9 (183.1)
M/L direction	***404.6 (163.4)* §**	311.3 (116.6)	498.0 (151.7)
LBP vs. Asympt **¤ *P* = 0.0007**		***287.8 (102.0)* ¤**	***469.9 (167.8)* ¤**

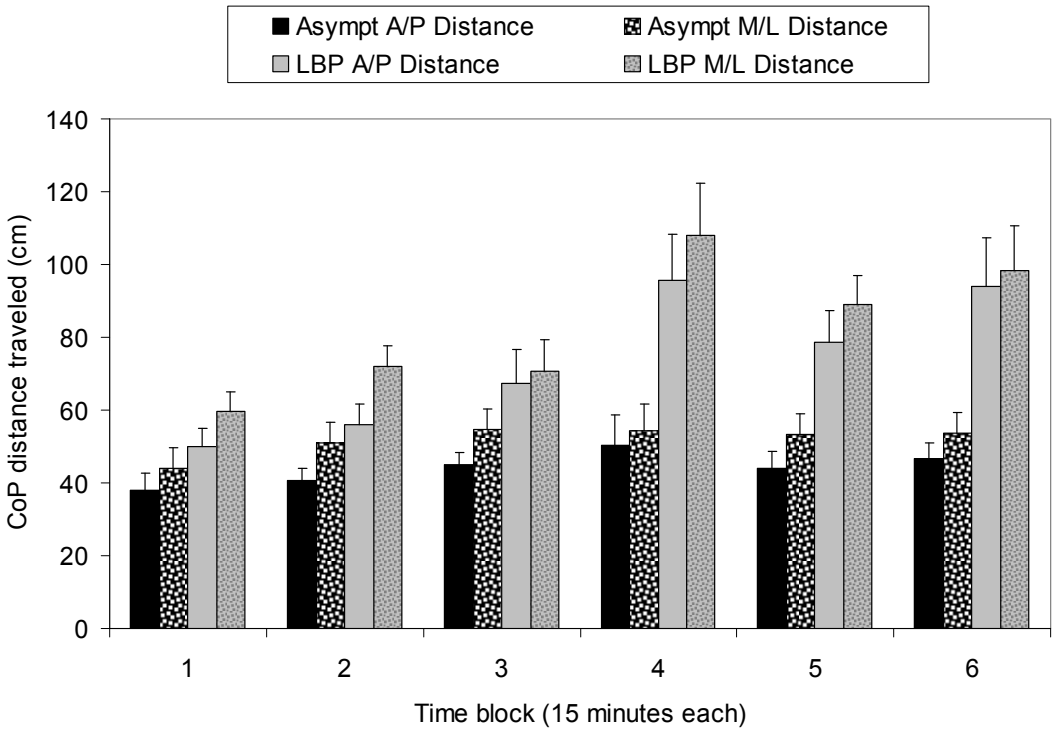


Figure 6.10: Total Centre of pressure (CoP) excursion in the anterior/posterior (A/P) and medial/lateral (M/L) directions for each 15 minute time block of the sitting period. There was a significant LBP*time interaction for the total CoP excursion in both directions ($P < 0.01$).

6.6 Discussion

Time-varying lumbar spine movement patterns allowed for the distinguishing between the LBP and asymptomatic groups during prolonged seated office work. The major finding of this study was that LBP sufferers moved more than asymptomatic individuals during 90 minutes of seated work and they reported increased LBP over time. LBP sufferers used as much as 80% of their lumbar spine RoM while sitting and demonstrated large, frequent shifts in lumbar spine posture and seat pan CoP. These movement patterns increased in amplitude and frequency over time along with LBP development. On the other hand, asymptomatic individuals generally exhibited a more static sitting pattern, using only 30% of their lumbar RoM with smaller fidget movements around a mean posture and reported little to no LBP development. For asymptomatic individuals, seated movements may serve a purpose to diminish feelings of discomfort from restlessness (Bhatnager et al., 1985; Jurgens, 1989), body stiffness, lack of circulation (Winkel and Jorgensen, 1986) or seat pressure (de Looze et al., 2003). Frequent shifts in lumbar spine posture could be a mechanism for redistributing the load to different tissues of the spine, particularly if some tissues are more vulnerable than others. However, increased movement did not completely eliminate pain in individuals with pre-existing LBP.

Both LBP and asymptomatic individuals in the current study demonstrated a decrease in lumbar spine RoM after 90 minutes of sitting, which is likely synonymous with an increase in lumbar spine stiffness. Previous research has suggested that increased spine stiffness after prolonged sitting could occur because spinal movement may increase spinal height through fluid flow into the disc (Beach et al., 2005), especially if a chair back rest is present (Van Dieen et al., 2001a). However, the increased stiffness in the LBP and asymptomatic individuals in the current study may be due to different mechanisms. Due to the less dynamic nature of LBP-free sitting, increased spinal stiffness could be due to changes in passive elastic properties of the muscles and passive tissues surrounding the joint. However,

chronic LBP patients generally exhibit higher back muscle activation, most likely aimed at avoiding painful tensile stresses placed on spinal structures (Van Dieen et al., 2003b). Even slight increases in trunk extensor activity associated with small postural changes (Andersson et al., 1974; Callaghan and Dunk, 2002) or low back pain disorders (Van Dieen et al., 2003b), could have negative effects on the muscles of the back, thus increasing lumbar spine stiffness. This study lacks information regarding muscular activity during prolonged sitting, which may have helped explain the impact of the movement patterns and subsequent post-exposure response seen in the different groups.

Other studies have quantified trunk and lumbar spine movements of asymptomatic populations using various methods. Two sitting strategies in an asymptomatic group were previously described based on the amplitude probability distribution function (APDF) of time-varying lumbar spine postures (Callaghan and McGill, 2001b). "Static" sitters used less than 10% of their total lumbar flexion RoM whereas "dynamic" sitters used up to 50% of their RoM, but no relationship between sitting strategies and discomfort was established. The results from our study demonstrated that asymptomatic individuals generally adopted the "static" sitting strategy, while LBP individuals were "dynamic" sitters. Vergara & Page (2000; 2002) noted that increases in lumbar discomfort reporting were associated with less frequent lumbar fidgeting and more frequent lumbar and pelvic shifts. They suggested that small movements around a mean posture help to reduce muscular strain while large changes in posture are a good indicator of the presence of discomfort. Based on their quantification methods, they documented large lumbar spine shifts (or "macromovements") that ranged from one change every four to eight minutes with amplitudes from 14 to 27°. "Micro-movements" occurred every 4.5 to 6.5 seconds, with amplitudes of 1 to 2.5°. In the present study, the definition of a shift was very similar to that of Vergara and Page (2002) but the shift amplitudes never exceeded 13° (or 40% RoM). This is likely due to the mathematical calculations of this study's method, which compared average values of two moving windows, thus perhaps masking any large peaks in lumbar spine posture. Therefore, large peaks in

lumbar spine movement could be occurring that may be more detrimental to spine health than beneficial as higher trunk velocity has been identified as a risk factor for LBP (Marras et al., 1995). Future work could be aimed at examining the efficacy of slower, more controlled movements in the relief of sitting-induced LBP.

One of the uncertainties of the algorithm used to identify shifts in the current study is the choice of the shift amplitude threshold of 5°. This value was chosen because it has been used as a threshold by other researchers to define large lumbar spine movements (Vergara and Page, 2000; Vergara and Page, 2002) and because flexion at the L4/5 joint contributes approximately 40% to the total flexion angle in sitting (Dunk et al., 2007). Based on this percentage, a 5° change in total lumbar spine angle will effect a 2° change at the L4/5 intervertebral joint (IVJ). While it is not fully understood how much of an angular change will affect the IVJ, 2° increment changes of a spinal segment have been shown to affect the mean intervertebral disc pressure (McNally and Adams, 1992). The algorithm employed in the current study to detect a fidget was very different from previous studies (Vergara and Page, 2000; Vergara and Page, 2002) where the signal was conditioned (i.e. high pass filtered) to isolate the high frequencies. Zero-crossings of the filtered signal were counted as "micromovements". Our method of detecting fidgets used the unaltered time-varying angle signal and the variability of the signal as a threshold to detect biologically meaningful changes in lumbar spine posture. Given the choice of the parameters to mathematically identify a fidget (i.e. fidget threshold, moving window length) there may have been higher frequency movements that were not detected. However, Duarte and Zatsiorsky (Duarte and Zatsiorsky, 1999) identified high frequency movement patterns in standing CoP, which they termed trembling. Trembling may have more pertinent implications for studying postural control mechanisms, which was not the focus of this study.

This is the first study to examine the postural responses of a LBP population over a prolonged period of time. Previous research has suggested that LBP sufferers assume sitting postures with more lumbar spine flexion and posterior pelvic tilt (O'Sullivan et al., 2006c). Contrary to these findings, which were developed based on static analyses of instantaneous seated postures, the development of LBP in the current study was not associated with any one posture. One implication of this finding is that short duration investigations of seated postures may not accurately represent the time varying biological responses to prolonged exposure. Every participant in the current study demonstrated a fidgeting movement pattern, where a fidget occurred on average every 40 to 50 seconds. However, only the LBP sufferers demonstrated a significant increase in the number of shifts over 90 minutes of seated work. In fact, 80% of the asymptomatic individuals did not shift their lumbar spine until after 45 minutes of sitting and fidgeting was the primary movement pattern observed. On the other hand, only 2 LBP sufferers did not shift during the first 45 minutes. These findings support the notion that more frequent shifts are associated with LBP development. It is difficult to address the issue of why individuals moved more over 90 minutes of sitting when so little is understood about why seated individuals move at all. The relationship between in-chair movement and discomfort is elusive since movement is necessary to avoid detrimental static postures. From a tissue perspective, prolonged static trunk postures can have severe detrimental effects on the various structures of the spine. Static flexion can lead to changes in the intervertebral disc (IVD), such as increased stresses in the annulus of the IVD (Adams et al., 1994; Hedman and Fernie, 1997) and a loss of disc height over time (Botsford et al., 1994; Hedman and Fernie, 1995). Furthermore, prolonged bouts of extreme static flexion have been shown to increase joint laxity due to creep of the posterior passive structures of the spine (McGill and Brown, 1992) and this creep has the potential to alter spine muscle activity patterns (Solomonow et al., 2003a). Prolonged seated work has also been shown to alter the passive stiffness of the *in vivo* lumbar spine (Beach et al., 2005). Movement during sitting may be necessary to promote disc nutrition through fluid flow (Adams and Hutton, 1983; Adams and Hutton, 1986; McMillan et al., 1996a), relieve potentially harmful stress

concentrations in the disc (McNally and Adams, 1992; Adams et al., 1994; Adams et al., 1996), vary muscle activity (Mclean et al., 2000; Mclean et al., 2001; Callaghan and McGill, 2001b) and help circulate blood to and from the muscles (Sjogaard et al., 1988).

It is difficult to prove a cause and effect relationship between seated spine movement and LBP because the etiology of sitting-induced LBP is generally unknown. LBP sufferers in the current study reported increasing pain scores in association with larger and more frequent shifts in spine posture over the duration of the sitting period. There are two plausible explanations as to why movement did not abolish pain in these individuals. First, LBP individuals may possess some underlying pathology that is aggravated by increased movement in sitting. LBP sufferers may demonstrate asymmetric movements (Al Eisa et al., 2006), they may be at their end range of motion already (O'Sullivan et al., 2006c), or have altered muscle activation and load sharing (Van Dieen et al., 2003a). Movement in sitting may lead to strain on tissues that are already vulnerable or reduce spinal stability (Van Dieen et al., 2003b). Secondly, LBP individuals may move preemptively in order to reduce or prevent expected LBP development. Pre-emptive movement may serve the purpose of shifting the load from vulnerable tissues to less vulnerable ones, in the effort to find a more comfortable position. However, in the current study, the LBP sufferers' seated spine movements increased in frequency and amplitude as time passed. The large shifts in spine flexion could forcefully place this group towards their end range of motion. Large shifts in extension could cause pinching of ligaments, facet capsule or nerve roots, particularly if disc height is lost. It is likely that these movements became more difficult to properly control because LBP patients may lack proper lumbar spine postural control (Radebold et al., 2001; O'Sullivan et al., 2003).

Mechanically induced spine movement using continuous passive motion devices has shown to effectively reduce sitting-induced LBP (van Deursen et al., 1999). The findings of the

current study are inconclusive with respect to motion reducing LBP, since a LBP control group whose motion was restricted would be needed to make such a conclusion. It is likely that large changes in posture observed in the current study are an indicator of pain in LBP sufferers and a method for preventing pain from becoming worse. This highlights the need to investigate the merits of mechanically induced movement vs. self-selected movement in LBP development. Little is known about the biomechanical effects of proactive or mechanically induced slow and controlled movements. Education regarding movement while sitting, as well as regularly scheduled standing stretch breaks, appears to minimize the increase of low back discomfort over time (Mclean et al., 2001; Fenety and Walker, 2002). Furthermore, regular breaks may reduce "static" sitting in asymptomatic individuals and promote muscular load sharing (Mclean et al., 2001; Fenety and Walker, 2002). At-work exercise programs are relatively effective at relieving neck/shoulder pain (Kietrys et al., 2007), and whole body discomfort (Fenety and Walker, 2002). More work is needed to determine whether specific exercises targeting the low back would be appropriate and effective at alleviating sitting-induced LBP. Low frequency, small amplitude continuous passive lumbar motion has also been effective in reducing pain in LBP sufferers over a period of prolonged sitting (Reinecke et al., 1994; van Deursen et al., 1999). However, the biomechanical effectiveness of mechanically induced movements, and how these movements can mitigate the detrimental effects of sitting, is largely unknown. Further work to understand the biomechanical effects of proactively induced slow and controlled movement may help to explain the paradox of the relationship between movement and discomfort or pain.

There were no patterns in the results of the muscular fatigue tests in the current study. This may support the idea that there may be variability in muscular load sharing between the different muscles of the back that may affect the fatigue response of all individuals in the current study (Van Dieen et al., 2003a; Van Dieen et al., 2008). McLean and colleagues (2000) found a cyclic pattern in the MPF of LES EMG. They suggested that using the slope of MPF over time may not be an appropriate means of quantifying muscle fatigue during

seated work because the frequency of EMG varies over time in a cyclic manner. This could potentially explain why other researchers have not found MPF decreases over 2 hours of sitting, but have observed the effect in back muscle that were activated at a constant level as low as 2% MVC for 30 minutes (Kingma and Van Dieen, 2008; Van Dieen et al., 2008). In the current study, there were no differences in MPF before and after the sitting period, but all participants demonstrated a drop in EMG amplitude of about 8% during the post-sitting RVE test. The RVE posture is commonly used in several laboratories in order to provide an EMG to moment relationship for EMG-driven models of lumbar spine loading (Norman et al., 1998). Thus, the RVE posture was chosen in the current study because it has been shown to be a reliable and repeatable method for obtaining muscle activity in the erector spinae muscles. One explanation is that there may have been a shift of activation and load sharing to other trunk muscles not measured in this study (i.e. the deep muscles of the spine). The lack of muscular fatigue demonstrated in the current study could suggest that both asymptomatic and LBP individuals could have adopted some sort of muscular load sharing that decreased the likelihood of developing back muscle fatigue. Another explanation could be that changing from a sitting to a standing posture would likely cause recruitment of other motor units, thus potentially masking any change in frequency content. Therefore, future work should ensure that the RVE be completed in a seated posture to mimic the working posture as closely as possible. It was the original plan to evaluate changes in EMG activity throughout the 90-minute sitting period, but the signals were contaminated with noise from other equipment, as outlined in Appendix F.

The time-varying changes in the lumbar spine and pelvic postures did not support the hypothesis that the lumbar spine would become more flexed and the pelvis more posteriorly rotated over time. Rather, the results showed that the pelvis became less posteriorly rotated and the lumbar spine had a small tendency to become less flexed for all participants. One explanation could be that individuals used the back rest more as the duration of the sitting period progressed. However, measured trunk postures as well as the position of the upper

body CoM did not strongly corroborate this thought. Given that there were no measurements of back rest use (i.e. contact switches, pressure mapping), it is plausible that whole upper body measurements are too coarse to detect subtle changes in the use of the back rest. Thus, the results may suggest that one of the purposes of movements during sitting could be to subtly change the lumbar spine and pelvic posture to a more upright position.

Gender differences were observed in pelvic posture, with all females having less posterior pelvic rotation than all males, regardless of LBP status. Males in the current study demonstrated less hamstring flexibility than females, which has been shown to affect lumbar spine posture due to the muscles' direct attachment to the ischial tuberosities (Bridger et al., 1989; Bridger et al., 1992), and is associated with more posterior rotation of the pelvis in sitting (Beach *et al.*, 2008). A gender effect was not found in lumbar spine posture in the current study. Previous research has shown that the intervertebral angles ranging from the sacrum to L3 do not differ between genders in seated postures (see Study #1, Chapter 3). It is plausible that the amount of flexion observed in seated postures is distributed across the entire spinal column in a gender-specific manner. Given that the thoracic spine angle was not measured in the current study, it is not possible to ascertain if this is where the gender differences in posture occur.

The postural responses and body positioning on the chair were different from expected. Previous research has found that males and females assumed different body positions while sitting in a chair with a back rest (Dunk and Callaghan, 2005). Specifically, males leaned back against the back rest while females tended to perch towards the front of the chair in an upright position. No such strong biologically relevant differences were observed in the current study. The difference between the current study and the previous one is that the chair in the current study had a fixed seat pan, thus eliminating any need to balance the upper body over a pivoting seat pan. It has been suggested that the smaller size and mass of females may

reduce their ability to maneuver a pivoting seat pan (Dunk and Callaghan, 2005). Previous research has also suggested that sitting on an unstable surface will induce more spinal motion but not affect lumbosacral spine kinematics or muscle activity (Gregory et al., 2006; O'Sullivan et al., 2006b; Kingma and Van Dieen, 2008). Additionally, individuals with LBP may have poor trunk postural control that would be exacerbated sitting on an unstable surface (Radebold et al., 2001; O'Sullivan et al., 2003). Thus, in the current study, the fixed seat pan (i.e. stable sitting surface) could explain why there were no observed effects of gender or LBP status on body positioning on the chair.

The results of this study suggest that lumbar spine movement drives the upper body posture while sitting, since CoP measurements (a surrogate measure for upper body movement) increased over time along with lumbar spine movement. Fenety and colleagues (2000; 2002) measured the seat pan centre of pressure (CoP) of visual display unit (VDU) operators who performed typing and screen reading tasks for 2 hours. The assumption is that seat pan CoP movement reflects movement of the entire upper body on the chair. They demonstrated approximately 100cm of CoP distance travelled in the first 15 minute time block. The CoP distance traveled increased over the 2 hours, with over 200cm of CoP distance in the last 15 minute time block. These values were for an asymptomatic population and are similar to the CoP distance documented in the LBP group in the current study. However, it is possible that the tasks performed by the VDU operators required more in-chair movement. It is not clear if the change in CoP distance travelled was due to greater magnitude or increased frequency of the CoP movement. A linear relationship between the position of seat pan CoP and the trunk inclination angle was demonstrated, suggesting that the movements of the CoP reflect trunk movement. However, the current study suggests that increases in CoP movement are due to increases in large lumbar spine shifts, not smaller movements. This is because the number of shifts tended to increase over time for the LBP group along with an increase in CoP distance traveled. This supports the idea that the upper body movements documented in the LBP group were driven by the lumbar spine.

6.7 Conclusions

The proposed methods for identifying shift and fidget movement patterns developed in this work were successful at differentiating between LBP and asymptomatic groups. The results highlight the fact that short duration investigations of seated postures do not accurately represent the time varying biological responses to prolonged exposure. The results from this study also support the theory that large changes in posture are a good indicator of the presence of discomfort in asymptomatic individuals (Vergara and Page, 2002) and pain in LBP sufferers. Frequent shifts in lumbar spine posture could be a mechanism for redistributing the load to different tissues of the spine, particularly if some tissues are more vulnerable than others. However, increased movement did not completely eliminate pain in individuals with pre-existing LBP. When one feels movement or a break is necessary, the level of discomfort or pain could be indicating that the process of irritation or damage to the tissues may have already begun. Thus, it is possible that proactively inducing movement may be more beneficial than self-selected movement. However, increased voluntary seated movement is not a mechanism that shows potential to reduce LBP development in individuals with sitting-induced LBP. This highlights the need for the development of other interventions in the workplace to help reduce LBP during seated work.

Chapter 7:
Do simulated seated movement patterns affect spine joint mechanics in an *in vitro* porcine model?

7.1 Synopsis

Background: The mechanical changes that occur in the intervertebral joint during seated postures have been attributed to the flexed posture of the lumbar spine along with prolonged static loading. This study examined the specific seated spine movement patterns observed in low back pain and asymptomatic populations using an in vitro model to quantify mechanical changes in the intervertebral joint associated with the development of low back pain. Three types of seated spine movement patterns were simulated: static profile – sitting with moderate joint flexion and no movement; shift profile – step-like changes in spine joint angle from moderate flexion with periods of static loading, and; fidget profile – small, frequent changes in spine joint angle about one moderately flexed posture.

Methods: This study was performed in two parts. For Parts 1 & 2, thirty-nine porcine cervical (C3/4) spinal units were assigned to one of three movement protocol groups: static, shift or fidget. All specimens underwent passive range of motion (RoM) tests to determine their physiologic flexion/extension range. Individualized angles (50% RoM) and loads (10% of predicted compressive strength) were used for each of the three 2-hour simulated sitting protocols and specimens underwent testing while force plate and motion analysis data were collected. X-rays were obtained before and after the 2 hour protocol with specimens locked in their 50% RoM position and post-protocol RoM tests were performed. Specimen and disc height loss were determined as well as pre- and post-test passive RoM characteristics. In Part 2, the location of force plate centre of pressure (CoP) with respect to the intervertebral disc was quantified throughout the 2-hour protocol for a subset of 15 specimens (n = 5 per group).

Findings: All specimens lost the same amount of disc height (1.58 mm (SD 0.3)) regardless of the amount of movement during the 2 hours. The average angular stiffness increased after 2 hours and there appeared to be a step-like pattern in the post-test RoM torque-angle curve, with regions of reduced stiffness around the pre-test flexion and extension physiological end ranges as well as the 50% flexion angle. The static group exhibited no change in laxity or stiffness around the 50% flexion angle. Shifting caused decreased stiffness and increased laxity in the mid-range. Fidgeting increased the stiffness with no change in laxity. The CoP measurements demonstrated that the load carried through the joint varied by a potential creep mechanism as seen in the static protocol or by changing postures as seen in the shift protocol. The CoP measurements for fidget protocol remained in the same region indicating less variation in the tissue load distribution throughout the 2-hour protocol.

Interpretation: Disc height loss and overall stiffness were independent of the variation in tissue loading caused by the different movement patterns. There is no clear-cut benefit of one movement protocol over the other. However, it is likely that the tissue load distribution changes throughout the 2-hour period accounted for the group differences in the mid-range of the passive test. The fidget protocol appeared to reduce the variation of load carriage through the joint and lead to a more uniform increase in stiffness across the entire RoM. While fidgeting is the movement pattern most likely adopted by individuals who do not develop LBP during sitting, it remains unknown whether fidgeting while sitting is, in fact, beneficial to the spine. Nevertheless, the observed disc height loss and changes in joint mechanics may help explain the increased risk of developing disc herniation and degeneration if exposure to sitting is cumulative over many years.

7.2 Introduction

While sitting is viewed as an occupational task with very few large fluctuations in loads on the different tissues, there is still a range of spine postures adopted and thus time-varying loading patterns. Callaghan and McGill (2001b) classified individuals into two different categories based on the amplitude probability distribution functions (APDF) of the time-varying lumbar spine flexion angle. "Static" sitters used less than 10% of their total lumbar flexion range of motion (RoM) whereas "dynamic" sitters used up to 50% of their range of motion (RoM), but no relationship between sitting strategies and discomfort was established. Vergara and Page (2000; 2002) suggested that static postures may provoke discomfort in sitting while small, quick movements alleviate it. While large movements have also been suggested to be an effect of discomfort (Vergara and Page, 2002), there is little information in the literature regarding how varying spine movement affects the mechanics of the intervertebral joint, specifically height changes and passive RoM at the motion segment level.

It has been shown that a one-hour exposure to sitting conditions caused significant changes in the mechanical properties of the lumbar intervertebral disc (IVD) that led to instability of the motion segment (Wilder et al., 1988), which may present an opportunity for disc herniations to occur. The mechanical changes that occur in the joint during seated postures have been attributed to the flexed posture of the spine along with prolonged static loading that will affect such factors as disc height (Adams et al., 1996), fluid flow (Adams and Hutton, 1983; Adams and Hutton, 1986), the stress profile of the disc (McNally and Adams, 1992; Adams et al., 1994), and ligament length changes (McNally and Adams, 1992). It is thought that alternating periods of activity and rest with posture changes will further boost the fluid exchange and diffusion thus improving lumbar disc nutrition (Adams and Hutton, 1986; McMillan et al., 1996a). However, none of the *in vitro* mechanical changes documented in the literature have been directly associated with *in vivo* spine movement patterns and the

development of low back discomfort or pain. A previous study in this thesis (See Study #4, Chapter 6) documented movement patterns in a low back population exposed to prolonged seated computer work and compared them to an asymptomatic population. The study demonstrated that LBP sufferers moved more than asymptomatic individuals during 90 minutes of seated work and they reported increased LBP over time. Sitting-induced LBP sufferers demonstrated large, frequent shifts in lumbar spine posture while asymptomatic individuals generally exhibited a more static sitting pattern, with smaller movements around a mean posture and reported little to no LBP development. Thus, the current study was conducted in order to examine the specific spine movement patterns documented in LBP and asymptomatic populations using an *in vitro* model so that associated mechanical changes in the intervertebral joint could be quantified. Three protocols were developed in order to simulate three types of seated spine movement patterns:

1) Static pattern – to simulate sitting with moderate joint flexion and no movement.
2) Shift pattern – step-like changes in spine joint angle from a moderate amount of flexion with shorter periods of static loading; this movement pattern simulated the shift pattern observed in LBP sufferers (see Chapter 6, Table 6.2).
3) Fidget pattern – small, frequent changes in spine joint angle about one moderately flexed posture; this movement pattern simulated the fidget pattern observed in asymptomatic individuals (see Chapter 6, Table 6.2).

7.2.1 Purpose

The main purpose of this study was to examine the three simulated seated spine movement patterns using a porcine cervical spine joint model in order to identify where the load is transferred through the joint during a two-hour test as well as IVD height loss and passive flexion/extension range of motion characteristics before and after the test.

7.2.2 Hypotheses

1) IVD height loss will be highest for the shift movement pattern since it combines static loading, flexed postures and large fluctuations in posture.
2) The passive range of motion (RoM) characteristics (i.e. flexion and extension end ranges, stiffness) will be altered after 2 hours of simulated sitting and will differ between the three movement protocol groups.
3) The mechanical changes highlighted in Hypotheses #1 and #2 will be quantifiable as changes in the location of the centre of pressure (CoP) of the specimen on a force plate during the two-hour simulated sitting protocol.

7.3 Methods

7.3.1 Overview of Experiments

This study was performed in two parts. Part 1 consisted of a total of 39 porcine cervical function spinal units (FSU) tested (n = 13 per movement protocol group) and examined specimen height loss and changes in passive RoM characteristics after 2 hours of simulated sitting. Part 2 tested a subset of 15 FSUs (n = 5 per movement protocol group) and measured specimen kinematics during the passive RoM tests and kinetics (i.e. CoP) during the 2 hour movement protocol. The experimental set-up and testing protocol used in Part 1 were also used in Part 2. In addition, Part 2 employed instrumentation and analyses to describe the kinematics and kinetics (i.e. CoP) of the FSU during the 2 hour movement protocol. A justification for the chosen sample size can be found in 0.

7.3.2 Parts 1 & 2: Specimen preparation

Twenty-four porcine cervical functional spinal units (FSU) were obtained from a common source to control for physical activity, diet and age. The cervico-thoracic spine segments (C1

to ~T9) were thawed at room temperature over night and the C3/4 FSU was isolated and used for this study. The surrounding musculature was removed in order to isolate the osseoligamentous structures consisting of two vertebrae, the IVD and all associated ligaments. The exposed endplates of the superior and inferior vertebrae were measured along the anterior-posterior (AP) and medial-lateral (ML) directions. The endplate area was approximated by calculating the area of an ellipse ($\pi/4$ x AP x ML) (Table 7.1). The average area of the superior and inferior endplates represented the area of the FSU and was used to predict the compressive tolerance of the FSU so that the loads applied could be normalized (Parkinson et al., 2005b) (Table 7.1).

Table 7.1: Specimen characteristics used for determining load and posture testing parameters. Thirty-nine specimens were tested in Part 1. A subset of 15 specimens was also tested in Part 2.

