AXIAL TWIST LOADING OF THE SPINE: MODULATORS OF INJURY MECHANISMS AND THE POTENTIAL FOR PAIN GENERATION

by

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Author’s Declaration Page

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.

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ABSTRACT

There are several reasons to research the effects of axial twist exposures and the resulting loading on the spine. The lack of consensus from the limited work that has previously examined the role of axial twist moments and motions in the development of spine injuries or generation of low back pain is the primary reason. From recently published works, axial twist moments appear to represent an increased risk for injury development when it acts in concert with loading about other physiological axes (i.e. flexion, extension, and compression). However, there is a large body of epidemiologic data identifying axial twist moments and/or motion as risk factors for low back disorders and pain, demonstrating the need for this series of investigations. It is likely that these combined exposures increase risk through altering the spine's load distribution (passive resistance) by modifying the mechanics, but this deduction and related causal mechanism need to be researched.

The global objective of this research was focused on determining whether there is evidence to support altered load distribution in the spine, specifically between the intervertebral disc and facets, in response to applied axial twist moments (when added in combination with one and two axes of additional loading). Also included was whether these modes of loading can modify spine mechanics and contribute and/or alter the development of damage and pain. This objective was addressed through one in-vivo (Drake and Callaghan, 2008a– Chapter #2) and three in-vitro (Drake et al., 2008– Chapter #4; Drake and Callaghan, 2008b– Chapter #5; Drake and Callaghan, 2008c– Chapter #6) studies that: (1) Quantified the amount of passive twist motion in the lumbar spine when coupled with various flexion-extension postures; (2) Documented the effects of flexion-extension postures and loading

history on the distance between the facet articular surfaces; (3) Evaluated the result of axial twist rotation rates on acute failure of the spine in a neutral flexion posture; and (4) Explored whether repetitive combined loading has the ability to cause enough deformation to the spine to generate pain.

Through the combination of findings previously reported in the literature and the outcomes of Drake and Callaghan (2008a– Chapter #2) and Drake et al. (2008– Chapter #4), a postural mediated mechanism was hypothesized to be responsible for governing the load distribution between the facet joints and other structures of the spine (i.e. disc, ligaments). Increased flexed postures were found to decrease the rotational stiffness by resulting in larger twist angles for the same applied twist moment in-vivo relative to a neutral flexion posture (Drake and Callaghan, 2008a– Chapter #2). This suggested there might be an increased load on the disc due to a change in facet coupling in these combined postures. Similarly, increased angles were observed in flexed and twisted postures for in-vitro specimens relative to a neutral flexion posture. These observed differences were found to correspond with altered facet joint mechanics. Specifically that flexed twisted postures increased the inter-facet spacing relative to the initial state of facet articulation (Drake et al., 2008– Chapter #4). These finding supported the postulated postural mechanism. Therefore, in a neutral posture the facet joints likely resisted the majority of any applied twist moment based on the limited range of motion and higher axial rotational stiffness responses observed. It was suspected that the changes in mechanics would likely cause a change in the load distribution however the magnitude of change in load distribution remains to be quantified.

Further support for this postulated postural mechanism comes from the mode of failure for specimens that were exposed to 10,000 cycles of 5° axial twist rotation while in a

static flexed posture (Drake and Callaghan, 2008c– Chapter #6), and neutrally flexed specimens exposed to 1.5° of rotation for 10,000 cycles reported in the literature. Without flexion, the failure patterns were reported to occur in the endplates, facets, laminae and capsular ligaments, but not the disc. However, with flexion the repetitive axial twist rotational displacements caused damage primarily to the disc. If the load distribution was unchanged, the higher axial rotation angle should have caused the specimen to fail in less cycles of loading, and the failure pattern should not have changed. Modulators of this hypothesized mechanism include the velocity of the applied twist moment and the effects these have on the failure parameters and injury outcomes. The three physiologic loading rates investigated in this work were not shown to affect the ultimate axial twist rotational failure angle or moment in a neutral flexion/extension posture, but were shown to modify flexion-extension stiffness (Drake and Callaghan, 2008b– Chapter #5). All of the flexion-extension stiffness values post failure, from a one-time axial twist exposure, was less than those from a repetitive combined loading exposure that has been established to damage the intervertebral disc but not the facets. Therefore, it is likely that the facet joint provides the primary resistance to acute axial twist moments when the spine is in a neutral flexion posture, but there appears to be a redistribution of the applied load from the facets to the disc in repetitive exposures.

The aforementioned studies determined there are changes in load distribution and load response caused by altered mechanics resulting from twist loading, but whether the exposures could possibly produce pain needed to be addressed. Previous research has determined that the disc has relatively low innervation in comparison to the richly innervated facet capsule and vertebra, with only the outer regions being innervated. Likewise, it is

assumed that pain could be directly generated as the nucleus pulposus disrupted the innervated outer annular fibres in the process of herniation. Also, direct compression of the spinal cord or nerve roots has been shown to occur from the extruded nucleus and result in the generation of pain responses. Additionally, the nucleus pulposus has been shown to be a noxious stimulus that damages the function and structure of nerves on contact. The other source of nerve root compression commonly recognized is a decrease in intervertebral foramina space, which was previously believed to only be caused through losses in disc height. However, decreased intervertebral foramina space due to repetitive motions appears to be a viable pain generating pathway that may not directly correspond to simply a loss of specimen or disc height (Drake and Callaghan, 2008c– Chapter #6). This is new evidence for combined loading to generate pain through spinal deformation. The objective of many traditional treatments for nerve root compression focus on restoring lost disc height to remove the nerve root compression. Unfortunately, nerve root compression caused by repetitive loading may not be alleviated through this approach.

This collection of studies was focused on determining whether altered load distribution in the spine, specifically between the intervertebral disc and facets, in response to applied axial twist loading (when added in combination with one and two axes of additional loading) was occurring, and examining how these modes of loading can contribute and/or alter the development of injury and pain. Therefore, findings generated from this thesis may have important implications for clinicians, researchers, and ergonomists.

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Authorship Contribution Acknowledgements

I hereby declare that my contribution to each journal publication that was produced from my thesis (currently in press, accepted, and submitted) was from my own work, and for each study that I was: responsible for the conception and/or design, preparation, collection and analysis of the data, and the writing and editing of the manuscripts.

I hereby declare that the contribution of author Dr. Jack P. Callaghan on each manuscript was to the conception and/or design, editing of the manuscripts, assisting with instrumentation, and securing external funding for the research.

I hereby declare that the contribution of author Dr. Howard Dobson in Chapter #4 on the manuscript titled “The influence of posture and loading on inter-facet spacing: An investigation using MRI on porcine spinal units”, was to the concept and/or design of the study, and editing of the manuscript.

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CHAPTER 1

INTRODUCTION

1.0 Overview

There are several reasons to research the effects of axial twist loading on the spine. There is a lack of consensus from the limited work that has been completed on how twist moments and twist motions affect the mechanical behaviour and injury mechanisms of the spine. There is a large body of epidemiologic data identifying axial twist motion and/or moment as risk factors for low back disorders. The injury statistics and gaps in the literature demonstrate the need to investigate the role axial twist loading plays in the development of spinal injuries and the generation of low back pain. From recently published works, twist moments appear to have an increased risk for injury development when acting in concert with loading on other physiological axes (i.e. flexion, extension, and compression).

It is important to note that the literature is neither clear nor consistent on the definition for the terms torsion and twisting. The cause of the misuse of these terms is likely the close coupling of axial twist motion and twist moment. Further, in the in-vivo spine, there is a very large change in twist moment for a very small change in twist motion, and so torsion is often used to refer to both the twist motion and its causal moment (Adams et al., 2002). In this document the terms torsion and twisting were not used. The motion will be referred to as twist motion, rotation, rotational displacement, or twist angle, and the moment will be referred to as twist moment, rotational moment, twist load (loading), and rotational load (loading).

1.1 Why examine axial twist motion/moment loading?

Epidemiological Evidence

Twist motions and moments are experienced daily by the lumbar spine while performing domestic, athletic, and industrial tasks. From industrial evidence, axial rotational (Kelsey et al., 1984; Marras et al., 1993; Punnet et al., 1991) and cumulative compressive loading (Jager et al., 2000; Kumar, 1990; Norman et al., 1998) have been identified in epidemiologic studies as significant risk factors for developing low back injuries and/or pain. To date, cumulative exposure to rotational loading has not been researched. Regarding the cause of lost work time, Frymoyer and Mooney (1986) stated that low back pain is second only to the common cold. Every year in the United States alone, an estimated 11-13 million people experience a low back pain episode (Marras, 2000). Many of these episodes are precipitated by occupational factors, and the financial cost to industry from low back compensation claims, estimated to be as high as $100 billions of dollars (US) annually (Marras, 2000; Webster and Snook, 1994; Spengler et al., 1986), is staggering. The consideration that activities involving twist motion have been reported to be connected with at least 18% of all claim compensation costs (Snook, 1978), presents convincing evidence that the role twist moments in the etiology of spine injury and pain needs to be investigated.

Pain Generating Potential

Damage to both the facet joints (McCall et al., 1979; Schwarzer et al., 1994; Schwarzer et al., 1995b) and intervertebral disc (Ito et al., 1998; Boos et al., 2000) from twist moment exposures have the potential to cause low back pain. Further, the prevalence of painful facets was found to be 40% in an elderly population (Schwarzer, et al., 1995b), with prevalence reported to be as high as 15% in a working population (Schwarzer, et al., 1994).

Also, pain attributed to disruption of the internal structure of the disc was cited at 40% in a low back pain population (Schwarzer, et al., 1995a). Additionally, twist moments have been implicated in causing disc degeneration (Krismer et al., 1996). However, the onset of intervertebral (IVD) injury or degeneration may not always coincide with the onset of pain (Boden et al., 1990; Jensen et al., 1994), nor is there always diagnostic evidence to correlate detectable damage with painful facet joints (Schwarzer et al., 1995c). However, in a study of people without low back pain using magnetic resonance imaging (MRI), Jensen et al. (1994) found that disc protrusion or bulging is not always correlated with clinical symptoms of pain. This suggests that either herniation or other IVD damage may not be the primary pathology affecting the spine to cause low back pain. Alternatively, people could be unwittingly accelerating the accumulation of irreversible damage to their spines before there is pain or diagnostic certainty of injury. Currently, the normal function of the intervertebral joint (IVJ), the intervertebral disc between two adjacent vertebrae, cannot be restored through surgical interventions or replacement techniques (Eijkelkamp et al., 2003). Since mechanical overloading of tissue is suspected to be the primary cause of low back injury (Pope and Novotny, 1993), the role of axial rotational moments in IVJ injury mechanisms need to be more fully explored. Applications of the known mechanisms can then be used to aide in the determination of tolerance limits and establishment of exposure guidelines to this type of loading are obviously warranted. A detailed description of specific pain producing pathways is provided in Chapter #3.

Controversy Regarding Mechanism Of Failure

Controversy over the role of twist moments and the resulting twist motion in IVD failure mechanics exists. Farfan (1969) and Farfan et al. (1970) found that twist moments can damage the IVD without causing any bony damage. Krismer et al. (1996) reported that intact annulus fibres resist axial rotation more than facet joints, and so twist moments are likely important in the development of disc degeneration. However, loading with similar combined axial rotational moments and compressive exposures has been reported to damage the facet joints but did not compromise the structural integrity of the IVD (Adams and Hutton, 1981; Pearcy and Hindle, 1991). The opinion of these researchers was that intact facets protect the IVD from twist moments, so facet failure must precede axial twist induced disc herniation. There are several limitations of these studies including the use of primarily static loading, fixed postures, and minimal cycles of repetition.

1.2 What type of loading should be assessed?

Posture and Repetition Considerations

In compressive testing, neutral postures rarely result in the herniation of the IVD, but instead cause endplate fractures, despite the application of up to 10,000 repeated loading cycles (Brinckmann et al., 1988; Lui et al., 1983). It has been established that non-neutral postures combined with repetitive loading are required to cause herniation (Adams and Hutton, 1983b, 1985; Callaghan and McGill, 2001; Drake et al., 2004; Gordon et al., 1991). Several researchers have investigated a range of maximum repeated cycles (from 1000 to > 1 million) of various two axes combined loading, on specimens in a variety of fixed postures (Adams and Hutton, 1983b; Brown, 1957; Goel et al., 1988; Gordon, 1991; Hansson et al.,

1987; Hardy et al., 1958). Despite speculation that three axes of combined loading could increase the vulnerability of the IVD to injury (Ahmed et al., 1990; Pearcy and Hindle, 1991), and that such complex modes of loading more closely mimics loading in-vivo (Pearcy and Tibrewal, 1984), three axes of combined loading, repetitive or acute, has rarely been investigated. Further, in the few studies that have been conducted, only one axis (usually compression) is dynamic while loads in the others are static. Although static loading occurs in-vivo, such as prolonged sitting, standing, kneeling, and forward bending (Frymoyer and Mooney, 1986), repetition is a key ingredient in generating in-vitro herniation of the IVD as previously described.

A repetitive loading experiment was conducted by Gordon et al. (1991) who herniated all 14 functional spinal units (FSU) with the application of two static positions (7° flexion and >3° axial twist) combined with repetitive compression (1334N), to an average of 40,000 loading cycles. Haberl et al. (2004) loaded specimens three times with combinations of 200N of compression, up to 6Nm of flexion or extension, and 12.5 Nm of static twist moment, but only reported the resulting kinematics. Callaghan and McGill (2001) have shown that IVD herniation is linked to highly repetitive dynamic flexion/extension motions with applied joint compression. Also, Drake et al. (2005) demonstrated that added static axial twist moment to the combined loading scheme of flexion/extension motion under joint compression increased the vulnerability of the FSU by changing the failure mechanism. These findings suggest twist loading coupled with combined loading reduces the exposure time required to cause IVD injury. Recently, an investigation using finite element analysis on the annular strains and nuclear pressure predicted that the risk of disc damage increased during load combinations of axial rotation and with flexion (Schmidt et al., 2007). Further,

Fazey et al. (2006) tracked the in-vivo deformation of the nucleus pulposus in response to flexion-extension postures alone and to flexion-extension coupled with axial rotation using magnetic resonance imaging and suggested different asymmetrical loading on the intervertebral disc results from altered deformations due to the addition of rotation. These results suggest a postural mechanism may be responsible for modulating how facet joints articulate thereby affecting the moment resisting capability of the facets and shifting the load distribution between the facets and the disc. So, although the injurious nature of axial twist loading has been illustrated, the mechanism of how chronic exposure to twist moments and motions contributes to intervertebral joint failure mechanics remains poorly understood.

Active Versus Passive Twist Moment Generation

In-vivo

In-vivo active rotational stiffness and maximal twist moment have been measured during voluntary axial rotation in both upright standing (Marras et al., 1993; McGill and Hoodless, 1990; Peach et al., 1998) and sitting (Kumar et al., 1996; Toren, 2001). The effect of the rotation velocity on the stiffness and moment were also examined (Kumar et al., 1996; Marras et al., 1993; McGill and Hoodless, 1990; Peach et al., 1998). From in-vitro research and the anatomy of the spine, it can be expected that in upright standing the facet joints would severely limit the degree of trunk rotation as well as carry a substantial portion of the rotational moment. The range of twist motion permitted has been shown to be a function of the gap distance between the articulating surfaces of the facets (Haberl et al., 2004). Once the trunk is flexed the facet joints may disengage thereby increasing the gap distance between articular surfaces and reduce their load carrying capability. The annulus would then be required to resist increased axial rotational twist load in these situations. There are little or no

data available on the mechanical role of the facet joints under combined twist and flexion/extension moments (Panjabi et al., 1989b). The behaviour of passive axial rotational stiffness in-vivo has been investigated in only upright standing (McGill et al., 1994) and seated (Bodén and Öberg, 1998) postures, so the passive rotational stiffness response of the spine to changes in the velocity of the twist motion and non-neutral flexed postures is unknown.

In-vitro

Gordon et al. (1991) showed no correlation between damage and biomechanical parameters (stiffness or hysteresis), and no differences prior to or following injury in an in-vitro model for a flexed, compressed, and rotated posture. However, increased FSU stiffness has been noted for increased IVD damage (Thompson et al., 2000), increased exposure to compression combined with flexion/extension motions (Callaghan and McGill, 2001; Yoganandan et al. 1994), and further increases when a twist moment was added to the combined exposures (Drake et al., 2005). The effects of dynamic rotational loading on the biomechanical responses of FSUs have not been assessed.

1.3 Thesis Objectives and Composition

Postulated Mechanism Of Injury

The application of static axial twist moment did not alter the number of specimens that ultimately herniated in a study conducted by Drake et al. (2005), so these researchers concluded that the twist moment was not the primary cause of herniation at the levels of loading tested in the study. The IVD is strongest at bearing compressive loads, but combined flexion and twist motions generate combinations of compressive, tensile and shear stresses

which the IVD may not be well suited to resist (Adams and Dolan, 1995; Green et al., 1993; Panjabi et al., 1984; Pearcy, 1993). With the use of MRI, Fazey et al. (2006) found that the addition of axial rotation to flexion/extension postures in-vivo caused asymmetrical movement of the nucleus pulposus, which led to their conclusion that the loading of the IVD must also be asymmetrical. It has been hypothesized that more water is lost during twist motions versus other types of movements (Au et al., 2001), possibly due to the potential opening of alternate layers of the annulus. Both herniation and water loss cause a decrease in disc height (Brown et al., 2002) and bulging of the posterior annulus (Adams and Dolan, 1995; Wenger and Schlegel, 1997). However, in-vivo and in-vitro approaches are neither able to examine the contributors to disc height loss independently (i.e. loading, water loss, and injury) in isolation, nor in controlled degrees of interaction. Recently, Schmidt et al. (2007) predicted that the risk of disc damage increased during combinations of axial rotation coupled with flexion using a finite element model to investigate annular strains and nuclear pressure. Using a photogrammetric method, Wenger and Schlegel (1997) measured an increased disc bulge in the posterolateral region with herniation. Adams and Dolan (1995) hypothesized that the strain on the posterior annulus increases as the annulus bulges, causing further bulging, annular delamination, and disc narrowing. As a result of this destructive cycle, the nucleus pulposus can begin to track through the weakened posterior annulus. In addition, the loss of disc height and the depressurization of the nucleus pulposus can increase the compressive load on the facets from 15% (Eijkelkamp et al., 2001) to 20% (Adams and Dolan, 1995) to upwards of 70% (Adams and Hutton, 1983a). Haberl et al. (2004) hypothesized that once the facets are in contact, the twist moment is translated to a coupled moment of flexion/extension and lateral bend. It is suspected that the IVD is required to

resist more tension when exposed to twist moments in midranges of flexion due to the increased initial spacing between the facet joints (Haberl et al., 2004; Pearcy, 1993; Pearcy and Hindle, 1991). As the facets begin to fail, the FSU is able to rotate further, which again increases the loads on the annulus, therefore contributing to IVD damage following the initiation of damage to the facets. Drake et al. (2005) observed approximately three degrees of creep in the specimens loaded with a static twist moment at the end of the testing schedule, which coincides with the reported amount of rotation where facets fail (Adams and Hutton, 1981). From these investigations it appears that the failure of the facet and IVD are not necessarily independent, but may suffer an interwoven path of damage that ultimately leads to the accelerated destruction of both structures. Without time varying data monitoring the load distribution between the IVD and facet joints, delineating this complex interaction can not be completed, nor can the assigned potential for pain generation and/or injury risk be identified.

This proposed failure mechanism for combined loading has been supported by the findings of several authors (Drake et al., 2005, Fazey et al., 2006; Schmidt et al., 2008). Specifically, the addition of 5Nm of static twist moment resulted in more severe facet damage, higher energy dissipation, higher specimen flexion-extension stiffness up to approximately 3000 cycles of loading (Drake et al., 2005). The low level of static twist moment (5Nm) applied, also accelerated IVD and facet damage when combined with compression and flexion/extension motions. The applied twist moment was lower than the physiologically in-vivo maximum predicted by Adams et al. (2002) to be 6-12 Nm for the human lumbar spine. Research generated in all of the studies of this thesis also support this proposed mechanism (Drake and Callaghan, 2008a– Chapter #2; Drake et al., 2008– Chapter

#4; Drake and Callaghan, 2008b– Chapter #5, 2008c– Chapter #6;). However, without established repetitive tolerance limits for FSUs, and the unknown effects of compression, and disc height, it remains difficult to evaluate the validity of this postural mechanism.

Global Thesis Questions

1. Does the passive contribution of the facet joints change to resisting twist moments over a large range of flexion postures? How do coupled postures affect facet joint mechanics?
2. Does the velocity of axial twist rotation affect injury outcomes, and twist angle or moment at failure?
3. Is it possible for combined loading to have the ability to cause enough FSU deformation to generate pain?

Thesis Composition and Specific Study Questions

This chapter provides a review of the pertinent literature to establish the context for the completed research. The in-vivo investigation, is presented in Drake and Callaghan (2008a– Chapter #2). Specific review of literature for this study has been included before the introduction of the expanded journal submission, followed by the study specific limitations and contributions. A literature review common for the in-vitro research, is presented in Chapter #3. Drake et al. (2008– Chapter #4; Drake and Callaghan (2008b– Chapter #5; 2008c– Chapter #6) describe the three in-vitro investigations. The outline of each in-vitro study chapter is: an expanded form of the journal submission, followed by study specific limitations and contributions. The limitations that were common to all of the studies are presented in Chapter #7, along with a discussion of the impact of the thesis research, the thesis conclusion, and possible future questions are provided. Completing Chapter #1 is the

presentation of focused study questions. All references used in the individual chapters have been included in a general reference list at the end of the document.

This thesis is comprised of one in-vivo and three in-vitro studies that provided the following important information and addressed the following study specific questions.

Study #1 (in-vivo): This study provided the link from the in-vitro studies to living participants. The passive range of motion was measured, the axial rotational stiffness was calculated, and coupling patterns of vertebral motion were assessed. (Drake and Callaghan, 2008a– Chapter #2)

1. What was the passive stiffness response of the lumbar spine during twist motions?
2. Did increased flexion cause a detectable change in the measured twist range of motion?

Study #2 (in-vitro): This study determined if the postural mechanism observed in Study #1 was due to a change in the initial distance separating the facets prior to the applied twist motion. Similar to Study #1, this work provided a link from the in-vitro studies to living participants. The effect of two types of loading history on facet mechanics was also examined in this study. The inter-facet spacing was quantified with the use of MRI technology, and the outcomes between the loading exposures were compared. (Drake et al, 2008– Chapter #4)

1. Did increased flexion cause uncoupling of the facet joints (increasing facet gap distance)?
2. What was the effect of loading history on the range of motion and inter-facet spacing?

Study #3 (in-vitro): The acute axial rotational failure limits that established a context for the behaviour of the FSU were provided by this study. The effects of three axial twist rotation rates on the failure measures were examined. The acute axial rotational tolerances were used to determine the values used in repetitive failure testing for Study #4. (Drake and Callaghan, 2008b– Chapter #5)

1. What was the effect of axial twist rotational load rate on the ultimate twist moment and angle in acute loading?
2. What was the effect of damage created by the different twist load rates investigated?

Study #4 (in-vitro): This study quantified the intervertebral foramina (IVF) size changes, in response to two combined loading scenarios. The progression of the changes over time was compared to the information regarding IVJ structural failure and the potential for pain generation. (Drake and Callaghan, 2008c– Chapter #6)

1. Did compressed repetitive flexion-extension loading cause larger occlusions of the IVF than static flexed repetitive axial twist loading?

2. Was there a difference in the mode of failure in the specimens from the different loading conditions?
3. Did changes in disc height accompany increased IVF occlusion and increased IVD damage (herniation)?

CHAPTER 2

STUDY #1: DO FLEXION/EXTENSION POSTURES AFFECT THE IN-VIVO PASSIVE LUMBAR SPINE RESPONSE TO APPLIED AXIAL TWIST MOMENTS?

Journal Article: In Press, Clinical Biomechanics.

2.1 Introduction

The injury potential and mechanical effects of combining axial rotation with non-neutral flexion/extension postures in-vivo remains poorly understood. In an epidemiologic investigation, twist rotation of the trunk was involved in over 60% of all back injuries documented (Manning et al., 1984). Marras et al. (1993) analysed over 400 industrial jobs in 48 different industries and determined that trunk axial twist velocity was the second highest, of five plausible biomechanical factors that had a significant odds ratio of 10.7, for identifying high risk group membership for low back disorders. Similarly, from complex motions measured in industry, Fathallah et al. (1998) found the highest risk of injury corresponded to the jobs involving end ranges of motion and large complex trunk motion velocities. Kelsey et al. (1984) reported that lifting 11.3kg or holding 4.5kg, more than five times per day when associated with trunk axial rotation (half of the time or more) tripled the risk for a prolapsed intervertebral disc (IVD), relative to handling the same loads less than five times per day with the body twisted less than one quarter of the time. However, the mechanism causing the increased risk of joint injury due to trunk rotation is unknown.

The application of a twist moment has been identified in in-vitro research as a modulator of injury, acting to accelerate annular injury formation under repetitive combined loading (Drake et al., 2005), and to reduce compressive failure tolerance in one-time loading

(Aultman et al., 2004). From the anatomy of the lumbar spine it would be expected that in a neutral flexion–extension posture the facet joints would limit the degree of twist angle as well as carry a substantial portion of the twist moment. This is supported by work that has shown that the range of twist motion permitted is a function of the gap distance between the articulating surfaces of the facets (Haberl et al., 2004; Shirazi-Adl, 1994).

Drake et al. (2005) postulated that the increased injury risk in coupled motions is likely due to the annulus being required to resist an increased portion of the axial twist moment generated, enabled by the increased separation within the facet joint from the flexion movements. Pearcy and Hindle (1991) concluded from their in-vitro work that although a twist moment in a neutral posture may not be sufficient to damage the IVD, they found an increased ability of the specimen to rotate when a combination of flexion and axial twist moments were applied, which may increase the risk of injury to the annulus. An investigation on the annular shear and fibre strains and nuclear pressure predicted that the risk of damage increased during load combinations of axial rotation coupled with flexion (Schmidt et al., 2007). Fazey et al. (2006) tracked the in-vivo deformation of the nucleus pulposus in response to flexion–extension postures alone and to flexion–extension coupled with axial rotation using magnetic resonance imaging (MRI), and suggested different asymmetrical loading on the IVD results from altered deformations due to the addition of rotation. These results suggest a postural mechanism may be responsible for modulating how the facet joints engage/disengage, subsequently decrease/increase the inter-facet spacing, thereby improving/reducing the moment resisting capability of the facets and shifting the load distribution to/from the facets from/to the IVD. In contrast Gunzburg et al. (1991) reported that axial rotation was found to be less when combined with flexion in their in-vitro and in-

vivo studies. The investigation of non-neutral postures in the sagittal plane combined with passive rotation in-vivo would provide a link to the in-vitro literature, and may reveal underlying mechanisms to injury and low back pain.

Several researchers have investigated the patterns of voluntary coupled motion (Hindle and Pearcy, 1989; Hindle et al., 1989; Pearcy, 1993; Pearcy and Tibrewal, 1984; Stokes et al., 1981; Russell et al., 1993). Generally, these authors suggest there is a possible increased risk to the posterior annulus during coupled postures. However, the passive response of lumbar spine tissues to applied axial twist moments has been primarily investigated in neutral postures: supine (Haughton et al., 2002; Ochia et al., 2006), upright standing (McGill et al., 1994) and sitting (Bodén and Öberg, 1998). Clinically, axial rotational instability has been implicated as a potential cause of low back pain (Ochia et al., 2006). Likewise, neutral axial rotation has been quantified with magnetic resonance imaging (Haughton et al., 2002) and computed tomography (Ochia et al., 2006). Since abnormal axial rotational motion is used to indicate instability, the objective of these studies was to investigate whether the non-invasive modalities could be used to detect and quantify the segmental twist motion (Haughton et al., 2002; Ochia et al., 2006). The outcomes demonstrated that both modalities can be used to quantify axial twist. Haughton et al. (2002) also reported larger rotations were associated with painful or abnormal disc appearance. Therefore, passive axial rotation in neutral postures has potential diagnostic value for detecting spinal instability and to determine possible treatment strategies (Haughton et al., 2002). Unfortunately, it is difficult to contextualize the outcomes of these studies, even when considering only healthy participants, without knowing the percent of total axial rotation motion applied, or without normal range of motion data for comparison with the pain

populations. Also, the flexion–extension posture of the participants does not to appear to have been controlled, documented, or monitored. This lack of normalization and/ or control could be factors confounding the data from other magnetic resonance and computed tomography low back pain diagnostic experiments, where differences between groups, if existing, could be concealed by subtle changes in posture yielding inconclusive study outcomes. The response to passive axial twist moments needs to be quantified on normal, healthy participants if abnormal passive coupled postures are to be used clinically as an indicator of spinal instability.

2.2 Literature Review

Active Rotation

Stiffness and Velocity

The effects of posture on the activation patterns of trunk electromyography (EMG) and twist moment and stiffness have been investigated for standing based postures (Marras et al., 1998; McGill et al., 1991b; McGill and Hoodless, 1990) and seated postures (Kumar, 1997; Torén, 2001). Parkinson et al. (2004) measured the flexion stiffness response of the lumbar spine to repetitive flexion motions in eight participants, and found a biphasic stiffness response. The trend in flexion-extension stiffness decreased over the first 30 minutes of the study, but following continued loading, returned to almost initial testing levels (Parkinson et al., 2004). Unrestrained in-vivo trunk axial rotation in seated postures at a self-selected velocity was studied by Kumar et al. (1996), who found higher axial twist moments could be generated isometrically as compared to isokinetically. McGill and Hoodless (1990) measured and modelled both maximal axial twist moments for upright standing, and ±30° of

pre-rotation, and dynamic axial twist moment generation at 30°/s and 60°/s. On 12 male participants (aged 21-31 years), Marras et al. (1998) observed that the muscle activation to generate the twist moment was a function of the posture and velocity of the motion. The muscle activation required to generate the twist moment caused increased co-contraction only in non-neutral or postures other than upright standing. Peach et al. (1998) documented the three dimensional range for kinematics of flexion/extension, lateral bending, and axial rotation movements, along with EMG of rectus abdominus, internal and external oblique, upper and lower erector spinae, latissimus dorsi, and multifidus muscles, in 17 and 7 university aged men and women respectively. The highest peak velocity for the performance of these movements was 11.0°/s, with the mean velocity 3.9°/s ±1.6 (Peach et al., 1998).

Range of Motion

There is a large range in the reported values of the observed motion obtained during active axial twist motion of the lumbar spine. Torén (2001) looked at the range of motion in a seated posture and documented approximately 7°-11° of rotation in the lumbar region. Russell et al. (1993) found that lateral bend and axial twist motions, flexion and lateral bending motions, and flexion and axial twist motions are strongly coupled, with greater coupling observed in younger as compared to older participants in their investigation using an electromagnetic system (Polhemus Navigation Sciences 3Space Isotrak system). Gregerson and Lucas (1967) found approximately 10° of rotation motion about the whole lumbar spine in young men performing with maximal effort, or when divided equally across the 5 lumbar FSUs about one degree to the left and right for each joint. Pearcy and Tibrewal (1984) reported two degrees of rotation for each lumbar joint in-vivo, but that L3/4 and L4/5 segments being slightly more mobile had up to 5 degrees of rotation. Whereas, during

unconstrained maximal twist efforts the peak axial twist range of motion was 16.6° ±4.7 and 15.6° ± 3.6 to the left and right respectively, as measured between L1 vertebrae and the sacrum via an electromagnetic tracking system (Peach et al., 1998).

Passive Rotation

McGill et al. (1994) investigated the passive stiffness of the lumbar torso in upright standing to measure axial twist motion, and supine position for flexion, extension, and lateral bending. For the passive angles ranging from 2° to 24°, the stiffness ranged from 0.13Nm/° to 0.64Nm/° (McGill et al., 1994). The focus of this paper was on the effect of belt wearing and breath holding but provides comparative data for passive axial twist motion in an upright posture. EMG was monitored to ensure the relaxation of the participants' muscles during the passive range of motion testing (McGill et al., 1994). Bodén and Öberg (1998) documented approximately 18° to 26° of motion in the lumbar region in a seated posture. Farfan et al. (1970) calculated in-vitro axial rotation stiffness values of 2.0Nm/° for lumbar FSUs. White and Panjabi (1990) reported the axial rotation moment stiffness coefficient of a representative FSU in the human lumbar region to be 1.36Nm/°, 2.08Nm/°, and 5.00Nm/° for flexion, extension, and axial rotation respectively.

2.3 Purpose

The purpose of this experiment was to quantify the amount of axial twist passive motion in the lumbar spine in various flexed postures, to assess whether flexion/extension postures can cause a detectable change in the in-vivo range of axial twist motion, lumbar spine axial twist stiffness, and to investigate the diurnal effect on these motions.

2.4 Hypotheses

1. Increasing trunk flexion will increase the range of axial rotation achieved.

   Findings supported Hypothesis #1.

2. End point stiffness will be reduced for increasingly flexed postures.

   Findings supported Hypothesis #2.

3. Maximal axial twist moment will not change with any of the postures.

   Findings supported Hypothesis #3.

2.5 Methods

Participants

Ten healthy male participants, 23.3 years (± 2.6), 1.79m (± 0.06), and 75.6kg (± 4.6), were recruited from a university population. Due to the configuration of the restraint apparatus required to immobilize the torso, only male participants were studied. All participants were healthy, engaged in average levels of physical activity, and had reported no occurrences of low back pain for at least the year preceding the study. Russell et al. (1992), Adams et al. (1987), and Parkinson et al. (2004) have shown a diurnal influence on spine mobility. Likewise, to examine whether time of day influenced the measured response, sessions in the morning and afternoon were incorporated into this study. The participants were given time to become accustomed to the harnesses, table, and procedures before data were collected in both the morning and the afternoon sessions. Informed consent was obtained from all participants prior to taking part in the study. The protocol was approved by the University of Waterloo's Research Ethics and Review Committee.

Apparatus

The passive axial twist moment–angle relationships were measured on a custom jig modified from the frictionless table that has been described in the work of Parkinson et al. (2004). The participant's upper body (T3 to T12) was fixed using an adjustable harness system of padded wooden plates (Figure 2.1). This upper harness was bolted to the wall through an aluminum bar. The bar could be adjusted for length to enable each participant to be positioned optimally on the cradle. Chain cross-ties were secured to the bar on either side to ensure a rigid structure. The participants knelt on a cradle, with their lower body fixed to the cradle via a second rigid harness system. The rigid harness systems minimized the contribution of the thorax and pelvis/lower limb to the rotations measured, leaving the lumbar spine as the isolated, moveable section. Prior to the start of data collection, the lumbar spine posture of a participant in the harness systems was compared to upright standing lumbar spine posture and the average difference within the infra-red emitting diodes location measured using an Optotrak system (Certus, Northern Digital Waterloo, Ontario, Canada) across all three axes and lumbar levels (L1 to L5) was 0.43mm (± 0.55). The cradle moved freely on the frictionless table while resting on 2500 nylon ball bearings (Salem Specialty Ball Incorporated, Canton, Connecticut, USA) located between the cradle and the table surface. Moments were applied to the cradle via a force couple until the participant stopped rotating despite the application of additional force on the cabling (verified via displayed feedback of angle and force data), or the participant instructed the researcher to stop applying the force (Figure 2.2). No participant required a premature termination of the force application protocol. The force was measured with a load cell located between the

participant and application of force to the cables. The axial twist reaction moment was calculated about the centre of the L4/5 joint.

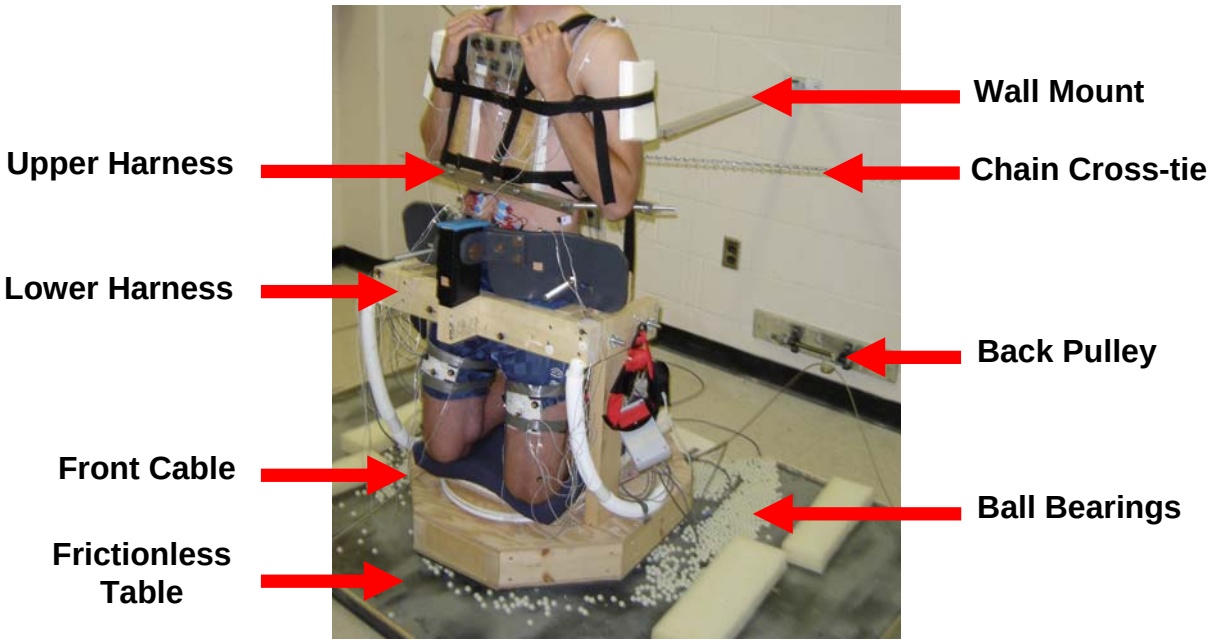

FIGURE 2.1 THE UPPER AND LOWER HARNESSES ISOLATED THE LUMBAR SPINE AS THE ONLY AREA FREE TO MOVE UNDER THE APPLIED ROTATIONAL MOMENT. THE UPPER HARNESS WAS BOLTED TO THE WALL THROUGH A NON-FLEXIBLE BAR WITH CHAIN CROSS-TIES SECURED TO THE BAR TO ENSURE A RIGID STRUCTURE. THE CRADLE MOVED FREELY ON THE FRICTIONLESS TABLE WHILE RESTING ON 2500 NYLON BALL BEARINGS BETWEEN THE CRADLE AND THE TABLE.

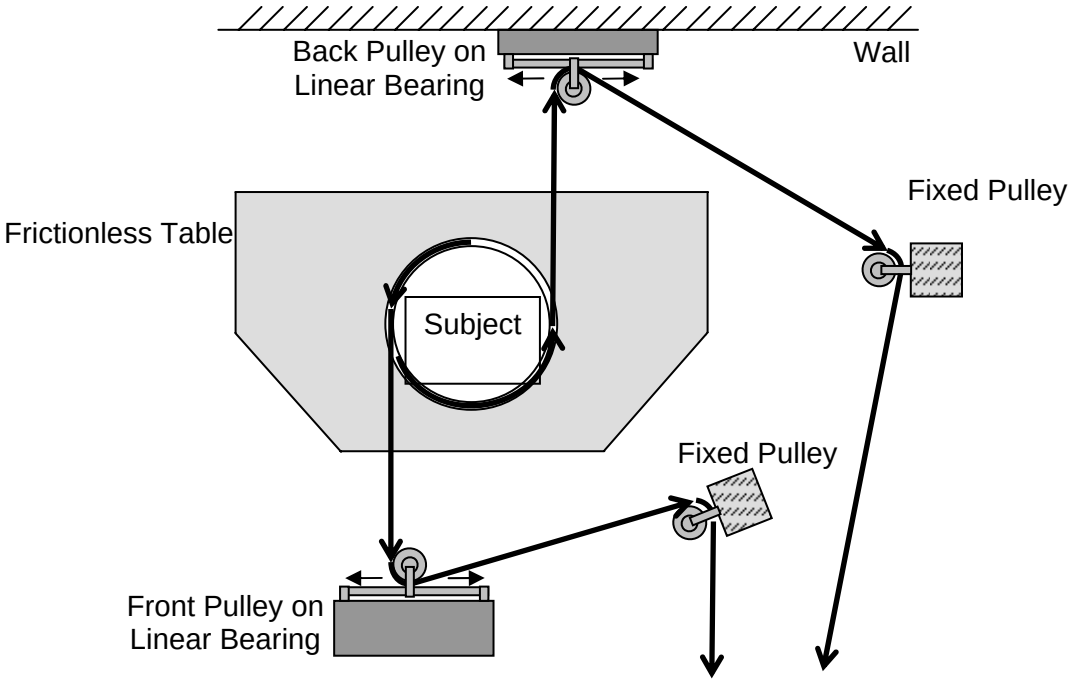

FIGURE 2.2 A SUPERIOR VIEW SCHEMATIC ILLUSTRATING THE FORCE PULLEY SYSTEM IN RELATION TO THE FRICTIONLESS TABLE. THE SUBJECT WAS POSITIONED ON THEIR KNEES OVER THE CENTRE OF THE CIRCULAR SURFACE. THE FRONT AND BACK PULLEYS WERE MOUNTED ON LINEAR BEARINGS TO PERMIT THE ENTIRE SYSTEM TO SHIFT LEFT AND RIGHT IF REQUIRED BY THE MOVEMENT OF THE PARTICIPANT DURING THE TRIALS. THE FORCE COUPLE THAT WAS GENERATED IS ILLUSTRATED, ALONG WITH THE DIRECTION OF CABLE MOVEMENT.