	Protocol Groups		
PART 1	**Shift (n = 13)**	**Fidget (n = 13)**	**Static (n = 13)**
Endplate Area (mm^2)	701.0 (55.0)	701.0 (55.0)	677.8 (50.7)
Predicted Compressive strength (kN)	10.2 (0.7)	10.2 (0.7)	9.9 (0.7)
Passive Range of Motion values (°)			
Total RoM	14.8 (2.2)	15.5 (1.7)	15.7 (1.7)
Flexion angle	11.8 (2.4)	11.4 (1.9)	11.8 (1.9)
Extension angle	-3.0 (2.0)	-4.1 (1.4)	-3.9 (2.3)
50% RoM angle	4.5 (1.8)	3.7 (1.5)	4.1 (1.8)
PART 2	**Shift (n = 5)**	**Fidget (n = 5)**	**Static (n = 5)**
Endplate Area (mm^2)	701.4 (48.2)	706.7 (54.2)	652.9 (73.4)
Predicted Compressive strength (kN)	10.2 (0.7)	10.3 (0.7)	9.5 (1.0)
Passive Range of Motion values (°)			
Total RoM	13.6 (2.4)	15.4 (1.5)	16.2 (1.3)
Flexion angle	10.4 (3.2)	11.2 (1.6)	11.2 (1.6)
Extension angle	-3.2 (2.1)	-4.2 (0.8)	-5.0 (2.5)
50% RoM angle	3.8 (2.1)	3.7 (1.2)	3.2 (1.9)

Prior to mounting, the anterior processes and exposed facets were trimmed to ensure that the endplates were in contact with the surface of custom aluminum cups used in the load application. Three small 10mm long screws were inserted into the anterior, left and right aspects of the inferior (C4) vertebral body just below the superior endplate in order to landmark points to be digitized for motion analysis of the AP and ML axes of the disc.

These landmarks were used in Part 2 of the study in order to define the IVD co-ordinate system (this will be described later in Section 7.3.7.1). In order to rigidly fix the specimen to the custom aluminum cups, screws were inserted into the anterior processes of the FSU and 18-gauge wires were looped around the posterior lamina and tightened. Once the specimens were fixed to the cups, non-exothermic dental plaster (Denstone, Miles, South Bend, USA) was poured around the specimen into the cup and allowed to dry in order to solidify the fixation of the screws and wires.

7.3.3 Parts 1 & 2: Instrumentation

The superior aluminum cup was mounted to a custom flexion/extension jig, which was fixed to the actuator of a servo-hydraulic materials testing machine (8872, Instron Canada, Toronto, ON, Canada) (Figure 7.1). Flexion/extension torque was applied to the jig through a rotational brushless servomotor (Kollmorgen, Model AKM23D-BNCNC-00, Danahar Motion, Illinois, USA) that was controlled through a PCI bus motion controller (model DMC1840, Galil Motion Control, Rocklin, USA). Torques were measured with a torque transducer (SensorData Technologies Inc., Model T120-106-1K, Sterling Heights, MI., USA) mounted in series with the servomotor. Throughout testing, Instron compressive load, vertical position of the actuator, servomotor angle and torque were sampled at 25Hz by the same system that controlled the rotational servomotor. The lower aluminum cup rested on a bearing table that allowed for unconstrained horizontal plane translations and axial rotation (about the table's vertical axis) (Figure 7.1).

7.3.4 Part 2: Instrumentation

The bearing table was rigidly fixed to and assumed to be parallel to a six-degree of freedom force plate (MC12-4000, Advanced Medical Technology Inc., Waterdown, Massachusetts, USA) that measured the forces and moments applied. The signals from the force plate were amplified (x1000) (MSA-6 mini-amp, Advanced Medical Technology Inc., Waterdown, Massachusetts, USA) and digitally sampled at a rate of 30Hz using a 12-bit analogue-to-

digital conversion system (National Instruments Corporation, Austin, TX, USA). Instron compressive load, vertical position of the actuator and servomotor torque were also sampled by this system so that data from the two systems could be synchronized in time.

An opto-electric motion tracking system (Polaris, Northern Digital Inc., Waterloo, ON, Canada) was used to locate the four corners of the force plate, the landmarks of the IVD and to track the motion of the FSU in space. A four-marker rigid body probe was used to digitize the 3-dimensional co-ordinates of the force plate corners as well as the three landmark screws inserted into the inferior (C4) vertebral body. Two more 4-marker rigid bodies were secured to the top and bottom cups so that cup motion could be tracked (Figure 7.1). The 3-D transformation analyses will be described in detail in Section 7.3.7. The motion data were digitally sampled at 30Hz and synchronized in time with the force plate data. The synchronization method consisted of an instrumented computer mouse that delivered a 5V pulse to initiate data collection of the force plate collection software when the Polaris recording software was initiated.

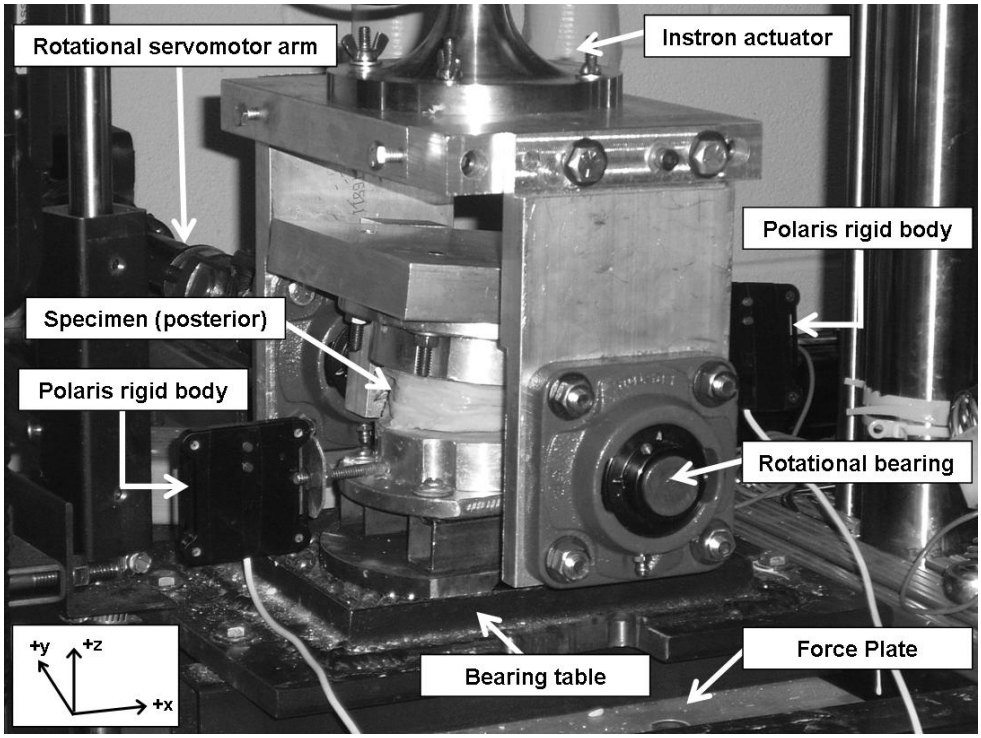


Figure 7.1: A mounted specimen in 50% range of motion (RoM). The force plate co-ordinate system (for Part 2) is indicated in the bottom left-hand corner of the figure.

7.3.5 Parts 1 & 2: Loading protocols

The FSU in the aluminum cups was mounted into the jig and preloaded at 300N for 15 minutes prior to testing (Callaghan and McGill, 1995; Yingling et al., 1999; Gunning et al., 2001; Callaghan and McGill, 2001a). During this time, the servomotor sought a position where no external flexion/extension torque was present and this position was taken as neutral. After this period, five cycles of passive flexion and extension were performed under 300N of compression in order to obtain flexion torque-angle curves. The torque was measured directly from the transducer in series with the rotational servomotor and the angle was the instantaneous position of the servomotor. Data from the last three cycles were used in the analyses. The "physiological" end-range angles for flexion and extension angles were obtained from the torque-angle curve by identifying the angle where the curve began to

deviate from linearity (Figure 7.2). The slope of the line followed the linear portion of the torque-angle curve, either on the "top" curve for flexion loading, or the "bottom" curve for extension loading (Figure 7.2). The starting point of these lines were where the torque-angle curve crossed the x-axis, at zero moment. Specimen-specific RoM was determined to be the range between end of physiological flexion and extension, and this value was used to normalize testing angles. After the passive test, the 50% RoM angle was determined by dividing the total RoM by 2 (i.e. flexion end-range minus extension end-range) and adding that value to the extension end-range angle (see Figure 7.2). The FSU was locked in its 50% RoM angle with wires wrapped around screw heads embedded in the front edges of the aluminum cups. The FSU was then removed from the jig and a sagittal plane X-ray was obtained from the specimen's right side. X-rays were developed using a digital X-ray system (Kodak DirectView CR500, Carestream, Toronto, ON, Canada).

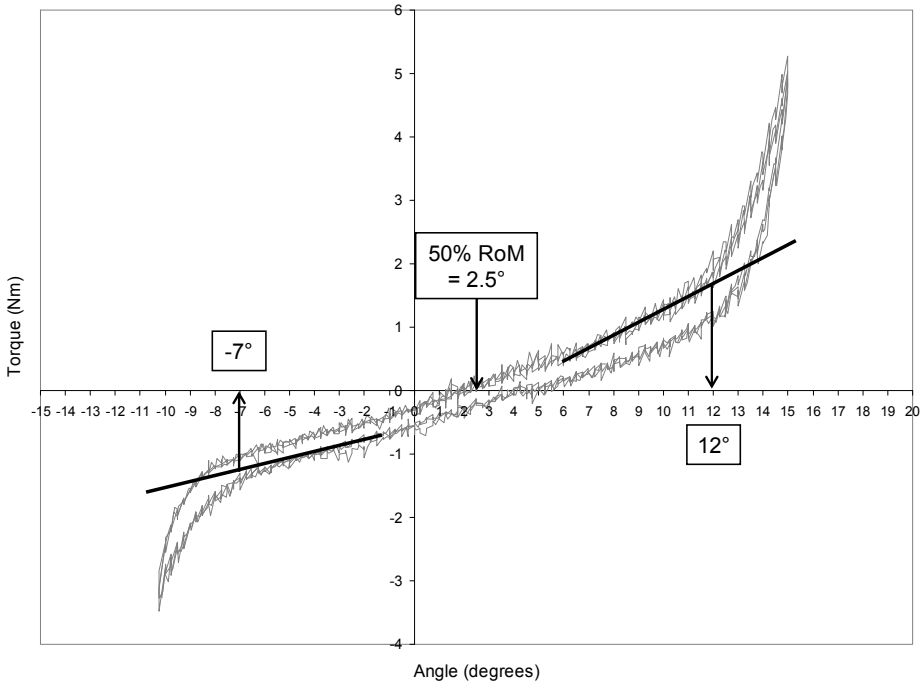


Figure 7.2: Torque-angle curve obtained from three cycles of a passive flexion/extension range of motion (RoM) test. Flexion and extension end ranges were determined to be the angles at which the slope deviated from linearity. The total RoM was the difference between these two angles, and the 50% RoM angle was calculated to be the angle in between the end ranges.

Three 2-hour movement protocols were designed to expose the FSU to simulated seated movement patterns (Figure 7.3). The "shift" and "fidget" profiles were chosen to represent two prolonged seated movement patterns previously observed in a low back pain and asymptomatic population, respectively (see Study #4, Chapter 6). The specimens in the static group were loaded at 50% RoM for 120 minutes (Figure 7.3A). The "fidget" protocol was a 5% RoM posture change alternating between flexing and extending followed by a return to 50% RoM every 50 seconds (Figure 7.3B). This pattern was repeated for 120 minutes. The "shift" protocol consisted of 8-minute time blocks at different postures starting at 50% RoM, then shifting to 30% RoM, back to 50% and then to 70% (Figure 7.3C). During each movement protocol, the specimen was statically compressed to 10% of its compressive strength and maintained at this level under load control. This value corresponds with seated joint compression values calculated from rigid link and single-muscle equivalent spine models previously calculated in our lab (unpublished data). Briefly, the load magnitude applied to the specimen was determined by normalizing the load calculated during sitting to a percentage of maximum compressive strength for individual human subjects using the regression equation proposed by Genaidy et al. (1993). The percent of maximum strength was then appropriately scaled to the porcine compressive strength based on the physical dimensions (i.e. endplate area) of the specimen (Parkinson et al., 2005b).

Throughout testing, specimens were wrapped in saline-soaked gauze and plastic wrap to reduce moisture loss. Upon completion of the 2-hour movement protocol, the specimen was locked into the 50% RoM posture, removed from the jig, X-rayed, and then re-mounted in order to perform a final passive RoM test.

7.3.5.1 Part 2: Passive RoM and 2 hour movement data collection protocols

Force plate and motion analysis data were collected during the 2-hour movement protocol. Synchronized recording was initiated for the force plate and motion analysis systems before

the 2 hour protocol was started. Thirty-minute time blocks of force plate and motion analysis data were recorded because of equipment data acquisition limitations. This meant that the recording systems were restarted three times throughout the 2 hour protocol. Collection restarting took approximately 10 seconds. Force plate and motion analysis data were also collected for the passive tests in addition to the torque and angle data. The last three consecutive RoM cycles were used in subsequent analyses.

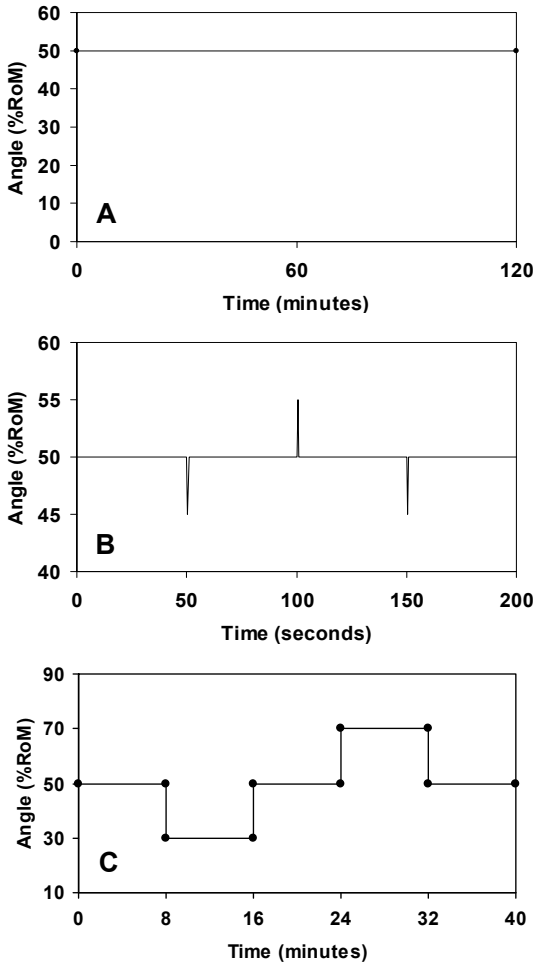


Figure 7.3: Schematic representation of the three movement protocols: A) static, B) fidget and C) shifts. Each protocol continued for 2 hours. Note that both the x- and y-axes are individually scaled for each graph for a close-up demonstration of the timing and amplitude of the movement protocol.

7.3.6 Parts 1 & 2: Data Analyses

Gross specimen height loss over 2 hours was determined as the change in position of the Instron actuator. Actuator position data were averaged over the first 10 seconds and the last 10 seconds of the movement protocol and the difference between these two values was calculated. Disc region-specific disc height loss was measured from the X-rays taken in the 50% RoM angle before and after the 2-hour movement protocol. Four points were digitized from each X-ray: the posterior and anterior corners of the superior (C3) and inferior (C4) endplates. The perpendicular distance was calculated for of the C4 anterior and posterior points to the line connecting the C3 anterior and posterior points (Figure 7.4).

The passive torque-angle curves from the passive RoM tests were obtained from the torque transducer and position of the rotational servomotor. The passive RoM curves were characterized in several different ways and will be explained further in the Results section. In addition to these methods, the average flexion-extension stiffness was calculated from the slope of the line connecting the pre-test flexion and extension end-range angles and corresponding torque values from the pre- and post-test passive RoM curves.

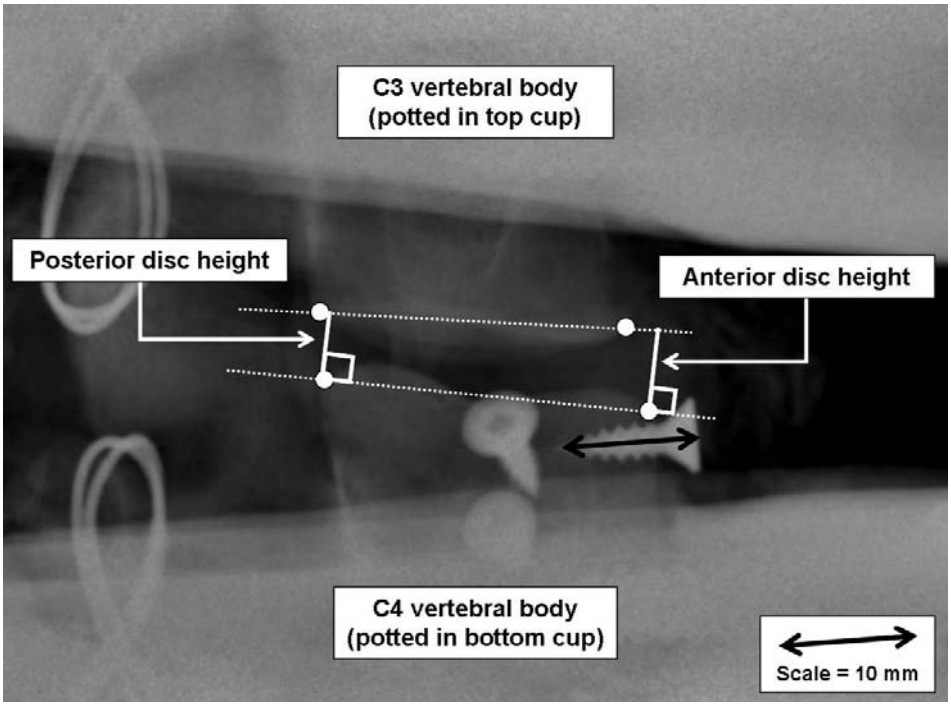


Figure 7.4: Pre-test X-ray of a specimen locked in 50% RoM. The anterior and posterior corners of the C3 and C4 vertebral body endplates were digitized (white dots). The perpendicular distances of the C4 posterior and anterior points to the C3 endplate line were calculated. The image was scaled using the screw inserted through the anterior surface of the C4 vertebral body.

7.3.7 Part 2: Force plate and kinematic data analyses

The forces and moments were continuously collected from the force plate throughout the 2-hour movement protocol and were used to calculate the centre of pressure (CoP) at the level of the IVD using the follow formulae (Robertson et al., 2004):

$$CoP_x = -\frac{(My + (c - \tau) * Fx)}{Fz} + a \qquad \textbf{(Equation 7.1)}$$

$$CoP_y = \frac{(Mz - (c - \tau) * Fy}{Fz} + b \qquad \textbf{(Equation 7.2)}$$

Where:

Fx, Fy, Fz, Mz, My = forces and moments measured from force plate
(a, b ,c) = true origin of the force plate (from specifications sheet)
τ = height of the origin of the IVD, where CoP will be located

The CoP represents a summation of all the forces applied to the force plate (Winter, 1990). In the current study, the CoP was a surrogate measure of how the compressive force translated through the specimen joint. For this purpose, the CoP was transformed so that it was aligned with the disc AP axis and thus a relation between the CoP movement and disc space could be made.

7.3.7.1 Co-ordinate system and transformation matrix definitions

The digitized anterior, right and left IVD landmark co-ordinates were used to reconstruct the posterior IVD point. The plane containing these three points was defined and the AP axis was determined to be the line perpendicular to the ML axis that runs through the anterior point. The posterior disc point was defined as lying along the AP axis, posterior to the anterior point at the measured AP disc width of the given specimen.

The IVD co-ordinate system was defined as the x-axis aligned with the AP IVD axis (positive towards anterior), the positive y-axis directed towards the left-side of the IVD and the positive z-axis directed vertically. The 4-marker rigid body fixed to the bottom cup was used to track the IVD during the motion trials. A 4 x 4 homogeneous transformation matrix that described the relationship between the IVD and the tracking marker was defined during the IVD landmark digitizing process. The Polaris motion analysis system output the position of the origin of the 4-marker rigid body and its orientation in the global co-ordinate system using quaternions. A quaternion is composed of four components – a scalar part (q_0), which represents a rotation about an axis in 3D space (a 3-part vector (q_1, q_2, q_3)) (Horn, 1987; Dumas et al., 2004). Quaternions were converted into the corresponding orthonormal matrix using the formula presented by Horn (1987).

The force plate co-ordinate system was defined using the digitized corners. Its axes were roughly aligned with the directions of the IVD co-ordinate system (Figure 7.4).

Summary of co-ordinate systems and 4 x 4 transformation matrices:

1) [T(g/d)] – position and orientation of the IVD in the global co-ordinate system; defined from digitized points.
2) [T(g/f)] – position and orientation of the force plate in the global co-ordinate system; defined from digitized points.
3) [T(g/m)] – position and orientation of the tracking rigid body marker attached to bottom cup in global co-ordinate system; defined from Polaris output during IVD digitizing process
4) [T(d/m)] – position and orientation of the tracking 4-marker rigid body in the IVD co-ordinate system; defined using the following formula:

[T(d/m)] = inverse[T(g/d)] * [T(g/m)] **(Equation 7.3)**

7.3.7.2 Computation of the CoP in the IVD co-ordinate system

The CoP was calculated in the force plate co-ordinate system and was transformed on a frame-by-frame basis into the IVD co-ordinate system using the following equation:

$$\mathbf{CoP_{IVD(n)} = [T(d/m)] * [T(m/g)]_n * [T(g/f)] * CoP_{f(n)}} \qquad \textbf{(Equation 7.4)}$$

Where:

$CoP_{IVD(n)}$ = CoP in the IVD co-ordinate system in frame "n"

$CoP_{f(n)}$ = CoP in the force plate co-ordinate system in frame "n"

$[T(m/g)_n]$ = the homogeneous transformation matrix from the global co-ordinate system to the tracking 4-marker rigid body; defined as the inverse of $T(g/m)_n$ obtained from the Polaris output of the rigid body.

[T(d/m)] = fixed relationship between IVD and tracking rigid body; defined above.

[T(g/f)] = fixed relationship between force plate and global co-ordinate system; defined above.

The z-component (or vertical component) of the CoP was assumed to be negligible and the CoP_{IVD} movement was projected onto the force plate X-Y plane.

7.3.7.3 Computation of specimen kinematics during passive RoM test

The x- and y-direction translations of the specimen on the bearing table were quantified during the passive RoM tests before and after the 2 hour movement protocol using the 4-marker rigid body marker attached to the bottom cup. The axial rotation of the bottom cup was ignored. These measurements gave an indication of the off-axis "coupled motion" of the specimen; in other words, the translational motion of the specimen during sagittal rotation would provide a measure of joint laxity.

The marker translations were transformed into the force plate co-ordinate system using the following calculation:

$$[T(f/m)]_n = \text{inverse}[T(g/f)] * [T(g/m)]_n \qquad \textbf{(Equation 7.5)}$$

Where:

$[T(f/m)]_n$ = the position and orientation of the tracking marker in the force plate co-ordinate system in frame "n"

The x- and y-positions of the bottom cup throughout the test were expressed as a change from the position at Time = 0. The x- and y-translations were extracted at three common angles throughout the passive RoM cycles: 10° of flexion, 4° of extension and the specimen-specific 50% RoM angle.

7.3.7.4 Data reduction of CoP variables during prolonged test

The CoP was located with respect to the anterior and posterior edges of the disc space, and its position was expressed as a percentage of a specimen's measured AP disc length. The CoP location was averaged over 1 second at time zero, 4 minutes in, then every 8 minutes (to match with the middle of each 8 minute shift cycle) in order to quantify the general time-varying trend for CoP direction of movement. In addition, the range of CoP movement and the total excursion of the CoP were calculated from the 2-hour collection period.

7.3.8 Parts 1 & 2: Statistical Analyses

Specimen height loss was assessed using a one-way analysis of variance (ANOVA) to test protocol group differences, and disc height loss was assessed with a two-way mixed general linear model (GLM) with a repeated measure on one factor to test group and disc region

(repeated measure) effects and interactions. All passive RoM variables were analyzed using a two-way repeated measures mixed GLM (group*time (pre vs. post; repeated measure)). One-way ANOVAs were performed to assess group differences in CoP location, range of movement and total excursion. Significant pair-wise differences were tested with Tukey's post hoc tests. All tests with $P < 0.05$ were considered statistically significant.

7.4 Part 1: Results

7.4.1 Disc Height Loss

Initial disc height, measured from X-rays in the 50% RoM posture, was significantly different between the anterior and posterior disc edges ($P < 0.0001$) for all specimens (posterior disc height = 4.32 mm (SD 0.83); anterior disc height = 5.27 mm (SD 0.96)). Gross height loss (measured from the actuator position change) over the 2-hour protocol was not significantly different between movement protocol groups (gross height loss for all specimens = 2.37 mm (SD 0.34)). When disc heights of the anterior and posterior edges of the disc were compared among the three protocols, all groups lost a similar amount of disc height (mean disc height loss = 1.58 mm (SD 0.3)). However, a significant effect of disc location ($P < 0.0001$) was observed. There was more disc height loss in the posterior region (1.81 mm (SD 0.37)) than in the anterior region (1.34 mm (SD 0.34)). The average amount of height lost across the disc (i.e. 1.58 mm) accounted for 67.3% (SD 11.8) of the total specimen height loss (i.e. 2.37 mm).

7.4.2 Passive Range of Motion

All specimens demonstrated a similar passive torque-angle response after 2 hours of the movement protocol **(**Figure 7.5). There appeared to be a step-like pattern in the torque-angle curve, with plateaus (or regions of reduced stiffness) around the pre-test flexion and extension physiological end range angles as well as at the 50% RoM angle (Figure 7.5). Several methods were utilized to quantify the torque-angle curve.

7.4.2.1 Total range of motion and average stiffness

The post-test end ranges (i.e. where the slope of the curve increases sharply) were determined to be the angle in flexion or extension where there was a sharp increase in slope from the plateaus around the flexion and extension pre-test end ranges (asterisks, Figure 7.5). The pre-test RoM for all specimens was 15.4° (SD 1.7) (Table 7.1) and was significantly lower then the post-test RoM (22.6° (SD 2.8); $P < 0.0001$). All specimen post-test flexion (17.4° (SD 2.3)) and extension (-5.3° (SD 2.2)) angles were greater than the pre-test angles (shown in Table 7.1). The pre-test average stiffness was similar for all three protocol groups (pre-test stiffness = 0.26 Nm/° (SD 0.07)), and was significantly different across all groups after the 2-hour loading protocol (post-test stiffness = 0.62 Nm/° (SD 0.17); main effect of time: $P < 0.0001$).

7.4.2.2 Angle at a given torque (Torque control):

Before the 2-hour protocol, the pre-test torque at maximum flexion and extension did not differ between the three protocol groups (max flexion torque = 2.4 Nm (SD 0.8); maximum extension torque = -1.5 Nm (SD 0.5) (Table 7.2). These pre-test torque values were used to determine the corresponding post-test flexion, extension and 50% RoM angles (Table 7.2). In other words, torque was kept constant and the corresponding angles were determined from the pre- and post-passive tests. It was found that the angle decreased in both flexion and extension for all specimens (Table 7.2). On average, the post-test flexion angle was 9.0° (SD 2.9) and the extension angle was 2.0° (SD 2.2). There was a significant group*time interaction for the 50% RoM angle ($P = 0.002$). The shift group's 50% RoM angle was 1.0° (SD 1.4) ***greater*** after 2 hours of loading ($P = 0.03$); the fidget group's 50% RoM angle was 1.3° (SD 1.5) ***less*** ($P = 0.003$) and the static group's angle stayed relatively the same (0.04° (SD 1.4)) after the 2 hours (Table 7.2). Furthermore, the static group post-test 50% RoM angle was significantly different than both the shift ($P = 0.003$) and the fidget ($P = 0.0002$) groups (Table 7.2).