Motion Capture Instrumentation

Kinematic motion was captured using 22 infra-red emitting diodes and an Optotrak system (Certus, Northern Digital Waterloo, Ontario, Canada). Groups of three and four active markers were fixed to the chest (cluster 2) and pelvis (cluster 3) rigid harness systems, respectively (Figure 2.3). Active markers on the right and left shoulder over the acromio-clavicular joints, and over the xiphoid process were cluster 1, while cluster 4 consisted of four active markers placed bilaterally over the iliac crests (at midline) and the anterior superior iliac spines. Three active marker clusters mounted on rigid plates were affixed to each leg at midfemur height (clusters 5 and 6), and one active marker was taped approximately 10cm proximal to the distal end of each femur (markers 7 and 8). The difference between clusters 1 and 2 gave the subject's torso versus harness translations and rotations, clusters 3 and 4 gave the subject's pelvis versus harness translations and rotations, and clusters 2 and 3 gave the lumbar spine three-dimensional rotations, which were corrected for participant relative to harness movement by the cluster 1:2 and 3:4 calculations. The relative motion between the participant and the harness systems was less than 1° for all axes. Clusters 5 and 6 were used to ensure there was no lower body movement relative to the lower harness, whereas the lower femur markers were required landmarks to build the lower limb in the model using Visual 3D software (Version 3.27, C-Motion Incorporated, Rockville, Maryland, USA) which was used to calculate three-dimensional lumbar spine motions.

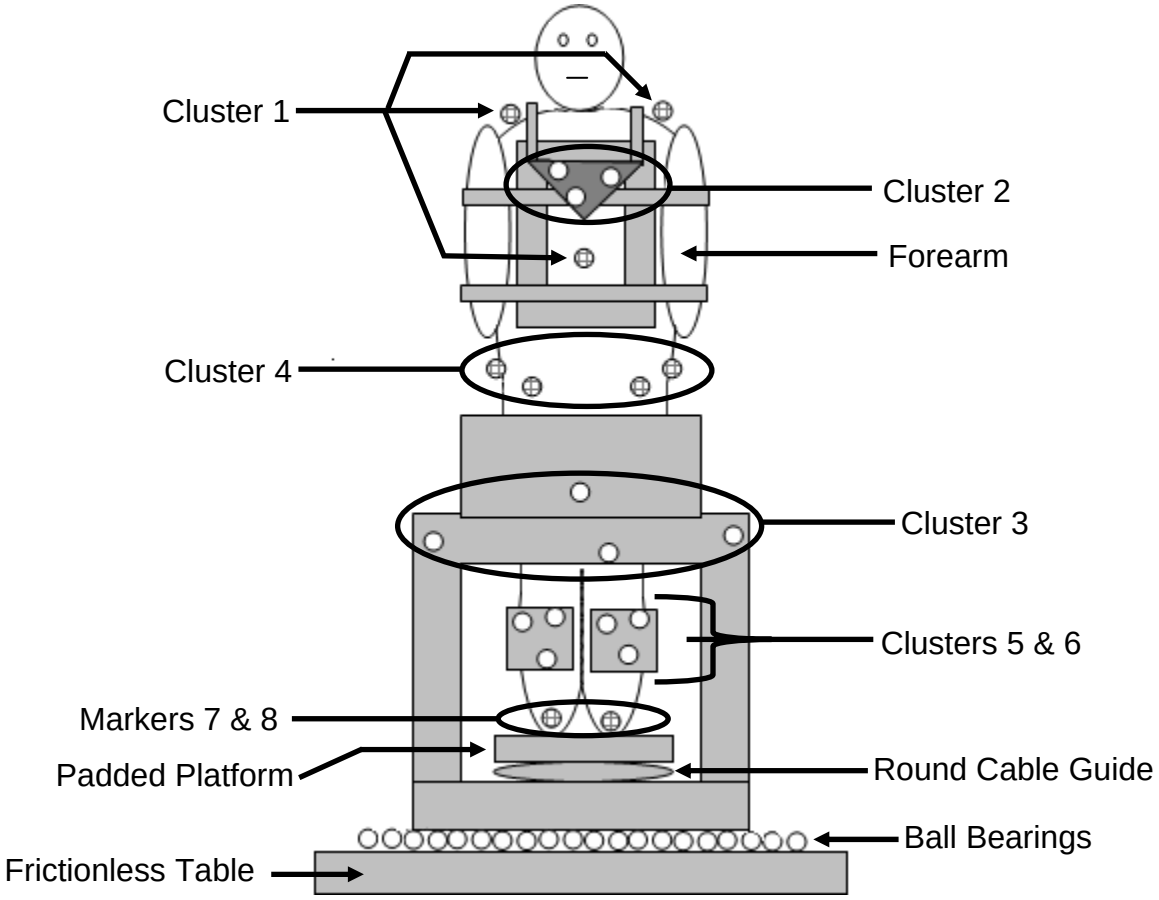

FIGURE 2.3 A FRONTAL PLANE SCHEMATIC SHOWING THE LOCATION OF HOW THE KINEMATIC MOTION WAS CAPTURED USING CLUSTERS OF INFRA-RED EMITTING DIODES. THE DIFFERENCE BETWEEN CLUSTERS 1 AND 2 GAVE THE SUBJECT'S TORSO VERSUS HARNESS TRANSLATIONS AND ROTATIONS, CLUSTERS 3 AND 4 GAVE THE SUBJECT'S PELVIS VERSUS HARNESS TRANSLATIONS AND ROTATIONS, AND CLUSTERS 2 AND 3 GAVE THE LUMBAR SPINE THREE-DIMENSIONAL ROTATIONS, WHICH WERE CORRECTED FOR PARTICIPANT RELATIVE TO HARNESS MOVEMENT BY THE CLUSTER 1 VERSUS 2 AND 3 VERSUS 4 CALCULATIONS.

Electromyography Instrumentation

EMG data were collected to ensure the measures were passive measures. The primary movers during upright and asymmetrical twist motion have been reported to be the internal and external obliques, latissimus dorsi, and erector spinae (Marras et al., 1998). Trunk muscle activity was monitored using 14 pairs of 20mm silver– silver chloride pre-gelled self-adhesive disposable electrodes (Blue Sensor, Medicotest Incorporated, Ølstykke, Denmark). The electrodes were attached bilaterally to the skin over the belly of the following muscles, with a centre-to-centre distance of 20mm: external and internal obliques, latissimus dorsi, and thoracic and lumbar erector spinae (McGill, 1991a). Reference electrodes were placed on the left and right clavicles, one for each of the two EMG amplifiers used (Model AMT-8, Bortec, Biomedical Limited, Calgary, Alberta, Canada). The raw EMG signals were band pass filtered from 10 to 1000Hz, and differentially amplified (common-mode rejection ratio 115dB at 60Hz, input impedance 10 GΩ). The EMG, lumbar kinematic, and moment data were synchronized. The EMG and force transducer were sampled at 2048Hz, and the position data at 64 Hz using a 16-bit analog-to digital conversion system (Optotrak Data Acquisition Unit II, Northern Digital Incorporated, Waterloo, Ontario, Canada). The average activation across all 14 channels, 10 participants, 21 trials, and two sessions was 0.3% (± 1.3) of maximum voluntary contraction, therefore, it was felt that participants did not actively assist or resist the passive axial twist motions.

Normalization Procedures and Trials

Prior to data collection, three maximum voluntary contraction (MVC) tasks were performed against isometric resistance, as well as a rest trial. These MVC trials permitted EMG normalization of the recorded muscles. The rest trial (representing baseline muscle

activation) and MVC protocols to obtain maximal muscle activation from the abdominal, erector spinae, and latissimus dorsi muscles have been previously reported in Drake et al. (2006).

To normalize the lumbar spine flexion/extension posture data, neutral-twist maximum voluntary flexion and extension trials were collected by instructing the participant to bend at the waist as far as comfortable while restrained by the lower body harness/cradle system. A sagittal plane view of the participant was projected on a 27-in. television, and the neutral and maximum flexion/extension postures (FEPs) were documented. Two additional positions (termed mild and mid) between the neutral and maximum positions for both flexion and extension were also documented on the screen. The starting position for each of the three repeats of the seven FEPs could then be replicated for the passive testing while being presented in a randomized order. The lumbar position in the neutral kneeling posture was defined as the zero position. For each trial the participant was instructed what posture to adopt, the position was checked on screen, and when the position was achieved and stable, the axial twist moment was applied. Since each of the seven FEPs was based on an individual's range of motion, the angles were not explicitly controlled. However, when tested with a two-way repeated measures ANOVA (posture, session) the FEPs were distinct from each other ($P < 0.0001$), were not different across sessions ($P = 0.652$) and had no session–posture interaction ($P = 0.172$). The FEPs, as shown in Figure 2.4, ranged from 93.2% ((± 1.3) 16.8) of maximum flexion to 93.4% ((± 1.3) 20.7) of maximum extension. Therefore, the FEPs were representative of the majority of the range of flexion–extension motion.

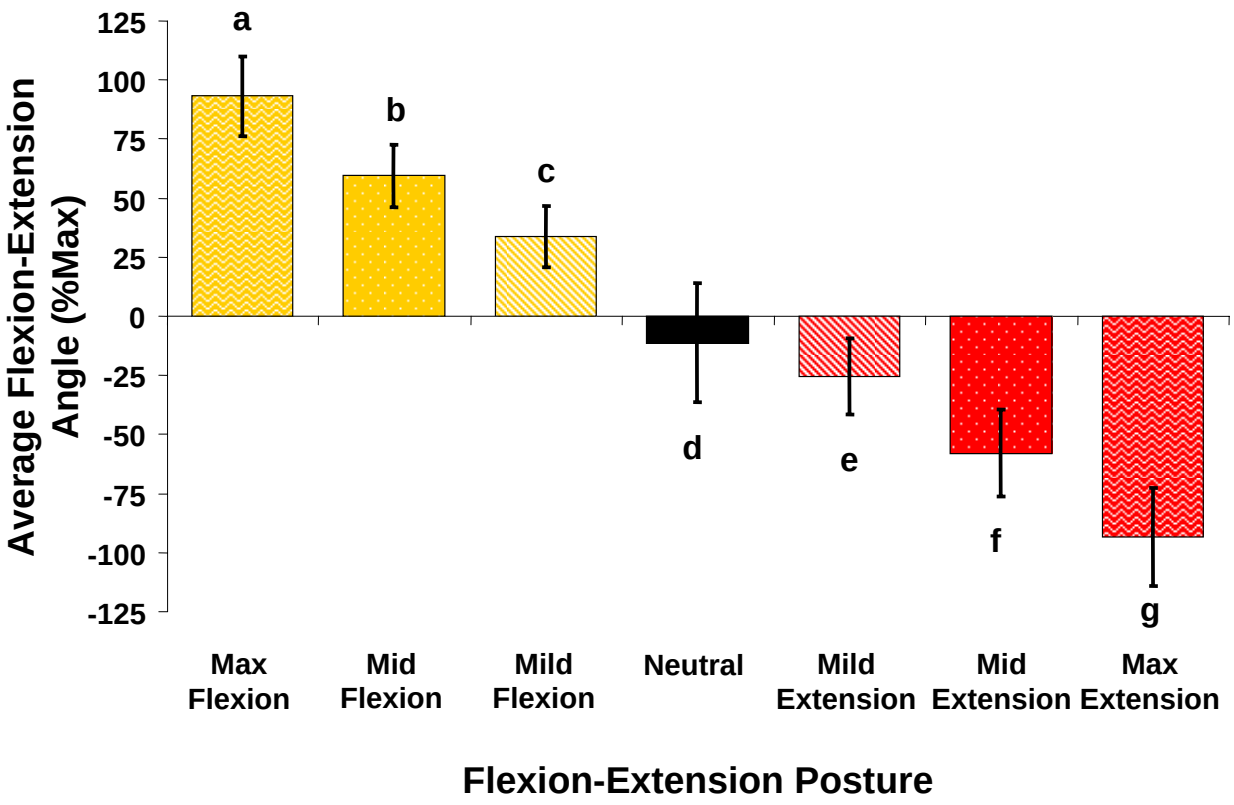

FIGURE 2.4 THE NEUTRAL AND SIX FLEXION AND EXTENSION POSTURES WERE DISTINCT FROM EACH OTHER ($P < 0.0001$), BUT WERE NOT DIFFERENT BETWEEN SESSIONS ($P = 0.652$). THE POSTURES RANGED FROM 93.2% (± 16.8) OF MAXIMUM-FLEXION TO 93.4% (± 20.7) OF MAXIMUM-EXTENSION. SIGNIFICANT DIFFERENCES IN THE POSTURES ARE DENOTED BY DIFFERENT LETTERS.

Trials

The participants performed the MVC and posture normalization trials at the beginning of both the 08:00h (morning) and 15:00h (afternoon) sessions. The participants wore the EMG electrodes between the sessions to minimize the variability introduced by slight changes in the electrode placement over the 14 muscle groups. All electrodes were replaced prior to the start of the afternoon session. The participants were asked to be at the morning session within 30 minutes of rising, and were to avoid any physical activity exceeding a moderate level for the preceding 24h. The participants rested for approximately 30 minutes, while being instrumented, prior to data collection. This rest was given to recover any changes that may have occurred between the time the participants arose to the start of the data collection (Adams et al., 1987). The EMG levels were monitored to indicate when the participants became comfortable with the passive experimental protocol and equipment, after which three trials in each posture were collected. The participants were passively rotated to the left from the seven randomized starting postures: maximum-flexion, mid-flexion, mild-flexion, maximum-extension, mid-extension, mild-extension, and neutral. The maximum-, mid-, and mild-flexion are within the ranges described as severe, mild, and neutral flexion, respectively by Punnett et al. (1991). The participants were smoothly rotated from the starting position until no additional range of motion was achieved with an increase in applied moment. Each trial was performed within a 10s period.

Data Processing and Statistical Analyses

Average axial twist stiffness, axial twist moment, axial twist angle, and lateral bend angle (off-axis motion) were calculated as the difference between the magnitude of the variable at the start of the trial and the value that occurred at the peak angle of axial rotation

achieved for each of the three repeats of the seven FEPs in both sessions. These data were normalized to the values in the neutral position for each session. The average axial twist stiffness was calculated from the axial twist moment divided by the peak axial twist angle for each repeat, FEP, and session. The flexion–extension postural data were normalized to the maximum flexion and extension angles, using the data collected from the maximal range of motion trials. The average velocity of the resultant motion was calculated from the peak axial twist angle divided by the time from the start of the trial and the maximal angle of axial rotation achieved for each of the three repeats of the seven FEPs in both sessions.

The EMG data were full wave rectified and passed through a second-order Butterworth filter, with a 2.5Hz cut-off frequency (Brereton and McGill, 1998), to produce a linear envelope signal for each of the 14 muscles monitored. The filtered signals were normalized to the maximum muscle activation determined from the MVCs and expressed as a percent of MVC. The peak EMG values from the 14 channels were averaged for each of the three repeats in the seven postures. The participants were instructed to remain relaxed and not to consciously activate any musculature. To assess the effort required to kneel in the cradle, the values with the rest bias removed were compared to those with the neutral relaxed kneeling posture bias removed. There was negligible difference between these methods since the effort required to kneel in the cradle was less than 1% MVC. The data reported has had the neutral relaxed kneeling posture bias removed.

Two-way repeated measure ANOVAs, for posture and session, were used to test the effect of the flexion–extension posture on the axial twist stiffness, axial twist moment, axial twist angle, lateral bend angle, and average rotational velocity. A Student–Newman–Keuls post hoc test was used to test any significant main effects of posture. A Least Square Means

was used to test any significant interaction effects between posture and session. The data had normal distributions and the residuals were acceptable, therefore, parametric statistical analyses were performed. In all statistical tests, the 95% (P = 0.05) level of confidence were used for rejection of the null hypothesis.

2.6 Results

The average normalized axial twist stiffness values (expressed as a percent of magnitudes from the neutral position) were smallest in the maximum-flexion posture at 81.0% (± 16.6), and largest in maximum-/mid-extension postures at 125.4% (± 24.4, P < 0.0001, Figure 2.5). The normalized peak axial twist angle was significantly different for the six non-neutral flexion and extension postures (P < 0.0001). The axial twist angle normalized to the rotation achieved in the neutral posture is shown in Figure 2.6, and ranged from 13.8% (± 8.9) greater rotation in the flexion-maximum posture to 23.8% (± 7.8) less rotation in the extension-maximum posture. The axial twist angle in the neutral posture was 39.4° (± 8.8), and ranged from 43.9° (± 8.9) in maximum-flexion to 30.1° (± 7.8) in maximum-extension. The average velocity of rotation was 28.6°/s (± 8.1) across all participants, postures, and the two sessions (P > 0.393). The normalized lateral bend (off-axis motion) that was coupled with the axial twist motion was significantly different between the maximum flexion posture at 11.4° (± 6.3), the flexion-mid/extension- maximum/extension-mid postures at 6.5° (± 4.5), and the extension-mid/flexion-mild/extension-mild postures at 4.4° (± 3.8, P < 0.0001, Figure 2.7). There was no effect of posture on the applied axial twist moment of 82.6Nm (± 18.1, P = 0.107). There were no significant differences caused by diurnal changes (P >0.201),

and also no session–posture interactions on average axial twist stiffness, axial twist moment, axial twist angle, or lateral bend angle ($P > 0.457$).

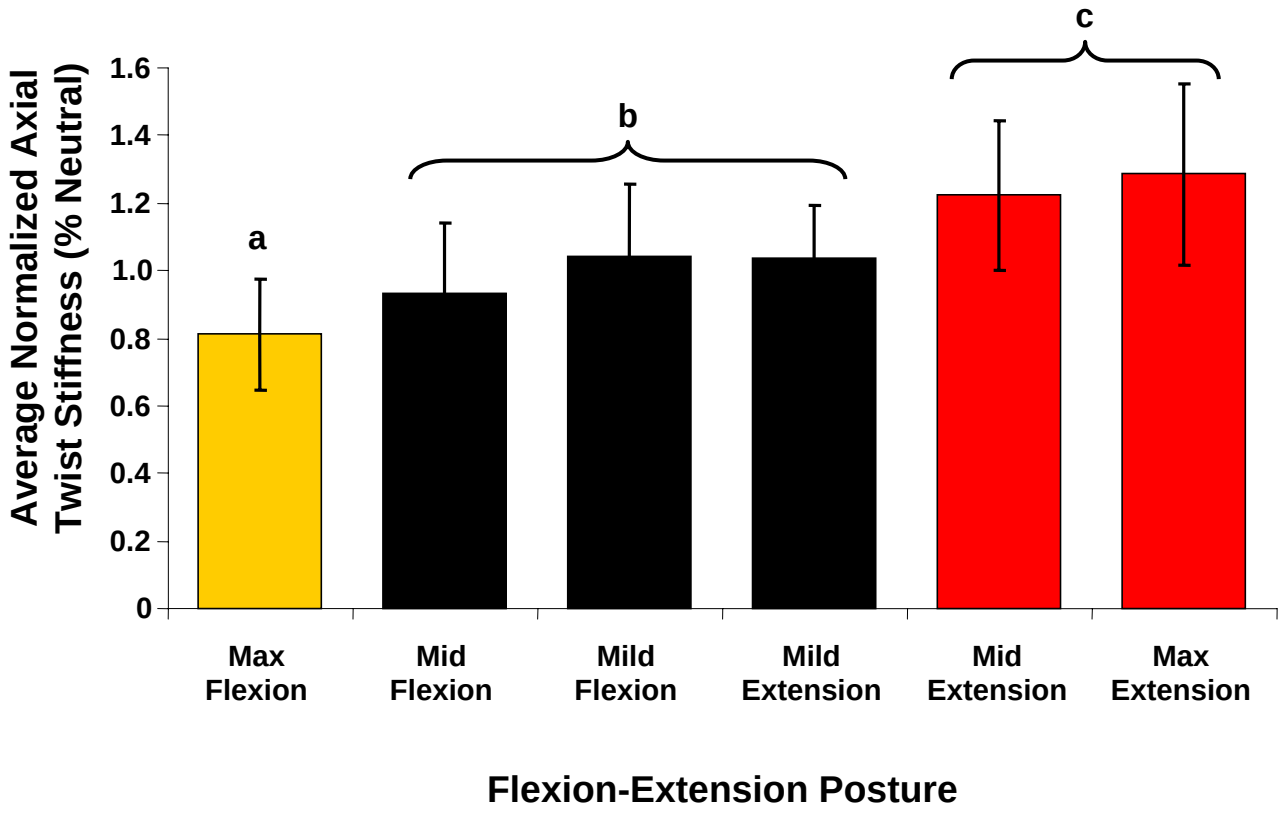

FIGURE 2.5 THE AVERAGE NORMALIZED AXIAL TWIST STIFFNESS VALUES FOR THE SIX FLEXION AND EXTENSION POSTURES. THE STIFFNESS VALUES WERE SMALLEST IN THE MAXIMUM-FLEXION POSTURE (81% ± 16.6) AND LARGEST IN THE MAXIMUM- AND MID-EXTENSION POSTURES AT 125.4% (± 24.4, $P < 0.0001$). SIGNIFICANT DIFFERENCES IN THE POSTURES ARE DENOTED BY DIFFERENT LETTERS.

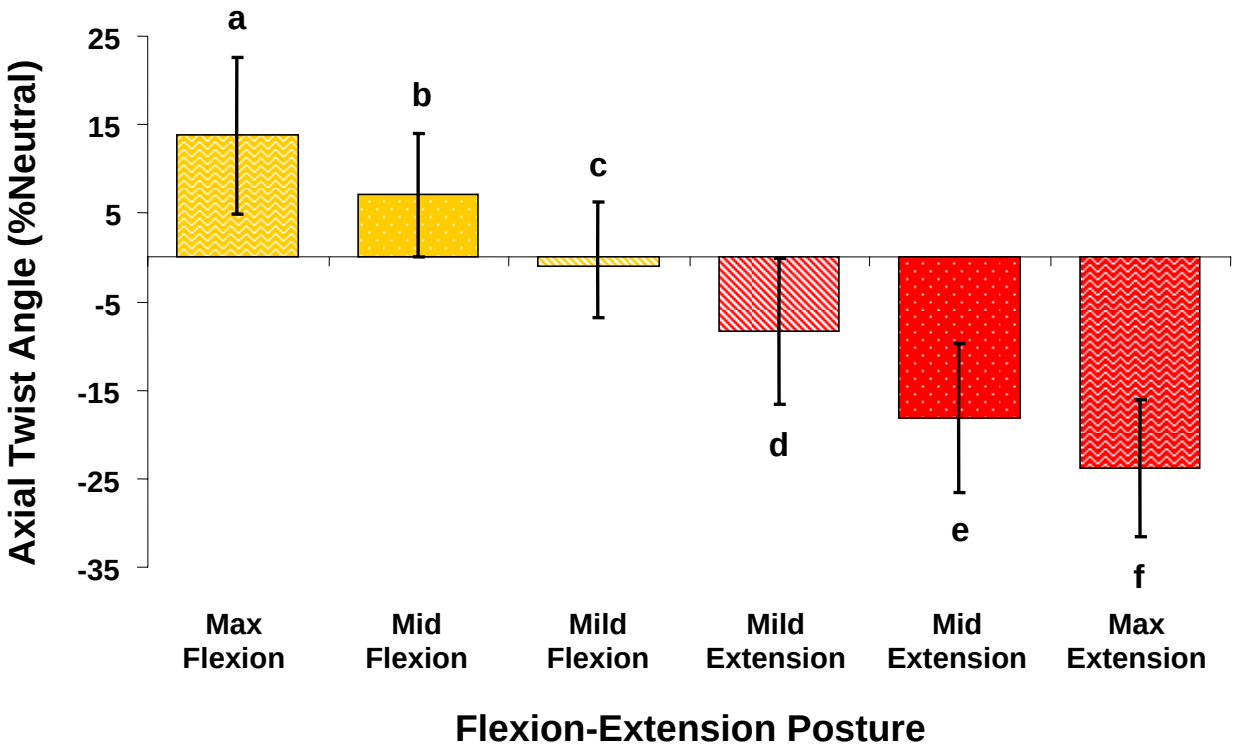

FIGURE 2.6 THE AVERAGE NORMALIZED AXIAL TWIST ANGLE VALUES WERE DIFFERENT FOR EACH OF THE SIX FLEXION AND EXTENSION POSTURES ($P < 0.0001$), AND RANGED FROM 13.8% (± 8.9) GREATER ROTATION IN THE MAXIMUM-FLEXION POSTURE TO 23.8% (± 7.8) LESS ROTATION IN THE MAXIMUM-EXTENSION POSTURE. SIGNIFICANT DIFFERENCES IN THE POSTURES ARE DENOTED BY DIFFERENT LETTERS.

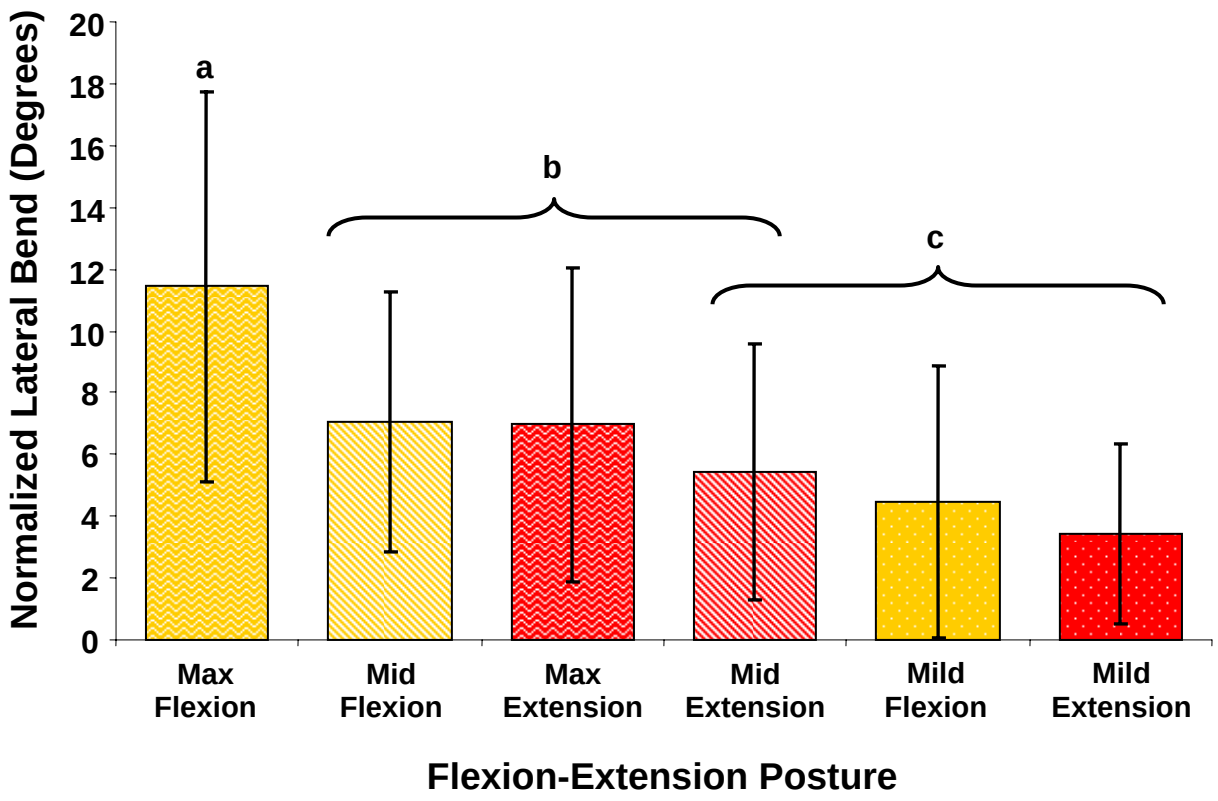

FIGURE 2.7 THE AVERAGE NORMALIZED LATERAL BEND ANGLE FOR EACH OF THE SIX FLEXION AND EXTENSION POSTURES WERE DISTINCT FROM EACH OTHER ($P < 0.0001$) AS INDICATED BY THE DIFFERENT LETTERS, BUT WERE NOT DIFFERENT ACROSS SESSIONS ($P = 0.333$).

2.7 Discussion

The magnitude of flexion or extension adopted by the participants influenced the axial twist stiffness measured in this study. Likewise, the amount of passive axial twist motion and the coupled off-axis lateral bend motion in the participants changed depending on the FEPs adopted. Whether the changes in axial twist and coupled motions with each FEP were due to a change in the initial interfacet spacing as suggested by Haberl et al. (2004) and Shirazi-Adl (1994) cannot be directly confirmed in this study.

However, the current findings support the results of in-vitro research that has identified axial twist moments as a possible modulator of injury in coupled postures (Drake et al., 2005; Pearcy and Hindle, 1991; Schmidt et al., 2007) since specimens in coupled postures had an increased ability to rotate (Pearcy and Hindle, 1991). Therefore, this work provides links between this passive in-vivo work, prior in-vitro research, and the proposed theories of how inter-facet spacing can alter axial twist motion. Important implications regarding the proper positioning of patients or research participants in imaging investigations are raised, as even mild changes in the FEP led to significant changes in both axial rotation and the coupled lateral bend motion measured. These factors should be considered prior to diagnosis and treatment of spinal instability, since abnormal axial rotation is one measure used clinically to indicate instability (Haughton et al., 2002; Ochia et al., 2006). Also, the use of non-neutral postures may be more revealing of underlying injury or instability if compared to the outcomes of the patient in a neutral posture.

A diurnal effect, if present, was not captured with the protocol used in this study. Although participants were requested to arrive within 30 min of rising, this could not be directly measured. Adams et al. (1987) found that allowing a 1:1 ratio of time after rising to

rest required prior to testing recovered any lost diurnal effects. Even if more rest time had been allotted in this study, the time to harness the participants in conjunction with the MVC protocols would have negated any recovered effects. Therefore, rather than a diurnal effect, repeatability was achieved with the afternoon session.

The inability to include female participants due to the restraint apparatus required to immobilize the torso and the limitation of using a university aged population may have influenced the findings in this study. There has been no effect of gender reported on voluntary lumbar spine motion for healthy participants (Dvorák et al., 1995; Gracovetsky et al., 1995; Peach et al., 1998). However, the effect of gender on passive coupled motions should be quantified using a similar approach as the current study to confirm the results reported in the voluntary motion studies. Gracovetsky et al. (1995) reported that while there was a decrease in the amount of active range of motion in older subjects, the coordination of the coupled motions was the same as that for younger subjects. Further investigation is required to determine if the passive range of motion in older adults is also reduced, and whether the movement patterns are similar to their younger counterparts.

McGill et al. (1994) investigated the passive stiffness of the lumbar torso in upright standing for axial rotation, and supine position for flexion, extension, and lateral bending. The focus of that research was on the effect of belt wearing and breath holding, but provides comparative data for passive axial twist motion in an upright posture. The peak axial twist angle was approximately 25–30° with 10Nm of torque applied (McGill et al., 1994). Bodén and Öberg (1998) documented approximately 18–26° of motion in the lumbar region in a seated posture. For 85 men in an upright standing posture Gomez et al. (1991) reported 36.6° (± 5.6) and 37.4° (± 4.0) of lumbar spine axial rotation to the left and right, respectively. The

axial twist angle in the neutral posture in this study is slightly higher than that of these studies at 39.5° (± 8.7), but may be due to the higher moments applied or to the different methods used. The average axial twist stiffness in the neutral posture was 2.19Nm/° which is similar to the 2.0Nm/° reported by Farfan et al. (1970) for human lumbar FSUs, but is lower than the 5.00Nm/° reported by White and Panjabi (1990) for a representative human lumbar FSU. However, the rotational stiffness found in this study is higher than the 0.13Nm/° to 0.64Nm/° for passive twist angles of 2°and 24°, respectively reported by McGill et al. (1994), possibly due to the isolation of the lumbar spine in this study compared to the whole torso. The axial twist moment that was applied in this study is similar to the 97Nm achieved isometrically by male participants tested by McGill and Hoodless (1990). The velocity of passive rotation in this study across all participants and postures was 28.6°/s (± 8.1), which is within the range used by other investigators for in-vivo investigations. Marras et al. (1998) used velocities of 10°/s and 20°/s to investigate in-vivo muscular responses, McGill (1992) used 30°/s to represent in-vivo dynamic activities, Kumar et al. (2003) used 10°/s, 20°/s, and 40°/s, and McGill and Hoodless (1990) used 30°/s and 60°/s in their in-vivo research.

A possible postural mechanism that modulates the relationship between the inter-facet spacing and the amount of axial twist motion or axial twist moment resistance, and by corollary the resulting loading on the facet joints and IVD was developed from the findings of this study and related literature. Once the trunk is flexed the facet joints may partially disengage which could increase gap distance between articular surfaces and thereby reduce their moment resisting capability, increase the amount of axial twist motion at that joint, and change the load distribution between the facet joints and the IVD. If the trunk is extended the facet joints may partially engage causing the opposite effects. Axial twist loading has been

shown to fracture the facets and accelerate annular injury formation when added to flexion–extension motions to create combined loading postures (Drake et al., 2005). Fazey et al. (2006), who tracked the in-vivo deformation of the nucleus pulposus using MRI, found that different asymmetrical loading on the IVD results due to the addition of rotation to flexion/extension postures. The postural mechanism hypothesis is further supported by van Dieën (1996) who reported that axial rotation caused an asymmetry in back muscle activation patterns, and possibly reduced spinal stability. The extra motion acquired in non-neutral postures found in this study suggests the participants may require higher muscle activation to control spine stability, and/or other passive structures (such as the intervertebral disc) may be required to resist the portion of the load that may no longer be carried by the facets in non-neutral flexion–extension postures. Pearcy (1993) found that when the spine is flexed in-vivo the facet joints permit greater twist motion, but postural muscle action in flexed postures restricts active twist motion. In addition to the increased muscle activation that may be required to assume the non-neutral postures, higher muscle co-activation to maintain spine stability can lead to further increases in spine compression due to muscle activation (Axler and McGill, 1997; Drake et al., 2006; Granata et al., 2005; Marras and Granata, 1995). Higher muscular demands would lead to increased rates of fatigue, and possibly increases in motor control errors during movement which may leave the passive structures vulnerable to loads beyond their capacities. This postulated postural mechanism supports epidemiological reports that have identified a higher risk of low back injury with jobs incorporating coupled postures involving twist motion (Kelsey et al., 1984; Marras et al., 1993; Manning et al., 1984).

This investigation of non-neutral postures with passive rotation in-vivo combined with in-vitro findings suggests that the mechanism of increased risk of injury under axial twist moment/motion is likely due to a change in the load distribution accompanying the resulting coupled motions/postures. Whether the changes in load distributions that occurred with coupled motions or postures are due to an altered inter-facet spacing was not directly measured. There is little or no data available on the mechanical role of the facet joints under combined axial twist and flexion/extension moments (Panjabi et al., 1989b) or motions. This study has generated comparison data for clinical investigations, but more importantly has highlighted the effect that small changes in flexion/extension posture can have on the twist motions of the lumbar spine, and likewise that the positioning of patients during diagnostic tests may affect subsequent diagnoses and treatment strategies. Therefore, care must be taken to ensure the desired posture is attained to not confound any observations, subsequent treatment assignments, or patient retesting following treatment.

2.8 Conclusion

The lumbar spine passive axial twist average stiffness and rotational range were modified by flexion/extension postures when axial twist moments were applied. The postural mechanism observed may be due to a change in the distance separating the facets prior to rotation. The data combined with information on how the spine responds at a tissue level to coupled motions can be used in determining spine rotational injury mechanisms. This information will be useful for determining the appropriate patient positioning during diagnostic tests, as well as influencing treatment regimes.

2.9 Limitations

The main limitations of this study are the use of surface measures to represent underlying bony motion, and the use of a university male study sample. The two rigid harness systems minimized the contribution of the thorax (T3 to T12) and pelvis/lower limb to the rotations measured, leaving the lumbar spine as the isolated, moveable section. The relative motion between the harness systems and the participant was less than 1° for all axes. The relative motion of the participant to the harness was removed by the placement of markers on each harness and on the participant's thorax and pelvis/lower limb (locations with minimal skin movement artifact during the motions). The inability to include female participants was due to the restraint apparatus required to immobilize the torso. There has been no effect of gender reported on voluntary three-dimensional lumbar spine motion for healthy participants (Dvorák et al., 1995; Gracovetsky et al., 1995; Peach et al., 1998). However, the effect of gender on passive coupled motions should be quantified using a similar approach as the current study to confirm the results reported in the voluntary motion studies. Gracovetsky et al. (1995) reported that while there was a decrease in the amount of active range of motion in older subjects, the coordination of the coupled motions was the same as that for younger subjects. Further investigation is required to determine if the passive range of motion in older adults is also reduced, and whether the movement patterns are similar to their younger counterparts.

2.10 Contributions

The main contribution of this investigation is the finding that axial twist range of motion and lumbar spine stiffness are modified by flexed-extended postures. When considered in concert with the in-vitro literature, a change in load distribution from the coupled motions/postures likely produces the observed outcomes. Also, the results of this study support the epidemiological evidence that reports an increased risk of injury when tasks include axial twist moment/motion. This study has clinical relevance for patient diagnostic positioning and evaluation since it shows that small changes in flexion/extension posture can have an affect on twist motions of the lumbar spine. Types of rehabilitation/treatment for spinal instabilities should also consider the effects of coupled postures to provide safe regimes. Therefore, to ensure posture does not confound clinical observations, especially in test-retest scenarios, careful control of posture must be used. This research generated a paper that is currently in press with the journal of Clinical Biomechanics.

CHAPTER 3

GENERAL LITERATURE REVIEW FOR IN-VITRO STUDIES

3.1 Overview

The studies in Drake et al. (2008– Chapter #4) and Drake and Callaghan (2008b– Chapter #5; 2008c– Chapter #6) all involve in-vitro methodology as the primary experimental approach, so there is a common literature base for these studies. This section provides the necessary background to assess the design, as well as the implications and contributions of these in-vitro studies to the body of scientific knowledge. The common literature portion introduces and discusses the anatomy, sources of pain, and known effects of posture. The main issues of in-vitro testing protocols, the use of animal models, post mortem changes, storage, testing considerations, and common methodological protocols are also included. Any pertinent literature that was unique to an individual study was included in the associated chapter.

3.2 Common In-vitro Literature Review

Spinal Segment Anatomy

Using various levels of detail, Figures 3.1, 3.2, 3.3, and 3.4 illustrate the importance of examining the changes to the neural spaces during the formation of an IVJ injury. A diagram of the location of the neural tissues in relation to an articulated FSU is depicted in Figure 3.1 (Agur and Lee, 1999, p.265), with an analogous human lumbar spine shown dissected in Figure 3.2 (McMinn and Hutchings, 1989, p.85). From these figures the potential of deformation associated with motion and loading to cause mechanical

compression of the neural tissues is obvious. A more detailed diagram showing the intimate nature of the neural and vascular tissues within the bony protection of the spinal canal and intervertebral foramina is illustrated in Figure 3.3 (Agur and Lee, 1999, p. 293). The immediate implications to surrounding tissues (i.e. skeletal muscle) of neural tissue impingement or compression can be understood from Figure 3.4 (Snell, 2000, p.831).