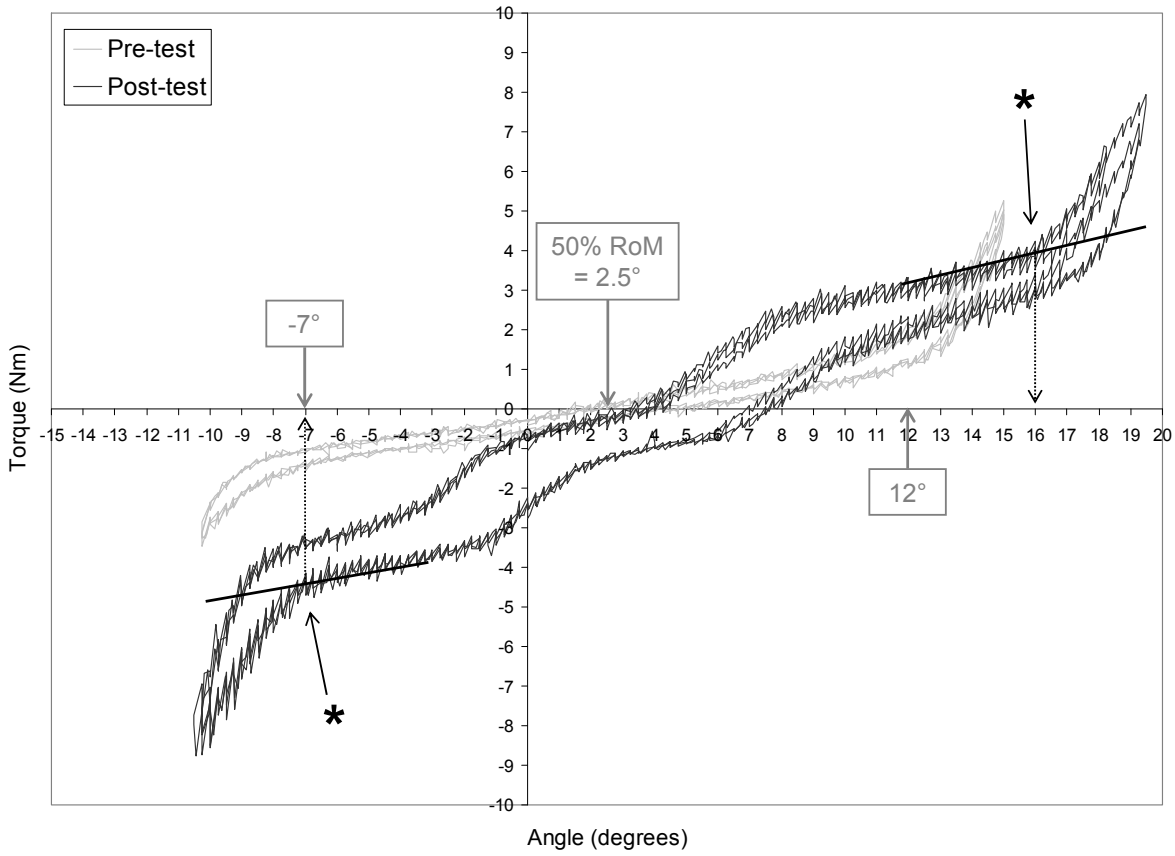


Figure 7.5: Torque-angle curve obtained during the pre- (light grey) and post-test (black) passive range of motion (RoM) trials. Each curve is comprised of 3 cycles. The grey boxes indicate the end range and 50% RoM angles determined from the pre-test. The asterisks (*) indicate the flexion and extension end range angles for the post-test.

Table 7.2: Pre-test end range angles (flexion, extension) and 50% RoM angle were used to determine pre-test torque values. The post-test end range and 50% RoM angles were then determined using the pre-torque values. There was a significant time effect for the flexion and extension angle for all groups ($P < 0.0001$). For the 50% RoM angle, there was a significant group*time interaction ($P = 0.009$) whereby the shift post-test 50% angle was greater ($P = 0.02$) and the fidget post-test 50% RoM angle was less ($P = 0.02$) than their respective pre-test angles. Significant differences ($P < 0.05$) are denoted by differing letters.

	FIDGET		SHIFT		STATIC	
FLEXION	**pre**	**post**	**pre**	**post**	**Pre**	**post**
Angle (degrees)	11.4 (1.9)	8.3 (3.1)	11.8 (2.4)	9.3 (3.3)	11.8 (1.9)	9.5 (2.5)
Pre Torque (Nm)	2.6 (0.8)		2.2 (0.7)		2.5 (0.8)	
EXTENSION						
Angle (degrees)	-4.0 (1.8)	1.1 (2.0)	-2.9 (2.2)	3.3 (1.5)	-3.3 (1.9)	2.0 (2.2)
Pre Torque (Nm)	-1.4 (0.4)		-1.3 (0.3)		-1.5 (0.8)	
50% RoM ANGLE						
Angle (degrees)	3.8 (1.7) [a]	***2.5 (2.7)*** [b]	4.9 (1.5) [a]	***6.2 (2.3)*** [c]	4.6 (1.6) [a]	4.9 (1.7) [a]
Pre Torque (Nm)	0.4 (0.4)		0.7 (0.5)		0.7 (0.8)	

7.4.2.3 Torque at a given angle (Position control)

The pre-test end range and 50% RoM angles were used to determine the torques at the given angle in both passive RoM curves. The main effect of protocol group was not significant at any of the three angles (flexion, extension, 50% RoM). However, even though the slope of the torque-angle curve leveled off around the end ranges (see Figure 7.5), more torque was required to reach the pre-test end range angles in both flexion and extension (Figure 7.6, $P < 0.0001$). Interestingly, the torque at the 50% RoM angle was similar before and after the 2-hour protocol (Figure 7.6).

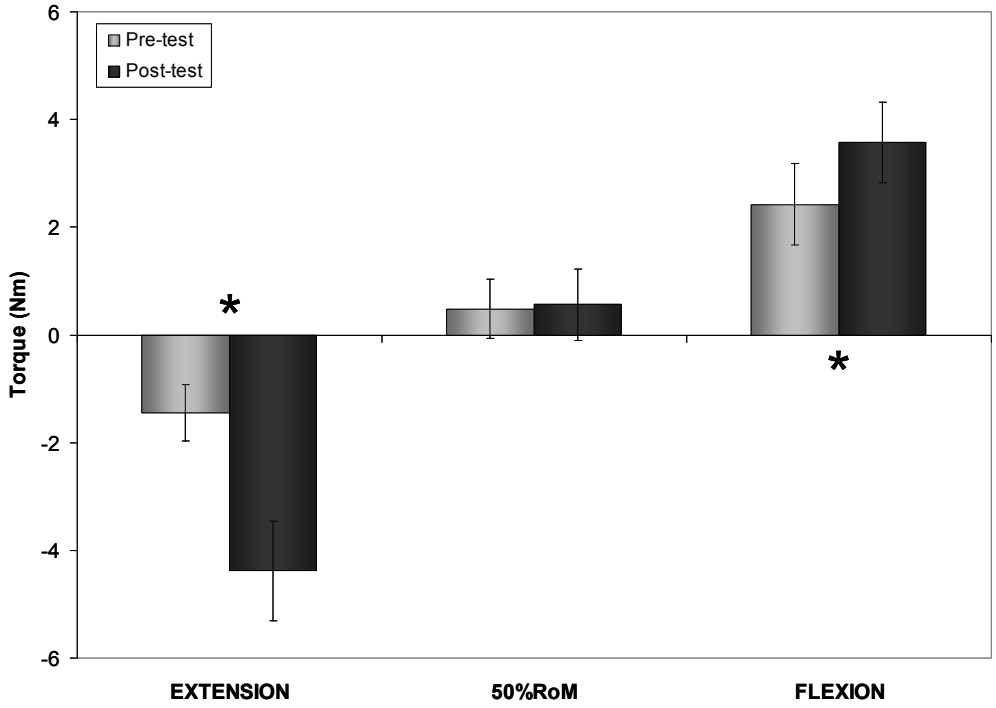


Figure 7.6: Torque values at 50% RoM, flexion and extension end range angles for the pre- and post-test passive range of motion (RoM) trials. At each angle, the pre-test torque was significantly different than the post-test torque (The asterisks (*) indicate that the pre-test and post-test values are significantly different ($P < 0.05$)).

7.4.2.4 Neutral zone:

The +/-1Nm neutral zone (NZ) (Panjabi, 1992) end ranges were determined before and after the 2-hour protocol. There were no differences in the angle range of the NZ between protocol groups, but all specimen NZ ranges decreased in width (pre-test NZ range = 10.7° (SD 2.6); post-test NZ range = 6.0° (SD 1.9); $P < 0.0001$).

7.5 Part 2: Results

7.5.1 Specimen kinematics during passive RoM tests

Upon observation of the individual specimen translation time histories, no distinct patterns of movement were noted. For example, some specimens translated towards the posterior in flexion and anterior in extension while others exhibited the opposite. For this reason, the resultant translations were calculated in order to identify combined movements in both the x- and y-directions regardless of direction. There were no significant effects or interaction of time or group for the amount of translation of the bottom cup quantified during the passive RoM test at 10° of flexion (Figure 7.7). The fidget and static specimens tended to translate less in flexion after the 2 hour protocol, but this difference was not significant (Figure 7.7). At 4° of extension, all specimens translated more after the 2 hour movement protocol (pre-test translation = 0.19 mm (SD 0.09); post-test translation = 0.33 mm (SD 0.22); $P = 0.0008$; Figure 7.7). For the 50% angle, there was a significant group*time interaction ($P = 0.05$; Figure 7.7): the shift specimens translated further in the post-test than they did in the pre-test (pre-test translation = 0.21 mm (SD 0.15); post-test translation = 0.45 mm (SD 0.30); $P = 0.01$) while the fidget and static specimen translations did not change (Figure 7.7).

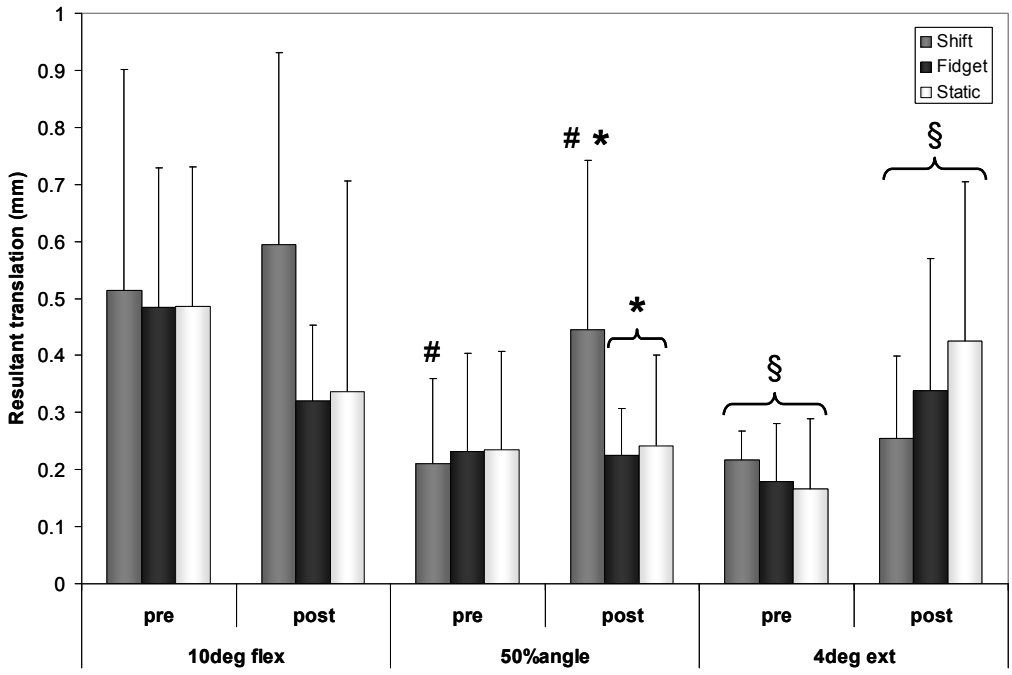


Figure 7.7: Resultant translation of the C4 vertebra (potted in the bottom cup) during the passive RoM test before (pre) and after (post) the 2 hour movement protocol. Resultant translations from zero were obtained to 10° of flexion, 4° of extension and the specimen-specific 50% RoM angle. There was a significant effect of time ($P = 0.008$) for translation in extension (denoted by "§"). For the 50% angle, there was a significant group*time interaction ($P = 0.05$): (#) indicates that the shift translation was significantly different before and after 2 hours ($P = 0.01$); (*) indicates that the shift post-test translation was significantly different from the fidget and static groups ($P < 0.02$).

7.5.2 Centre of pressure (CoP) measurements

Each of the three movement protocol groups had very distinct CoP movement patterns (Figure 7.8). Both the static and fidget CoP started to move towards the posterior edge of the disc and then gradually tracked anterior over the 2 hours (Figure 7.8A, B). The fidgets every 50 seconds are apparent in the CoP excursions and the size of the excursions appeared to increase over time (Figure 7.8B). The shift CoP moved towards the posterior when the specimen shifted to 30% flexion and towards the anterior in 70% flexion (Figure 7.8C). These CoP excursions increased over time and even moved outside of the disc space (Figure

7.8C). It is also interesting to note the direction of the CoP excursions within each 8 minute shift: the CoP tended to move back towards the 50% RoM CoP position (Figure 7.8C). The general shift CoP movement trend is shown as an average of all 5 specimens in Figure 7.9. Of note are the differences between the fidget and static groups: the static group CoP drifted towards the anterior while the fidget group CoP stayed in relatively the same position throughout the 2-hour protocol (Figure 7.9).

The range of CoP movement in the AP axis throughout the 2-hour movement protocol was significantly smaller for the fidget group than the other two groups (Figure 7.10; $P < 0.05$). The CoP for the fidget group stayed within the posterior third of the IVD while the shift CoP moved from outside the posterior edge of the IVD through the posterior half of the IVD A/P length (Figure 7.9 and Figure 7.10). The static group CoP drifted towards the centre of the disc along the AP axis (Figure 7.10). Interestingly, while the range of the fidget CoP was significantly smaller than that of the shift group, the total CoP excursion for the fidget group was similar (fidget CoP excursion = 216.5 mm (SD 20.7); shift CoP excursion = 210.1 mm (SD 50.2)). The total CoP excursion in the static group was significantly less than the other two groups ($P < 0.05$).

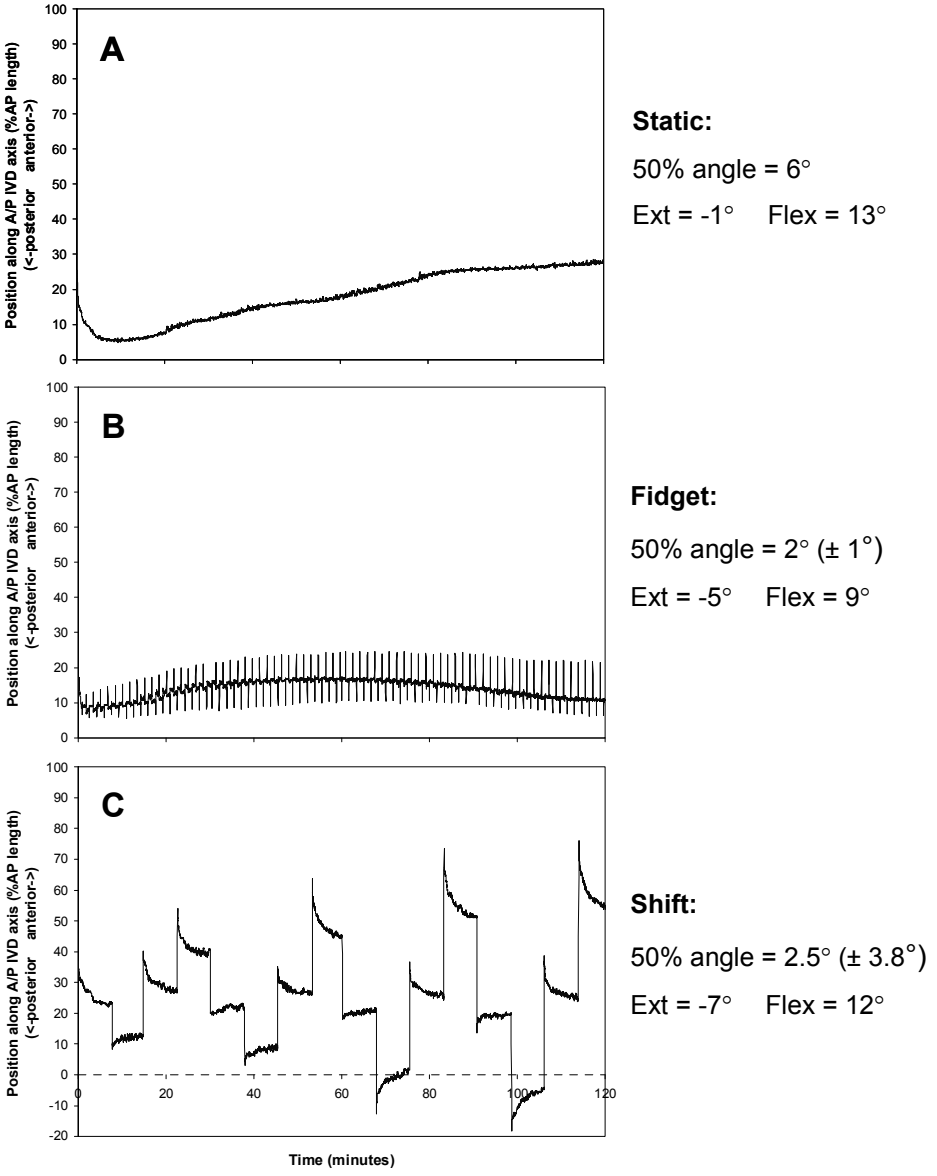


Figure 7.8: Representative time-varying anterior/posterior centre of pressure (CoP) excursion of specimens in the static (A), fidget (B) and shift (C) movement protocol groups. On the y-axis, "0" indicates the posterior edge of the disc. The values on the right side of the figure indicate the motion parameters for the representative specimens used: the pre-test flexion (Flex) and extension (Ext) are indicated, as well as the experimental 50% RoM angle used. The number in brackets beside the fidget and shift 50% angle values refers to the fidget and shift magnitude, respectively.

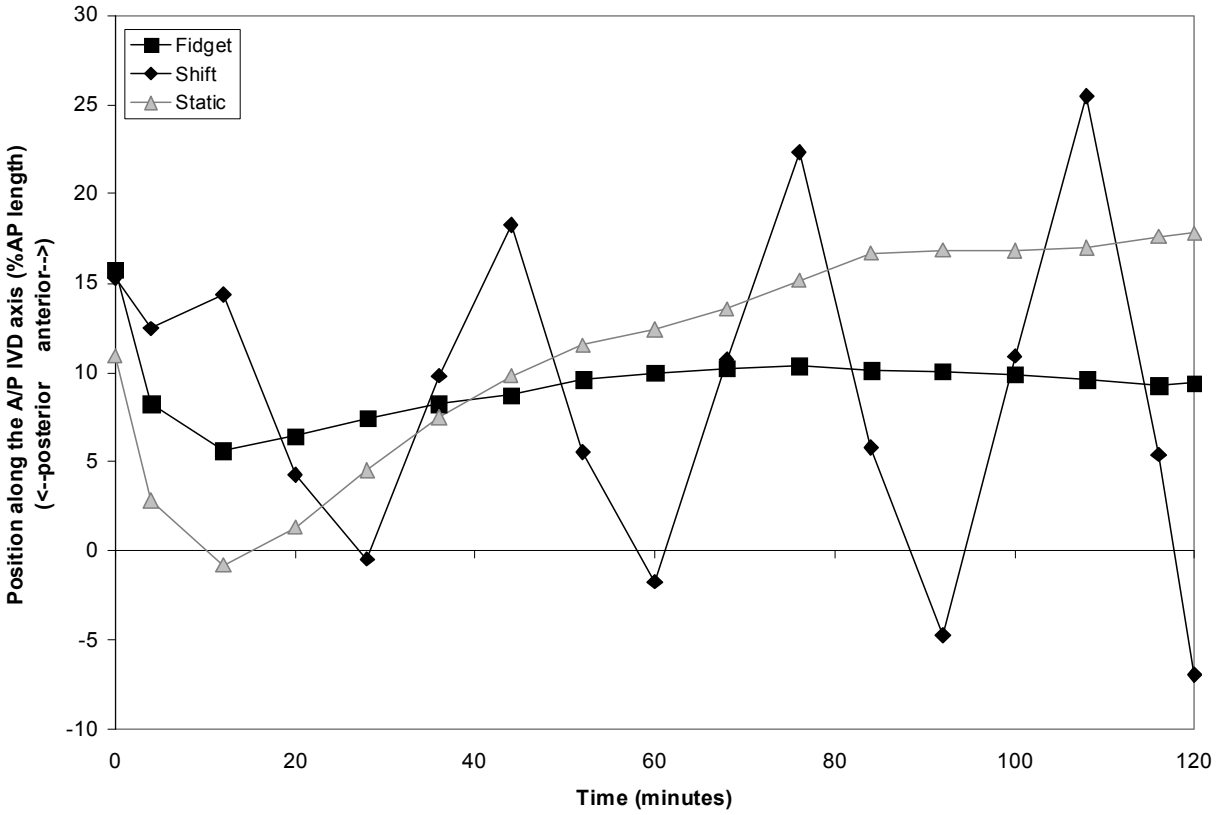


Figure 7.9: Mean position of the centre of pressure (CoP) every 8 minutes. The y-axis indicates the position along the anterior/posterior (A/P) axis of the intervertebral disc (IVD) as a percentage of AP disc length. Each point represents an average of 5 specimens within each group.

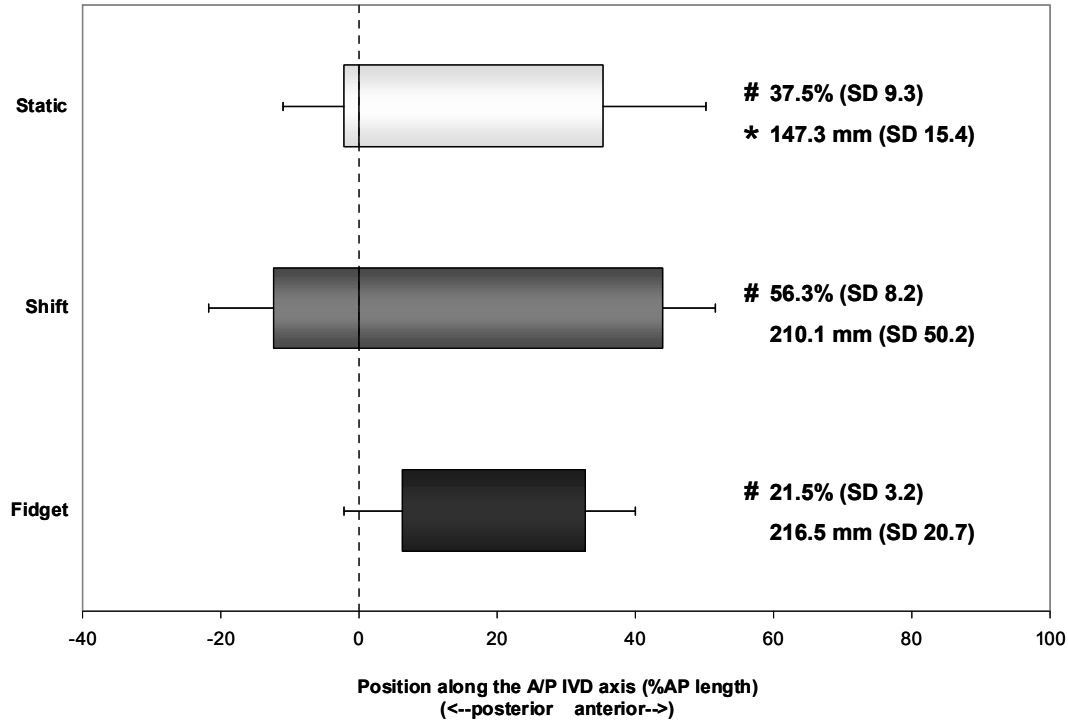


Figure 7.10: The location of the CoP within the intervertebral disc (IVD) along the anterior/posterior (A/P) axis. The range of CoP movement (top number on right) is expressed as a percent (%) of A/P length and the total CoP excursion (bottom number on right) is in millimeters (mm).

- denotes that the range of CoP movement was significantly different between all three groups ($P < 0.05$)
* - denotes that the static CoP excursion was significantly different from the other two groups ($P < 0.05$)

7.6 Discussion

Three protocols were developed in order to simulate three types of seated spine movement patterns. The static protocol simulated 2 hours of sitting in one posture. The shift protocol simulated infrequent but large changes in posture, similar to the seated movements observed in a group of LBP sufferers. The fidget protocol replicated small, frequent movements about one posture, demonstrated by a group of asymptomatic individuals. The results of this study

indicate that all FSUs lost the same amount of disc height and became stiffer after 2 hours regardless of the amount of movement around one posture. However, the type of movement affected the joint stiffness and laxity around the angle at which the FSU spent the majority of its time during the simulated sitting. Specifically, the static group exhibited no change in laxity or stiffness in the mid-range of the passive RoM. Shifting caused decreased stiffness and increased laxity in the mid-range. Fidgeting increased the stiffness with no change in laxity. The CoP measurements demonstrated that the load carried through the joint varied by a potential creep mechanism as seen in the static protocol or by changing postures as seen in the shift protocol. The CoP measurements for fidget protocol remained in the same region indicating less variation in the tissue load distribution throughout the 2-hour protocol. Disc height loss and overall stiffness were independent of the variation in CoP (i.e., variation in tissue loading). However, it is likely that the tissue load distribution changes throughout the 2-hour period accounted for the group differences in the mid-range of the passive test (and will be discussed later on in this section). The CoP results will be discussed in concert with the disc height loss and changes in joint mechanics to provide insight into the benefits and consequences of the different sitting behaviours observed in LBP and asymptomatic populations.

The results of this study need to be discussed within the context of the *in vitro* model's strengths and limitations. A porcine cervical spine model was used instead of human cadaveric specimens in the current study. The porcine cervical spine has generally been deemed an acceptable model and has been used by a number of researchers (Keller et al., 1990; Ekstrom et al., 1996; Yingling et al., 1997; Gunning et al., 2001; Callaghan and McGill, 2001a; Dickey and Gillespie, 2003; Ekstrom et al., 2004; Parkinson et al., 2005b). This model has been shown to have similar anatomy and function to the human lumbar spine (Yingling et al., 1999; McLain et al., 2002). Detailed dissection of the porcine disc has shown that the fiber orientation and general structure make it a suitable model for the human disc (Tampier et al., 2007). Total RoM documented in this study was 15.4° (SD 1.7) and

falls within the flexion/extension range of 16° to 19° for the human L4/5 joint *in vivo* (Pearcy et al., 1984; Dvorak et al., 1991; Kanayama et al., 1996; Frobin et al., 1996) and *in vitro* (Wilke et al., 1997). While it is understood that that the porcine model is simply a surrogate for the human lumbar spine, it offers the advantage of control over factors such as age, size, genetics, diet and level of physical activity, thus reducing the variability of the results (Yingling et al., 1999; Callaghan and McGill, 2001a; Parkinson et al., 2005b). Obtaining pigs from same source is not specifically controlling for activity level. However, all the animals were housed with standard conditions that would allow for similar levels of activity (e.g. roaming around housing enclosure). Beyond this standardization, there will still be a certain amount of variability among the activity levels of the animals. However, this housing standardization is considered to be an acceptable method of controlling for activity level. This control over activity level is much tighter than what would be expected in human subjects or cadaveric tissue. The specimens used in this study were frozen and allowed to thaw overnight before testing. Frozen storage has been shown to have little effect on the creep response of the human lumbar disc (Dhillon et al., 2001) and the stiffness response, displacement at failure and failure mode of porcine spine segments (Callaghan and McGill, 1995). Callaghan and McGill (1995) did find that frozen storage increased the energy absorbed to failure and the tolerated ultimate compressive load. Although acute failure mechanics appear to be more affected by frozen storage than other mechanical properties of the spine, the potential effects due to frozen storage were consistent in the current study as all specimens were stored and thawed in the same manner.

It is generally understood that fluid flow plays an important role in the mechanical behaviour of the disc (Adams and Hutton, 1983; Kraemer et al., 1985; Keller et al., 1990; McMillan et al., 1996a; Costi et al., 2002; van der Veen et al., 2005; van der Veen et al., 2007). However, fluid flow cannot be directly measured *in vitro* or *in vivo*. Therefore, changes in disc height and stiffness are used to monitor changes in mechanical behaviour. Van der Veen et al. (2005; 2007) examined static and dynamic compressive loading of specimens in an

environment designed to mimic physiological conditions (i.e. submersion in a saline solution at body temperature). They concluded that disc height loss and increased stiffness are independent of loading type, despite the physiological environment. This is surprising because dynamic loading would allow for periods of low load, and thus potential for fluid inflow. Nevertheless, these results are consistent with the findings of the current study that disc height loss and stiffness are independent of postural changes, suggesting that even in a physiological environment, fluid flow into the disc cannot be validly studied *in vitro*. Despite this limitation, an *in vitro* model is a very useful way to study the isolated effects of the different manipulations on the mechanical changes of the joint. *In vivo* measurements at the IVJ level are very complex and not very accessible. Several research groups have used stadiometry (or precise height measurements) to examine changes in height after various activities *in vivo*. It has been suggested that most changes in height occur in the spine (Leivseth and Drerup, 1997). Some groups have reported height loss after exposure to sitting (Magnusson et al., 1990; McGill et al., 1996b; van Deursen et al., 2005) while others have observed height gain (Althoff et al., 1992; Van Dieen et al., 2001a). Factors such as sitting posture, prior loading history, and movement during sitting all affect the load on the spine and subsequent height changes (Althoff et al., 1992; Leivseth and Drerup, 1997; Magnusson et al., 1990; McGill et al., 1996; van Deursen et al., 2000; Van Dieen et al., 2001). It is very difficult to control for these factors *in vivo*, thus leading to the conflicting results. An *in vitro* model is the most logical experimental model to use in order to be able to isolate the effects of these different factors on the mechanical properties of the spine. All FSUs in the current study were subject to the same pre-load protocol which effectively equalizes the hydration level between specimens. Therefore, the effects of spine movement from postural changes on the mechanical changes of the joint can be studied in isolation.