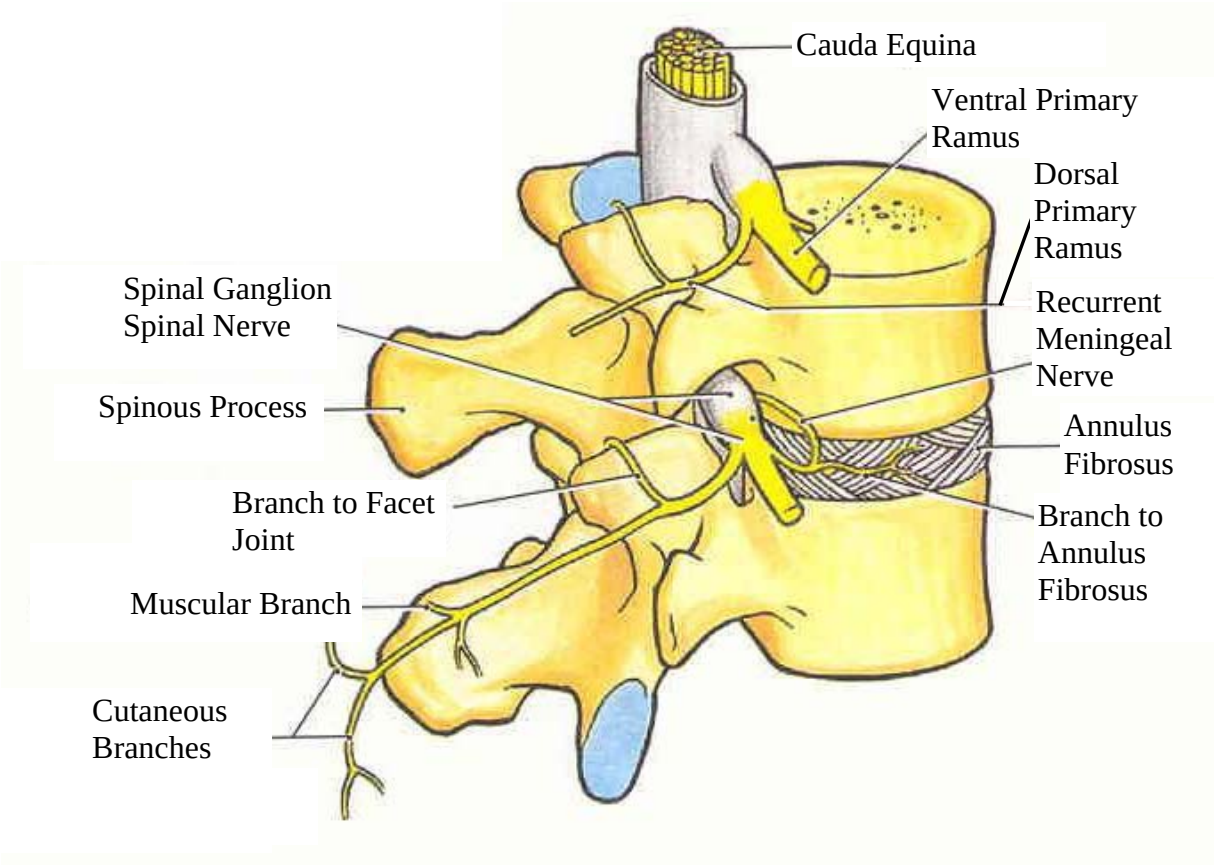

FIGURE 3.1 A DIAGRAM OF THE NEURAL ROOT BRANCHING OFF OF THE SPINAL CORD, TRAVELLING THROUGH THE INTERVERTEBRAL FORAMINA BETWEEN TWO ADJACENT VERTEBRAE OF AN INTACT FSU, AND INNERVATING THE ANNULUS FIBROSUS AND FACET JOINT. (MODIFIED FROM AGUR, A.M.R., LEE, M.J., 1999. GRANT'S ATLAS OF ANATOMY. 10TH EDITION. LIPPINCOTT WILLIAMS & WILKINS, PHILADELPHIA, PP. 265.)

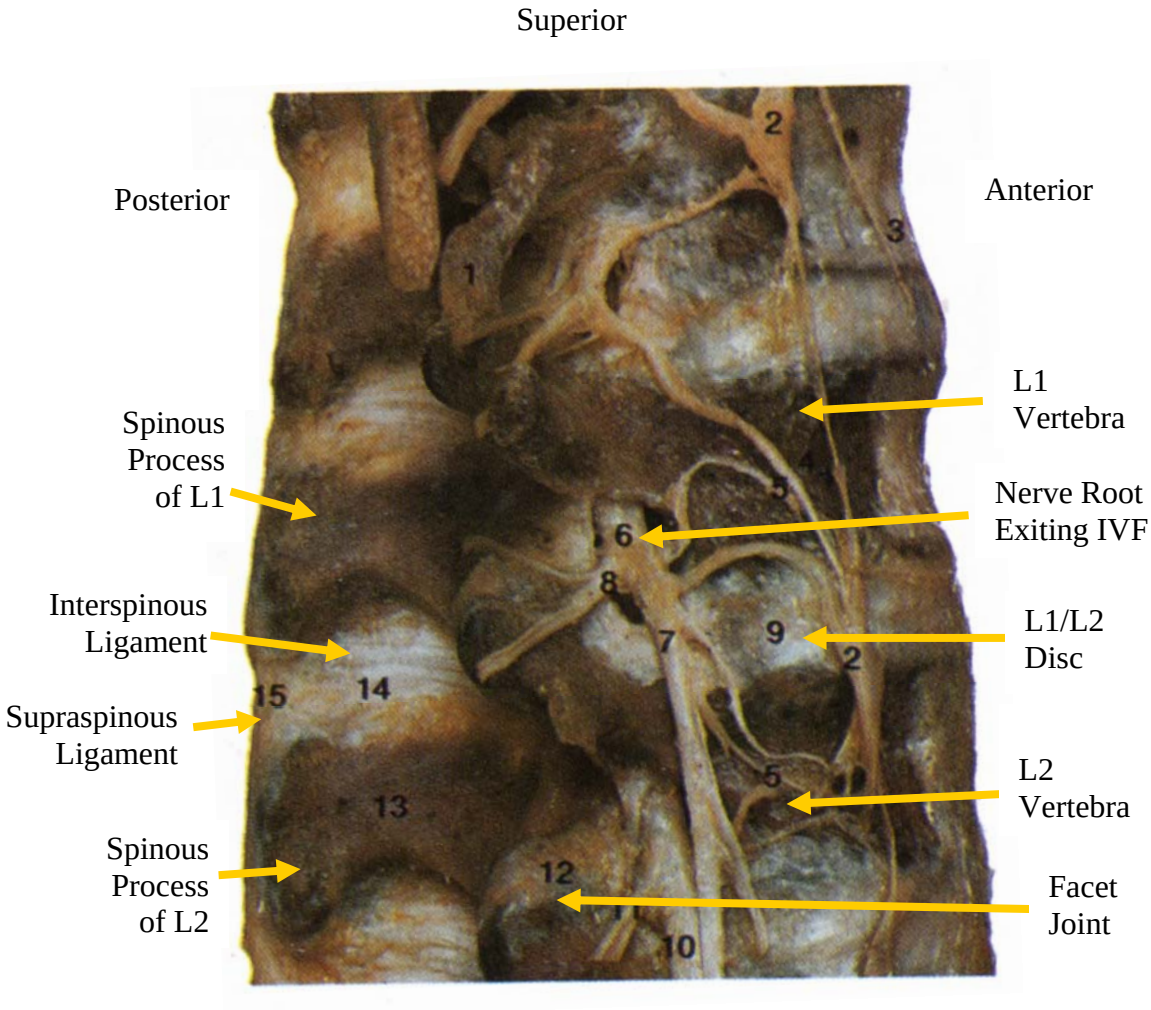

FIGURE 3.2 THE VERTEBRAL COLUMN IN THE UPPER LUMBAR REGION, FROM THE RIGHT SIDE, SHOWING THE LUMBAR NERVES EXITING THROUGH THE INTERVERTEBRAL FORAMINA (IVF) AND INNERVATING THE FACET JOINT AND INTERVERTBRAL DISC. NOTE: L1 AND L2 REFER TO THE 1ST AND 2ND LUMBAR VERTEBRAE RESPECTIVELY. (MODIFIED FROM MCMINN, R.M.H., AND HUTCHINGS, R.T., 1989. COLOR ATLAS OF HUMAN ANATOMY. 2ND EDITION, YEAR BOOK MEDICAL PUBLISHERS, INC., CHICAGO, PP. 85.)

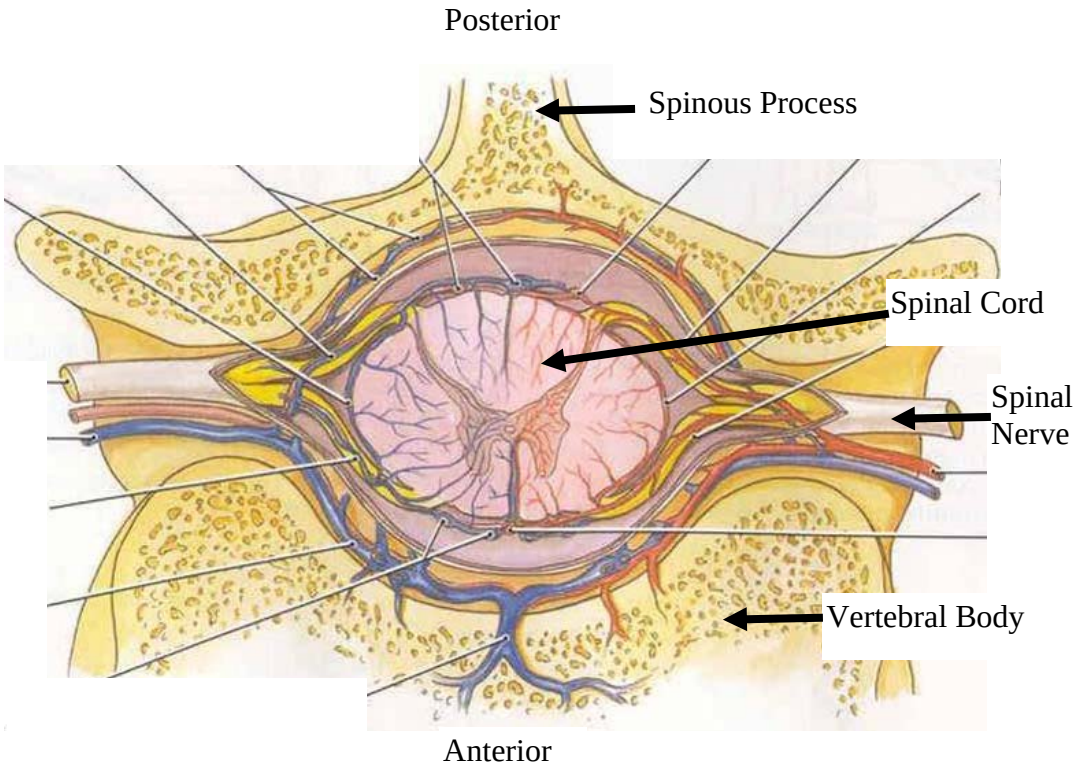

FIGURE 3.3 A DETAILED DIAGRAM SHOWING THE PROXIMITY OF THE NEURAL AND VASCULAR TISSUE WITH RESPECT TO THE VERTEBRAE. THE NERVES, ARTERIES, AND VEINS ARE SHOWN IN YELLOW, RED, AND BLUE RESPECTIVELY. IN THIS VIEW, A SMALL PORTION OF THE TOP OF THE VERTEBRAE HAS BEEN REMOVED, AND THE POSTERIOR ELEMENTS LEVELLED TO THE SAME HEIGHT. (MODIFIED FROM AGUR, A.M.R., LEE, M.J., 1999. GRANT'S ATLAS OF ANATOMY. 10TH EDITION. LIPPINCOTT WILLIAMS & WILKINS, PHILADELPHIA, PP. 293.)

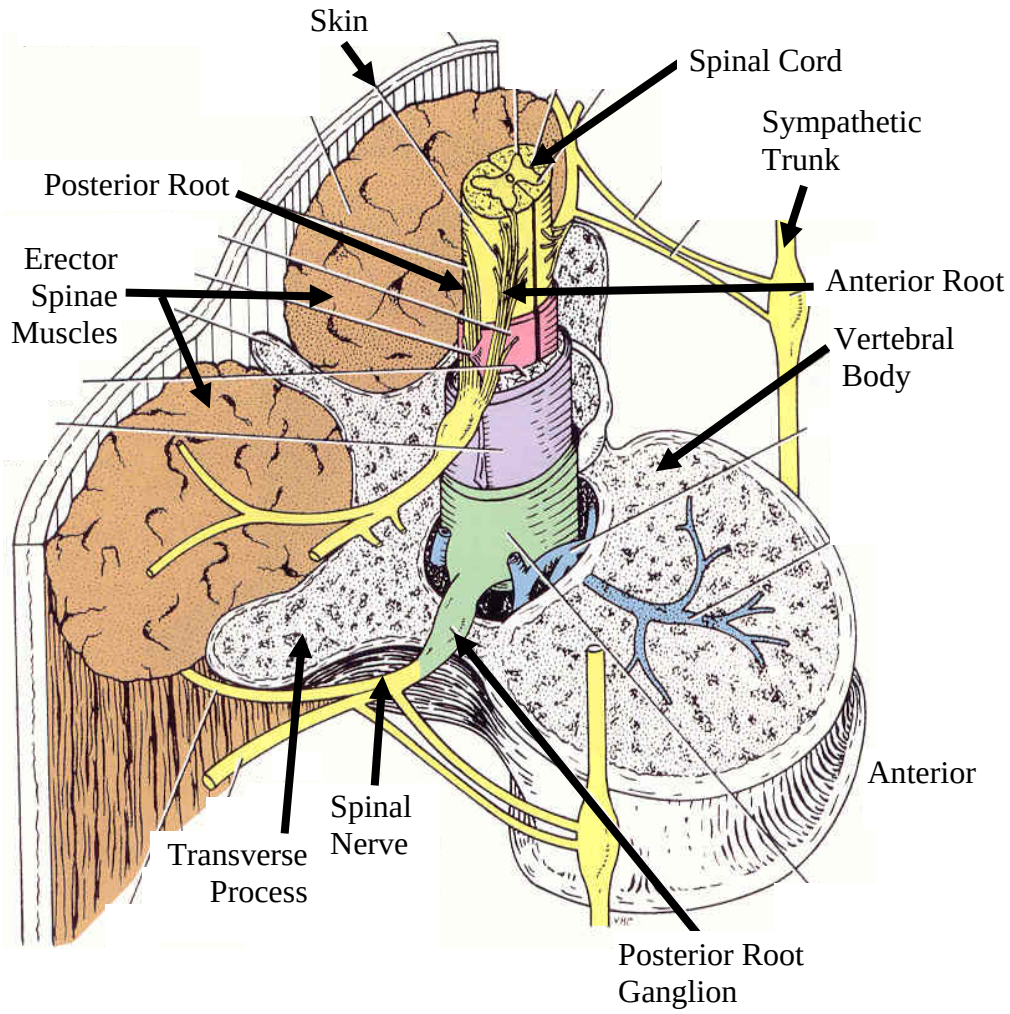

FIGURE 3.4 A DIAGRAM OF THE NERVE ROOTS AS THEY BRANCH OFF OF THE SPINAL CORD AND INNERVATE THE SURROUNDING TISSUES. (MODIFIED FROM SNELL, R.S. 2000. CLINICAL ANATOMY FOR MEDICAL STUDENTS. 6TH EDITION, LIPPINCOTT WILLIAMS & WILKINS, PHILADELPHIA, PP. 831.)

Sources of Pain

Due to the powerful effects that autologous nucleus pulposus material has on the structure and function of nerve roots upon contact when injected into the neural space in the absence of mechanical deformation or injury (Chen et al., 2003; Cornefjord, et al., 1996; Kayama et al., 1998; Olmarker et al, 1993, 1995, 1996; Otani et al., 1997, 1999) it is important to address the loading that is capable of generating such contact (i.e. herniations). These observed effects include morphologic and functional injury, such as decreased nerve conduction velocity (Otani et al., 1997), increases in vascular permeability, and membrane structural changes (Kanayama et al., 1998; Olmarker et al., 1993, 1995, 1996). The hypothesized pathophysiologic pathway has been hypothesized to be neurotoxic, vascular, inflammatory, or immunologic in nature (Cornefjord, et al., 1996; Olmarker et al, 1993, 1995, 1996).

The outer layers of the annulus and the facet joint capsule are innervated by a network of nociceptors (pain fibres) and mechanoreceptors (Bogduk et al., 1981; Roberts et al., 1995; Yamashita et al., 1993.). The nerves that supply the disc are not-uniformly distributed and are scarce (Bogduk et al., 1981). Under normal conditions the nociceptors have high mechanical thresholds, but in pathologic conditions the depolarization threshold can be reduced to points of spontaneous discharge or in response to very low levels of stress and strain (Cavanaugh et al., 1996).

Winkelstein et al. (2004) and Hubbard et al. (2008) confirmed neural tissue compression as a pain generating pathway with their experimental approach using an in-vivo rat model. These researchers showed that nerve root compression (via suture ligation) results in increased pain behaviours in rats. From the use of this animal model, the contribution of

both chemical and mechanical sources to pain generation was identified (Hubbard et al., 2008; Winkelstein et al., 2004). Possible causes of compression of the spinal cord and/or nerve roots include herniation, facet hypertrophy, ligamentum flavum bulges, vertebral body height loss, IVD height loss, and disc degeneration (Nuckley et al., 2002). Although the level of mechanical strain imposed on the nerve roots required to generate pain behaviours has been examined, the mechanism of how the possible contributing factors interplay to initiate depression of the mechanical thresholds are largely unknown.

Facet Joints Mechanics

Normally, partitioning experiments are conducted to attribute the load born by the facet joints during loading (Farfan, 1969; Farfan et al., 1970; Adams and Hutton, 1981; Panjabi et al., 1984). Lorenz et al. (1983) directly measured the pressure and contact area between facets of cadaveric FSUs, and calculated the load on the facets. These researchers loaded the spinal units with compression alone (approximately 200N, 400N, 700N, and 1400N), and compression combined with extension (6° to 8°), in a normal state and following facetectomy on the left side. The smallest two compressive loads were chosen to provide initial loading response of the facets, and the higher two levels corresponded to the reclining position and upright seated postures from Nachemson et al. (1966). They found that the absolute facet load remained relatively constant with increasing compressive loads, staying between approximately 50N to 175N for a neutral posture, and 100N and 275N for the combination posture across the L2/3 and L4/5 segments. However, the facet pressures were high ranging from 100 N/cm^2 to $350N/cm^2$ while the contact area decreased with increasing compression due to the irregular geometry of the facets. These authors hypothesized that degenerative changes to the facets are caused by pressure versus load.

Thompson et al. (2003) stated that the facet joints are the major passive contributor to limit spinal joint rotation, whereas the IVD's role is to stabilize the joint under exposures to twist moments that occur once a facet joint becomes compromised. The facets are important for maintaining the normal load on the IVD, as their removal increased the region of little or no resistance to motion in the middle of the IVD's range of motion (neutral zone). Therefore, the facets were not removed for these studies.

The range of twist motion permitted in the spine has been calculated as a function of the distance between the articulating surfaces of the facets using kinematic data and computed tomography reconstructions (Fazey et al., 2006), but the tracking of the facet joint in coupled postures has not been reported in detail. From finite element modelling, Schmidt et al. (2007) reported that the primary factor in determining the relative effectiveness of the facet joints in resisting rotation was the amount of inter-facet spacing. Drake and Callaghan (2008a– Chapter #2) found larger axial twist angles can be achieved when coupled with forward flexion, and Fazey et al. (2006) found the addition of axial rotation to flexion/extension postures caused asymmetrical loading of the intervertebral disc. An investigation on the annular strains and nuclear pressure predicted that the risk of disc damage increased during load combinations of axial rotation coupled with flexion (Schmidt et al., 2007). Although the load distribution was not directly quantified in this study, the change in load distribution can be inferred from the change in the spine mechanics reported.

Facet Tropism

To the author's knowledge, the relationship between of facet tropism and exposures to twist loading have not been examined. Facet tropism is defined as the difference between the angle of the left and right facet for a given vertebrae, and is usually measured relative to

the bisection of the sagittal or frontal plane (Ahmed et al., 1990; Boden et al., 1996; Grogan et al, 1997; Karacan et al, 2004; Lee et al, 2006; Masharawi et al., 2004; Ko and Park, 1997). Generally, spinal levels are considered to have: no facet tropism; if the asymmetry is less than 5° (Grogan et al., 1997) or 6° (Boden et al., 1996); mild tropism, if it is greater than 5° to 7° or greater than 6° to10°; moderate, if it is greater than 7° to 15° or greater than 10° to16°; and severe, if it is greater than 15° or greater than 16° as described by Grogan et al. (1997) and Boden et al. (1996). Although there is evidence that suggests there is a causal link between facet tropism and the development of disc degeneration and/or herniation (Karacan et al., 2004), the majority of the research has reported there is no association between increased facet tropism and disc degeneration and/or herniation (Ahmed et al., 1990; Boden et al., 1996; Grogan et al, 1997; Karacan et al, 2004; Lee et al, 2006; Masharawi et al., 2004). For this study the classification suggested by Boden et al. (1996) was used, since the majority of human facet joint angles have been reported to have less than 7° asymmetry (Grobler et al., 1993). Therefore, measures of facet tropism were included in Chapter #5 to see if there was a relationship to acute twist loading failure variables.

Postural Effects On Neural Spaces

The effects of posture and load combinations on the spinal cord and nerve roots have really only been addressed for the cervical spine region, but it has been shown that the size of the neural spaces is dependent on posture (Harrison et al., 1999). Accordingly, any source of pain due to mechanical changes that may occur over the course of complex combined loading, such as neural space occlusions, or abnormal facet joint loading, needs to be investigated. Panjabi et al. (1983) tested human cadaveric FSU in three dimensions, and measured the intervertebral foramina, revealing that both posture and degree of disc

degeneration alter neural space area. These researchers found that the size of the IVF was 185mm^2 and 108mm^2 for non-degenerated and degenerated specimens respectively, and so degeneration may play a role in the compression of nerve roots during physiologic ranges of motion (Panjabi et al., 1983). The effect of posture on the size of the IVF was increased 24% with flexion and decreased 20% with extension (Panjabi et al., 1983). Nuckley et al. (2002) flexed the spine in 1Nm increments up to 4Nm for flexion/extension and lateral bending, and up to 3Nm for axial twist, holding each increment for 3 seconds in cervical spinal segments, while tracking any deformations in the IVF using custom occlusion transducers and an Optotrak motion analysis system. The IVF was significantly changed in rotation (5-10%) and combined simulated physiological motions of bending with extension (19-20%) (Nuckley et al., 2002). In contrast, Norwicki et al. (1990) reported no statistical differences in IVF diameter or cross sectional area after loading human cadaveric functional spinal segments (ranging from T12 to S1) with approximately 3000N of axial compression (IVF cross sectional area 20.2mm^2). To the author's knowledge, no measures of the IVF have been made for repeated loading protocols, or time varying changes during the development of known IVJ injuries.

Animal Model

The use of humans in-vitro specimens in experiments to understand the mechanisms of injury and pain in the low back, and to develop safety guidelines/treatment strategies for the prevention/treatment (or even reversal) of low back disorders is ideal, but not always possible (Alini et al., 2008). Due to the invasiveness and health hazards associated with some of the methods required to investigate the mechanisms, animal models are often substituted

to be surrogates for humans. For in-vitro approaches, human materials are not always used since cadavers are expensive and can be difficult to obtain (Alini et al., 2008; Yoganandan, et al., 1996). Typically, donors are from elderly populations and were sick at the time of death, where as young donors, if obtained, usually sustained violent trauma or were terminally ill (Alini et al., 2008; Yingling et al., 1999). Further, Alini et al. (2008) stress that even when suitable human tissues become available, the lengthy legal gymnastics required to obtain the tissues for research purposes makes it difficult to complete research in a timely fashion or even at all (i.e. at an institution without a medical school). The use of an animal model provides a homogeneous specimen population and control over variables such as age, diet, weight, level of physical activity, and disc degeneration, but will not usually have direct transference of the results to live humans.

Accordingly, there are many types of animals documented in the literature as viable models for human tissue (Alini et al.,2008), including cats (Claude et al., 2003), mice/rats (Elliott and Sarver, 2004; Hubbard et al., 2008; Kuga and Kawabuchi, 2001; Winkelstein and DeLeo, 2004; Winkelstein et al., 2002), rabbits (Cavanaugh et al., 1995; Cavanaugh et al., 1996), ovine (sheep- Kettler et al., 2000; Costi et al., 2002; Thompson et al., 2003; goats- Smit, 2002), dogs (Otani et al., 1997, 1999; Buttermann et al., 1991,1992), bovine (Simunic et al., 2004), and porcine (Callaghan and McGill, 2001; Cornefjord et al., 1996; Gardner-Morose and Stokes, 2003; Gunning et al., 2001; Indahal et al., 1997; Kaigle et al., 1997; Kayama et al., 1998; Olmarker et al., 1993, 1995, 1996, 1997; Oxland et al., 1991; Panjabi et al., 1989a; van Deursen et al., 2001; Yingling et al., 1997, 1999). Additional studies and animal models have been reviewed in Alini et al. (2008). The human lumbar spine supports large compressive forces due to the weight of the upper body and head, and surrounding

musculature. The loading experienced by pig cervical vertebrae is also highly compressive given the need to support a cantilevered head (Alini et al., 2008; Yingling et al., 1999). Yingling et al. (1999) reported porcine cervical vertebrae resembled human lumbar vertebrae geometrically and functionally, while Oxland et al. (1991) found anatomical similarities. The notable differences, for mechanical testing, between the vertebrae were that the pig vertebrae were smaller and had anterior processes (Alini et al., 2008; Oxland et al., 1991; Yingling et al., 1999). However, Oxland et al. (1991) stated that these processes did not appear to serve any mechanical role (e.g. load carriage). In the investigation of the modulators of axial twist loading, the facet angle of the animal model may be of particular importance. Yingling et al. (1999) observed that the facet angle in the cervical porcine is closer to human lumbar vertebrae than porcine lumbar specimens. Further, Gardner-Morse and Stokes (2003) reported that porcine specimens have similar rotational stiffness to that of human specimens, making them a reasonable analogue for human lumbar vertebrae. Alini et al. (2008) support the use of animal models for mechanistic research when human specimens cannot be obtained. Therefore, due to the similarities between porcine and human specimens, the fact that porcine specimens are readily availability and cost-effective, and given the mechanistic research performed, a porcine cervical spine model was used for the in-vitro studies.

Post Mortem Changes, Storage, And Testing Considerations

Dead Versus Live

Regarding post mortem changes, Adams and Dolan (1996) mentioned any changes observed were small with respect to the variability inherent between specimens. The primary difference between loading living and dead tissue is that in living tissue there is an inflammatory response, and a feedback based system leading to recovery periods (sleep/rest).

Since physiological repair mechanisms of microfractures in bone (Brinckmann et al., 1988), and synthesis of proteoglycans (Urban et al., 1978) and collagen (Adams and Hutton, 1982), take much longer than two weeks (several weeks to > 1 year), more than 6000 cycles of loading are easily accumulated in shorter time periods. Also, testing conditions on dead tissue are usually limited to examining constrained motion(s), and often with static loads.

Storage

Due to the logistics associated with obtaining testing materials immediately post mortem and equipment/facility constraints, the frozen storage of specimens is unavoidable for most experiments. To be cost effective, immediately following harvest the supplier freezes the spines and preserves them in frozen storage until the order (typically 30 spines) is completed. Frozen storage facilitates the investigation of the injury mechanisms resulting from cumulative loading, since the protocols require the application of tens of thousands of loading cycles and several hours. Callaghan and McGill (2001) were the first researchers to demonstrate the reliable reproduction of cumulative trauma in-vitro, loading specimens up to 86,400 cycles taking 24 hours. To preserve the tissue from the time of slaughter to testing, the material obtained immediately following death is sealed in doubled polyethylene bags and stored at -20°C. Many researchers have investigated the effect of freezing on the biomechanical response of tissue. In a matched comparison study between fresh and frozen porcine specimens, Callaghan and McGill (1995) found that the frozen storage of the vertebral specimens significantly increased the ultimate compressive load (24%) and energy absorbed to failure (33%), but did not affect the stiffness and displacement at failure. No duration of frozen storage was found to affect the load-deformation for axial rotation, anterior shear, or lateral bending (Panjabi et al., 1985), and Dhillon et al. (2001) reported that

frozen storage does not significantly alter the creep response of human lumbar discs. Further, Nachemson (1960) reported only a 0.6% decrease on intradiscal pressure for human tissues kept in cold (morgue, ~0°C) and frozen (-25°C) storage. Conversely, Bass et al. (1997) measured a 25% decrease in IVD swelling pressure, with an osmometer, and they stated that swelling pressure was a significant contributor to the increase in creep from 0.7-110mm for fresh specimens to 0.7-140mm for frozen specimens (trial one to trial five). Although these researchers applied five cycles of 20 minute static axial loaded and 40 minutes unloaded, they only applied 100N of compressive force.

Hydration

The in-vivo study of Nachemson and Morris (1964) that used a needle mounted pressure transducer, revealed spinal tissue is constantly exposed to some level of compressive load, finding approximately 150N at L3/4 when participants were administered muscle relaxants. The removal of segments results in a loss of loading, and with no facility to oppose cellular osmotic gradients the water diffuses from high areas of concentration to areas of low concentration. The freezing/thawing process results in additional swelling of tissues. Preloading the tissue has been reported to return water content to a physiological range (Adams and Dolan, 1996) and to restore the IVD load-deformation response (Gardner-Morse and Stokes, 2003), thereby countering these post mortem changes. The magnitude of the preload must be selected high enough to reverse the post mortem swelling, but low enough not to damage the tissue. Gunning et al. (2001) demonstrated that the level of hydration in porcine motion segments affected the compressive failure tolerance, so the magnitude of preload must also be able to produce approximately the same level of hydration with high fidelity across a specimen population.

Another concern regarding specimen hydration is the potential for change over the course of testing. Costi et al. (2002) compared the effects of testing lumbar spine segments in a saline bath and exposed to the air on the load-deformation response, and concluded that the specimens were stiffer when tested in a saline bath. Further, the temperature of the testing environment can alter hydration and testing response of the tissue. Hasberry and Pearcy, (1986) showed the elastic mechanical properties of ligaments changed slightly at body temperature, but Adams and Dolan (1996) commented that these changes likely reflect small thermal expansion of tissue and so should have little effect on the function of the whole motion segment assuming the discs and ligaments expand by roughly the same amount. Researchers appear to test predominantly at room temperature, i.e. 21°C (e.g. Adams and Dolan, 1996; Nachemson et al., 1979), or at body temperature, i.e. 37°C (e.g. Brinckmann et al., 1988; Callaghan and McGill, 2001).

The use of a 15 minute, 260N preload on specimens wrapped in saline soaked plastic-backed cloth by Callaghan and McGill (2001) produced no significant differences in the final axial creep obtained of 26 specimens. These results were duplicated in 52 and 18 specimens in Aultman et al. (2004) and Drake et al. (2005) respectively, despite variability in the load deformation response occurring in approximately the first 5-8 minutes of loading, suggesting 260N of preload is able to reliably normalize the condition of the specimens. In these studies, 300N was programmed into the compressive axis, but due to jig design a portion of the compressive load could not applied to the specimen. This has been corrected through the improvement of the jig. Given that the objective in the in-vitro testing of the proposed research is to compare the response from different types of loading, a preload value of 300N

was applied to all specimens tested, which is equivalent to the 260N applied in the different test configuration of a previous system.

Specimen Length

The number of vertebrae and IVDs included from the specimen will affect the mechanical response. In a comparison study, Kettler et al. (2000) demonstrated that single-motion segment specimens (two vertebrae and the intervening IVD) had an increased flexion-extension range of motion and decreased neutral zone (the displacements, translations and/or rotations, between the neutral position and the initiation of resistance to motion; White and Panjabi, 1990) compared to the multi-motion segment, without the application of a preload prior to testing. These authors attribute the changes in motion to the loss of the multi-articular supraspinous ligament. Yingling et al. (1997) found no effect on compressive failure outcomes between two and three vertebrae spinal segments. Dickey and Kerr (2003) found an increase in the range of motion and neutral zone, but no difference in stiffness response about the L3/4 IVD for the single-motion segment (L3/4) versus the multi-motion segment (L1 to L5) specimens. However, for experiments aimed at relating a specific structural change (e.g. IVD or facet damage) to a measured biomechanical response (e.g. stiffness, creep, hysteresis), single-motion segments should be used to isolate the response to a single controlled location. Therefore, single-motion segments will be used for all of the in-vitro studies proposed in this thesis.

Axial Twist Loading Modulators

Load Type: Acute Twist Loading Failure

The twist angle at failure reported for FSUs has been shown to reach angles up to 22.6° (Farfan et al., 1970), whereas the normal range of rotation from maximal efforts in-

vivo has been reported to be quite limited to values in the range of 2° to 5° (Adams and Hutton, 1981; Gregersen and Lucas, 1967; Gunzburg et al., 1991; Pearcy and Tibrewal, 1984; White and Panjabi, 1990). There were several methodological differences in testing protocols that contributed to this large range of results, including variable magnitudes of compression, twist rotation rate, and twist moment loading method.

The compression facet was reported to yield after 1-2° from the application of approximately 459.4 ± 150.2Nm of axial twist moment (Adams and Hutton, 1981). However, this study generated the twist moment by applying compression to a rotatable platform, thereby continuously increasing the compressive load to generate greater rotation. The FSUs used by these researchers ranged in age from 18-77 years. Adams and Hutton (1981) concluded the compression facet was the structure primarily responsible for the resistance of applied twist moment and twist motion.

On specimens ranging in age from 27-86 years, Farfan (1969) and Farfan et al. (1970), applied an axial twist moment to the FSUs by hanging increasing magnitudes of weight (acting through a pulley). Weights were added at 30 and 60 second intervals to create 1.5° and 3.6° deflections respectively in the FSU, both with and without posterior elements intact (all structures posterior to the facets). Failure was defined as the point where an increase in weight elicited no further deflection in the specimen for these studies, and at this point the maximum moment was recorded. In Farfan (1969), the compressive loads applied were approximately 427N, and the axial twist moment at failure was approximately 32.7Nm (range 26.0-39.5Nm). Farfan et al. (1970) reported that the facet joints and IVD failed after 12° and 16° respectively from subsequent partitioning trials for non-degenerated intact FSUs. However, these researchers inserted a rigid bar through the FSU which prevents tracking of

the facet joint with applied loads. The rod was placed at the geometric centre of the specimen, so as to impose an axis of twist motion constant for the sequential partitioning performed (Farfan et al., 1970). Although Farfan et al. (1970) reported a failure twist moment of up to 20Nm for the facet joints, the compression magnitude was omitted. Farfan (1969) and Farfan et al. (1970) postulate that annular tears are more likely the result of axial twist loading than compression since this damage to the IVD was found without injury to bony structures. These circumferential tears of the peripheral layers of the annulus resembled annular changes found in natural disc degeneration which were reported by these authors to characterize the failure produced from forced rotation.

Although these were appropriate methods at the time these investigations were performed, none of these studies were able to smoothly apply a constant ramping twist moment or twist angle in a continuous manner (from zero to failure), and were unable to abruptly stop loading upon detection of failure. The values of the twist moment and twist angles at failure reported in Chapter #5 were quantified under a constant exposure of 1500N of compression, and the system control was such that the damage inflicted could be related directly to the loads applied. Using positional control enabled repeatable applications of the three axial twist rotation rates used Chapters #5 and #6. The failure twist moment and twist angle measures were obtained from the moment-angle curve in Chapter #5. The failure moment and angle were identified when further application of load caused the stiffness to fall toward zero (Adams et al., 2002; White and Panjabi, 1990) as is shown in Figure 3.5.

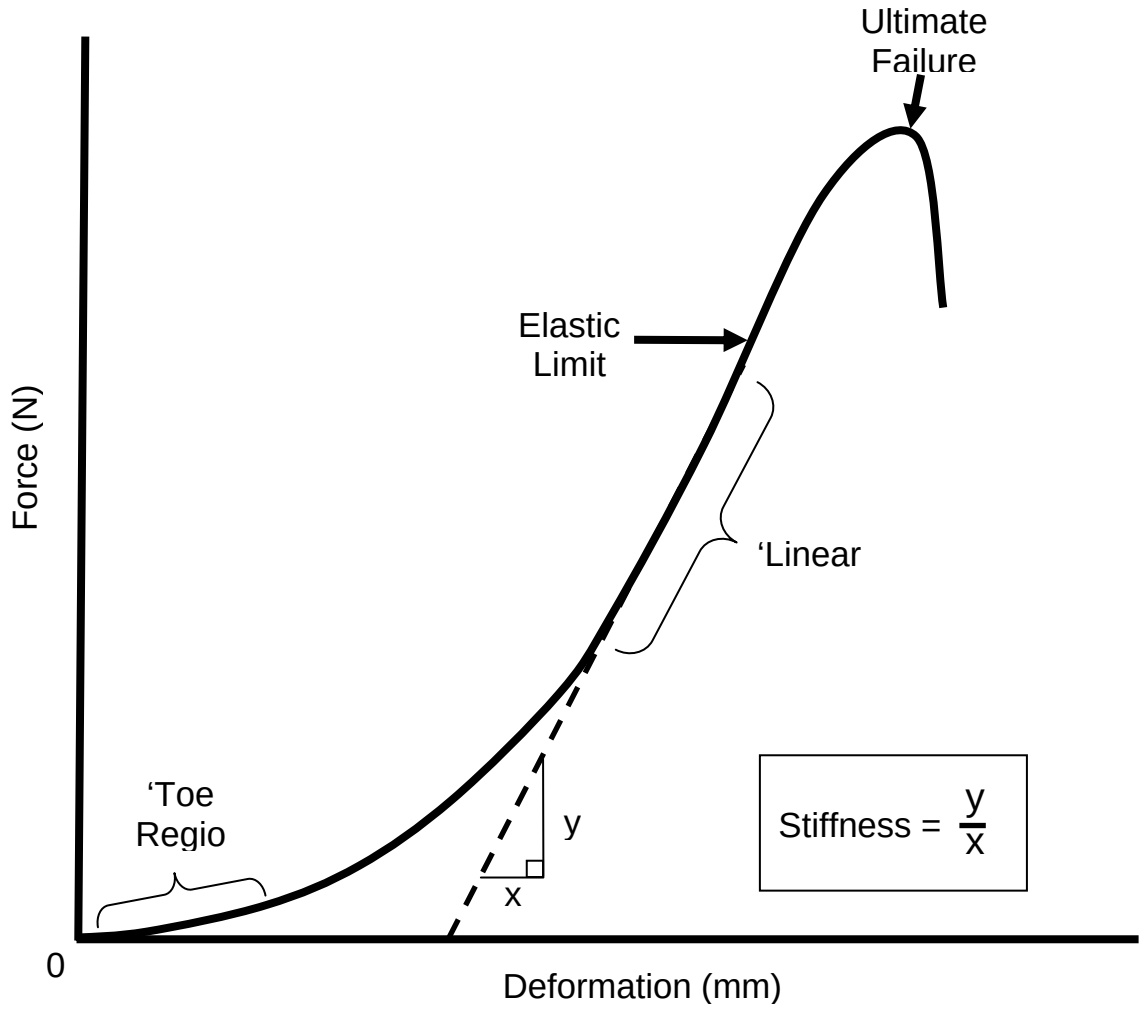

FIGURE 3.5 THE IDENTIFICATION OF THE ULTIMATE FAILURE POINT FROM A FORCE-DEFORMATION CURVE. ULTIMATE FAILURE OCCURS AT THE POINT WHERE THE STIFFNESS FALLS TOWARD ZERO. (MODIFIED FROM ADAMS, M.A., BOGDUK, N., BURTON, K., DOLAN, P., 2002. THE BIOMECHANICS OF BACK PAIN. CHURCHILL LIVINGSTONE, TORONTO, PP. 7.)

Repetitive Twist Loading

The information in the literature on the failure limits for repetitive or fatigue twist loading is sparse. Farfan et al. (1970) used constant cyclic angular rotations (step wise deformation) and showed the torque response leveled off at approximately 3000 cycles of

loading. A study conducted by Liu et al. (1985) examined repetitive twist moment and angle failure limits for neutral flexion postures using moment control (cyclically loading of the specimen to a constant level of rotational moment and record the resulting rotation) and positional control (cyclically loading of the specimen to a constant angular rotational displacement and collect the resulting twist moment). These authors loaded mostly Grade 3 and 4 human specimens ranging from 24 to 89 years of age. Regardless of the control mode employed, the control variable was applied as a sinusoid function at 0.5Hz, with a 440N compressive load, and in a chamber that was used to maintain an environment of 100% humidity. For moment control, four magnitudes of loading were examined ±11.3Nm (n=5), ±22.6Nm (n=5), ±33.9Nm (n=4), and 45.2Nm (n=5), to failure or a maximum of 10,000 cycles (~5.5 hours). The twist angle was measured at intervals of 100 cycles. For the positional control, a constant rotation of ±1.5° was applied to five specimens, since the failure tests in torque control identified 1.5° as the critical threshold in determining injury. That is all specimens with an initial rotational displacement less than 1.5° after 100 cycles of loading did not fail after 10,000 cycles of loading. Whereas specimens with initial rotational displacements larger than 1.5° after 100 cycles of loading failed before 10,000 cycles of loading. The different torques applied revealed that the higher the initial load, the larger the angular deformations relative to the number of cycles completed. The axial rotation increased to approximately ±6° at 2500 cycles and leveled off with the application of the 33.9Nm load, whereas for 11.3Nm there was less than 0.5° over the 10,000 cycle test duration. The positional control testing revealed that the torque started high, at -8Nm to +10 Nm, and after 3000 cycles had decreased to ±4Nm for the bilateral twist motion applied for the duration of testing. Liu et al. (1985) reported that the initial gap in a neutral posture

between normal facet articular surfaces is approximately 1.5mm. These authors comment that this distance and resulting angular deformation with applied of axial twist moments were too small to initiate disc degeneration, but as disc degeneration progresses, twist moments could contribute to the damage of the IVJ.