All FSUs in the current study lost the same amount of disc height, regardless of the amount of movement over the 2 hour protocol. The mean amount of disc height loss for all specimens in the current study was approximately 1.58mm, or 33%. It is generally

understood that disc hydration, and thus disc height, varies under the influence of loading (Adams and Hutton, 1983; Kraemer et al., 1985; Keller et al., 1990; McMillan et al., 1996a; Costi et al., 2002; van der Veen et al., 2005; van der Veen et al., 2007). Furthermore, creep deformation plays an early role in changes in disc height (Botsford et al., 1994). Therefore, it can be assumed that disc height loss is due to both creep deformation and fluid flow (Adams and Hutton, 1983; van der Veen et al., 2005; 2007). Reversible disc volume changes of approximately 8-20% have been reported to occur diurnally (Adams et al., 1987; Botsford et al., 1994). Reilly et al. (1984) reported a 19mm loss in sitting height over the course of a day. Magnusson et al. (1990) reported 4.53 mm (SD 2.51) of total sitting height loss during 5 minutes of static sitting and suggested that most spine shrinkage may be achieved within 5 minutes of loading. It is difficult to compare the *in vitro* results to *in vivo* observations because stadiometry does not specifically isolate disc height loss. Spine imaging studies offer the means to provide information about disc height changes, but no study has examined *in vivo* disc height loss after continuous exposure to 2 hours of sitting. The results from the current study demonstrated a difference between gross height loss and isolated disc height loss across all specimens; in particular, disc height accounted for approximately 67% of total specimen loss. A difference between the gross height and isolated disc height loss has been demonstrated by other research groups (van der Veen et al., 2005). Thus, even in low level loading conditions, spine height loss is a combined reduction in disc and vertebral body height. Nevertheless, the results from the current study support the idea that the postures and loads observed in sitting cause disc height loss regardless of the amount of movement.

In the current study, the average stiffness across the entire passive RoM increased after 2 hours of simulated sitting regardless of the amount of movement. Increased joint stiffness was observed in other *in vivo* examinations of prolonged sitting where all participants had less flexion RoM after sitting (see Study #4, Chapter 6; (Callaghan and McGill, 2001b; Beach et al., 2005)). Furthermore, several other research groups using isolated FSUs *in vitro* have demonstrated increased compressive and angular stiffness accompanied by a decrease

in disc height after prolonged loading protocols (Yoganandan et al., 1994; Callaghan and McGill, 2001a; Ching et al., 2003; Johannessen et al., 2004; Parkinson and Callaghan, 2007; van der Veen et al., 2007). The observed changes in stiffness likely result from fluid outflow and creep deformation of the annulus, as 18 hours of recovery (immersion in bath) after cyclic loading has shown to restore these properties in ovine spinal units (Keller et al., 1990; Johannessen et al., 2004). With fluid loss out of disc, the pressure in the nucleus drops suggesting an internal redistribution from load bearing nucleus to load bearing annulus (McNally and Adams, 1992; Adams et al., 1996; McMillan et al., 1996a; McMillan et al., 1996b; Van Dieen et al., 2001b). Alterations to the collagen network organization and delamination of the annulus fibrosus may also affect the stiffness, but these microscopic changes are speculative and remain largely unstudied.

Another research group has reported that disc height loss was associated with a reduced resistance to bending but their experimental set-up differed from the one used in the current study (Adams et al., 1987; McNally and Adams, 1992; Adams et al., 1996). The current study's set-up allowed the specimen to translate on the horizontal plane on top of a ball bearing table. This designed permitted the axis of rotation to moving within the specimen during loading (Callaghan and McGill, 2001a). It is thought that this design feature allows for more physiological loading of the joint throughout its RoM (Parkinson et al., 2005a; Rousseau et al., 2006). With this in mind, the regions of reduced slope observed at the flexion and extension end-ranges during the post-test passive RoM can be explained by translation of the specimen and increased laxity in the joint (the 50% RoM stiffness plateau will be discussed in a later section). Indeed, increased translation of the bottom cup was observed in flexion for all specimens, which are indicative of laxity in the joint as it approached its end range (see Figure 7.7). Increased translations were not seen in extension, but the region of reduced stiffness in extension observed in this study supports the notion that alterations in the location of the axis of rotation affect the angular stiffness of the joint (Rousseau et al., 2006). The translations observed in the current study may be a result of

ligament laxity due to disc height loss (Adams et al., 1994). However, it would be very difficult to verify if these regions of reduced stiffness occurred *in vivo* as excessive segmental rotations and translations are likely limited by proper neuromuscular control in a healthy spine.

Of particular interest in the current study is the stiffness plateau at the 50% angle, the angle at which the joint spent the majority of its time in each loading protocol (see Figure 7.5). The observed group differences can be explained in reference to the variation in tissue loading, measured by CoP, throughout the 2-hour test. The CoP was a surrogate measure of how the compressive force translated through the specimen joint. The 2-hour movement protocol was position-controlled indicating that the angles were keep constant (for the static protocol) or consistently varied (for the fidget and shift protocols) throughout the testing period. It was observed that the CoP location varied with posture whereby flexion and extension caused CoP movement in the anterior and posterior directions, respectively. A closer observation of the static CoP trace (see Figure 7.8a) revealed that during the first 10 minutes of flexion, the CoP drifted posteriorly. This can be explained by the posterior migration of the nucleus pulposus, which would likely take close to 10 minutes to move due to the viscoelastic and poroelastic properties of the disc (Alexander et al., 2007). The gradual tracking of the CoP towards the anterior edge of the disc was likely due to deformation of the anterior annulus via creep in flexion and an internal redistribution from a load-bearing nucleus to a load-bearing annulus (McNally and Adams, 1992; Adams et al., 1996; Race et al., 2000). The gradual tracking of the CoP towards the anterior region of the disc in the static protocol coupled with the posterior tracking of the nucleus could explain the no change in stiffness or laxity in the mid-range for this group. The fidget group CoP tended to remain in the same position throughout the 2 hours of testing, except for small movements that coincided with the fidget postural changes (see Figure 7.8b). This observation indicates that fidgeting causes the load-bearing capacity of the disc to remain more central even though the specimens lost disc height. This group exhibited increased stiffness with no change in laxity in the mid-range of

the passive RoM. It is possible that this load centralization mechanism led to a more uniform increase in stiffness across the entire range of motion. The CoP of the shift group moved through the greatest range over the 2-hour loading period and the CoP location when the specimen was in its 50% posture became more variable as the test progressed (see Figure 7.9). A closer examination of the torques required to move the specimen to the appropriate angle during the shift protocol indicated that more torque was necessary as the 2-hour test progressed. Thus, as the specimen became stiffer over time, the same angular change in posture caused a larger change in the tissue load distribution, even sending it outside of the disc space (see Figure 7.8c). Both the anterior and posterior regions of the annulus would be affected by the postural changes, and the facet joints would be loaded in the extension movement as the disc lost height. It is possible that the peripheralization of the tissue loading due to shifting could lead to radial bulging and delamination of the annular layers of the disc (Adams and Hutton, 1985). The observed decrease in stiffness and increase in laxity in the mid-range could be explained by these changes in the disc accompanied by disc height loss. This mechanism could be similar to the cyclic flexion/compression mechanism described in the initiation of disc herniation (Adams and Hutton, 1985; Callaghan and McGill, 2001a; Tampier et al., 2007).

Given that all three specimen groups differed substantially in the time-course of the CoP, it is surprising that group responses did not vary in the amount or location of disc height loss. However, all specimens lost more posterior disc height than anterior, likely because the posterior annulus has a tendency to dehydrate at a faster rate in flexion (Adams and Hutton, 1983). Nevertheless, the gross disc height loss measurements were not sensitive enough to detect more subtle effects that can exist from the different movement patterns. It is possible that there were microscopic changes that could redistribute the load by changing the disc pressure or the stresses within the disc (Adams and Hutton, 1983; Hedman and Fernie, 1997). The pressure in the disc increases in flexion, attributable to the tension of the stretched posterior ligaments and is reduced in extension, mainly because of bony contact of the facet

joints that resists compressive forces (McNally and Adams, 1992; Adams et al., 1994). Creep loading decreases disc pressure and stress peaks begin to appear in the annulus throughout the RoM (Adams et al., 1990; Adams et al., 1996; McMillan et al., 1996a), and may be exacerbated by large shifts in spine posture throughout a prolonged bout of sitting. The annulus is more easily dehydrated when the spine is flexed (Adams and Hutton, 1983). However, flexed sitting postures increase diffusion of small solutes into the posterior annulus of the IVD and rectify the imbalance resulting from an erect posture (Adams and Hutton, 1986). It is possible that the fidgeting movement pattern would increase fluid exchange in the disc but not affect gross disc height. The constant variation in posture may act to continuously cause fluid to move in and out of the disc. In fact, this has been demonstrated *in vivo* in canine intervertebral discs (Holm and Nachemson, 1983). Increased fluid exchange will boost the transport of metabolites out of the disc and nutrients in because the disc relies mainly on fluid flow for nutrition (Adams and Hutton, 1986; McMillan et al., 1996b). Also, previous research has shown that prolonged static compression tended to decrease collagen production and cause an inflammatory response in the disc and endplates while an optimal level of dynamic loading may have the opposite effect (Wang et al., 2007). Future studies should be aimed at examining microscopic changes in the structures of the disc and the vertebral body during simulated sitting in addition to examining variables such as fluid flow, disc pressure and facet joint contact forces.

The neutral zone (NZ) was first described by Panjabi (1992) as the region in spinal motion where there is little or no resistance to motion; it represents the laxity around the neutral position of a motion segment. In the current study, the range of the ±1Nm NZ decreased for all specimens after 2 hours of simulated sitting. This may seem like a beneficial change as the existence of a NZ implies that spinal joint has no intrinsic stability and must rely on the muscles to control the movement around the neutral position (Thompson et al., 2003). However, the creation of slope plateaus in the passive RoM in the current study could indicate that there were more regions of laxity throughout the joint's RoM. According to this

suggestion, prolonged sitting changes the passive mechanics of the joint thereby making it clinically unstable (Panjabi, 2003) and more susceptible to abnormal, uncontrolled motion if proper muscular control fails (Cholewicki and McGill, 1992). Laxity in lumbar spine ligaments can potentially cause neuromuscular disorders exhibiting symptoms such as altered muscle activation and spasms elicited by collagen fibre micro-damage and relayed reflexively by pain receptors (Van Dieen et al., 2003b; Solomonow, 2004). Furthermore, disc height loss can alter the morphology of the neural foramina resulting in nerve impingement that elicits pain (Winkelstein and DeLeo, 2004). The increased laxity from shifting, accompanied by disc height loss demonstrated in the current study provides some insight into why individuals exhibiting spinal shift movement patterns develop LBP during seated work (see Study #4, Chapter 6). An underlying pathology may be exacerbated by the increased laxity around the spinal joint, leading to more shifting to find a non-painful posture during prolonged sitting.

The reason for performing this study in two parts is the following: the study was conducted in two separate time blocks. In the first time block, 8 specimens per group were tested (total n = 24) and the results analyzed. From these analyses, it was determined that there were no group differences in disc height loss or average stiffness across the RoM. The group differences in stiffness at the 50% flexion angle were the same as the ones reported in the final version of this study. In the second time block, 5 specimens per group were tested (total n = 15) and analyzed separately from the first 24. The results from these 15 specimens were similar to the first 24 and thus, the 39 specimens were pooled and presented in the final version of this chapter (Part 1 of the study). CoP measurements were collected and calculated for all 39 specimens, in both the first and second time block. However, the CoP measurements in the first group of 24 could not be expressed in relation to the location of the specimen disc because of errors and incorrect assumptions that were made with the equipment and data collection protocol. Because the bottom cup of the mounted specimen was unconstrained and could translate horizontally on the bearing table, movements of CoP

attributed to postural change could not be separated from movements due to bottom cup translation. The data collection errors were corrected for the second group of specimens, and the CoP measurements for these 15 could be correctly calculated and located with respect to the disc (reported in Part 2 of the study). Regardless of whether the CoP was located with respect to the disc or not, similar patterns of CoP movement were observed (see Figure 7.8). However, the difference and improvement of CoP calculations for the second group of 15 specimens was that any movement of the bottom cup was accounted for by tracking the CoP with respect to the specimen disc and any CoP movement could be attributed to postural and mechanical changes within the joint over time.

7.7 Conclusions

The results of this study indicate that regardless of the amount of spine movement around one posture, the passive range of motion of a joint changed substantially after 2 hours of low level loading. These changes, along with disc height loss, could have greater consequences for a low back pain population who may be more susceptible to abnormal muscular control and clinical instability. However, small movements around a posture (i.e. fidgeting) may mitigate the changes in the passive stiffness around the seated flexion angle. While fidgeting is the movement pattern most likely adopted by individuals who do not develop LBP during sitting (see Study #4, Chapter 6), it remains unknown whether fidgeting while sitting is, in fact, beneficial to the spine. LBP developers exhibit large shifts in spine posture during sitting, which appear to lead to greater laxity in the joint potentially exacerbating pre-existing pathologies. It is possible that large movements (i.e. shifting) or long periods of static loading could cause detrimental alterations in spine joint mechanics that are mitigated on a microscopic level by fidgeting while sitting. Future studies should be aimed at examining microscopic changes in the structures of the disc and the vertebral body during simulated sitting in addition to examining variables such as fluid flow, disc pressure and facet joint contact forces. Nevertheless, while the observed disc height loss and changes in joint

mechanics were observed after only 2 hours of simulated sitting in the current study, these results may help explain the increased risk of developing disc herniation and degeneration if exposure to sitting is cumulative over many days, months and years (Kelsey, 1975; Wilder et al., 1988; Videman et al., 1990).

Chapter 8:
General Discussion

8.1 Summary of Major Findings & Novel Contributions

Study #1 - Evidence of a pelvis driven flexion pattern: Are the joints of the lower lumbar spine fully flexed in seated postures?

Findings: The L5/S1 joint was at 60% of full flexion in upright sitting and contributed the most to total lumbar spine flexion in this posture. All three lower lumbo-sacral joints approached their total range of motion in slouched sitting. There were no gender differences in IVJ angles in any of the seated postures. An integration of the findings related to gender differences is further discussed in Section 8.2.1.

Novel Contributions: This is the first study to define the lumbar IVJ angles with respect to their available RoM in seated postures. Gender differences observed in sitting were not explained by differences in the lower 3 lumbar IVJ angles. However, the IVJs can approach the same angle as the flexion angles seen in standing flexion during seated postures. Since standing flexion has been identified as a risk factor in occupational settings, this is an important finding because it places slouched sitting in the same "risk factor" category as prolonged standing flexion. The results of this study have identified slouched sitting as a posture that could lead to increased loading of the passive tissues surrounding the lower lumbo-sacral IVJs.

Study #2 - The feasibility of using accelerometers to measure lumbo-sacral spine angles in standing and seated postures

Findings: Accelerometers used to measure external lumbar spine and pelvic angles over-predicted the internal lower lumbar spine angle in standing flexion and under-predicted the

internal angle in seated postures. However, the process of removing the "upright standing bias" and expressing angles as a change from upright helped to reduce the error between internal and external measure of spine angle. Both the internal and external measurement methods tracked ***changes*** in L3/Sac posture in a similar manner, but only within standing or seated postural changes. The accelerometer placed on the sacrum tracked changes in sacral orientation accurately over a wide range of pelvic rotations. A trend towards gender differences was noted in external measures of lower lumbar spine angle while no significant gender effect was observed for internal angles. However, significant gender differences were observed in sacral rotation with both measurements.

Novel Contributions: The comparison of internal and external angles is not new. However, the wide range of spine postures examined in this study has not been studied before. The identification of posture- and gender-specific responses is noteworthy and highlights the need for caution when interpreting results from external measurement devices. This study provided an in-depth analysis of the current methods for treatment of data from external devices and how these methods affect the outcomes. In addition, results from this study and Study #1 (Chapter 3) indicated that standing forward flexion may not be the best way to elicit maximum lumbar RoM. Recommendations were provided for improving lumbar spine normalization procedures and the use of external measurement devices in sitting research (see Chapter 4, Section 4.6.5).

Study #3 – Individual differences in lumbar vertebral body and pelvic movement patterns during a seated flexion motion

Findings: Individuals performed a slouched seated flexion movement using a number of different motion strategies. The pelvis did not initiate the slouching motion and a disordered sequence of vertebral rotation was observed at the initiation of the movement. Three vertebral body motion patterns were identified based on how the VBs moved with respect to each other and these motion patterns affected the IVJ angles attained during the slouching

motion. Gender differences were noted in the amount of pelvic rotation necessary to perform the slouching motion.

Novel contributions: The observation of a disordered sequence of motion in seated asymptomatic individuals is different than what has been previously observed in standing flexion (Kanayama et al., 1996; Okawa et al., 1998; Harada et al., 2000). This outcome suggests that there may be a high demand on the neuromuscular control system when performing a seated slouching motion. A "normal" motion pattern in seated slouching was not identified, yet the different motion pattern groups may help identify a subset of individuals who are predisposed to LBP based on their spine movement patterns. A technical contribution of this study was the development of the image registration program in collaboration with Dr. Jeff Orchard. This was a novel application of an intensity-based least-squares optimization algorithm for tracking rigid body motion.

Study #4 – Time-varying postural responses during prolonged sitting differentiate low back pain developers from matched asymptomatic controls

Findings: The development of LBP in this study was not associated with any one lumbar spine or pelvic posture. In fact, LBP sufferers moved more than asymptomatic individuals during 90 minutes of seated work and they reported increased LBP over time. LBP sufferers used as much as 80% of their lumbar spine RoM while sitting and demonstrated large, frequent shifts in lumbar spine posture and seat pan CoP. These movement patterns increased in amplitude and frequency over time along with LBP development. Seat pan CoP measurements (a surrogate measure for upper body movement) increased over time along with lumbar spine movement, suggesting that lumbar spine movement drives the upper body posture while sitting. On the other hand, asymptomatic individuals generally exhibited a more static sitting pattern, using only 30% of their lumbar RoM with smaller fidget movements around a mean posture and reported little to no LBP development. Gender differences were observed in pelvic posture, with all females having less posterior pelvic rotation than all males, regardless of LBP status.

Novel contributions: This was the first study to examine sitting-induced LBP sufferers over a prolonged period of time during a simulated occupational task. The novel application of an algorithm to quantify time-varying lumbar spine movement patterns allowed for the distinguishing between the LBP and asymptomatic groups during prolonged seated office work. The responses observed in the current study may be more representative of those in the field and highlight the fact that short duration investigations of seated postures do not accurately represent the time varying biological responses to prolonged exposure. The existing research has observed the biological responses of LBP sufferers in short durations performing non-occupational tasks (Cholewicki and McGill, 1996; Radebold et al., 2001; Maigne et al., 2003; O'Sullivan et al., 2003; Dankaerts et al., 2004; Dankaerts et al., 2006a; Dankaerts et al., 2006b; O'Sullivan et al., 2006c; Gombatto et al., 2008). Previous research has suggested that LBP sufferers assume sitting postures with more lumbar spine flexion and posterior pelvic tilt (O'Sullivan et al., 2006c). Contrary to these findings, which were developed based on static analyses of instantaneous seated postures, the development of LBP in the current study was not associated with any one posture. The "quality" of the spine movement pattern identified in the LBP group may be more detrimental to spine health than beneficial. The LBP sufferers' seated spine movements increased in frequency and amplitude as time passed. The large shifts in spine flexion could forcefully place this group towards their end range of motion. Large shifts in extension could cause pinching of ligaments, facet capsule or nerve roots, particularly if disc height is lost. It is also likely that these movements became more difficult to properly control because LBP patients may lack proper lumbar spine postural control (Radebold et al., 2001; O'Sullivan et al., 2003).

Study #5 – Do simulated seated movement patterns affect spine joint mechanics in an in vitro porcine model?

Findings: All specimens lost the same amount of disc height and became stiffer throughout the joint's passive RoM regardless of the amount of spine movement around one posture. The passive range of motion of a joint changed substantially after 2 hours of low level

loading, regardless of the amount of spine movement. The static group exhibited no change in laxity or stiffness in the mid-range of the passive RoM. Shifting caused decreased stiffness and increased laxity in the mid-range. Fidgeting increased the stiffness with no change in laxity. The load carried through the joint (as measured by CoP) was varied either by changing postures as in the shift protocol or by potentially a creep mechanism seen in the static protocol. Fidgeting appeared to reduce the variation of load carriage through the joint. However, it is possible that this focused load carriage may lead to a more uniform increase in stiffness across the entire passive RoM. Small, frequent movements around one posture may mitigate joint mechanics in a way that could be deemed protective.

Novel Contributions: This *in vitro* model was a novel way to study the effects of spine movement from postural changes during 2 hours of simulated sitting on the mechanical properties of the joint. The innovative use of a force plate and motion analysis technology to track CoP with respect to the specimen allowed for a non-invasive measurement of the effects of postural changes on the tissue load distribution throughout the joint. The creation of regions of reduced stiffness in the passive RoM indicates that prolonged sitting changes the mechanics of the joint in such a way that makes it clinically unstable and more susceptible to abnormal, uncontrolled motion if proper muscular control fails. LBP developers exhibit large shifts in spine posture during sitting (see Study #4), which appears to lead to greater laxity in the joint potentially exacerbating pre-existing pathologies. Large movements (i.e. shifting) or long periods of static loading could cause detrimental alterations in spine joint mechanics that are mitigated on a microscopic level by fidgeting while sitting. While the disc height loss and changes in joint mechanics were observed after only 2 hours of simulated sitting regardless of the amount of movement, these results may help explain the increased risk of developing disc herniation and degeneration if exposure to sitting is cumulative over many days, months and years.

8.2 Synthesis of Findings

The five studies in this thesis all provide new information regarding lumbar spine movement and time-varying responses to seated exposures. The IVJ rotations measured in Study #1 along with the lumbar spine movement patterns documented in Study #4 were used to develop the simulated sitting profiles used in Study #5 (Figure 8.1). Studies #1 and 3 provided new information regarding sitting-specific vertebral positions and motion patterns (Figure 8.1). Study #5 provided an *in vitro* simulation of the movement patterns observed in Study #4 and developed a tissue-based rationale for the detrimental effects of prolonged sitting on the spinal joint and the potential for pain generation. Study #2 provided a verification of the external lumbar spine measurement methodology used in Studies #3 and #4. The external measurement devices were sensitive to changes in angle within seated postures and were also consistent when used at different time-points in different populations.

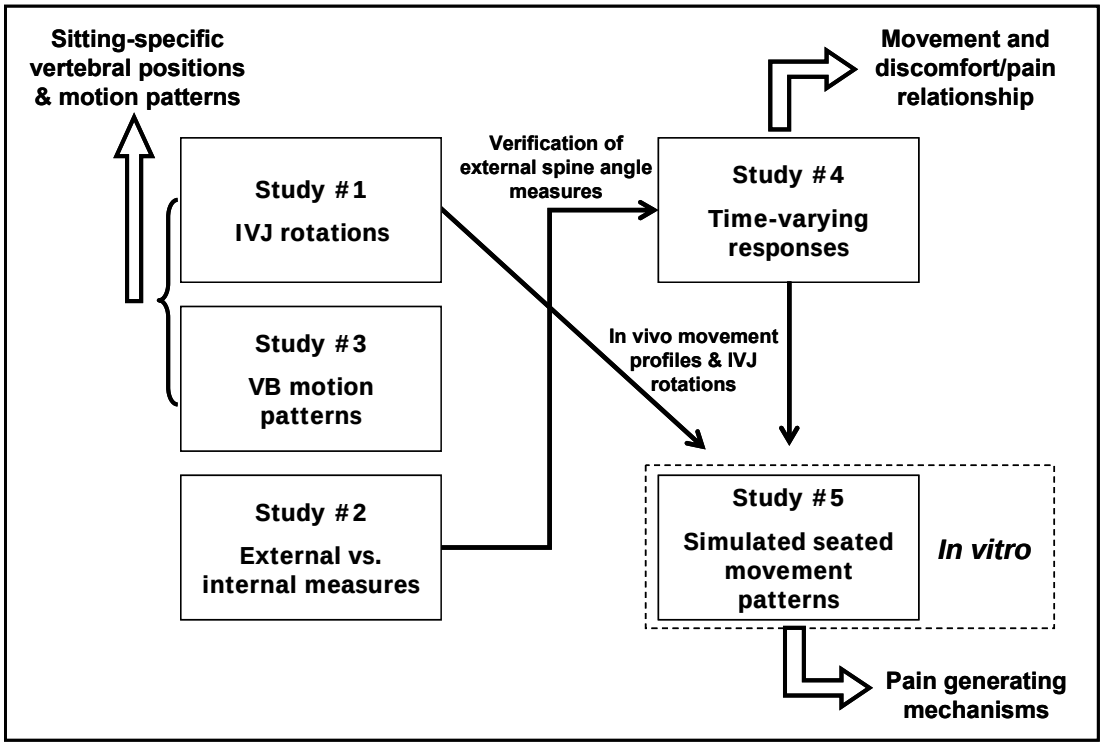


Figure 8.1: Flowchart highlighting the connections between the studies involved in this work.

8.2.1 How do the lumbar spine and pelvis move during sitting?

Study #1 showed that the lower 3 lumbar joints approached their total range of motion in slouched sitting. Study #2 demonstrated that an accelerometer placed on the sacrum tracks changes in pelvic rotation well. Therefore, pelvic rotation was used in conjunction with videofluoroscopy to examine dynamic slouching in Study #3. Study #3 confirmed that the seated slouching motion was not initiated by the pelvis and there was a disordered sequence of VB movement at the initiation of rotation. Individuals performed the slouching movement using a variety of different VB motion strategies, and these motion patterns affected the IVJ angles attained during the slouching motion. The pelvis contributes to seated slouching but does not initiate the motion suggesting that individuals use their back muscles to initiate this motion. The observed disordered sequence of vertebral motion may indicate that different joints approach their end range before the completion of the slouching movement. The different motion pattern groups may help direct the identification of a subset of individuals who are predisposed to LBP based on their spine movement patterns. Given that clinicians use hip flexion to rotate the pelvis and reduce L5/S1 flexion the next step to further investigates this issue would be to see if training individuals to initiate slouching with their pelvis makes the sequence of rotation ordered. Because pelvic accelerometers are acceptable for tracking pelvic rotation they would be useful for this type of clinical intervention that modifies movement.

Several gender-specific responses were observed and need to be addresses. There were no gender differences in IVJ angles in any of the seated postures examined in Study #1. This means that these IVJs in both men and women go through the same change from upright standing. However, the absolute IVJ angles presented in Appendix B indicated that the female L3/4 and L4/5 IVJs always had more lordosis than the corresponding male IVJs. The lower 3 male VBs were more rectangular in the sagittal plane, while the female VBs had more posterior wedging, likely allowing for more lordotic IVJ angles. Pelvic orientation

differs both across gender and standing or sitting posture (Study #2). For males, standing flexion is accomplished more by the lumbar spine than females because the pelvis does not rotate as much but the lumbar spine angle is greater in standing flexion. Female standing forward flexion is accomplished by more pelvic rotation, suggesting that females tend to execute standing forward flexion by rotation at the hips (and thus the pelvis) rather than flexing the lumbar spine. When moving from upright standing to upright sitting, the male pelvis goes through more posterior rotation than the female pelvis (Study #2). However, females achieved a slouching motion with more posterior rotation of the pelvis than males (Study #3). The gender differences observed in pelvic rotation are not manifested in the IVJ angles in static postures, yet more posterior pelvic rotation to accomplish the slouching motion in females suggests that the majority of flexion could occur at the L5/S1 IVJ, concentrating the stress at this joint throughout the motions.

8.2.1.1 Consistency of Posture

Previous research shows that individuals can reliably assume upright standing postures from varying degrees of forward flexion and they can return to the same upright sitting posture from slouched sitting (Bullock-Saxton, 1993; Swinkels and Dolan, 1998; Goh et al., 1999; Swinkels and Dolan, 2000; Dunk et al., 2005; Dolan and Green, 2006). When given verbal and visual cues, individuals are able to imitate seated postures with the same curvature direction in the thoraco-lumbar and lumbar regions, specifically, slouched, flat-back and long lordosis sitting (Claus et al., 2009). However, manual facilitation was required to achieve the proposed "clinically ideal" sitting posture where the thoraco-lumbar angle is kyphosed and the lumbar angle is lordosed (Claus et al., 2009). In the first three studies the same consistent instructions about the adopted seated postures were given to all participants. The participants otherwise adopted their most natural posture. Based on the existing literature, it was assumed that participants could consistently adopt the same posture repeatedly. The results from Study #3 indicate that while each participant rotated their pelvis to achieve the slouched posture, the lower lumbar spine was flexed to varying degrees and the initiation of vertebral

body rotation did not follow an ordered pattern. A specific intervention could be used to train participants to initiate the movement with their pelvis, yet research shows that this movement pattern would likely require extensive instruction and training to achieve (Claus et al., 2009).

8.2.1.2 Directed vs. Self-selected Postures

Slouched sitting can happen in one of two ways. The first is through rounding the spine while maintaining the upper body centre of mass over the ischial tuberosities as seen in the first three studies of this thesis (Callaghan and Dunk, 2002). The second is through rounding the spine and leaning against a back rest with the upper body CoM behind the ischial tuberosities (Snijders et al., 2004). The sitting postures observed in this thesis were either specified through verbal instructions (Studies #1 to #3) or self-selected by the participants for a typical office workspace (Study #4). In addition the participants in Studies #1 to #3 sat on a stool without a backrest, while the participants in Study #4 used a standard office chair with a backrest. Because of these differences, it is difficult to make direct comparisons of the lumbar spine and pelvic angles. However, by comparing angle results, certain hypotheses can be made about directed versus self-selected seating postures while sitting in a chair with a backrest. For the purpose of this inter-study comparison, the total lumbar spine angle (measured by the L1 and sacral accelerometers) and pelvic angle (measured by the sacral accelerometer) were used (Figure 8.2 & Figure 8.3). Female participants in both the asymptomatic and LBP groups in Study #4 sat with more posterior pelvic rotation than they did in Study #2 (where the sitting postures were directed). These females also appeared to sit closer to a slouched sitting lumbar spine posture. The male participants rotated their pelvis to a position similar to the slouched sitting posture, but the male participants' lumbar spine was closer to an upright sitting posture.