Load Rate

The FSU is a viscoelastic structure which means the response to loading (deformation) is dependent on the speed at which the load is applied. Likewise, more than one loading or displacement rate needs to be investigated in a given study if the effects are to be quantified. In earlier studies, axial moments were applied to FSUs by incrementally hanging static loads until the desired axial rotation was obtained (Farfan, 1969; Farfan et al., 1970). This protocol was repeated on specimens both with and without posterior elements (all structures posterior to the facets), in an attempt to partition the resistance of the twist moment to specific structures (Farfan et al., 1970). Whereas Adams and Hutton (1981) applied twist moments via a rotating plate loaded with compression at a rate of 333N/s. None of these studies described above were able to smoothly apply a ramping torque in a continuous manner (from 0N to failure), and were unable to abruptly stop loading upon detection of discrete changes in the slope of the moment-deformation curve. These aspects of experimental design are important when examining load rate. One objective is to 'catch' the failure as quickly as possible, which allows for more accurate assignment of physical findings of damage to measured time-varying variables (Gunning et al., 2001; Yingling et al., 1997). Marras et al. (1998) showed that velocity (10°/s and 20°/s for approximately T12 to S1, or 5 levels ~2°/s and 4°/s for a FSU) generated influences on the magnitude of axial twist

rotation and the ability to generate twist moments. In a study of compressive tolerance, Yingling et al. (1997) applied 5 compressive loading rates (100N/s, 1000N/s, 3000N/s, 10,000N/s, and 16,000N/s) to porcine FSUs, and found the compression and stiffness of the specimens increased in dynamic (>100N/s) as compared to the quasi-static (100N/s) condition. These authors reported the axial displacement at failure decreased with increasing load rate, and resulted in a change in the observed injury patterns. No studies have looked at the effects of load rate on the failure response to acute twist moments.

Compression

The specific effect of various compression magnitudes were not addressed in this thesis. However, the knowledge generated from this thesis has provided the foundation for these studies to be carried out in combined loading scenarios in the future. A review of pertinent literature was included in this document to provide the appropriate context, since compression magnitude must be considered when interpreting the findings of in-vitro literature.

Aultman et al. (2004) investigated the effects of static twist moment magnitude on compressive failure load and found the twist moments decreased the maximum compression tolerance value for the specimens. Likewise, it has been postulated that the magnitude of compression decreases the initial distance between the facets, and would likely affect the tracking and load carriage of the facets.

Acute failure of FSUs resulting from a one time twist load exposure has been examined by Farfan (1969), Farfan et al. (1970), and Adams and Hutton (1981). There were several limitations associated with the testing protocols that likely affected the results. The FSUs ranged in age from 27 to 86 years across experiments. Variable compression values

were applied as shown in Table 3.1 by Farfan (1969), whereas no reference to applied compression was made in Farfan et al. (1970). Farfan et al. (1970) reported a maximal twist loading of 14Nm (Range: 6.9Nm-20Nm) for the facet joints, 10Nm (Range: 8Nm-12Nm) for the facet capsules, and 31Nm (Range: 17Nm to 54Nm) for the IVD. Farfan (1969) and Farfan et al. (1970) postulate that annular tears are more likely the result of twist than compression loading since this damage to the IVD was found without injury to bony structures. However, in Farfan et al. (1970) a steel rod was placed at the geometric centre of the specimen, so to impose an axis of rotation constant for this partitioning study. Therefore, the behaviour of the FSU could be considerably altered.

TABLE 3.1: ADAPTED FROM TABLE I IN FARFAN (1969). THE VALUES HAVE BEEN CONVERTED TO SI UNITS (FARFAN REPORTED COMPRESSIVE LOAD IN POUNDS-PER-SQUARE-INCH, AND MOMENTS IN INCH-POUNDS). NOTE: 'TMAX' REPRESENTS THE MOMENT AT THE DEFINED POINT OF FAILURE.

Type of Specimen	Number of FSUs	Compressive Load (N)	Average Area (cm^2) (Range)	Average Tmax (Nm) (Range)
Intact FSU	8	---	23.2 (18.7-26.5)	23.7 (20.3-33.9)
Intact FSU	6	275.8	21.9 (20.0-23.2)	31.6 (20.3- 45.2)
Intact FSU	5	427.4	27.1 (20.6-27.7)	32.7 (26.0- 39.5)
Isolated IVD	9	---	21.9 (18.7-28.4)	18.1 (6.8- 29.4)
Isolated IVD	5	275.8	21.9 (20.0-23.2)	16.9 (9.0 -22.6)
Isolated IVD	6	320.5	14.8 (15.5-21.3)	13.5 (7.9 -19.2)

Adams and Hutton (1981) loaded human FSUs, aged 18 to 77 years with variable levels of compression (up to 1000N), repeating the loading after cutting structures (in order from supraspinous and interspinous ligaments, compression facet, to the tension facet) to

partition out the resistance to the applied axial twist moments (Table 3.2). From this study the authors concluded the compression facet was the structure primarily responsible for the resistance of the axial twist load, with the disc playing a minor role. They concluded that twist moments were unimportant in the development of disc degeneration and herniation. However, the magnitude of the compression load and applied twist moment were not held constant, so it is difficult to assess the differences in the failure of specimens due to applied twist moments, and the effect of disc degeneration.

TABLE 3.2: ADAPTED FROM ADAMS AND HUTTON (1981). THE MEAN AND STANDARD DEVIATION FOR WERE CALCULATED PER DEGREE OF DISC DEGENERATION. NOTE: 'COMP.' REPRESENTS COMPRESSION.

# FSU	Grade	Applied Moment (Nm)	Percent Torque Resisted By Each Structure				Moment on Disc (Nm)	Applied Comp. (N)
			Disc	Facet - Comp.	Facet - Tension	SSL/ ISL		
		18.04	31	63	2.4	3.4	6.18	459.4
5	1	(5.36)	(22.71)	(21.48)	(2.88)	(5.98)	(5.37)	(150.22)
		30.528	40.4	49.8	6.6	3.1	12.73	624.5
10	2	(15.05)	(12.29)	(15.21)	(8.91)	(2.89)	(6.83)	(325.41)
		16.285	39.83	53.17	3.00	3.67	6.87	377.83
6	3	(4.52)	(26.81)	(26.81)	(4.38)	(2.73)	(6.33)	(135.36)
		31.605	33	50.5	12.25	4.25	10.445	571.5
4	4	(10.04)	(16.33)	(14.57)	(4.35)	(2.99)	(6.17)	(191.27)

3.3 Common In-Vitro Methodology

Dissection and Fixation

The C3/4 porcine cervical motion segments were obtained from a common source to control for physical activity, diet, and age prior to death. The spine segments (~C1 to T6) were thawed for a minimum of 12 hours at room temperature in the double polyethylene bags in which they were frozen. Once thawed the desired single-motion cervical segment or FSU (C3/4) were obtained by slicing through the C2/3 and C4/5 IVDs and other soft tissues

remaining at these levels. The level of degeneration (Galante, 1967) and structural dimensions (anterior to posterior and medial to lateral endplate distances) were obtained from the transected superior and posterior IVDs. The specimens were further dissected, removing the overlying skeletal muscle, to leave an osteo-ligamentous structure. The FSUs were fixed into custom aluminum or ultra high molecular weight polyethylene (UHMWPE, A-K & Lippert Plastics, Kitchener, Ontario, Canada) cups using an appropriate number of steel 18 gauge or 14 gauge copper wire looped around the laminae and anterior processes, stainless steel or brass screws 0.5cm into the vertebral bodies, and finally embedded in non-exothermic dental plaster. The non-ferrous metals and plastics were used to mount the specimens in Chapter #4 to achieve a fixation that was compatible with the magnetic resonance imaging scanner. The specimens were wrapped with saline soaked plastic-backed cloth and plastic wrap to ensure continuous hydration. Approximately $0.7cm^3$ of barium sulfate radio-opaque mixed with blue dye (Coomassie Brilliant Blue G-mix: 0.25% dye, 2.5%MeOH, 97.25% distilled water) was injected into the IVD prior to testing for the studies in Chapter #5 and #6, but for Chapter #4 the barium sulfate was omitted. There are no adverse effects of injecting this mixture into the disc. This generalized mounting method and solution have proved effective for permitting the documentation of nuclear material tracking using radiography (Callaghan and McGill, 2001).

Loading

Prior to testing, each FSU was preconditioned with 300N of compressive force using a 2-axis servo hydraulic testing system (Chapter #4; Model 8511, Instron Canada, Burlington, Ontario, Canada) or a 3-axis servo-hydraulic dynamic testing system (Chapters #5 and #6; Model 8872, Instron Canada, Burlington, ON, Canada) for 15 minutes to counter

any swelling that had occurred postmortem. A brushless servomotor (Kollmorgen/Danaher Motion AMK23D servo motor and S20260-VTS servo amplifier, Radford, Virginia, USA) with a 70:1 Gear Head (Bayside PS60-070-LB) produced the flexion/extension moments on the 3-axis system. Through the use of moment transducer feedback, the flexion/extension moments, applied in concert with the axial compression, the servomotor was programmed to find a zero flexion-extension moment during the preloading phase so the angular position at the end of this phase was taken as the zero position for each FSU and was within the neutral zone defined by Panjabi et al. (1989). In order to monitor the non-linear tracking of the nucleus pulposus through the annulus during the repetitive loading studies, X-rays were taken pre/post loading, as well as every 1000 cycles during testing.

A compressive load of 1500N was applied in all studies during the testing conditions. This load was selected to enable comparison with previous literature findings (Callaghan and McGill, 2001; Drake et al., 2005). The compressive load for all failure tests was 1500N, which is approximately 14% of the ultimate compressive load tolerance of C3/4 porcine cervical spine segments (Gunning et al. 2001, Parkinson et al., 2005). The rationale behind using a 1472N compressive load combined with moderately repetitive flexion –extension motions has been shown to reliably produced disc herniations (Callaghan and McGill, 2001), in less than 6000 cycles of loading (Drake et al., 2005). Also, 1500N of compression is 56% below the NIOSH action limit of 3400N, and so would be considered a low injury risk exposure in industry (Ferguson et al., 2005; Waters et al., 1993).

Post-Loading Protocol

Once testing was completed the specimens were removed from the cups and X-rays were taken of the intact and sectioned (through the disc) specimens. The condition of the

specimen was inspected for fractures and posterior accumulation of the dye stained nucleus pulposus material, and then photographed. Radiographic determination of nuclear extrusion and annular bulge were described as herniation since they can not always be distinguished radiographically (Adams and Hutton, 1982). The facets were visually inspected and manually tested for resistance to pressure, and confirmation of fracture was achieved radiographically.

3.4 Summary

The purposes of the three in-vitro studies were to investigate the role of combined loading as a modulator of facet mechanics, failure responses, and potential for pain generation. This section provided the introduction and discussion of the literature and methods relevant to all of the in-vitro studies. The anatomy, sources of pain, and known effects of posture were presented along with common methodological protocols and testing considerations for the in-vitro experimentation. Also, the main issues regarding the use of animal models, post mortem changes, and storage were reviewed.

CHAPTER 4

STUDY #2: THE INFLUENCE OF POSTURE AND LOADING ON INTER-FACET SPACING: AN INVESTIGATION USING MRI ON PORCINE SPINAL UNITS.

Journal Submission: Accepted, Spine.

4.1 Introduction

This investigation was generated from the findings in Chapter #2 where the magnitude of flexion or extension adopted by the participants influenced the resulting axial twist stiffness, amount of passive axial twist motion and the coupled off-axis lateral bend motion. The research question deals with whether the changes in axial twist and coupled motions seen in each flexed-extended posture in-vivo were due to a change in the initial interfacet spacing. The possibility of such a mechanism was suggested by Haberl et al. (2004) and Shirazi-Adl (1994) but could not be directly confirmed in the in-vivo study. The direct quantification of the inter-facet joint spacing occurring in coupled postures has not been previously reported in detail.

Rotation has frequently been identified as a risk factor for the development of low back injury in epidemiological investigations (Kelsey et al., 1984; Manning et al., 1984; Marras et al., 1993). However, the mechanism responsible for this increased risk has not been directly quantified. Increased axial rotation motion is used clinically to indicate instability and has been implicated as a potential cause of low back pain (Haughton et al., 2002; Ochia et al., 2006; Vitzthum et al., 2000). Recently, it has been demonstrated that larger axial twist angles can be achieved when coupled with forward flexion in-vivo (Drake and Callaghan, 2008a– Chapter #2). The range of twist motion permitted in the spine has

been calculated as a function of the distance between the articulating surfaces of the facets using kinematic data and computed tomography reconstructions (Haberl et al., 2004), but the tracking of the facet joint in coupled postures has not been reported in detail. Whether the reason epidemiological studies identify axial twist as a risk factor for low back pain is due to a shift in the load distribution from the facet joint to the intervertebral disc or other spinal tissues has not been determined. Using finite element modelling (Shirazi-Adl, 1994), it was reported that the primary factor in determining the relative effectiveness of the facet joints in resisting rotation was the amount of inter-facet spacing. Changes in facet joint spacing in different postures could lead to errors in diagnosis and subsequent treatments prescribed when magnetic imaging technologies are used to diagnose spinal instability. Can posture or loading history cause a difference in the mechanics of the spine? This question is relevant to address concepts such as instability, spine mechanics, and injury mechanisms, and can be explored using an in-vitro approach combined with current medical imaging technologies. In-vitro investigations permit the necessary control and accessibility necessary to examine the facet joint behaviour when the spine is in coupled postures. Therefore, quantifying the effect of posture and loading history on inter-facet spacing would provide important information in helping to resolve some of these issues.

The primary structure responsible for resisting rotation is disputed in the literature, with research supporting the argument for either the intervertebral disc (Farfan, 1969; Farfan et al., 1970; Krismer et al., 1996) or the facet joints (Adams and Hutton, 1981; Pearcy and Hindle, 1991). Recent work has investigated the passive response of lumbar spine tissues in-vivo to applied axial twist moments in flexed and extended postures (Drake and Callaghan, 2008a– Chapter #2). The normalized angle of passive rotation and passive rotational stiffness

was larger in a flexed posture and was reduced if an extended posture was assumed (Drake and Callaghan, 2008a– Chapter #2) Pearcy and Hindle (1991) concluded from their in-vitro work that although twist loading in a neutral posture may not be sufficient to damage the intervertebral disc, there was an increased ability of the specimens to rotate when a combination of flexion and axial twist moments were applied. This additional rotation may increase the risk of injury to the annulus. However, Gunzburg et al. (1991) reported a decrease in axial rotation when combined with flexion in their in-vitro and in-vivo studies. An investigation on the annular strains and nuclear pressure predicted that the risk of disc damage increased during load combinations of axial rotation coupled with flexion (Schmidt et al., 2007). Fazey et al. (2006) tracked the in-vivo deformation of the nucleus pulposus in response to flexion-extension postures alone and to flexion-extension coupled with axial rotation using magnetic resonance imaging and suggested different asymmetrical loading on the intervertebral disc results from altered deformations due to the addition of rotation. These results suggest a postural mechanism may be responsible for modulating how facet joints articulate thereby affecting the moment resisting capability of the facets and shifting the load distribution between the facets and the disc.

4.2 Purpose

Given the consistent identification of coupled postures as contributing to an increased risk of low back injury, the primary objective of this study was to quantify the effect of posture on the distance between the facet articular surfaces (inter-facet spacing). Since loading history has an effect on the mechanical behaviour of tissue, the secondary objective was to examine the effect of loading history on the posture-facet spacing relationship.

Increased axial twist motion is used clinically to indicate instability and has been implicated as a potential cause of low back pain. Recently, it has been demonstrated that larger twist angles can be achieved when coupled with forward flexion in-vivo. These findings suggest a postural mechanism may be responsible for modulating how the facet joints articulate, thereby affecting the moment resisting capability of the facets and altering the load distribution between the facet joints and the disc. The effects of flexion-extension postures and loading history on the distance between the facet articular surfaces require quantification.

4.3 Hypotheses

1. Applying flexion to the specimens will increase the inter-facet gap distance.

   Findings supported Hypothesis #1.

2. Applying flexion to the specimens will increase the amount of axial rotation permitted.

   Findings supported Hypothesis #2.

3. Repetitively loaded specimens will have increases in flexion-extension and axial twist motions.

   Findings supported Hypothesis #3.

4.4 Methods

Specimen Preparation and Fixation

Four porcine cervical spine segments (C3/4) that were frozen immediately following death, were obtained from a common source to control for age, weight, diet, and physical

activity. The spines were thawed at 24°C for 12 hours and were dissected to osteoligamentous FSU. The preparation and fixation methods used in this investigation were modified from previously published protocols which used aluminum cups, steel wire, and stainless steel screws for fixation (Aultman et al., 2004; Callaghan et al., 2001; Drake et al., 2005; Tampier et al., 2007). The specimens were injected with approximately 0.5mL of a blue dye (Coomassie Brilliant Blue Gmix: 0.25% dye, 2.5% MeOH, 97.25% distilled and deionized water) to aid in the documentation of any nucleus movement upon final dissection of the specimen. Non-ferrous metals and ultra high molecular weight polyethylene (UHMWPE, A-K & Lippert Plastics, Kitchener, Ontario, Canada) were substituted for aluminum to achieve a fixation that was compatible with the magnetic resonance imaging scanner. The specimens were secured into the custom UHMWPE cups using 14 gauge copper wire and brass screws (Figure 4.1).

Loading Protocols

The loading protocols used in this study have been previously published (Callaghan et al., 2001; Drake et al., 2005). Briefly, all of the specimens were preloaded for 15 minutes with a 300N compressive force using a custom servo-hydraulic dynamic testing system (Model 8511, Instron Canada, Burlington, Ontario, Canada) to counter any swelling that had occurred postmortem. The servomotor was programmed to find a zero flexion-extension moment during the preloading phase so the angular position at the end of this phase was taken as the zero position for each FSU and was within the neutral zone defined by Panjabi et al. (1989a) Following the preloading phase, each specimen was exposed to 1500N of compressive force and was flexed and extended to the limits of the neutral zone five times at a rate of 0.5°/s. From the last three moment-angle profiles the boundaries of the elastic zone

were estimated in order to compare the properties of the different specimens and to set the flexion-extension angle for the repetitive loading. The specimens were randomly assigned to receive either preloading only or preloading and dynamic repetitive flexion/extension motions. A limit of 5000 flexion-extension cycles was selected as it has been shown that this amount of loading is highly likely to cause at least the initiation of herniation, and full herniations in the majority of cases (Drake et al., 2005).

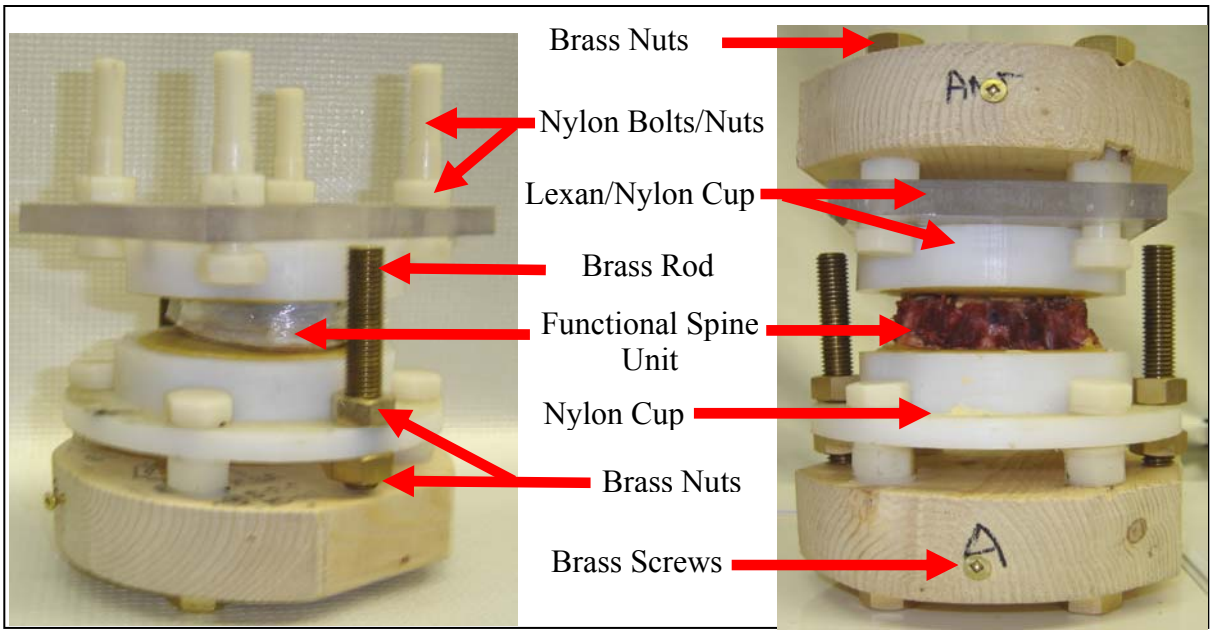

FIGURE 4.1 THE C3/4 SPECIMENS WERE SECURED TO CUSTOM ULTRA HIGH MOLECULAR WEIGHT POLYETHYLENE CUPS USING COPPER WIRES, BRASS SCREWS, AND NON-EXOTHERMIC DENTAL PLASTER. A SECOND FIXATION SYSTEM CONSISTING OF NYLON AND BRASS FASTENERS, PLASTIC ZIP TIES, AND COPPER WIRE WAS USED TO APPLY THE SIX POSTURES.