These comparisons would indicate that when a backrest is present without a large lumbar support, like the chair used in Study #4, none of the participants chose to assume a lordotic

lumbar spine posture. The combination of a posteriorly rotated pelvis and a slouched lumbar spine would suggest that the lower lumbar IVJs approach their end range of motion (see Study #1, Chapter 3). Alternatively, posterior rotation of the pelvis with less lumbar flexion could be more akin to the slouched posture described by Snijders et al. (2004). In this posture, a greater load is placed on the iliolumbar ligaments and the L5/S1 joint, in particular. The presence of a backrest is beneficial for reducing muscle activity and loads on the spine (Andersson et al., 1979; Van Dieen et al., 2001a). However, recent chair design research has highlighted the importance of designing a back rest that accommodates anterior rotation of the pelvis and provide enough lumbar support to encourage a lordotic lumbar spine (Makhsous et al., 2003; Carcone and Keir, 2007; De Carvalho et al., 2007). Furthermore, a dynamic back rest would continuously move with and support spine movement through a range of motions, and would be a potential intervention to evaluate for the LBP population studied in Study #4 of this thesis.

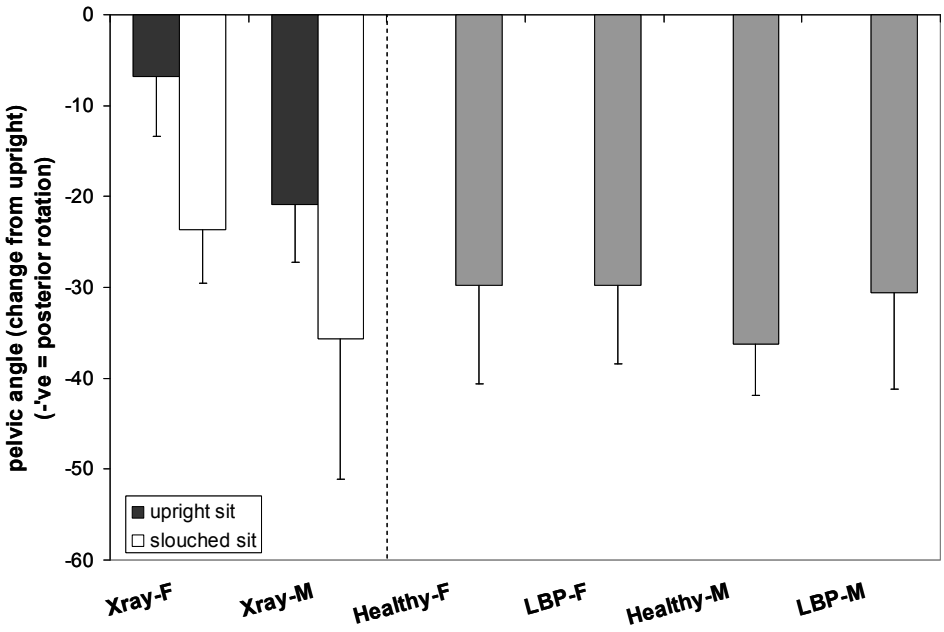


Figure 8.2: Pelvic rotation angle measured using accelerometers from female (F) and male (M) participants from Study #1-2 (Xray) and Study #4 (Healthy/LBP). The last 4 grey bars are average angles from the first 5 minutes of sitting for each group in Study #4.

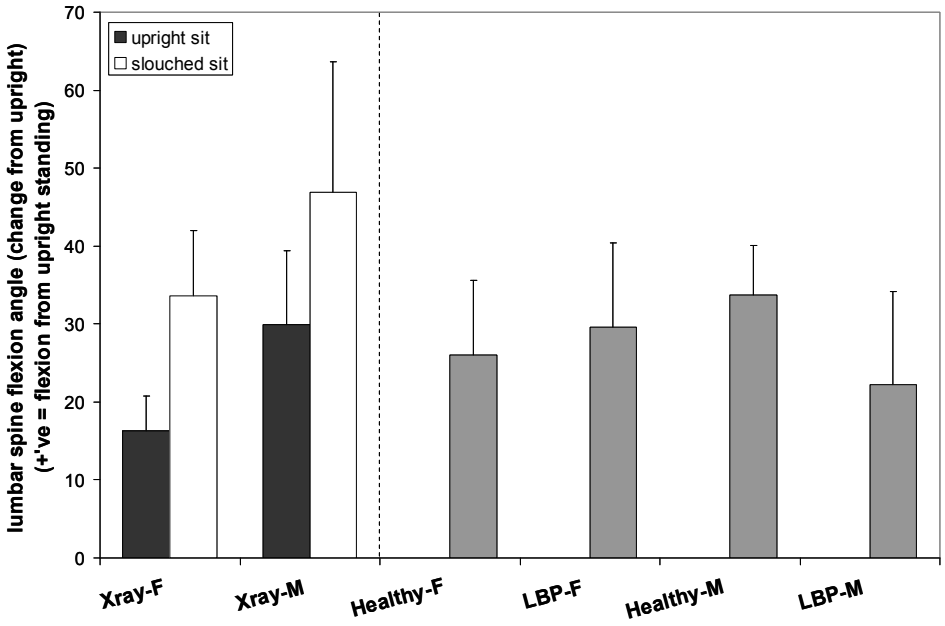


Figure 8.3: Lumbar spine flexion angle measured using accelerometers from female (F) and male (M) participants from Study #1-2 (Xray) and Study #4 (Healthy/LBP). The last 4 grey bars are average angles from the first 5 minutes of sitting for each group in Study #4.

8.2.2 How can lumbar spine movement and postures explain LBP and injury associated with prolonged sitting?

From a physiological standpoint, the results from Study #1 showed that the lower 3 lumbar IVJs can approach the same angle as the flexion angles seen in standing flexion during seated postures. More importantly, since standing flexion has been identified as a risk factor in occupational settings, this is an important finding because it places slouched sitting in the same "risk factor" category as prolonged standing flexion.

From a physiological standpoint, the results from Study #1 showed that the lower 3 lumbar IVJs exceeded 80% RoM in slouched sitting. For flexion angles greater than 75% RoM, high tensile forces can be generated in the posterior ligaments of the spine (Adams et al., 1994). Sustained static flexion will increase joint stiffness (Studies #4 and #5) and drastically change the passive RoM characteristics of the IVJ (Study #5), likely because of disc height

loss and creep in the posterior passive structures of the spine. This creep has the potential to alter spine muscle activity patterns (Solomonow et al., 2003a) and laxity in lumbar spine ligaments can potentially cause neuromuscular disorders exhibiting symptoms such as altered muscle activation and spasms (Van Dieen et al., 2003b; Solomonow, 2004). Higher intervertebral disc pressures *in vivo* have also been demonstrated in sitting compared to standing (Andersson et al., 1975; Sato et al., 1999) and over 6 mm of posterior migration of the L4/5 and L5/S1 nucleus pulposus can occur in upright and flexed sitting (Alexander et al., 2007). The posterior migration of the nucleus corroborated with the initial movement of the CoP towards the posterior edge of the disc space, observed in Study #5. The posterior annulus is at a higher risk for weakening and herniation because it is the narrowest part of the disc and least able to sustain large compressive loads (Adams et al., 1996). Posterior migration of the nucleus combined with disc height loss and joint laxity seen in Study #5 help explain the increased risk of developing disc herniation and degeneration if exposure to sitting is cumulative over days, months and years.

Several variables were examined in order to distinguish sitting aggravated LBP sufferers from asymptomatic individuals in Study #4. These groups did not differ in how factors such as hamstring flexibility or erector spinae muscle fatigue were affected by prolonged sitting. Individuals with LBP demonstrated less lumbar range of motion (RoM) than the asymptomatic group, but <u>all</u> individuals had less RoM after 90 minutes of sitting. While sitting, the development of LBP in this study was not associated with any one lumbar spine posture. The distinguishing factor between these two groups was their respective time-varying lumbar spine movement patterns. The results of this study highlight the fact that short duration investigations of seated postures do not accurately represent the biological responses to prolonged exposure. Individuals with sitting-induced LBP and those without pain differ in how they move during seated work and this will have different impacts on the tissues of the lumbar spine.

LBP sufferers moved more than asymptomatic individuals during 90 minutes of seated work and they reported increased LBP over time. The movement patterns observed in LBP sufferers were such that they tended to move their spines towards the extreme end of flexion. Over time, spine movements increased in amplitude and it is likely that these movements became more difficult to properly control because of the likelihood of a disordered order of VB rotation (Study #3) and increased laxity demonstrated in the mid-range (Study #5). Furthermore, as the joint became stiffer over time and the disc lost height, large shifts in spine angle can lead to more variable tissue loading (Study #5). Frequent shifts in lumbar spine posture could be a mechanism for redistributing the load to different tissues of the spine, particularly if some tissues are more vulnerable than others. Despite the apparent benefits of movement on the health of the spine, increased movement did not completely eliminate pain in individuals with pre-existing LBP.

Fidgeting is the movement pattern most likely adopted by individuals who do not develop LBP during sitting (Study #4), but this movement pattern does not diminish disc height loss and increased stiffness across the joint (Study #5). However, small movements around a posture (i.e. fidgeting) may mitigate the changes in the passive stiffness by causing a more uniform increase in stiffness with no increase in laxity. It has also been suggested that small movements around a mean posture help to reduce muscular strain (Vergara and Page, 2000; 2002). Still, it remains unknown whether fidgeting while sitting is protective to the spine and decreases the risk for developing LBP. It is possible that fidgeting positively affects fluid flow, disc pressure and the stress distribution in the disc because the tissue load distribution remains centralized over 2 hours of sitting (Study #5). More work is needed to probe the cause-and-effect relationship between movement and LBP.

8.3 Future Considerations and Directions

8.3.1 Trunk muscle activation during sitting and low back pain

It is well documented that individuals with LBP have altered neuromuscular responses to various movement tasks (Van Dieen et al., 2003a). Examining hip muscle activation patterns in individuals who developed LBP from prolonged standing indicated that agonist-antagonist co-activation may predispose some individuals to develop LBP (Nelson-Wong et al., 2008). Therefore, the activation patterns of the various muscles of the trunk during sitting could provide useful information when comparing individuals who experience LBP during sitting. However, the amplitudes of the trunk EMG signals are quite low in seated postures and EMG techniques are very susceptible to noise contamination from various sources as experienced in Study #4 (Chapter 6) that examined postural responses to prolonged sitting (see Appendix G). Thus, a future study examining trunk muscle activation patterns during prolonged sitting may need to limit the other instrumentation used to collect accompanying data. Previous research has examined a large number of trunk muscles and used pattern recognition techniques to quantify temporal muscle activation patterns and differentiate between healthy and LBP groups (Hubley-Kozey and Vezina, 2002). A similar investigation into the trunk muscle activation patterns associated with seated spine movement would provide important information regarding possible neuromuscular impairments associated with LBP development during prolonged sitting.

8.3.2 Quantifying tissue loads in seated postures

The investigations in this thesis were mainly kinematic and it is possible to only speculate on the resultant loads applied to the tissues of the spine. Mathematic models of the spine have had limited use predicting loads on the spine during seated tasks, mainly because of the complex combination of external forces acting on the body, including gravitational forces, reaction forces from the seat, backrest and floor (obtained from a chair instrumented with load cells) together with possible external forces due to the task (Eklund and Corlett, 1987).

Furthermore, the sequence of motion of the vertebral joints is an important assumption in dynamic and quasi-static biomechanical models, since joint position will affect the distribution of forces to individual muscles and ligaments. A model that includes sitting specific biomechanical information may allow for an improved estimate of forces at the L4/5 joint, which in turn would provide an important contribution towards the understanding of low back injury and pain generation as well as the mechanical response to this exposure (i.e. disc height loss). However, in the case of sitting, the cumulative tissue loading may be more important and technologies to document microscopic tissue damage may provide a clearer understanding of the cause and effect relationship of LBP and sitting.

8.3.3 Animal models of pain

Many of the results of the studies in this thesis have been discussed in the context of their implications for pain-generating potential. For example, we now know the IVJs of the lower lumbar spine approach their end range in seated postures (see Study #1, Chapter 3) and that sustained end range of flexion may have detrimental effects on the tissues surrounding a spinal joint, but the actual effect of this phenomenon on pain generation is not directly measurable in humans. The move towards *in vivo* animal model research can allow for the study of biomechanical and neurophysiological determinants of chronic pain from spinal injuries. It is possible to explore the direct effects of biomechanical exposures, such as end range spinal flexion, on tissue loading, pain behavioural outcomes, and neurophysiological responses using *in vivo* or *in situ* animal preparations. Examining human biomechanical exposures and applying similar exposures to *in vivo* animal models will help expand our knowledge of the relationship between tissue loading, injury and the physiology of pain.

8.4 Summary

8.4.1 Why do we slouch and why does it not hurt?

Slouching is a passive movement and requires less muscle activity than maintaining a certain amount of lordosis in upright sitting, as described above. On the other hand, upright, seated postures require muscle activity to maintain. In fact, "neutral" lordosis, similar to upright standing, is difficult to maintain in sitting, even after training and/or manual facilitation (Claus et al., 2009). Therefore, slouching likely occurs naturally because it is less physiologically demanding. Also described above (in Section 8.2.1) is the notion that office chair design may not accommodate a lordotic posture, especially for females. Thus, individuals may be forced into a slouching posture because the chair does not easily allow for any other postural adaptations. A flexed posture may be beneficial because it can unload the facet joints, redistribute the load to a different part of the IVD and allow for fluid to flow into the posterior part of the annulus. Any healthy joint in the body, including the spine, operates within its normal RoM without pain. Moving into a slouched posture does not cause immediate pain because the joints remain within their normal range (up to 80% of RoM according to Study #1). As discussed above in Section 8.2.2, sustaining slouched posture can have detrimental effects on the spine. Furthermore, prolonged spine flexion can exacerbate pre-existing conditions such as disc herniation, segmental instability and neuromuscular disorders.

8.4.2 Thesis Conclusions

The five studies in this thesis all provide new information regarding lumbar spine movement and time-varying responses to seated exposures. This work has highlighted that seated postures place the joints of the lumbar spine towards their end range of motion, which is considered to be risky for pain/injury in a number of tissue sources. In-depth analyses of both internal and external measurements of spine postures identified different seated motion patterns and self-selected seated postures that may increase the risk for developing LBP. The

model of seated LBP/discomfort development used in this thesis provided evidence that large lumbar spine movements do not reduce pain in individuals with pre-existing LBP. Tissue-based evidence demonstrated that 2 hours of sitting substantially affects IVJ mechanics and may help explain the increased risk of developing disc herniation and degeneration if exposure to sitting is cumulative over many days, months and years.

When one feels that movement or a break is necessary, the level of discomfort or pain could be indicating that the process of irritation or damage to the tissues has already begun. Increased seated voluntary movement is not a mechanism that shows potential to reduce LBP development in individuals with sitting-induced LBP. There is a need for other interventions in the workplace to help reduce LBP during seated work.

1) Participants could be trained to initiate the slouching movement with their pelvis, in order to ensure an ordered initiation of vertebral body rotation and less demand on the neuromuscular system.
2) Pelvic accelerometers are good for tracking pelvic orientation and can be used as a feedback tool when modifying movement patterns.
3) Encouraging the implementation of chairs with back rests that accommodate anterior rotation of the pelvis and provide enough lumbar support to encourage a lordotic lumbar spine.
4) Ensuring training for the proper use of ergonomic chairs. Specifically, a dynamic back rest would continuously move with and support spine movement through a range of motions, and would be a potential intervention to evaluate for the LBP population studied in Study #4 of this thesis.
5) Proactively inducing slow and controlled movement may help to alleviate LBP. Intermittent use of a lumbar support, or a device that provides continuous passive motion could be offered as an intervention for LBP sufferers.

Appendix A:
Letter of Copyright Permission

Chapter 3 of this thesis was reprinted, with permission from Elsevier, from "Clinical Biomechanics, Vol. 24, Iss. 2, Nadine M. Dunk, Angela E. Kedgley, Thomas R. Jenkyn, Jack P. Callaghan, Evidence of a pelvis-driven flexion pattern: Are the joints of the lower lumbar spine fully flexed in seated postures? pp. 164-168, 2009".

See the following pages for Letter of Copyright Permission obtained from Elsevier.

ELSEVIER LICENSE
TERMS AND CONDITIONS

Jan 11, 2010

This is a License Agreement between Nadine Dunk ("You") and Elsevier ("Elsevier") provided by Copyright Clearance Center ("CCC"). The license consists of your order details, the terms and conditions provided by Elsevier, and the payment terms and conditions.

All payments must be made in full to CCC. For payment instructions, please see information listed at the bottom of this form.

Supplier	Elsevier Limited The Boulevard,Langford Lane Kidlington,Oxford,OX5 1GB,UK
Registered Company Number	1982084
Customer name	Nadine Dunk
Customer address	6 Hardy Street Guelph, ON N1E 5G5
License Number	2346120887807
License date	Jan 11, 2010
Licensed content publisher	Elsevier
Licensed content publication	Clinical Biomechanics
Licensed content title	Evidence of a pelvis-driven flexion pattern: Are the joints of the lower lumbar spine fully flexed in seated postures?
Licensed content author	Nadine M. Dunk, Angela E. Kedgley, Thomas R. Jenkyn, Jack P. Callaghan
Licensed content date	February 2009
Volume number	24
Issue number	2
Pages	5
Type of Use	Thesis / Dissertation
Portion	Full article
Format	Electronic
You are an author of the Elsevier article	Yes
Are you translating?	No
Order Reference Number	
Expected publication date	Jan 2010
Elsevier VAT number	GB 494 6272 12
Permissions price	0.00 USD
Value added tax 0.0%	0.00 USD

Total 0.00 USD

Terms and Conditions

INTRODUCTION

1. The publisher for this copyrighted material is Elsevier. By clicking "accept" in connection with completing this licensing transaction, you agree that the following terms and conditions apply to this transaction (along with the Billing and Payment terms and conditions established by Copyright Clearance Center, Inc. ("CCC"), at the time that you opened your Rightslink account and that are available at any time at http://myaccount.copyright.com.proxy.lib.uwaterloo.ca).

GENERAL TERMS

2. Elsevier hereby grants you permission to reproduce the aforementioned material subject to the terms and conditions indicated.

3. Acknowledgement: If any part of the material to be used (for example, figures) has appeared in our publication with credit or acknowledgement to another source, permission must also be sought from that source. If such permission is not obtained then that material may not be included in your publication/copies. Suitable acknowledgement to the source must be made, either as a footnote or in a reference list at the end of your publication, as follows:

"Reprinted from Publication title, Vol /edition number, Author(s), Title of article / title of chapter, Pages No., Copyright (Year), with permission from Elsevier [OR APPLICABLE SOCIETY COPYRIGHT OWNER]." Also Lancet special credit - "Reprinted from The Lancet, Vol. number, Author(s), Title of article, Pages No., Copyright (Year), with permission from Elsevier."

4. Reproduction of this material is confined to the purpose and/or media for which permission is hereby given.

5. Altering/Modifying Material: Not Permitted. However figures and illustrations may be altered/adapted minimally to serve your work. Any other abbreviations, additions, deletions and/or any other alterations shall be made only with prior written authorization of Elsevier Ltd. (Please contact Elsevier at permissions@elsevier.com)

6. If the permission fee for the requested use of our material is waived in this instance, please be advised that your future requests for Elsevier materials may attract a fee.

7. Reservation of Rights: Publisher reserves all rights not specifically granted in the combination of (i) the license details provided by you and accepted in the course of this licensing transaction, (ii) these terms and conditions and (iii) CCC's Billing and Payment terms and conditions.

8. License Contingent Upon Payment: While you may exercise the rights licensed immediately upon issuance of the license at the end of the licensing process for the transaction, provided that you have disclosed complete and accurate details of your proposed use, no license is finally effective unless and until full payment is received from you (either by publisher or by CCC) as provided in CCC's Billing and Payment terms and conditions. If full payment is not received on a timely basis, then any license preliminarily

granted shall be deemed automatically revoked and shall be void as if never granted. Further, in the event that you breach any of these terms and conditions or any of CCC's Billing and Payment terms and conditions, the license is automatically revoked and shall be void as if never granted. Use of materials as described in a revoked license, as well as any use of the materials beyond the scope of an unrevoked license, may constitute copyright infringement and publisher reserves the right to take any and all action to protect its copyright in the materials.

9. Warranties: Publisher makes no representations or warranties with respect to the licensed material.

10. Indemnity: You hereby indemnify and agree to hold harmless publisher and CCC, and their respective officers, directors, employees and agents, from and against any and all claims arising out of your use of the licensed material other than as specifically authorized pursuant to this license.

11. No Transfer of License: This license is personal to you and may not be sublicensed, assigned, or transferred by you to any other person without publisher's written permission.

12. No Amendment Except in Writing: This license may not be amended except in a writing signed by both parties (or, in the case of publisher, by CCC on publisher's behalf).

13. Objection to Contrary Terms: Publisher hereby objects to any terms contained in any purchase order, acknowledgment, check endorsement or other writing prepared by you, which terms are inconsistent with these terms and conditions or CCC's Billing and Payment terms and conditions. These terms and conditions, together with CCC's Billing and Payment terms and conditions (which are incorporated herein), comprise the entire agreement between you and publisher (and CCC) concerning this licensing transaction. In the event of any conflict between your obligations established by these terms and conditions and those established by CCC's Billing and Payment terms and conditions, these terms and conditions shall control.

14. Revocation: Elsevier or Copyright Clearance Center may deny the permissions described in this License at their sole discretion, for any reason or no reason, with a full refund payable to you. Notice of such denial will be made using the contact information provided by you. Failure to receive such notice will not alter or invalidate the denial. In no event will Elsevier or Copyright Clearance Center be responsible or liable for any costs, expenses or damage incurred by you as a result of a denial of your permission request, other than a refund of the amount(s) paid by you to Elsevier and/or Copyright Clearance Center for denied permissions.

LIMITED LICENSE

The following terms and conditions apply only to specific license types:

15. **Translation**: This permission is granted for non-exclusive world **English** rights only unless your license was granted for translation rights. If you licensed translation rights you may only translate this content into the languages you requested. A professional translator must perform all translations and reproduce the content word for word preserving the integrity of the article. If this license is to re-use 1 or 2 figures then permission is granted for non-exclusive world rights in all languages.

16. **Website**: The following terms and conditions apply to electronic reserve and author websites:
Electronic reserve: If licensed material is to be posted to website, the web site is to be password-protected and made available only to bona fide students registered on a relevant course if:
This license was made in connection with a course,
This permission is granted for 1 year only. You may obtain a license for future website posting,
All content posted to the web site must maintain the copyright information line on the bottom of each image,
A hyper-text must be included to the Homepage of the journal from which you are licensing at http://www.sciencedirect.com.proxy.lib.uwaterloo.ca/science/journal/xxxxx or the Elsevier homepage for books at http://www.elsevier.com.proxy.lib.uwaterloo.ca , and
Central Storage: This license does not include permission for a scanned version of the material to be stored in a central repository such as that provided by Heron/XanEdu.

17. **Author website** for journals with the following additional clauses:

All content posted to the web site must maintain the copyright information line on the bottom of each image, and
he permission granted is limited to the personal version of your paper. You are not allowed to download and post the published electronic version of your article (whether PDF or HTML, proof or final version), nor may you scan the printed edition to create an electronic version,
A hyper-text must be included to the Homepage of the journal from which you are licensing at http://www.sciencedirect.com.proxy.lib.uwaterloo.ca/science/journal/xxxxx , As part of our normal production process, you will receive an e-mail notice when your article appears on Elsevier's online service ScienceDirect (www.sciencedirect.com). That e-mail will include the article's Digital Object Identifier (DOI). This number provides the electronic link to the published article and should be included in the posting of your personal version. We ask that you wait until you receive this e-mail and have the DOI to do any posting.
Central Storage: This license does not include permission for a scanned version of the material to be stored in a central repository such as that provided by Heron/XanEdu.

18. **Author website** for books with the following additional clauses:
Authors are permitted to place a brief summary of their work online only.
A hyper-text must be included to the Elsevier homepage at
http://www.elsevier.com.proxy.lib.uwaterloo.ca

All content posted to the web site must maintain the copyright information line on the bottom of each image
You are not allowed to download and post the published electronic version of your chapter, nor may you scan the printed edition to create an electronic version.
Central Storage: This license does not include permission for a scanned version of the material to be stored in a central repository such as that provided by Heron/XanEdu.

19. **Website** (regular and for author): A hyper-text must be included to the Homepage of the journal from which you are licensing at http://www.sciencedirect.com.proxy.lib.uwaterloo.ca

/science/journal/xxxxx. or for books to the Elsevier homepage at http://www.elsevier.com.proxy.lib.uwaterloo.ca

20. **Thesis/Dissertation**: If your license is for use in a thesis/dissertation your thesis may be submitted to your institution in either print or electronic form. Should your thesis be published commercially, please reapply for permission. These requirements include permission for the Library and Archives of Canada to supply single copies, on demand, of the complete thesis and include permission for UMI to supply single copies, on demand, of the complete thesis. Should your thesis be published commercially, please reapply for permission.

21. **Other Conditions**: None

v1.6

Gratis licenses (referencing $0 in the Total field) are free. Please retain this printable license for your reference. No payment is required.

If you would like to pay for this license now, please remit this license along with your payment made payable to "COPYRIGHT CLEARANCE CENTER" otherwise you will be invoiced within 30 days of the license date. Payment should be in the form of a check or money order referencing your account number and this license number 2346120887807.
If you would prefer to pay for this license by credit card, please go to http://www.copyright.com.proxy.lib.uwaterloo.ca/creditcard to download our credit card payment authorization form.

Make Payment To:
Copyright Clearance Center
Dept 001
P.O. Box 843006
Boston, MA 02284-3006

If you find copyrighted material related to this license will not be used and wish to cancel, please contact us referencing this license number 2346120887807 and noting the reason for cancellation.

Questions? customercare@copyright.com or +1-877-622-5543 (toll free in the US) or +1-978-646-2777.

Appendix B:
Study Sample Size Justifications

Study #1 (Chapter 3)

Mean intervertebral joint (IVJ) angles measured in standing and sitting were obtained from the literature in order to estimate the required sample size (Table A1).

The formula used to calculate sample size for a comparison between two independent means is as follows:

$$n = \frac{2 * (z_{(1-\frac{\alpha}{2})} + z_{(1-\beta)})^2 * s^2}{\delta^2}$$

where: n = number of samples in each group

z = z-values for a 95% confidence level (α=0.05) and 80% power (β=0.20)

s = pooled standard deviation of a known population

δ = hypothesized difference between the two means

The data from the literature (Table A1) were taken from studies designed with repeated measures. As in the case of Study #1 where IVJ measurements are compared between two different postures in the same individual, paired t-tests would be used to evaluate the difference between two dependent means. However, it is necessary to know the variation in the differences of the paired observations in order to determine the appropriate sample size. This information is not available from the studies in the literature. Therefore, the sample size calculations for Study #1 were approached as t-tests with two independent means, using the "pooled standard deviation" for the IJV measurements. That is:

$$s^2 = \frac{s_1^2 + s_2^2}{2}$$

where s_1^2 and s_2^2 are the reported variances of the two IJV measurements from the same study with the same degrees of freedom (i.e. same precision).

This approach provided a conservative estimate of the necessary sample size, as a repeated measures analysis would have greater power. (personal communication with Dr. Jock MacKay, Associate Professor, Department of Statistics and Actuarial Science, University of Waterloo).

Assuming an alpha of .05 and a beta of .20, the corresponding z-values are 1.96 and 0.84, respectively. Using the means and standard deviations supplied in Table A1 and the sample size equation above, the estimated sample size for each IVJ comparison was calculated as follows:

For example, using the standing and sitting L3/4 IVJ angles from Andersson et al. (1979) (Table A1)

$$n = \frac{2 * (1.96 + 0.84)^2 * (4.0)^2}{(89.6 - 81.6)^2}$$

n = 3.92, rounded up to 4

Table A1: Means and standard deviations (in brackets) from two similar studies in the literature. The estimated sample size was computed for each comparison using the formula $n = \frac{2 * (z_{(1-\frac{\alpha}{2})} + z_{(1-\beta)})^2 * s^2}{\delta^2}$**, where the z-values are 1.96 and 0.84 when α=0.05 and β=0.20.**

	L3/4	L4/5	L5/S1
From (Andersson et al., 1979)			
Standing	89.6 (4.8)	94.2 (3.6)	58.4 (1.8)
Sitting	81.6 (3.0)	84.0 (2.6)	53.8 (2.6)
Difference between means (δ)	8.0	10.2	4.6
"Pooled" SD (s)	4.0	3.1	2.2
δ^2	64.0	104.0	21.2
s^2	8.0	9.6	4.8
Computed sample size	***4***	***2***	***4***
From (McGregor et al., 2001)			
Sitting flexed	4.9 (1.8)	5.5 (1.2)	5.6 (1.4)
Sitting extended	7.2 (2.1)	8.4 (3.4)	6.7 (2.2)
Difference between means (δ)	2.3	2.9	1.1
"Pooled" SD (s)	2.0	2.6	1.8
δ^2	5.3	8.4	1.2
s^2	4.0	6.8	3.2
Computed sample size	***12***	***13***	***45***

Based on these sample size calculations, at least 45 participants in each group would be needed to have enough power (at least 80%) to detect a difference between the means of the L5/S1 IVJs. Because of the limits of time and resources and the exploratory nature of the current study, the mean of these sample size estimates of 13.3 (rounded up to 14) was used. Because gender differences were to be examined, it was decided that 15 participants per gender group would be collected. One female and 2 males were excluded from the subsequent analyses because the x-ray images were of insufficient quality to enable precise digitizing of the appropriate landmarks.

Study #2 (Chapter 4)

The proposed sample size for this study (15 males, 15 females) was chosen based on the justification provided for Study #1. Three females and 4 males were excluded from the analyses because the accelerometer data files were unusable.

Study #3 (Chapter 5)

The proposed sample size for this study (15 males, 15 females) was chosen based on the justification provided for Study #1. Other studies using x-ray and fluoroscopy to examine intervertebral kinematics have collected data on up to 20 subjects (i.e. Pearcy et al., 1984; Descarreaux et al., 2003; Teyhen et al., 2005).

Only 10 females and 9 males had videofluoroscopy trials that were adequate for analysis. Trials were excluded if: the relevant vertebrae were not in view at both the beginning and end of the motion, which was necessary for the registration method where a template image is used in the tracking process, or; the image quality was very poor due to noise from changes in image contrast, soft tissue scatter or motion of other internal anatomical structures. Based on a previous study that found significant gender differences in various kinematic variables during sitting, it was decided that these numbers were sufficient to explore any gender effects in the current study.

Study #4 (Chapter 6)

It was decided to test eight participants per group (gender (2) x LBP status (2)) because results from a previous study with very similar methodology testing similar variables observed significant differences between gender groups with the same number of participants per group (Dunk and Callaghan, 2005).

Study #5 (Chapter 7)

Pilot data from 15 specimens (5 per group) were used to compute the necessary sample size to detect a difference in disc height loss. This variable was chosen for two reasons: 1) it was considered an important outcome variable, and; 2) it was possible to estimate a "clinically significant" difference in disc height loss from the literature.

A "clinically significant" difference (δ) in disc height between groups of 0.5mm was chosen. The mean initial disc height of these 15 specimens was 4.8mm and 0.5mm is approximately 10% of this height. Reversible height changes of approximately 8-20% have been reported to occur diurnally (Adams et al., 1987; Botsford et al., 1994).

An a priori sample size calculation was performed using data for total specimen height loss from 15 pilot specimens. Based on the pilot data, the pooled standard deviation (s) for all specimens was 0.3. The formula used to calculate sample size for a comparison between two independent means is as follows:

$$n = \frac{2 * (z_{(1-\frac{\alpha}{2})} + z_{(1-\beta)})^2 * s^2}{\delta^2}$$

where: n = number of samples in each group

z-values = z-values for a 95% confidence level (α=0.05) and 80% power (β=0.20)

s = pooled standard deviation of a known population

δ = hypothesized difference between the two means

Assuming an alpha of .05 and a beta of .20 and using the sample size equation above:

$$n = \frac{2 * (1.96 + 0.84)^2 * (0.3)^2}{(0.5)^2}$$

$$n = 5.64, \text{ or } 6$$

Eight specimens per group were collected because the time and resources were available.

Appendix C:
Supplementary data: Intervertebral joint angles expressed as absolute angles

Introduction

The intervertebral joint (IVJ) angles presented in Chapter 3 were expressed in several different ways in order to examine how each joint moved between postures. For thoroughness, the absolute IVJ angles are presented here, along with a few other analyses that highlight gender differences not previously reported in Chapter 3. The results presented in this Appendix were referred to in Chapter 4 and were discussed in more detail in the General Discussion (Chapter 8)

Methods

Data from the same sample of 27 participants (13 males, 14 females) used in Study #1 (Chapter 3) are reported. One static X-ray image was obtained for standing, half flexion, full flexion, upright sitting and slouched sitting. The top two corners of the sacrum and four corners of the L3 to L5 vertebral bodies (VB) were manually digitized in each X-ray image. IVJ angles were calculated as the angle between the sagittal "mid-plane" lines of adjacent VBs, or the L5 "mid-plane" and the top of the sacrum. The data reported here are the absolute angles, where a negative angle indicates IVJ lordosis and a positive angle indicates IVJ kyphosis. An additional variable was calculated and was called the vertebral body wedge ratio (Ha/Hp). Ha/Hp was calculated as:

(anterior VB height ÷ posterior VB height)

Results

There were significant main effects for posture and joint ($P < 0.0001$), and the interaction between these two factors was also significant ($P < 0.0001$). All IVJs remained lordotic, except for the L3/4 IVJ in half and full standing flexion (Figure B1). Additionally, the L3/4 IVJ appeared to have parallel endplates (mean IVJ angle = 0°) in slouched sitting. A

significant gender*joint interaction also existed ($P = 0.03$), where the female L3/4 and L4/5 IVJs always had more lordosis than the corresponding male IVJs (Figure B2). This effect is possibly explained by the fact that the lower 3 VBs for males had a lower wedging ratio (Ha/Hp = 1.03 (0.11)) than females (Ha/Hp = 1.11 (0.13)) (Table B1). In other words, the male VBs were more rectangular in the sagittal plane, while the female VBs had more posterior wedging, likely allowing for more lordotic IVJ angles.

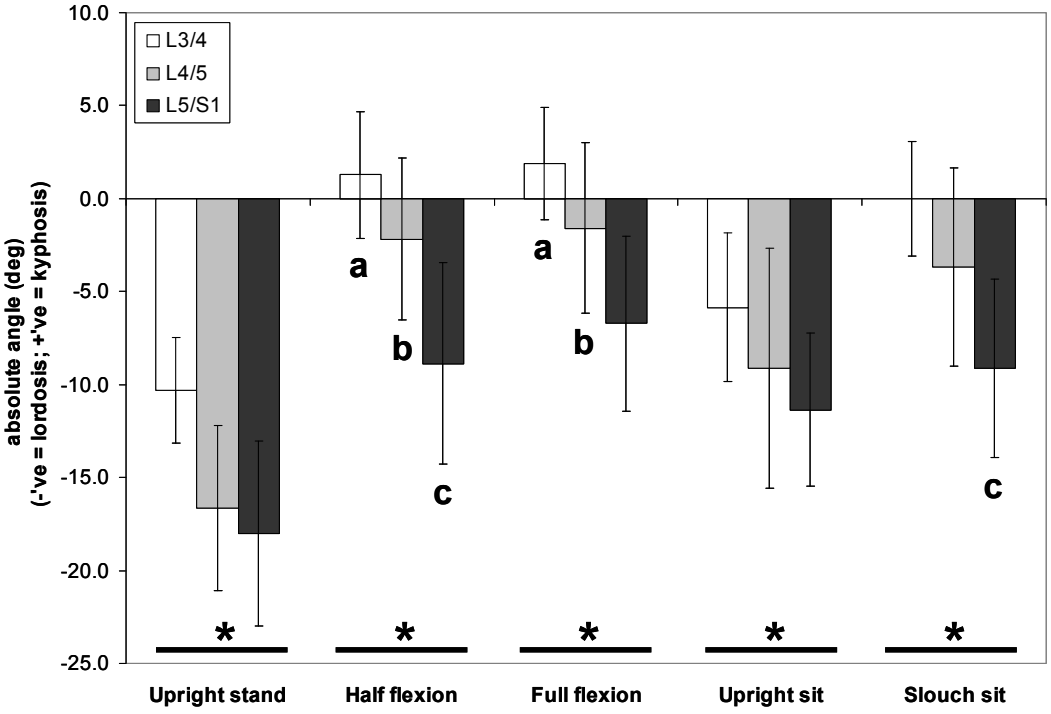


Figure B1: Absolute intervertebral joint angles in 5 different postures. There was a significant joint*posture interaction ($P < 0.0001$). The asterisk (*) indicates that all 3 IVJs were significantly different from each other within a given posture ($P < 0.01$).
'a' indicates that for L3/4, only the half and full flexion postures were NOT significantly different (all other comparison, $P < 0.05$)
'b' indicates that for L4/5, only the half and full flexion postures were NOT significantly different (all other comparison, $P < 0.02$)
'c' indicates that for L5/Sac, only the half flexion and slouched sitting postures were NOT significantly different (all other comparison, $P < 0.0005$)

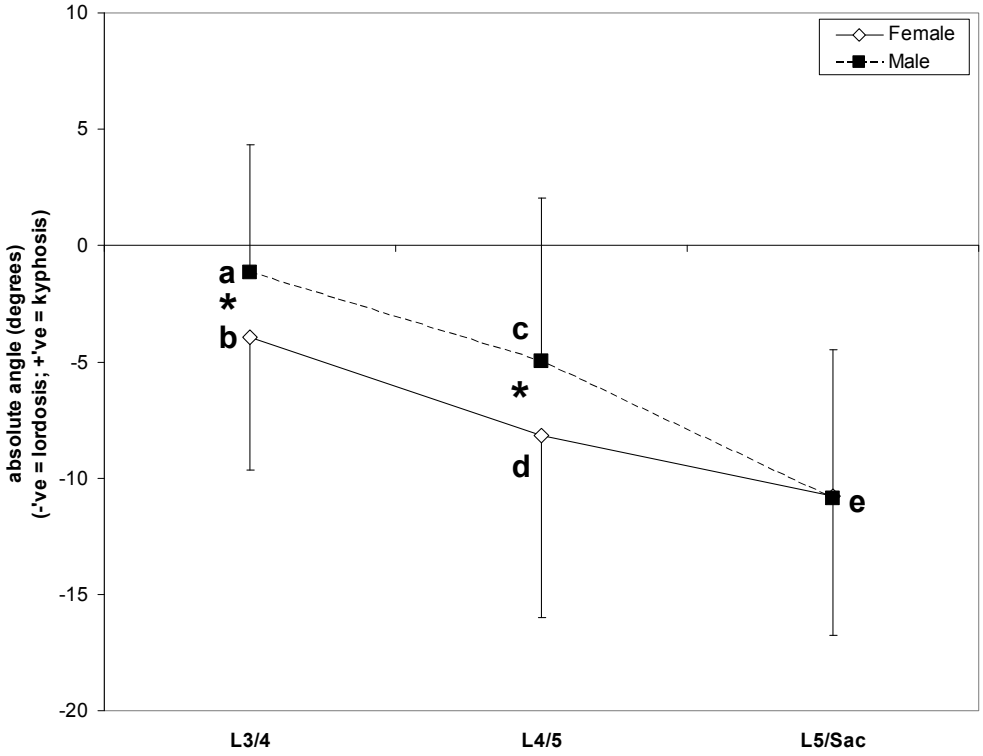


Figure B2: Plot of the significant gender*joint interaction for absolute IVJ angles ($P = 0.03$). Different letters indicate significantly different comparisons ($P < 0.006$). The asterisks (*) highlight the existing gender differences, where female L3/4 and L4/5 always had more lordosis than the corresponding male IVJs.

Table B1: Vertebral body "wedge" ratio (Ha/Hp), calculated as (anterior VB height ÷ poster VB height). A value greater than 1 indicates posterior wedging. The effects of gender ($P = 0.02$) and VB ($P < 0.0001$) were significant, but the interaction was not.

	L3	L4	L5	Combined across VBs (gender effect)
Female	1.04 (0.05)	1.07 (0.08)	1.22 (0.14)	1.11# (0.13)
Male	0.96 (0.08)	1.00 (0.08)	1.12 (0.12)	1.03# (0.11)
Combined across gender (joint effect)	1.00 (0.08)	1.04 (0.08)	1.17* (0.14)	

* - denotes that L5 wedging was significantly different from L3 and L4 ($P < 0.05$)
# - denotes that overall male and female VB wedging was significantly different ($P = 0.02$)

Appendix D:
Verification of the performance of an intensity-based image registration program used for tracking vertebral bodies in fluoroscopic image sequences

Various tests were performed in order to verify the performance of the image registration program described in Study #3 (Chapter 5).

Methods: Test #1 – Phantom spine model

Instrumentation

A phantom spine model was designed to approximate two human vertebral bodies and consisted of two metal square cutouts (5 x 5 cm, 0.5 cm thick) separated by 1.5 cm and attached in parallel to a piece of wood. The wood was fixed to the arm of an electrogoniometer (5000 ohm potentiometer, Model 6639S-001-502, Bourns Precision, Riverside, CA, USA) that was calibrated so that the angular excursion of the arm could be determined (Figure C1). The electrogoniometer signal was digitized at a rate of 30 Hz using a 12-bit (NI 6024E, National Instruments Corporation, Austin, TX, USA) analog-to-digital conversion system and synchronized in time with the fluoroscopy video using an electromechanical contact switch placed on the fluoroscope control foot pedal (see Chapter 5, Section 5.3.3 for full description of fluoroscope).

Data Collection and Analyses

Fluoroscopic images, electrogoniometer and contact switch signals were collected as the spine model was moved through the field of view from left to right (Figure C1). Object-to-intensifier distance was 16 cm, which corresponded to the average distance of a human's spine-to-intensifier distance. A second trial was collected where a plastic jug (20 cm in

diameter) filled with water was placed between the X-ray source and the model to simulate soft tissue scatter (Cholewicki et al., 1991).

"Vertebral body" angular displacements were determined in three different ways: 1) manual digitization of the 4 corners of each "VB" in each frame of the fluoroscopic image sequence; 2) angular displacement of the electrogoniometer, and; 3) the VBs were tracked using the fluoroscopic image registration program described in Chapter 5, Section 5.3.4 For each frame of the two trials, the four corners of each square were manually digitized and the mid-plane angle was determined (similar to the method described in Chapter 3). The angle between the mid-plane and the horizontal was determined using the dot product.

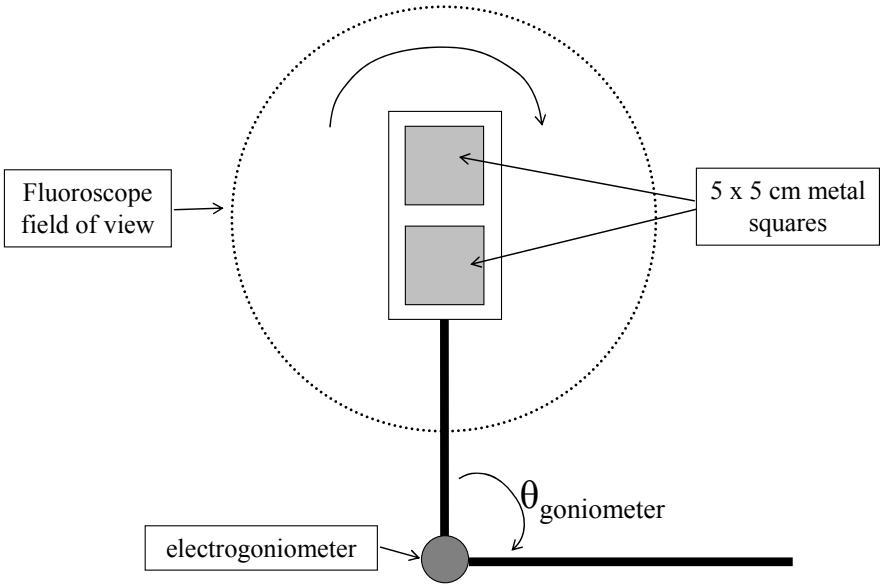


Figure C1: Schematic diagram of the experimental set-up with phantom spine model. The 5x5 cm metal squares were used to simulate two vertebral bodies of the phantom spine model. The goniometer measured the angular displacement of the phantom through the fluoroscope field of view.

Methods: Test #2 – Hand-digitized outputs

VB angular motion data was obtained using the registration program and compared with hand-digitized VB angular motions from 7 participants (4 females, 3 males). In order to digitize the fluoroscopic trial, the video sequence was down-sampled to 6 Hz and each frame was converted to an image using VideoMach (www.gromada.com), yielding approximately 17 frames per trial. The four corners of the L3, L4 and L5 VBs were digitized and angle between the "mid-plane" line and the horizontal was calculated. VB angular motion time series were filtered with a low-pass Butterworth filter (cutoff frequency = 1 Hz). Registration program outputs were also filtered and downsampled to 6 Hz in order to directly compare the two outputs. The mean absolute difference and the correlation between the two angle outputs were calculated for each VB in each trial.

$$M.A.Diff = \frac{1}{n}\sum_{i=1}^{n}\left|\theta_i^{digitized} - \theta_i^{reg-program}\right|$$

Results

Since the phantom spine model did not allow for rotation between the two "vertebrae", any calculated relative rotation could be attributed to error (distortion error, and/or errors accumulated during the image registration). The mean angular differences between the two vertebrae over the entire trial were 0.4° (SD 0.2°) and 0.5° (SD 0.3°) for the no scatter and scatter trials, respectively.

The mean absolute difference and correlations were calculated between 6 paired combinations of VB angle measurements; each measurement method (registration, digitizing or goniometer) was compared for each VB (L4 or L5) (Table A1). If the goniometer angle measurement was considered the "gold standard", it appeared as though the registration method performed better than the digitizing method for both the no scatter and scatter conditions. Interestingly, it appears as though the errors were smaller when scatter was induced. However, this is likely due to the contrast auto-adjustment feature of the

fluoroscope, which kept more uniform contrast (spatial and temporal) across the image sequence when scatter was induced.

When the hand-digitized trials were compared to the VB angular displacements from the registration program, mean absolute differences for each VB across all participants were: L3 = 0.8° (SD 0.4); L4 = 1.6° (SD 0.9); L5 = 1.1° (SD 0.3). In most cases, the registration program over-predicted the rotation of the VB. The mean correlations across all participants were 0.91 (SD 0.09), 0.94 (SD 0.09) and 0.98 (SD 0.02) for the L3, L4 and L5 VBs, respectively. These numbers indicate that although the absolute angle values differed between the two tracking methods, they both followed the movement of the VB in the same way.

Table C1: Mean absolute difference and correlation coefficients between the combinations of measurement methods.

	Registration vs. Goniometer		**Digitizing vs. Goniometer**		**Registration vs. Digitizing**	
	L4-gon	**L5-gon**	**L4-gon**	**L5-gon**	**L4**	**L5**
Trial 1 – no scatter						
Mean absolute error (degrees)	1.0 (0.4)	0.7 (0.3)	1.8 (0.8)	1.2 (0.5)	0.7 (0.3)	0.6 (0.2)
Correlation co-efficient	0.9978	.9981	0.9967	0.9968	0.9999	0.9998
Trial 2 – with scatter						
Absolute mean error (degrees)	0.3 (0.1)	0.6 (0.3)	0.4 (0.4)	1.0 (0.5)	0.3 (0.2)	1.7 (0.6)
Correlation co-efficient	0.993	0.9981	0.9980	0.9995	0.9993	0.9963

Discussion

The distinguishing component of a standard fluoroscopy system is the image intensifier (II) – image intensifiers are far more sensitive than a standard 400-speed screen-film cassette and can produce images using less radiation (Cholewicki et al., 1991; Bushberg et al., 2002). Image intensifier technology requires electrons to be focused using an input screen with a curved surface, thus resulting in "pin cushion" distortion in the output image.

For the fluoroscope used in this study, distortion errors were tested using a grid of points spaced evenly 15 mm apart in both directions. The average error due to distortion was 1.598 pixels, which corresponded to 0.564 mm. The maximum error was 3.918 pixels (or 1.384 mm), and occurred close to the outer edge of the field of view. Since the phantom model in the present analysis did not allow for rotation between the two "vertebrae", any calculated relative rotation could be attributed to error (distortion error, and/or errors accumulated during the image registration). The highest calculated rotation occurred when the phantom approached the edge of the field of view. Since this error was less than 1°, and given that the calibration errors were generally less than 1mm, it is concluded that error due to geometrical distortion is minimal and has little effect on the performance of the registration method.

Because of the lower exposure levels used in fluoroscopy, the contrast resolution is lower thus producing images with relatively low signal to noise ratio. Contrast resolution is increased when higher exposure rates are used (by increasing the mA and kV) but the disadvantage is a higher patient radiation dose (Bushberg et al., 2002). The thickness of the patient will also affect the quality of the output image because thicker regions will attenuate more X-rays so fewer strike the II and generate less light (Bushberg et al., 2002). Different regions of tissue thickness and density will mean that the brightness of the image will vary spatially across an individual frame, or temporally from frame to frame. Motion blurring can also occur when the tissue moves during the collection of photons for a single frame (it takes a finite amount of time to collect a frame) (Bushberg et al., 2002). The registration method used in the current study may be susceptible to errors from motion blurring; however, the signal-to-noise ratio was improved by multi-frame averaging where the three most recently registered frames were used as a template for the registration of the proceeding frame. The mean absolute differences between the hand digitized angles and the registration program outputs were higher for the L4 and L5 VBs. It is likely that these two VBs are most affected by poor image quality and large contrast differences caused by tissues (i.e. intestines, iliac crests) or pockets of air passing in front of the VB. However, poor image quality will affect

the accuracy of both hand-digitizing and an intensity-based registration program, discussed in the next paragraph.

The image registration method eliminates the onerous task of manually digitizing vertebral body landmarks frame by frame through a video sequence. This approach does not require frame-by-frame user decisions about landmark location, making it more automatic. One of the benefits of using the image registration method is that the video could be analyzed at 30Hz, rather than needing to be down-sampled to reduce the analysis time for manual digitization. Furthermore, the intensity-based registration approach can offer higher accuracy for tracking objects because it takes into account all available information from the user-defined region of interest, rather than relying on the user's visual location and selection of VB landmarks. Hand-digitizing of videofluoroscopy sequences is also susceptible to errors from poor image quality and should not be considered the "gold standard" in terms of accurate representation of VB angular displacement. While trials can be digitized multiple times in order to reduce random error associated with this process (Cholewicki et al., 1991), this method is still extremely laborious, time-consuming and potentially harmful for the individual performing the hand-digitizing. The automated registration program performance used in this study can be, and has been, verified: the phantom spine model analysis and comparison with hand-digitized outputs shows adequate performance of the program. Additionally, the motion parameters determined by the image registration program could be applied to the initial video sequence. This process essentially changed the frame of reference from the fluoroscope field of view to the VB being tracked. This resulted in a corrected video where the tracked VB remained fixed and all other image details rotated around it. The VB could be visually assessed to ensure that it remained stationary, thus verifying the tracking performance of the program. Both the hand-digitizing method and the automated registration program are susceptible to errors; in reality, the true answer may lie somewhere in between the outputs from the two methods. However, because of the steps taken to verify

the registration program output, I am convinced that the registration program's performance is adequate.

Appendix E:

Oswestry Disability Index 2.0

Could you please complete this questionnaire. It is designed to give us information as to how your back (or leg) trouble has affected your ability to manage in everyday life.

Please answer *every section*. Mark *one box only* in each section that most closely describes you *today*.

Section 1: Pain intensity
[ ] I have no pain at the moment.
[ ] The pain is very mild at the moment.
[ ] The pain is moderate at the moment.
[ ] The pain is fairly severe at the moment.
[ ] The pain is very severe at the moment.
[ ] The pain is the worst imaginable at the moment.

Section 2: Personal care (washing, dressing, *etc.*)
[ ] I can look after myself normally without causing extra pain.
[ ] I can look after myself normally but it is very painful.
[ ] It is painful to look after myself and I am slow and careful.
[ ] I need some help but manage most of my personal care.
[ ] I need help every day in most aspects of self care.
[ ] I do not get dressed, wash with difficulty, and stay in bed.

Section 3: Lifting
[ ] I can lift heavy weights without extra pain.
[ ] I can lift heavy weights but it gives extra pain.
[ ] Pain prevents me from lifting heavy weights off the floor but I can manage if they are conveniently positioned, *e.g.*, on a table.
[ ] Pain prevents me from lifting heavy weights but I can manage light to medium weights if they are conveniently positioned.
[ ] I can lift only very light weights.
[ ] I cannot lift or carry anything at all.

Section 4: Walking
[ ] Pain does not prevent me walking any distance.
[ ] Pain prevents me walking more than 1 mile.
[ ] Pain prevents me walking more than a quarter of a mile.
[ ] Pain prevents me walking more than 100 yards.
[ ] I can only walk using a stick or crutches.

[ ] I am in bed most of the time and have to crawl to the toilet.

Section 5: Sitting
[ ] I can sit in any chair as long as I like.
[ ] I can sit in my favorite chair as long as I like.
[ ] Pain prevents me from sitting for more than 1 hour.
[ ] Pain prevents me from sitting for more than half an hour.
[ ] Pain prevents me from sitting for more than 10 minutes.
[ ] Pain prevents me from sitting at all.

Section 6: Standing
[ ] I can stand as long as I want without extra pain.
[ ] I can stand as long as I want but it gives me extra pain.
[ ] Pain prevents me from standing for more than 1 hour.
[ ] Pain prevents me from standing for more than half an hour.
[ ] Pain prevents me from standing for more than 10 minutes.
[ ] Pain prevents me from standing at all.

Section 7: Sleeping
[ ] My sleep is never disturbed by pain.
[ ] My sleep is occasionally disturbed by pain.
[ ] Because of pain I have less than 6 hours' sleep.
[ ] Because of pain I have less than 4 hours' sleep.
[ ] Because of pain I have less than 2 hours' sleep.
[ ] Pain prevents me from sleeping at all.

Section 8: Sex life (if applicable)
[ ] My sex life is normal and causes no extra pain.
[ ] My sex life is normal but causes some extra pain.
[ ] My sex life is nearly normal but is very painful.
[ ] My sex life is severely restricted by pain.
[ ] My sex life is nearly absent because of pain.
[ ] Pain prevents any sex life at all.

Section 9: Social life
[ ] My social life is normal and causes me no extra pain.
[ ] My social life is normal but increases the degree of pain.
[ ] Pain has no significant effect on my social life apart from limiting my more energetic interests, *e.g.*, sport, *etc.*
[ ] Pain has restricted my social life and I do not go out as often.
[ ] Pain has restricted social life to my home.
[ ] I have no social life because of pain.

Section 10: Traveling

[ ] I can travel anywhere without pain.
[ ] I can travel anywhere but it gives extra pain.
[ ] Pain is bad but I manage journeys over 2 hours.
[ ] Pain restricts me to journeys of less than 1 hour.
[ ] Pain restricts me to short necessary journeys under 30 minutes.
[ ] Pain prevents me from traveling except to receive treatment.

Scoring the ODI

For each section of six statements the total score is 5; if the first statement is marked, the score is 0; if the last statement is marked, it is 5. Intervening statements are scored according to rank. If more than one box is marked in each section, take the highest score.

If all 10 sections are completed the score is calculated as follows: if 16 (total scored) out of 50 (total possible score) x 100 = 32%.
If one section is missed (or not applicable) the score is calculated: Example: 16 (total scored)/45 (total possible score) x 100 = 35.5% Therefore, the final score may be summarized as: (total score/(5 3 number of questions answered) x 100%. The authors suggest rounding the percentage to a whole number for convenience.

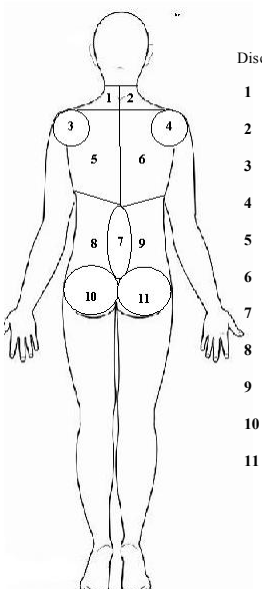
Appendix F:

Visual Analog Scale for discomfort/pain measurement

RATING OF PERCEIVED DISCOMFORT SCALE

Part A: Please make a mark on each line corresponding to the areas in which you are currently feeling discomfort

Part B: Please check the appropriate descriptive word box corresponding to the areas marked in Part A that describes your current level of discomfort



	No Discomfort — Worst Discomfort Imaginable	Pain	Tiredness	Soreness	Numbness	Sharp	Ache	Pressure	Other	
1	_____	☐	☐	☐	☐	☐	☐	☐	☐	_____
2	_____	☐	☐	☐	☐	☐	☐	☐	☐	_____
3	_____	☐	☐	☐	☐	☐	☐	☐	☐	_____
4	_____	☐	☐	☐	☐	☐	☐	☐	☐	_____
5	_____	☐	☐	☐	☐	☐	☐	☐	☐	_____
6	_____	☐	☐	☐	☐	☐	☐	☐	☐	_____
7	_____	☐	☐	☐	☐	☐	☐	☐	☐	_____
8	_____	☐	☐	☐	☐	☐	☐	☐	☐	_____
9	_____	☐	☐	☐	☐	☐	☐	☐	☐	_____
10	_____	☐	☐	☐	☐	☐	☐	☐	☐	_____
11	_____	☐	☐	☐	☐	☐	☐	☐	☐	_____

Appendix G:
Study #4: Noise-contaminated erector spinae EMG signal processing issues and decisions

The goal of Study #4 (Chapter 6) was to examine the time-varying biological responses of individuals with sitting-induced LBP and compare them to asymptomatic controls. It was the intention to investigate erector spinae muscle activity because LBP patients have demonstrated altered muscle responses in seated postures (Radebold et al., 2001; Dankaerts et al., 2006a).

During the 90 minute sitting period, data were collected simultaneously from the seat pan pressure mat, OPTOTRAK motion capture system, accelerometers and erector spinae EMG electrodes. The following concern was noted in the EMG signal: there were large spikes in the EMG with a frequency of 4Hz that appeared much larger on the right side of the body. These spikes corresponded to the 4 Hz sampling frequency of the pressure mats, and the circuitry for the pressure mats was located on the participant's right side (Figure 1). When data were collected without the pressure mat, the spikes were not present in the signal. Data collection continued with all systems because it was thought that the noise could be dealt with during the data analysis stage.

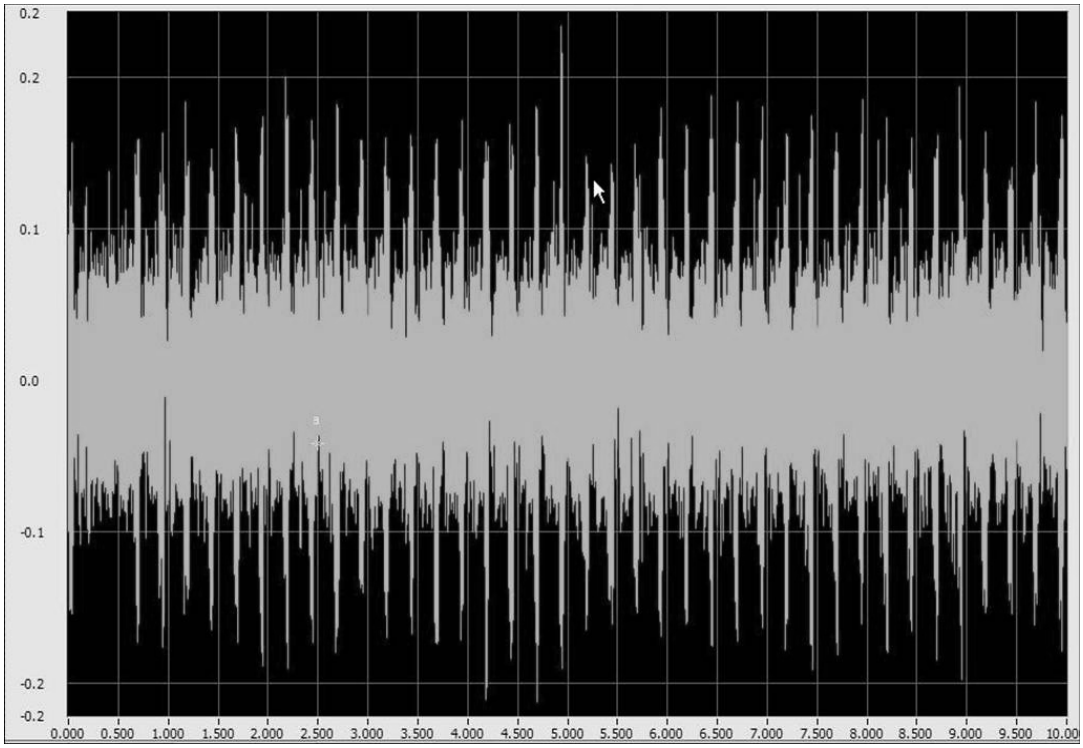


Figure F1: Raw EMG collected concurrently with the pressure mapping system from the right upper erector spinae muscle. Note the spikes that correspond to the 4 Hz collection frequency of the pressure system.

Several steps were taken to reduce this noise contamination:

1. Fast Fourier transforms (FFT) were performed on several data sets to examine how the noise was affecting the frequency spectrum of the contaminated EMG (Figure 2).

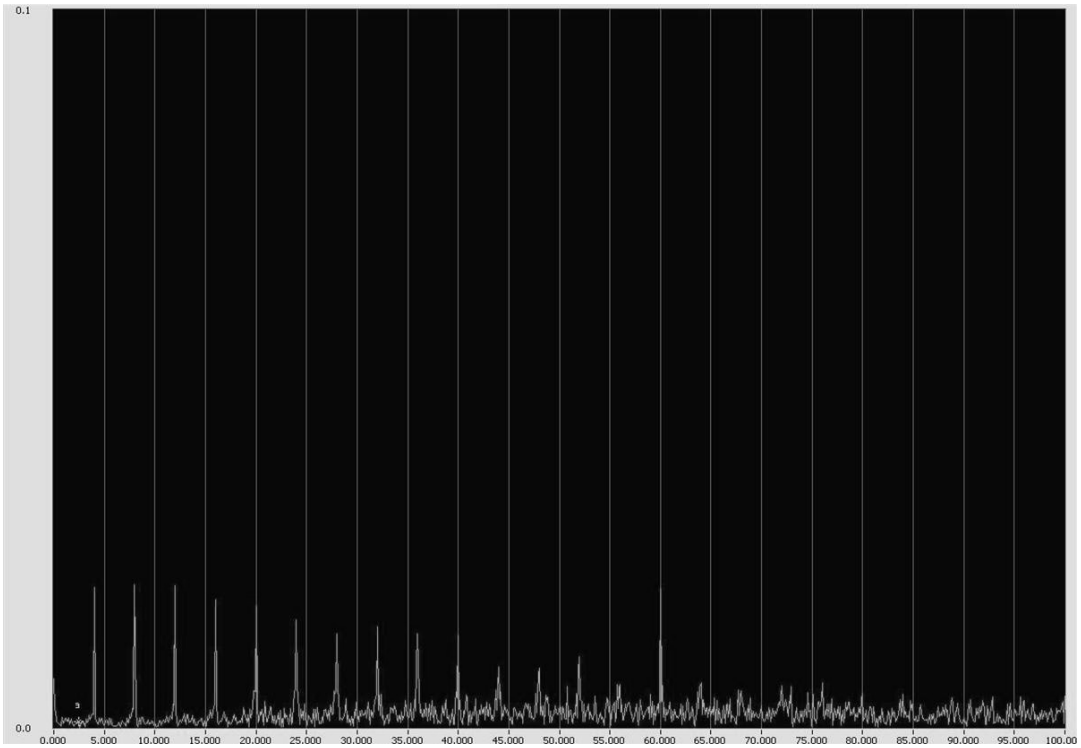


Figure F2: Example of the EMG power spectral density of the signal in Figure F1 that was contaminated with 4Hz noise from the pressure mapping system.

2. The contaminated raw EMG data were filtered using a zero lag 4th order low-pass Butterworth filter with a cut-off frequency of 350 Hz. This cut-off frequency was chosen for the following reasons:

    a. It visually reduced the amplitude of the 4 Hz spikes in the raw EMG.

    b. A residual analysis was performed according to Winter (1990): Based on the power spectral density of the contaminated EMG signal (Figure 2), the cut-off frequencies between 300 and 450 Hz were used in increments of 10 Hz. The root mean square (RMS) difference between the filtered and unfiltered signals over the cut-off range was computed and plotted (Figure 3). The signal distortion started to decrease after about 350 Hz.

c. It has been suggested that the bandwidth of erector spinae EMG is between 5 and 300 Hz and that the signal above 300 Hz is small and varies little during muscle contractions (Dolan et al., 1995)

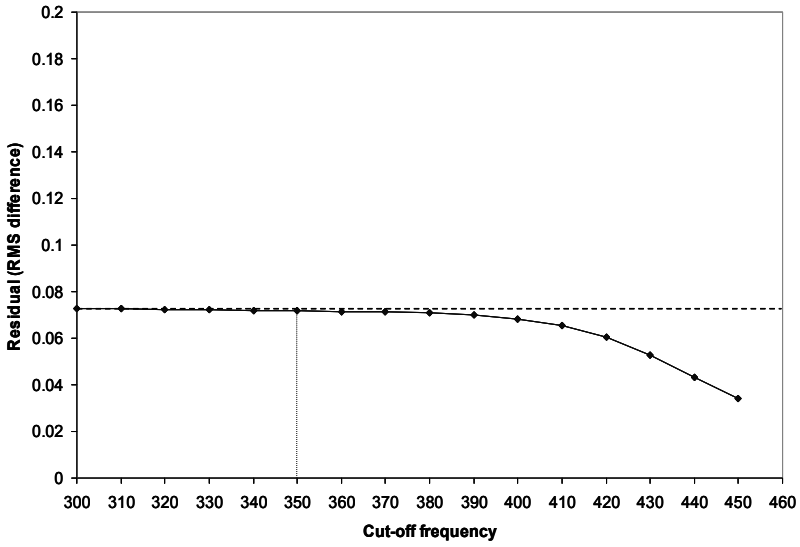


Figure F3: Plot of the residual between the filtered and unfiltered EMG signal as a function of the filter cutoff frequency. The signal distortion (i.e. residual) was reduced after about 350 Hz.

The final EMG processing steps were as follows:

1. Zero lag 4th order high pass Butterworth filter (cut-off frequency = 30 Hz) – to reduce heart rate artifact (Drake and Callaghan, 2006).
2. Zero lag 4th order low pass Butterworth filter (cut-off frequency = 350 Hz) – to reduce pressure mat sampling frequency contamination.
3. Linear envelop: full-wave rectify raw EMG signal; single pass 2nd order low pass Butterworth filter (cut-off frequency = 2.5 Hz) (Brereton and McGill, 1998).
4. The signal was normalized to the corresponding muscle's submaximal voluntary contraction (see Chapter 6, Section 6.4.4)

Cross-correlation analyses have been previously used as a method of describing coordination of muscle activation (Nelson-Wong et al., 2008). Cross-correlation quantifies the common signals present in two time-varying waveforms (i.e. two different EMG recording sites), and one of the goals of Study #4 was to examine the coordination of the erector spinae muscle activation during prolonged sitting. Cross-correlation techniques are also ideal for identifying noise in a signal and were used in this case to verify if the EMG signal processing steps described above were successful in reducing the noise in the signal.

As can be seen in Figure 4, the cross-correlation between the lower ES muscles revealed that these recording sites had a 4 Hz periodic signal in common. The 4 Hz signal is not noticeable in the upper ES cross-correlation function likely because these electrodes were further away from the source of the noise (i.e. the seat pan pressure mat). However, it is possible that the increase in the strength of the cross-correlation at phase shift (τ) = 0 seconds is falsely larger because of the presence of the common noise signal.

The noise in the EMG signal was present in both the amplitude and frequency domains. Signal processing steps reduced but did not eliminate the amplitude of the noise. The signal was still present in the frequency domain as demonstrated by the cross-correlation techniques. While muscle activation and coordination data would add significantly to the findings of Study #4, it was decided that the EMG data were too unreliable for inclusion in the study results.

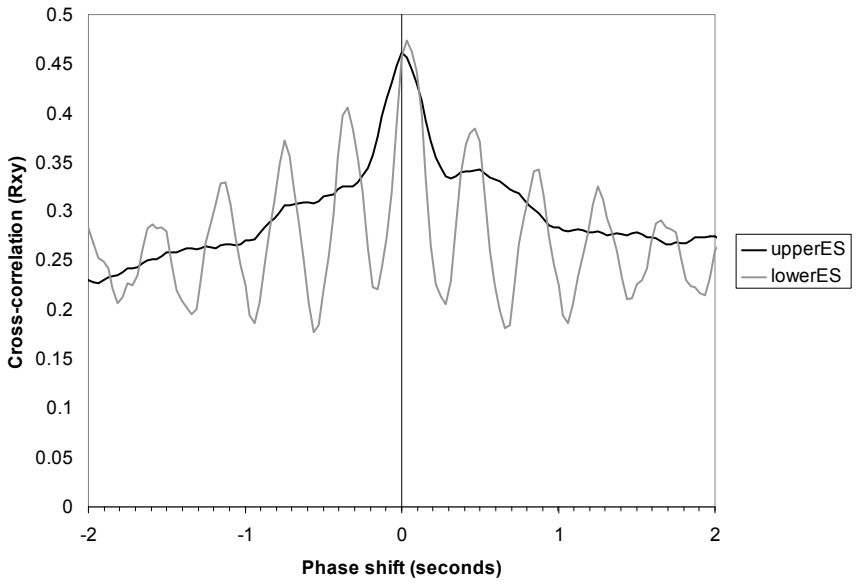


Figure F4: Normalized cross-correlation function for two signal correlations. UpperES is the cross-correlation between the right and left upper erector spinae EMG signals. LowerES is the cross-correlation between the right and left lower erector spinae EMG signals.

Bibliography

Adams, M.A., Dolan, P., Hutton, W.C., 1987. Diurnal variations in the stresses on the lumbar spine. Spine 12, 130-137.

Adams, M.A., Dolan, P., Hutton, W.C., Porter, R.W., 1990. Diurnal changes in spinal mechanics and their clinical significance. J Bone Joint Surg. Br. 72, 266-270.

Adams, M.A., Dolan, P., Marx, C., Hutton, W.C., 1986. An electronic inclinometer technique for measuring lumbar curvature. Clin Biomech 1, 130-134.

Adams, M.A., Hutton, W.C., 1983. The effect of posture on the fluid content of lumbar intervertebral discs. Spine 8, 665-671.

Adams, M.A., Hutton, W.C., 1985. Gradual disc prolapse. Spine 10, 524-531.

Adams, M.A., Hutton, W.C., 1986. The effect of posture on diffusion into lumbar intervertebral discs. J Anat. 147, 121-134.

Adams, M.A., McMillan, D.W., Green, T.P., Dolan, P., 1996. Sustained loading generates stress concentrations in lumbar intervertebral discs. Spine 21, 434-438.

Adams, M.A., McNally, D.S., Chinn, H., Dolan, P., 1994. Posture and the compressive strength of the lumbar spine. Clin Biomech 9, 5-14.

Al Eisa, E., Egan, D., Deluzio, K., Wassersug, R., 2006. Effects of pelvic asymmetry and low back pain on trunk kinematics during sitting: a comparison with standing. Spine 31, E135-E143.

Alexander, L.A., Hancock, E., Agouris, I., Smith, F.W., MacSween, A., 2007. The response of the nucleus pulposus of the lumbar intervertebral discs to functionally loaded positions. Spine 32, 1508-1512.

Althoff, I., Brinckmann, P., Frobin, W., Sandover, J., Burton, K., 1992. An improved method of stature measurement for quantitative determination of spinal loading. Application to sitting postures and whole body vibration. Spine 17, 682-693.

Andersson, B.J., Jonsson, B., Ortengren, R., 1974. Myoelectric activity in individual lumbar erector spinae muscles in sitting. A study with surface and wire electrodes. Scand. J. Rehabil. Med. Suppl 3, 91-108.

Andersson, B.J., Ortengren, R., Nachemson, A.L., Elfstrom, G., Broman, H., 1975. The sitting posture: an electromyographic and discometric study. Orthop Clin North Am 6, 105-120.

Andersson, E., Oddsson, L., Grundstrom, H., Thorstensson, A., 1995. The role of the psoas and iliacus muscles for stability and movement of the lumbar spine, pelvis and hip. Scand. J. Med. Sci. Sports 5, 10-16.

Andersson, E.A., Oddsson, L.I.E., Grundstrom, H., Nilsson, J., Thorstensson, A., 1996. EMG activities of the quadratus lumborum and erector spinae muscles during flexion-relaxation and other motor tasks. Clin. Biomech. 11, 392-400.

Andersson, G.B.J., Murphy, R.W., Ortengren, R., Nachemson, A.L., 1979. The influence of backrest inclination and lumbar support on lumbar lordosis. Spine 4, 52-58.

Beach, T.A., McDonald, K.A., Coke, S.K., Callaghan, J.P., 2008. Gender responses to automobile and office sitting - Influence of hip, hamstring and low-back flexibility on seated postures. Ergonomics Open Journal 1, 1-9.

Beach, T.A., Parkinson, R.J., Stothart, J.P., Callaghan, J.P., 2005. Effects of prolonged sitting on the passive flexion stiffness of the in vivo lumbar spine. Spine J. 5, 145-154.

Bernmark, E., Wiktorin, C., 2002. A triaxial accelerometer for measuring arm movements. Appl. Ergon. 33, 541-547.

Bhatnager, V., Drury, C.G., Schiro, S.G., 1985. Posture, postural discomfort, and performance. Hum. Factors 27, 189-199.

Blangsted, A.K., Vedsted, P., Sjogaard, G., Sogaard, K., 2005. Intramuscular pressure and tissue oxygenation during low-force static contraction do not underlie muscle fatigue. Acta Physiol Scand. 183, 379-388.

Borg, G., 1998. Borg's Perceived Exertion and Pain Scales. 1st ed. Human Kinetics, Champaign, IL.

Botsford, D.J., Esses, S.I., Ogilvie-Harris, D.J., 1994. In vivo diurnal variation in intervertebral disc volume and morphology. Spine 19, 935-940.

Breen, A., Allen, R., Morris, A., 1989. A digital videofluoroscopic technique for spine kinematics. J Med Eng Technol 13, 109-113.

Brereton, L.C., McGill, S.M., 1998. Frequency response of spine extensors during rapid isometric contractions: effects of muscle length and tension. J. Electromyogr. Kinesiol. 8, 227-232.

Bridger, R.S., Orkin, D., Henneberg, M., 1992. A quantitative investigation of lumbar and pelvic postures in standing and sitting: Interrelationships with body position and hip muscle length. Int J Ind Ergon 9, 235-244.

Bridger, R.S., Wilkinson, D., Van Houweninge, T., 1989. Hip joint mobility and spinal angles in standing and in different sitting postures. Hum Factors 31, 229-241.

Brinckmann, P., Frobin, W., Biggemann, M., Hilweg, D., Seidel, S., Burton, K., Tillotson, M., Sandover, J., Atha, J., Quinnell, R., 1994. Quantification of overload injuries to thoracolumbar vertebrae and discs in persons exposed to heavy physical exertions or vibration at the workplace. Part I. The shape of vertebrae and intervertebral discs. Clin Biomech 9, S1-S83.

Bullock-Saxton, J., 1993. Postural alignment in standing: A repeatability study. Austr. J. Physiother. 39, 25-29.

Bushberg, J.T., Seibert, J.A., Leidholdt Jr, E.M., Boone, J.M., 2002. The essential physics of medical imaging. 2nd ed. Lippincott Williams & Wilkins, Philadelphia, PA.

Callaghan, J.P., Dunk, N.M., 2002. Examination of the flexion relaxation phenomenon in erector spinae muscles during short duration slumped sitting. Clin Biomech 17, 353-360.

Callaghan, J.P., McGill, S.M., 1995. Frozen storage increases the ultimate compressive load of porcine vertebrae. J Orthop Res 13, 809-812.

Callaghan, J.P., McGill, S.M., 2001a. Intervertebral disc herniation: studies on a porcine model exposed to highly repetitive flexion/extension motion with compressive force. Clin. Biomech. (Bristol. , Avon.) 16, 28-37.

Callaghan, J.P., McGill, S.M., 2001b. Low back joint loading and kinematics during standing and unsupported sitting. Ergonomics 44, 280-294.

Campbell-Kyureghyan, N., Jorgensen, M., Burr, D., Marras, W., 2005. The prediction of lumbar spine geometry: method development and validation. Clin. Biomech. (Bristol. , Avon.) 20, 455-464.

Carcone, S.M., Keir, P.J., 2007. Effects of backrest design on biomechanics and comfort during seated work. Appl. Ergon. 38, 755-764.

Cavanaugh, J.M., 1995. Neural mechanisms of lumbar pain. Spine 20, 1804-1809.

Cavanaugh, J.M., Ozaktay, A.C., Yamashita, H.T., King, A.I., 1996. Lumbar facet pain: biomechanics, neuroanatomy and neurophysiology. J. Biomech. 29, 1117-1129.

Cavanaugh, J.M., Ozaktay, A.C., Yamashita, T., Avramov, A., Getchell, T.V., King, A.I., 1997. Mechanisms of low back pain: a neurophysiologic and neuroanatomic study. Clin Orthop Relat Res , 166-180.

Chaffin, D.B., 1969. A computerized biomechanical model - Development of and use in studying gross body actions. J Biomech 2, 429-441.

Chen, J.C., Chan, W.P., Katz, J.N., Chang, W.P., Christiani, D.C., 2004. Occupational and personal factors associated with acquired lumbar spondylolisthesis of urban taxi drivers. Occup Environ Med 61, 992-998.

Chen, Y.L., 2000. Predicting the vertebral inclination of the lumbar spine. Ergonomics 43, 744-751.

Chen, Y.L., Lee, Y.H., 1997. A non-invasive protocol for the determination of lumbosacral vertebral angle. Clin. Biomech. (Bristol. , Avon.) 12, 185-189.

Cheng, X.G., Sun, Y., Boonen, S., Nicholson, P.H., Brys, P., Dequeker, J., Felsenberg, D., 1998. Measurements of vertebral shape by radiographic morphometry: sex differences and relationships with vertebral level and lumbar lordosis. Skeletal Radiol 27, 380-384.

Ching, C.T., Chow, D.H., Yao, F.Y., Holmes, A.D., 2003. The effect of cyclic compression on the mechanical properties of the inter-vertebral disc: an in vivo study in a rat tail model. Clin. Biomech. (Bristol. , Avon.) 18, 182-189.

Cholewicki, J., McGill, S.M., 1992. Lumbar posterior ligament involvement during extremely heavy lifts estimated from fluoroscopic measurements. J Biomech 25, 17-28.

Cholewicki, J., McGill, S.M., 1996. Mechanical stability of the in vivo lumbar spine: implications for injury and chronic low back pain. Clin. Biomech. (Bristol. , Avon.) 11, 1-15.

Cholewicki, J., McGill, S.M., Wells, R.P., Vernon, H., 1991. Method for measuring vertebral kinematics from videofluoroscopy. Clin Biomech 6, 73-78.

Claus, A.P., Hides, J.A., Moseley, G.L., Hodges, P.W., 2009. Is 'ideal' sitting posture real? Measurement of spinal curves in four sitting postures. Man. Ther. 14, 404-408.

Correia, H., Balseiro, S., De Areia, M., 2005. Sexual dimorphism in the human pelvis: testing a new hypothesis. Homo 56, 153-160.

Costi, J.J., Hearn, T.C., Fazzalari, N.L., 2002. The effect of hydration on the stiffness of intervertebral discs in an ovine model. Clin. Biomech. (Bristol. , Avon.) 17, 446-455.

Dankaerts, W., O'Sullivan, P., Burnett, A., Straker, L., 2006a. Altered patterns of superficial trunk muscle activation during sitting in nonspecific chronic low back pain patients: importance of subclassification. Spine 31, 2017-2023.

Dankaerts, W., O'Sullivan, P., Burnett, A., Straker, L., 2006b. Differences in sitting postures are associated with nonspecific chronic low back pain disorders when patients are subclassified. Spine 31, 698-704.

Dankaerts, W., O'Sullivan, P.B., Burnett, A.F., Straker, L.M., Danneels, L.A., 2004. Reliability of EMG measurements for trunk muscles during maximal and sub-maximal voluntary isometric contractions in healthy controls and CLBP patients. J. Electromyogr. Kinesiol. 14, 333-342.

Davis, D.S., Quinn, R.O., Whiteman, C.T., Williams, J.D., Young, C.R., 2008. Concurrent validity of four clinical tests used to measure hamstring flexibility. J. Strength. Cond. Res. 22, 583-588.

De Carvalho, D.E., Dunk, N.M., Callaghan, J.P., 2007. Gender differences in spinal posture and user positioning on a prototype seat pan. In: 31st Annual Meeting of the American Society of Biomechanics. Stanford, California: American Society of Biomechanics, August 23-25.

de Looze, M.P., Kuijt-Evers, L.F., van Dieen, J., 2003. Sitting comfort and discomfort and the relationships with objective measures. Ergonomics 46, 985-997.

Dhillon, N., Bass, E.C., Lotz, J.C., 2001. Effect of frozen storage on the creep behavior of human intervertebral discs. Spine 26, 883-888.

Dickey, J.P., Gillespie, K.A., 2003. Representation of passive spinal element contributions to in vitro flexion-extension using a polynomial model: illustration using the porcine lumbar spine. J. Biomech. 36, 883-888.

Dolan, K.J., Green, A., 2006. Lumbar spine reposition sense: the effect of a 'slouched' posture. Man. Ther. 11, 202-207.

Dolan, P., Adams, M.A., Hutton, W.C., 1988. Commonly adopted postures and their effect on the lumbar spine. Spine 13, 197-201.

Drake, J.D., Callaghan, J.P., 2006. Elimination of electrocardiogram contamination from electromyogram signals: An evaluation of currently used removal techniques. J. Electromyogr. Kinesiol. 16, 175-187.

Duarte, M., Zatsiorsky, V.M., 1999. Patterns of center of presure migration during prolonged unconstrained standing. Motor Control 3, 12-27.

Dumas, R., Aissaoui, R., de Guise, J.A., 2004. A 3D generic inverse dynamic method using wrench notation and quaternion algebra. Comput. Methods Biomech. Biomed. Engin. 7, 159-166.

Dunk, N.M., Brown, S.H.M., Callaghan, J.P., 2004. Distribution of tissue loads supporting the low back during the flexion relaxation phenomenon in sitting and standing postures. In: The 13th Proceedings of the Canadian Society for Biomechanics. Halifax, NS: Canadian Society for Biomechanics, August 4-7.

Dunk, N.M., Callaghan, J.P., 2005. Gender-based differences in postural responses to seated exposures. Clin Biomech 20, 1101-1110.

Dunk, N.M., Kedgley, A.E., Jenkyn, T.R., Callaghan, J.P., 2007. Seated lumbar intervertebral angles using fluoroscopy. Stanford, California: 31st Annual Meeting of the American Society of Biomechanics, August 23-25.

Dunk, N.M., Lalonde, J., Callaghan, J.P., 2005. Implications for the use of postural analysis as a clinical diagnostic tool: reliability of quantifying upright standing spinal postures from photographic images. J Manipulative Physiol Ther. 28, 386-392.

Dvorak, J., Panjabi, M.M., Chang, D.G., Theiler, R., Grob, D., 1991. Functional radiographic diagnosis of the lumbar spine. Flexion-extension and lateral bending. Spine 16, 562-571.

Eklund, J., Corlett, E.N., 1987. Evaluation of spinal loads and chair design in seated work tasks. Clin Biomech 2, 27-33.

Ekstrom, L., Holm, S., Holm, A.K., Hansson, T., 2004. In vivo porcine intradiscal pressure as a function of external loading. J Spinal Disord Tech. 17, 312-316.

Ekstrom, L., Kaigle, A., Hult, E., Holm, S., Rostedt, M., Hansson, T., 1996. Intervertebral disc response to cyclic loading--an animal model. Proc. Inst. Mech. Eng [H.] 210, 249-258.

Fagan, A., Moore, R., Vernon, R.B., Blumbergs, P., Fraser, R., 2003. ISSLS prize winner: The innervation of the intervertebral disc: a quantitative analysis. Spine 28, 2570-2576.

Fairbank, J.C., Pynsent, P.B., 2000. The Oswestry Disability Index. Spine 25, 2940-2952.

Fenety, A., Walker, J.M., 2002. Short-term effects of workstation exercises on musculoskeletal discomfort and postural changes in seated video display unit workers. Phys. Ther. 82, 578-589.

Fenety, P.A., Putnam, C., Walker, J.M., 2000. In-chair movement: validity, reliability and implications for measuring sitting discomfort. Appl. Ergon. 31, 383-393.

Frobin, W., Brinckmann, P., Leivseth, G., Biggemann, M., Reikeras, O., 1996. Precision measurement of segmental motion from flexion-extension radiographs of the lumbar spine. Clin Biomech 11, 457-465.

Frymoyer, J.W., Pope, M.H., Costanza, M.C., Rosen, J.C., Goggin, J.E., Wilder, D.G., 1980. Epidemiologic studies of low back pain. Spine 5, 419-423.

Gatton, M.L., Pearcy, M.J., 1999. Kinematics and movement sequencing during flexion of the lumbar spine. Clin. Biomech. (Bristol. , Avon.) 14, 376-383.

Genaidy, A.M., Waly, S.M., Khalil, T.M., Hidalgo, J., 1993. Spinal compression tolerance limits for the design of manual material handling operations in the workplace. Ergonomics 36, 415-434.

Goh, S., Price, R.I., Leedman, P.J., Singer, K.P., 1999. Rasterstereographic analysis of the thoracic sagittal curvature: a reliability study. Journal of Musculoskeletal Research 3, 137-142.

Gombatto, S.P., Norton, B.J., Scholtes, S.A., Van Dillen, L.R., 2008. Differences in symmetry of lumbar region passive tissue characteristics between people with and people without low back pain. Clin Biomech. (Bristol. , Avon.) .

Grados, F., Fardellone, P., Benammar, M., Muller, C., Roux, C., Sebert, J.L., 1999. Influence of age and sex on vertebral shape indices assessed by radiographic morphometry. Osteoporos Int 10, 450-455.

Gray, H., 2000. Anatomy of the Human Body. Available from www.bartleby.com/107/ (accessed on 10/20/08).

Gregory, D.E., Dunk, N.M., Callaghan, J.P., 2006. Stability ball versus office chair: Comparison of muscle activation and lumbar spine posture during prolonged sitting. Hum Factors 48, 142-153.

Grieco, A., 1986. Sitting posture: an old problem and a new one. Ergonomics 29, 345-362.

Gunning, J.L., Callaghan, J.P., McGill, S.M., 2001. Spinal posture and prior loading history modulate compressive strength and type of failure in the spine: a biomechanical study using a porcine cervical spine model. Clin Biomech. (Bristol. , Avon.) 16, 471-480.

Halbertsma, J.P., Goeken, L.N., Hof, A.L., Groothoff, J.W., Eisma, W.H., 2001. Extensibility and stiffness of the hamstrings in patients with nonspecific low back pain. Arch. Phys. Med Rehabil. 82, 232-238.

Hansson, G.A., Asterland, P., Holmer, N.G., Skerfving, S., 2001. Validity and reliability of triaxial accelerometers for inclinometry in posture analysis. Med Biol. Eng Comput. 39, 405-413.

Harada, M., Abumi, K., Ito, M., Kaneda, K., 2000. Cineradiographic motion analysis of normal lumbar spine during forward and backward flexion. Spine 25, 1932-1937.

Hartvigsen, J., Leboeuf-Yde, C., Lings, S., Corder, E.H., 2000. Is sitting-while-at-work associated with low back pain? A systematic, critical literature review. Scand. J. Public Health 28, 230-239.

Hedman, T.P., Fernie, G.R., 1995. In vivo measurement of lumbar spinal creep in two seated postures using magnetic resonance imaging. Spine 20, 178-183.

Hedman, T.P., Fernie, G.R., 1997. Mechanical response of the lumbar spine to seated postural loads. Spine 22, 734-743.

Higuchi, K., Sato, T., 2002. Anatomical study of lumbar spine innervation. Folia Morphol. (Warsz.) 61, 71-79.

Holm, S., Nachemson, A., 1983. Variations in the nutrition of the canine intervertebral disc induced by motion. Spine 8, 866-874.

Horn, B.K.P., 1987. Closed-form solution of absolute orientation using unit quaternions. Journal of the Optical Society of America A 4, 629-642.

Hubley-Kozey, C.L., Vezina, M.J., 2002. Differentiating temporal electromyographic waveforms between those with chronic low back pain and healthy controls. Clin. Biomech. (Bristol. , Avon.) 17, 621-629.

Jensen, C.V., Bendix, T., 1992. Spontaneous movements with various seated-workplace adjustments. Clin Biomech 7, 87-90.

Johannessen, W., Vresilovic, E.J., Wright, A.C., Elliott, D.M., 2004. Intervertebral disc mechanics are restored following cyclic loading and unloaded recovery. Ann. Biomed. Eng 32, 70-76.

Jurgens, H.W., 1989. Body movement during observation tasks. Ann. Physiol Anthropol. 8, 33-35.

Kanayama, M., Abumi, K., Kaneda, K., Tadano, S., Ukai, T., 1996. Phase lag of the intersegmental motion in flexion-extension of the lumbar and lumbosacral spine. An in vivo study. Spine 21, 1416-1422.

Kasahara, S., Miyamoto, K., Takahashi, M., Yamanaka, M., Takeda, N., 2008. Lumbar-pelvic coordination in the sitting position. Gait Posture 28, 251-257.

Keegan, J.J., 1953. Alterations of the lumbar curve related to posture and seating. J Bone Joint Surg Am 35-A, 589-603.

Kell, R.T., Farag, M., Bhambhani, Y., 2004. Reliability of erector spinae oxygenation and blood volume responses using near-infrared spectroscopy in healthy males. Eur. J. Appl. Physiol 91, 499-507.

Keller, T.S., Holm, S.H., Hansson, T.H., Spengler, D.M., 1990. The dependence of intervertebral disc mechanical properties on physiologic conditions. Spine 15, 751-761.

Kelsey, J.L., 1975. An epidemiological study of the relationship between occupations and acute herniated lumbar intervertebral discs. Int J Epidemiol 4, 197-205.

Keyserling, W.M., Sudarsan, S.P., Martin, B.J., Haig, A.J., Armstrong, T.J., 2005. Effects of low back disability status on lower back discomfort during sustained and cyclical trunk flexion. Ergonomics 48, 219-233.

Kietrys, D.M., Galper, J.S., Verno, V., 2007. Effects of at-work exercises on computer operators. Work 28, 67-75.

Kingma, I., Van Dieen, J.H., 2008. Static and dynamic postural loadings during computer work in females: Sitting on an office chair versus sitting on an exercise ball. Appl. Ergon.

Konig, A., Vitzthum, H.E., 2001. Functional MRI of the spine: different patterns of positions of the forward flexed lumbar spine in healthy subjects. Eur Spine J 10, 437-442.

Kraemer, J., Kolditz, D., Gowin, R., 1985. Water and electrolyte content of human intervertebral discs under variable load. Spine 10, 69-71.

Kramer, M., Dehner, C., Hartwig, E., Volker, H.U., Sterk, J., Elbel, M., Weikert, E., Gerngross, H., Kinzl, L., Willy, C., 2005. Intramuscular pressure, tissue oxygenation

and EMG fatigue measured during isometric fatigue-inducing contraction of the multifidus muscle. Eur. Spine J. 14, 578-585.

Kumazawa, T., Mizumura, K., 1977. Thin-fibre receptors responding to mechanical, chemical, and thermal stimulation in the skeletal muscle of the dog. J Physiol 273, 179-194.

Lee, K.E., Thinnes, J.H., Gokhin, D.S., Winkelstein, B.A., 2004. A novel rodent neck pain model of facet-mediated behavioral hypersensitivity: implications for persistent pain and whiplash injury. J. Neurosci. Methods 137, 151-159.

Lee, R.Y., Wong, T.K., 2002. Relationship between the movements of the lumbar spine and hip. Hum. Mov Sci. 21, 481-494.

Leivseth, G., Drerup, B., 1997. Spinal shrinkage during work in a sitting posture compared to work in a standing posture. Clin. Biomech. (Bristol. , Avon.) 12, 409-418.

Liao, M.H., Drury, C.G., 2000. Posture, discomfort and performance in a VDT task. Ergonomics 43, 345-359.

Lin, F., Parthasarathy, S., Taylor, S.J., Pucci, D., Hendrix, R.W., Makhsous, M., 2006. Effect of different sitting postures on lung capacity, expiratory flow, and lumbar lordosis. Arch Phys Med Rehabil 87, 504-509.

Lis, A.M., Black, K.M., Korn, H., Nordin, M., 2007. Association between sitting and occupational LBP. Eur. Spine J 16, 283-298.

Magnusson, M., Hult, E., Lindstrom, I., Pope, M.H., Hansson, T., 1990. Measurement of time-dependent height-loss during sitting. Clin Biomech 5, 137-142.

Magnusson, M.L., Pope, M.H., 1998. A review of the biomechanics and epidemiology of working postures (It isn't always vibration which is to blame!). Journal of Sound and Vibration 215, 965-976.

Magora, A., 1972. Investigations of the relation between low back pain and occupation. Industrial Medicine 41, 5-9.

Maigne, J.Y., Lapeyre, E., Morvan, G., Chatellier, G., 2003. Pain immediately upon sitting down and relieved by standing up is often associated with radiologic lumbar instability or marked anterior loss of disc space. Spine 28, 1327-1334.

Main, C.J., Watson, P.J., 1999. Psychological aspects of pain. Man. Ther. 4, 203-215.

Makhsous, M., Lin, F., Hendrix, R.W., Hepler, M., Zhang, L.Q., 2003. Sitting with adjustable ischial and back supports: biomechanical changes. Spine 28, 1113-1121.

Marras, W.S., Davis, K.G., 2001. A non-MVC EMG normalization technique for the trunk musculature: Part 1. Method development. J. Electromyogr. Kinesiol. 11, 1-9.

Marras, W.S., Granata, K.P., 1997. The development of an EMG-assisted model to assess spine loading during whole-body free-dynamic lifting. J Electromyogr. Kinesiol. 7, 259-268.

Marras, W.S., Lavender, S.A., Leurgans, S.E., Fathallah, F.A., Ferguson, S.A., Allread, W.G., Rajulu, S.L., 1995. Biomechanical risk factors for occupationally related low back disorders. Ergonomics 38, 377-410.

May, S., Donelson, R., 2008. Evidence-informed management of chronic low back pain with the McKenzie method. Spine J 8, 134-141.

McGill, S., Juker, D., Kropf, P., 1996a. Quantitative intramuscular myoelectric activity of quadratus lumborum during a wide variety of tasks. Clin. Biomech. (Bristol. , Avon.) 11, 170-172.

McGill, S.M., 2002. Low back disorders: Evidence-based prevention and rehabilitation. ed. Human Kinetics, Champaign, IL.

McGill, S.M., 2007. Low back disorders: Evidence-based prevention and rehabilitation. 2nd ed. ed. Human Kinetics, Champaign, IL.

McGill, S.M., Brown, S., 1992. Creep response of the lumbar spine to prolonged full flexion. Clin Biomech 7, 43-46.

McGill, S.M., Grenier, S., Kavcic, N., Cholewicki, J., 2003. Coordination of muscle activity to assure stability of the lumbar spine. J. Electromyogr. Kinesiol. 13, 353-359.

McGill, S.M., Hughson, R.L., Parks, K., 2000. Lumbar erector spinae oxygenation during prolonged contractions: implications for prolonged work. Ergonomics 43, 486-493.

McGill, S.M., Norman, R.W., 1986. Partitioning of the L4-L5 dynamic moment into disc, ligamentous, and muscular components during lifting. Spine 11, 666-678.

McGill, S.M., van Wijk, M.J., Axler, C.T., Gletsu, M., 1996b. Studies of spinal shrinkage to evaluate low-back loading in the workplace. Ergonomics 39, 92-102.

McGregor, A.H., Anderton, L., Gedroyc, W.M., Johnson, J., Hughes, S.P., 2001. Assessment of spinal kinematics using open interventional magnetic resonance imaging. Clin Orthop Relat Res 392, 341-348.

McLain, R.F., Yerby, S.A., Moseley, T.A., 2002. Comparative morphometry of L4 vertebrae: comparison of large animal models for the human lumbar spine. Spine 27, E200-E206.

Mclean, L., Tingley, M., Scott, R.N., Rickards, J., 2000. Myoelectric signal measurement during prolonged computer terminal work. J. Electromyogr. Kinesiol. 10, 33-45.

Mclean, L., Tingley, M., Scott, R.N., Rickards, J., 2001. Computer terminal work and the benefit of microbreaks. Appl. Ergon. 32, 225-237.

McMillan, D.W., Garbutt, G., Adams, M.A., 1996a. Effect of sustained loading on the water content of intervertebral discs: implications for disc metabolism. Ann. Rheum. Dis. 55, 880-887.

McMillan, D.W., McNally, D.S., Garbutt, G., Adams, M.A., 1996b. Stress distributions inside intervertebral discs: the validity of experimental "stress profilometry'. Proc. Inst. Mech. Eng [H.] 210, 81-87.

McNally, D.S., Adams, M.A., 1992. Internal intervertebral disc mechanics as revealed by stress profilometry. Spine 17, 66-73.

Mense, S., 1977. Nervous outflow from skeletal muscle following chemical noxious stimulation. J Physiol 267, 75-88.

Miller, R., Brown, W., 2004. Steps and sitting in a working population. Int. J. Behav. Med. 11, 219-224.

Morl, F., Blickhan, R., 2006. Three-dimensional relation of skin markers to lumbar vertebrae of healthy subjects in different postures measured by open MRI. Eur. Spine J. 15, 742-751.

Moseley, G.L., Hodges, P.W., Gandevia, S.C., 2002. Deep and superficial fibers of the lumbar multifidus muscle are differentially active during voluntary arm movements. Spine 27, E29-E36.

Nachemson, A., 1966. The load on lumbar disks in different positions of the body. Clin Orthop Relat Res 45, 107-122.

Nelson, J.M., Walmsley, R.P., Stevenson, J.M., 1995. Relative lumbar and pelvic motion during loaded spinal flexion/extension. Spine 20, 199-204.

Nelson-Wong, E., Gregory, D.E., Winter, D.A., Callaghan, J.P., 2008. Gluteus medius muscle activation patterns as a predictor of low back pain during standing. Clin. Biomech. (Bristol. , Avon.) 23, 545-553.

Ng, J.K., Richardson, C.A., Kippers, V., Parnianpour, M., 2002. Comparison of lumbar range of movement and lumbar lordosis in back pain patients and matched controls. Journal of Rehabilitation Medicine 34, 109-113.

Norman, R., Wells, R., Neumann, P., Frank, J., Shannon, H., Kerr, M., 1998. A comparison of peak vs cumulative physical work exposure risk factors for the reporting of low back pain in the automotive industry. Clin Biomech (Bristol. , Avon.) 13, 561-573.

Nuckley, D.J., Konodi, M.A., Raynak, G.C., Ching, R.P., Mirza, S.K., 2002. Neural space integrity of the lower cervical spine: effect of normal range of motion. Spine 27, 587-595.

O'Sullivan, P., Dankaerts, W., Burnett, A., Chen, D., Booth, R., Carlsen, C., Schultz, A., 2006a. Evaluation of the flexion relaxation phenomenon of the trunk muscles in sitting. Spine 31, 2009-2016.

O'Sullivan, P., Dankaerts, W., Burnett, A., Straker, L., Bargon, G., Moloney, N., Perry, M., Tsang, S., 2006b. Lumbopelvic kinematics and trunk muscle activity during sitting on stable and unstable surfaces. J Orthop Sports Phys. Ther. 36, 19-25.

O'Sullivan, P.B., Burnett, A., Floyd, A.N., Gadsdon, K., Logiudice, J., Miller, D., Quirke, H., 2003. Lumbar repositioning deficit in a specific low back pain population. Spine 28, 1074-1079.

O'Sullivan, P.B., Grahamslaw, K.M., Kendell, M., Lapenskie, S.C., Moller, N.E., Richards, K.V., 2002. The effect of different standing and sitting postures on trunk muscle activity in a pain-free population. Spine 27, 1238-1244.

O'Sullivan, P.B., Mitchell, T., Bulich, P., Waller, R., Holte, J., 2006c. The relationship beween posture and back muscle endurance in industrial workers with flexion-related low back pain. Man. Ther. 11, 264-271.

Okawa, A., Shinomiya, K., Komori, H., Muneta, T., Arai, Y., Nakai, O., 1998. Dynamic motion study of the whole lumbar spine by videofluoroscopy. Spine 23, 1743-1749.

Orchard, J., 2003. Simultaneous registration and activation detection for fMRI. Simon Fraser University , Ph.D. Dissertation.

Otun, E.O., Anderson, J.A., 1988. An inclinometric method for continuous measurement of sagittal movement of the lumbar spine. Ergonomics 31, 303-315.

Pal, P., Milosavljevic, S., Sole, G., Johnson, G., 2007. Hip and lumbar continuous motion characteristics during flexion and return in young healthy males. Eur. Spine J. 16, 741-747.

Panjabi, M., Chang, D., Dvorak, J., 1992. An analysis of errors in kinematic parameters associated with in vivo functional radiographs. Spine 17, 200-205.

Panjabi, M.M., 1992. The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis. J. Spinal Disord. 5, 390-396.

Panjabi, M.M., 2003. Clinical spinal instability and low back pain. J. Electromyogr. Kinesiol. 13, 371-379.

Parkinson, R.J., Beach, T.A., Callaghan, J.P., 2004. The time-varying response of the in vivo lumbar spine to dynamic repetitive flexion. Clin. Biomech. (Bristol. , Avon.) 19, 330-336.

Parkinson, R.J., Brown, S.H.M., Flynn, J., Noble, J., Powell, M.L., Callaghan, J.P., 2005a. Is unconstrained loading of spinal segments more physiologic? In: 24th Annual Meeting of the Canadian Biomaterials Society. Waterloo, Ontario: Canadian Biomaterials Society, May 26-28.

Parkinson, R.J., Callaghan, J.P., 2007. The role of load magnitude as a modifier of the cumulative load tolerance of porcine cervical spinal units: progress towards a force weighting approach. Theoretical Issues in Ergonomics Science 8, 171-184.

Parkinson, R.J., Durkin, J.L., Callaghan, J.P., 2005b. Estimating the compressive strength of the porcine cervical spine: an examination of the utility of DXA. Spine 30, E492-E498.

Pearcy, M., Portek, I., Shepherd, J., 1984. Three-dimensional x-ray analysis of normal movement in the lumbar spine. Spine 9, 294-297.

Pearcy, M.J., 1993. Twisting mobility of the human back in flexed postures. Spine 18, 114-119.

Pearcy, M.J., Whittle, M.W., 1982. Movements of the lumbar spine measured by three-dimensional X-ray analysis. J. Biomed. Eng 4, 107-112.

Pearsall, D.J., Reid, J.G., Livingston, L.A., 1996. Segmental inertial parameters of the human trunk as determined from computed tomography. Ann. Biomed. Eng. 24, 198-210.

Pope, M.H., Goh, K.L., Magnusson, M.L., 2002. Spine ergonomics. Annu. Rev. Biomed. Eng 4, 49-68.

Race, A., Broom, N.D., Robertson, P., 2000. Effect of loading rate and hydration on the mechanical properties of the disc. Spine 25, 662-669.

Radebold, A., Cholewicki, J., Polzhofer, G.K., Greene, H.S., 2001. Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain. Spine 26, 724-730.

Randolph, J.A., 2000. Carpal tunnel syndrome. Testing the sensitivity and validity of four "localized discomfort" instruments. AAOHN. J 48, 385-394.

Reilly, T., Tyrrell, A., Troup, J.D., 1984. Circadian variation in human stature. Chronobiol. Int. 1, 121-126.

Reinecke, S.M., Hazard, R.G., Coleman, K., 1994. Continuous passive motion in seating: a new strategy against low back pain. J Spinal Disord 7, 29-35.

Rhalmi, S., Yahia, L.H., Newman, N., Isler, M., 1993. Immunohistochemical study of nerves in lumbar spine ligaments. Spine 18, 264-267.

Robertson, D.G.E., Caldwell, G.E., Hamill, J., Kamen, G., Whittlesey, S.N., 2004. Research Methods in Biomechanics. 1st ed. Human Kinetics, Champaign, IL.

Rousseau, M.A., Bradford, D.S., Hadi, T.M., Pedersen, K.L., Lotz, J.C., 2006. The instant axis of rotation influences facet forces at L5/S1 during flexion/extension and lateral bending. Eur. Spine J. 15, 299-307.

Sato, K., Kikuchi, S., Yonezawa, T., 1999. In vivo intradiscal pressure measurement in healthy individuals and in patients with ongoing back problems. Spine 24, 2468-2474.

Shum, G.L., Crosbie, J., Lee, R.Y., 2005. Effect of low back pain on the kinematics and joint coordination of the lumbar spine and hip during sit-to-stand and stand-to-sit. Spine 30, 1998-2004.

Shum, G.L., Crosbie, J., Lee, R.Y., 2007. Three-dimensional kinetics of the lumbar spine and hips in low back pain patients during sit-to-stand and stand-to-sit. Spine 32, E211-E219.

Singer, J.C., Dunk, N.M., Callaghan, J.P., 2007. Evaluation of surface marker systems to determine pelvic and lumbar kinematics during sitting. Barrie, ON: 4th Annual Ontario Biomechanics Conference, March 9-11.

Sjogaard, G., 1990. Exercise-induced muscle fatigue: the significance of potassium. Acta Physiol Scand. Suppl 593, 1-63.

Sjogaard, G., Savard, G., Juel, C., 1988. Muscle blood flow during isometric activity and its relation to muscle fatigue. Eur. J Appl. Physiol Occup. Physiol 57, 327-335.

Snell, R.S., 1995. Clinical Anatomy for Medical Students. 5th ed. Little, Brown and Company (Inc.), Boston.

Snijders, C.J., Hermans, P.F., Niesing, R., Spoor, C.W., Stoeckart, R., 2004. The influence of slouching and lumbar support on iliolumbar ligaments, intervertebral discs and sacroiliac joints. Clin Biomech (Bristol. , Avon.) 19, 323-329.

Solomonow, M., 2004. Ligaments: a source of work-related musculoskeletal disorders. J. Electromyogr. Kinesiol. 14, 49-60.

Solomonow, M., Baratta, R.V., Banks, A., Freudenberger, C., Zhou, B.H., 2003a. Flexion-relaxation response to static lumbar flexion in males and females. Clin. Biomech. (Bristol. , Avon.) 18, 273-279.

Solomonow, M., Zhou, B.H., Baratta, R.V., Burger, E., 2003b. Biomechanics and electromyography of a cumulative lumbar disorder: response to static flexion. Clin. Biomech. (Bristol. , Avon.) 18, 890-898.

Swinkels, A., Dolan, P., 1998. Regional assessment of joint position sense in the spine. Spine (Phila Pa 1976.) 23, 590-597.

Swinkels, A., Dolan, P., 2000. Spinal position sense is independent of the magnitude of movement. Spine (Phila Pa 1976.) 25, 98-104.

Tague, R.G., 1992. Sexual dimorphism in the human bony pelvis, with a consideration of the Neandertal pelvis from Kebara Cave, Israel. Am J Phys Anthropol 88, 1-21.

Takayanagi, K., Takahashi, K., Yamagata, M., Moriya, H., Kitahara, H., Tamaki, T., 2001. Using cineradiography for continuous dynamic-motion analysis of the lumbar spine. Spine 26, 1858-1865.

Tampier, C., Drake, J.D., Callaghan, J.P., McGill, S.M., 2007. Progressive disc herniation: an investigation of the mechanism using radiologic, histochemical, and microscopic dissection techniques on a porcine model. Spine 32, 2869-2874.

Teyhen, D.S., Flynn, T.W., Childs, J.D., Kuklo, T.R., Rosner, M.K., Polly, D.W., Abraham, L.D., 2007. Fluoroscopic video to identify aberrant lumbar motion. Spine 32, E220-E229.

Thompson, R.E., Barker, T.M., Pearcy, M.J., 2003. Defining the Neutral Zone of sheep intervertebral joints during dynamic motions: an in vitro study. Clin. Biomech. (Bristol. , Avon.) 18, 89-98.

Tissot, F., Messing, K., Stock, S., 2005. Standing, sitting and associated working conditions in the Quebec population in 1998. Ergonomics 48, 249-269.

van der Veen, A.J., Mullender, M., Smit, T.H., Kingma, I., Van Dieen, J.H., 2005. Flow-related mechanics of the intervertebral disc: the validity of an in vitro model. Spine 30, E534-E539.

van der Veen, A.J., Van Dieen, J.H., Nadort, A., Stam, B., Smit, T.H., 2007. Intervertebral disc recovery after dynamic or static loading in vitro: is there a role for the endplate? J. Biomech. 40, 2230-2235.

van Deursen, L.L., Patijn, J., Durinck, J.R., Brouwer, R., Erven-Sommers, J.R., Vortman, B.J., 1999. Sitting and low back pain: the positive effect of rotary dynamic stimuli during prolonged sitting. Eur Spine J 8, 187-193.

van Deursen, L.L., van Deursen, D.L., Snijders, C.J., Wilke, H.J., 2005. Relationship between everyday activities and spinal shrinkage. Clin. Biomech. (Bristol. , Avon.) 20, 547-550.

Van Dieen, J.H., Cholewicki, J., Radebold, A., 2003a. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. Spine 28, 834-841.

Van Dieen, J.H., de Looze, M.P., Hermans, V., 2001a. Effects of dynamic office chairs on trunk kinematics, trunk extensor EMG and spinal shrinkage. Ergonomics 44, 739-750.

Van Dieen, J.H., Kingma, I., Meijer, R., Hansel, L., Huiskes, R., 2001b. Stress distribution changes in bovine vertebrae just below the endplate after sustained loading. Clin. Biomech. (Bristol. , Avon.) 16 Suppl 1, S135-S142.

Van Dieen, J.H., Selen, L.P., Cholewicki, J., 2003b. Trunk muscle activation in low-back pain patients, an analysis of the literature. J Electromyogr. Kinesiol. 13, 333-351.

Van Dieen, J.H., Westebring-van der Putten EP, Kingma, I., de Looze, M.P., 2008. Low-level activity of the trunk extensor muscles causes electromyographic manifestations of fatigue in absence of decreased oxygenation. J Electromyogr Kinesiol In Press.

Van Herp, G., Rowe, P., Salter, P., Paul, J.P., 2000. Three-dimensional lumbar spinal kinematics: a study of range of movement in 100 healthy subjects aged 20 to 60+ years. Rheumatology (Oxford) 39, 1337-1340.

Vergara, M., Page, A., 2000. System to measure the use of the backrest in sitting-posture office tasks. Appl. Ergon. 31, 247-254.

Vergara, M., Page, A., 2002. Relationship between comfort and back posture and mobility in sitting- posture. Appl. Ergon. 33, 1-8.

Vergara, M., Page, A., Sancho, J.L., 2006. Analysis of lumbar flexion in sitting posture: Location of lumbar vertebrae with relation to easily identifiable skin marks. Int J Ind Ergon 36, 937-942.

Videman, T., Nurminen, M., Troup, J.D.G., 1990. Lumbar spinal pathology in cadaveric material in relation to history of back pain, occupation, and physical loading. Spine 15, 728-740.

Wall, B.F., Hart, D., 1997. Revised radiation doses for typical X-ray examinations. Report on a recent review of doses to patients from medical X-ray examinations in the UK by NRPB. National Radiological Protection Board. Br J Radiol 70, 437-439.

Wang, D.L., Jiang, S.D., Dai, L.Y., 2007. Biologic response of the intervertebral disc to static and dynamic compression in vitro. Spine 32, 2521-2528.

Wearing, S.C., Smeathers, J.E., Yates, B., Sullivan, P.M., Urry, S.R., Dubois, P., 2005. Errors in measuring sagittal arch kinematics of the human foot with digital fluoroscopy. Gait Posture 21, 326-332.

Wilder, D.G., Pope, M.H., 1996. Epidemiological and aetiological aspects of low back pain in vibration environments - an update. Clin Biomech 11, 61-73.

Wilder, D.G., Pope, M.H., Frymoyer, J.W., 1988. The biomechanics of lumbar disc herniation and the effect of overload and instability. J Spinal Disord 1, 16-32.

Wilke, H., Neef, P., Hinz, B., Seidel, H., Claes, L., 2001. Intradiscal pressure together with anthropometric data--a data set for the validation of models. Clin. Biomech. (Bristol. , Avon.) 16 Suppl 1, S111-S126.

Wilke, H.J., Kettler, A., Claes, L.E., 1997. Are sheep spines a valid biomechanical model for human spines? Spine 22, 2365-2374.

Wilke, H.J., Neef, P., Caimi, M., Hoogland, T., Claes, L.E., 1999. New in vivo measurements of pressures in the intervertebral disc in daily life. Spine 24, 755-762.

Willemsen, A.T., van Alste, J.A., Boom, H.B., 1990. Real-time gait assessment utilizing a new way of accelerometry. J Biomech. 23, 859-863.

Winkel, J., Jorgensen, K., 1986. Evaluation of foot swelling and lower-limb temperatures in relation to leg activity during long-term seated office work. Ergonomics 29, 313-328.

Winkelstein, B.A., DeLeo, J.A., 2004. Mechanical thresholds for initiation and persistence of pain following nerve root injury: mechanical and chemical contributions at injury. J. Biomech. Eng 126, 258-263.

Winter, D.A., 1990. Biomechanics and motor control of human movement. 2nd ed. Wiley and Sons, Inc., Toronto.

Winter, D.A., Quanbury, A.O., Hobson, D.A., Sidwall, H.G., Reimer, G., Trenholm, B.G., Steinke, T., Shlosser, H., 1974. Kinematics of normal locomotion--a statistical study based on T.V. data. J. Biomech. 7, 479-486.

Womersley, L., May, S., 2006. Sitting posture of subjects with postural backache. J Manipulative Physiol Ther. 29, 213-218.

Wong, A., Dunk, N.M., Callaghan, J.P., 2009. A systematic approach to feature tracking of lumbar spine vertebrae from fluoroscopic images using complex-valued wavelets. Computer Methods in Biomechanics and Biomedical Engineering In Press.

Wong, K.W., Leong, J.C., Chan, M.K., Luk, K.D., Lu, W.W., 2004. The flexion-extension profile of lumbar spine in 100 healthy volunteers. Spine 29, 1636-1641.

Wong, W.Y., Wong, M.S., 2008. Detecting spinal posture change in sitting positions with tri-axial accelerometers. Gait Posture 27, 168-171.

Yahia, L., Rhalmi, S., Newman, N., Isler, M., 1992. Sensory innervation of human thoracolumbar fascia. An immunohistochemical study. Acta Orthop. Scand. 63, 195-197.

Yang, K.H., King, A.I., 1984. Mechanism of facet load transmission as a hypothesis for low-back pain. Spine 9, 557-565.

Yingling, V.R., Callaghan, J.P., McGill, S.M., 1997. Dynamic loading affects the mechanical properties and failure site of porcine spines. Clin Biomech 15, 301-305.

Yingling, V.R., Callaghan, J.P., McGill, S.M., 1999. The porcine cervical spine as a model of the human lumbar spine: an anatomical, geometric, and functional comparison. J Spinal Disord 12, 415-423.

Yoganandan, N., Cusick, J.F., Pintar, F.A., Droese, K., Reinartz, J., 1994. Cyclic compression-flexion loading of the human lumbar spine. Spine 19, 784-790.

Yoshitake, Y., Ue, H., Miyazaki, M., Moritani, T., 2001. Assessment of lower-back muscle fatigue using electromyography, mechanomyography, and near-infrared spectroscopy. Eur. J. Appl. Physiol 84, 174-179.