Upon the completion of the loading protocol using the servo-hydraulic testing system, the specimens were secured in a secondary fixation system using wooden blocks, nylon (polyamide 6/6) hexagon cap screws, washers, and nuts (Spaenaur Inc., Kitchener, Ontario,

Canada). Brass threaded rods, brass screws, 14 gauge copper wires, and plastic zip-ties were used to secure the specimens (Figure 4.1) in the six postures: neutral, flexed, extended, neutral-twisted, flexed-twisted, and extended-twisted.

Following the loading protocol and secondary fixation the amount of rotation in the neutral, flexed and extended postures was measured between the top cup versus the bottom cup with the application of 4.96 Nm (±0.13) of axial twist moment (Digital Torque Gauge DTGHS, Chatillon Force Measurement, Digital Measurement Metrology, Brampton, Ontario, Canada). The resulting angular motion was recorded using a potentiometer (Model 6639S-001-502, resistance 5 KΩ ± 15%, linearity± 2.0%, Bourns, Electrosonic, Mississauga, Ontario, Canada). The resolution of the potentiometer was 0.032°. Both the potentiometer and torque transducer were analog-to-digital converted at 15 samples/s.

Imaging and post-imaging protocols

The secondary fixation system fit the standard human ankle magnetic resonance imaging coil (8 channel phased array, diameter 12 cm), which is a specialized antenna that encircles the specimen and enables high resolution close range imaging (Figure 4.2). The imaging specifications were an axial scan with 0.5mm transverse T1 weighted slices using a 1.5T GE Scanner (Excite II Software, Signa model, R3581, General Electric Healthcare, Milwaukee, WI, USA) following protocols established by the Ontario Veterinary College at the University of Guelph (Guelph, Ontario, Canada). The fixation methods and fit in the coil enabled repeatable positioning within the scanner. The positioning was confirmed using a three plane localizer scan. The twisted postures were obtained by the application of approximately 5Nm of axial twist moment (Digital Torque Gauge DTGHS, Chatillon Force Measurement, Digital Measurement Metrology, Brampton, Ontario, Canada). The order of

the six postures was shuffled and there was a 15 minute unloaded neutral posture recovery phase between each successive scan and posture per specimen. The scans were coded by the technologist to blind the researcher to the data and enable unbiased measurements to be recorded. Once imaging was completed the specimens were removed from the cups and X-rays were taken of the intact and sectioned (through the disc) specimens. The condition of the specimen was inspected for fractures and posterior accumulation of the nucleus pulposus material, indicative of herniation.

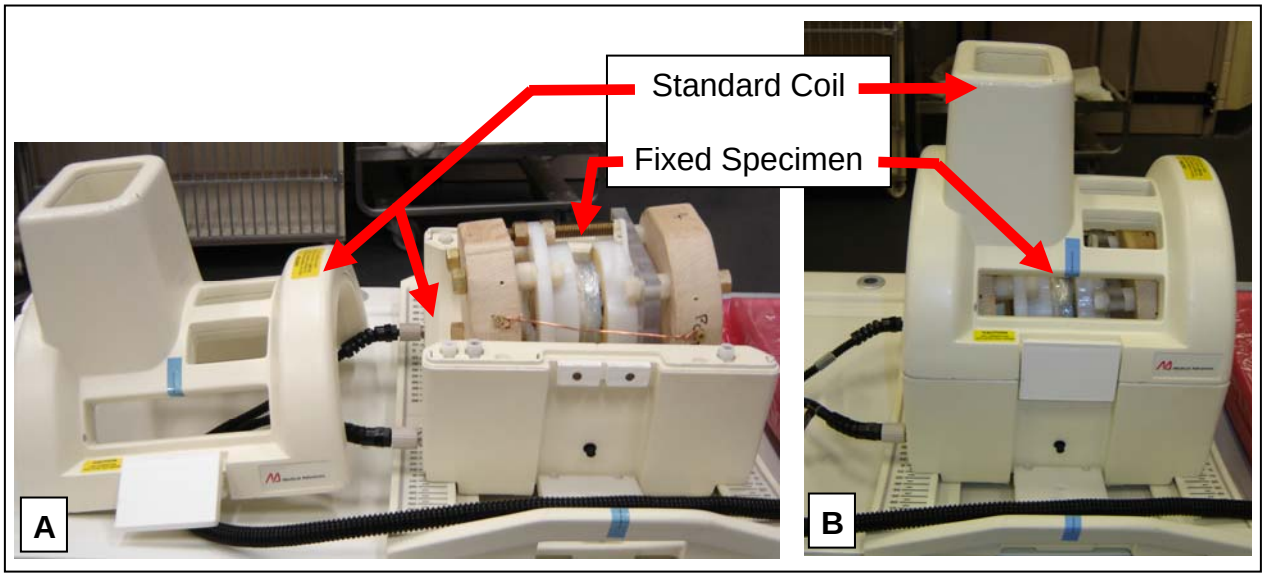

FIGURE 4.2 (A) THE SECONDARY FIXATION SYSTEM FIT THE STANDARD HUMAN ANKLE MAGNETIC RESONANCE IMAGING COIL (8 CHANNEL PHASED ARRAY, DIAMETER 12 CM). (B) THE COIL, WHICH IS A SPECIALIZED ANTENNA THAT ENCIRCLES THE SPECIMEN, ENABLES HIGH RESOLUTION CLOSE RANGE IMAGING.

Data reduction & statistical analyses

The inter-facet distances were measured using Linux Advantage Workstation (software version 4.2, General Electric Healthcare, Milwaukee, WI, USA). Facet gap measurements

were taken at the same depth (slice) for each specimen at the mid-joint level. The distance was measured as the perpendicular distance to the anterior facing facet at the centre of the articular surface of the right facet joint (tension side) as illustrated in Figure 4.3. To normalize the inter-facet distance measurements the percent difference of inter-facet distance was calculated for each posture relative to the neutral posture for each specimen.

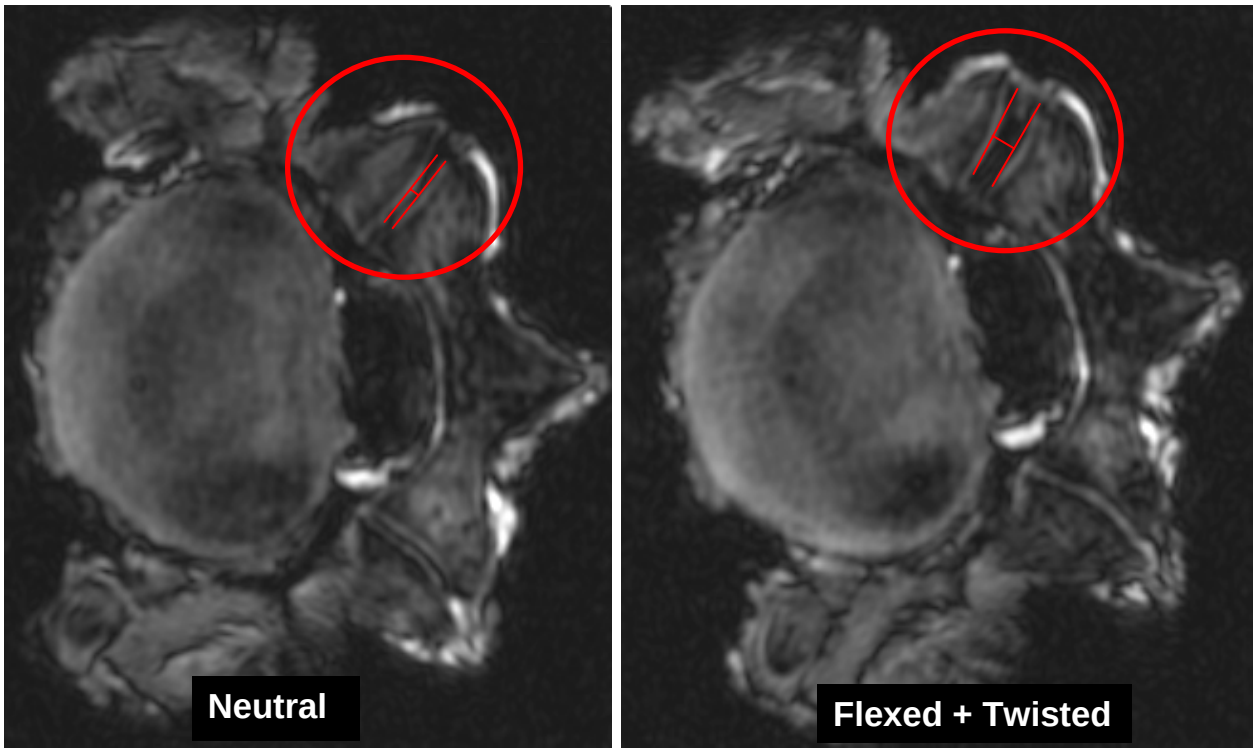

FIGURE 4.3 FACET GAP MEASUREMENTS WERE TAKEN AT THE SAME DEPTH (SLICE) FOR EACH SPECIMEN AS THE PERPENDICULAR DISTANCE TO THE ANTERIOR FACING FACET AT THE CENTRE OF THE ARTICULAR SURFACES OF THE RIGHT FACET JOINT (TENSION SIDE). IN THE IMAGE ON THE LEFT THE SPECIMEN IS IN THE NEUTRAL POSTURE AND IS IN THE FLEXED-TWISTED POSTURE IN THE RIGHT IMAGE.

The peak axial twist angles measured from the application of the axial twist moment and calculated axial twist stiffness values were compared for each of the four specimens in the flexed-twisted, neutral-twisted, and extended-twisted postures. To estimate the cross-sectional area of the C3/4 disc the dimensions of the exposed endplates of the FSU (superior C3 and inferior C4) were measured and used in the area of an ellipse equation: $\pi/4*A*B$ where A is the anterior-posterior length, and B is the medial-lateral width of the exposed disc. Two-way mixed measures ANOVAs (posture) were used to test the effect of load condition and posture on the axial twist angle, inter-facet spacing, and axial twist stiffness variables. A Student–Newman–Keuls post hoc test was used to test any significant main effects of posture. A Least Square Means was used to test any significant interaction effects between posture and load condition. In all statistical tests, the 95% ($p=0.05$) level of confidence was used for rejection of the null hypothesis.

4.5 Results

The normalized percent change of inter-facet spacing was significantly different with respect to posture ($P = 0.0008$) and loading condition ($P = 0.030$) and there was no interaction of these main effects ($P = 0.514$). The largest inter-facet distance occurred when the specimen was flexed and twisted at 138.5% ±54.4 relative to the neutral posture and was smallest in the extended posture (-26.7% ±0; Figure 4.4). The specimens that underwent only preloading had less inter-facet space difference relative to the neutral posture than the repetitively loaded and damaged specimens. The average twist angle was significantly effected by the posture ($P = 0.015$) and loading ($P = 0.049$), while there was no interaction of these main effects ($P > 0.242$). The average twist angles ranged from 7.52° ±1.3 in the flexed

and twisted posture in the loaded specimens to 4.05°±1.0 in the extended and twisted posture in the preloaded only specimens.

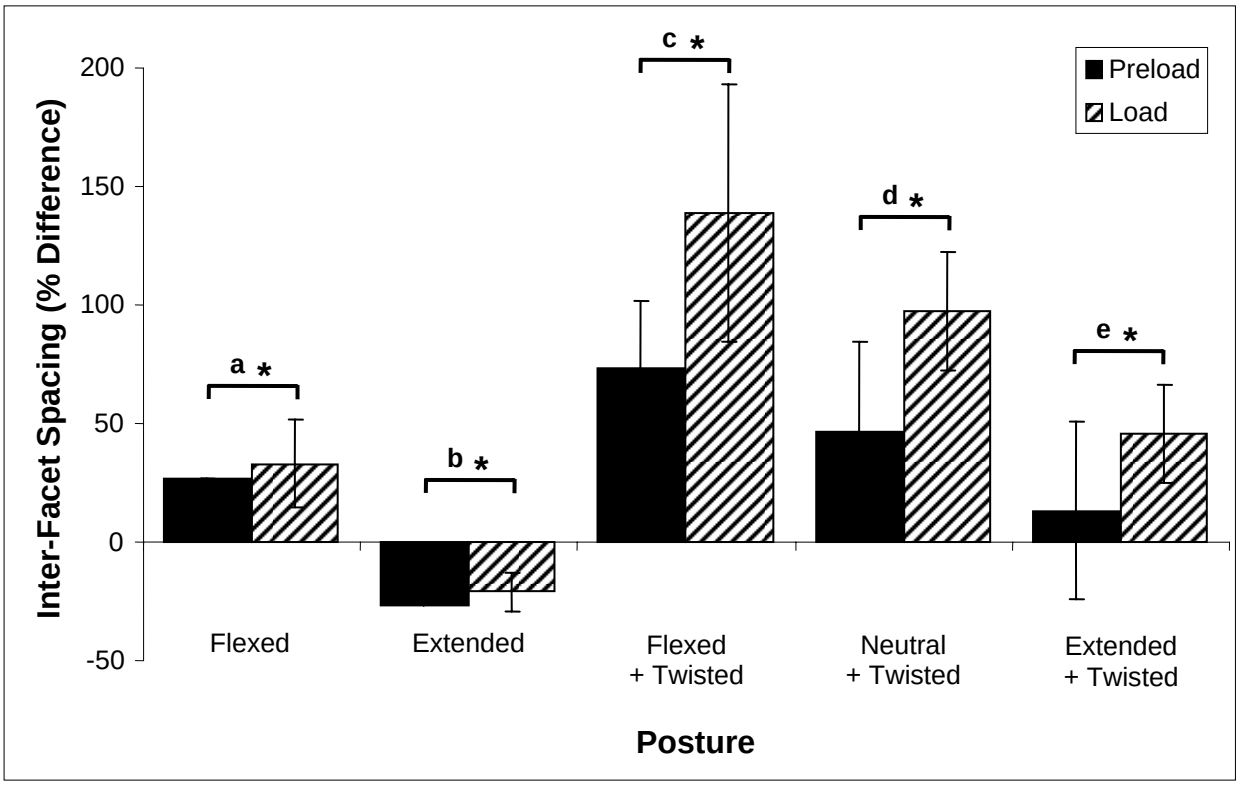

FIGURE 4.4 THE AVERAGE NORMALIZED PERCENT DIFFERENCE OF INTER-FACET DISTANCE FOR EACH OF THE SIX POSTURES RELATIVE TO THE NEUTRAL POSTURE FOR EACH SPECIMEN. SIGNIFICANT DIFFERENCES ARE DENOTED BY DIFFERENT LETTERS FOR THE POSTURES AND BY ASTERISKS (*) FOR THE LOADING CONDITIONS.

Specimens that were repetitively loaded had larger axial twist angles than did the specimens that were only preloaded (Figure 4.5). The twist angle was largest in flexed postures and smallest in extended postures. The average axial twist stiffness was effected by posture ($P = 0.031$) but not loading condition ($P = 0.062$). The stiffness values ranged from 0.65Nm/° ±0.09 in the flexed and twisted posture in the loaded specimens to 1.25Nm/° ±0.30 in the extended and twisted posture in the preloaded only specimens (Figure 4.6). The average stiffness values for the specimens in the neutral posture were 0.96Nm/° ±0.02 and 0.92Nm/°±0.07 for the preload and loaded conditions respectively. There was no statistical difference in the cross sectional area for the four specimens ($P = 0.404$), and the average area was 767.6mm^2 ±87.4. The average boundaries of the elastic zone across the four specimens were 17.25° ±0.96 for flexion and -2.25° ±0.50 for extension. Neither of the two preload condition specimens had any visible migration of the nucleus pulposus and had no damage to the annular fibres. One of the repetitively loaded specimens had herniation initiation and a vertebral fracture, whereas the other had a complete herniation.

4.6 Discussion

Flexed postures and repetitive loading modified facet joint mechanics by increasing inter-facet distance, thereby permitting increased rotational motion in porcine spines. Specimens that underwent only preload conditioning and were positioned in extended postures had the smallest inter-facet spacing and twist angles. These findings support the hypothesis that a postural mechanism is responsible for modulating facet joint articulation.

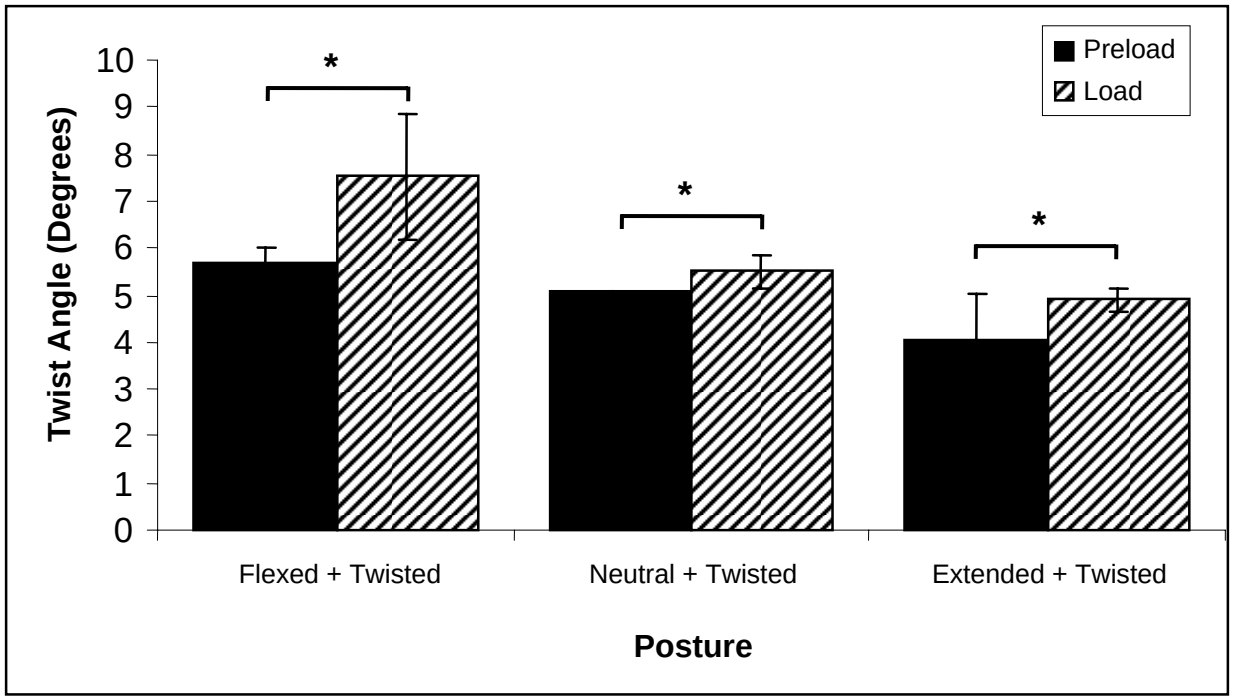

FIGURE 4.5 THE AVERAGE TWIST ANGLE FOR EACH OF THE TWISTED POSTURES FOR EACH LOAD CONDITION. SIGNIFICANT DIFFERENCES WERE FOUND FOR LOADING HISTORY AND ARE DENOTED BY ASTERISKS (*).

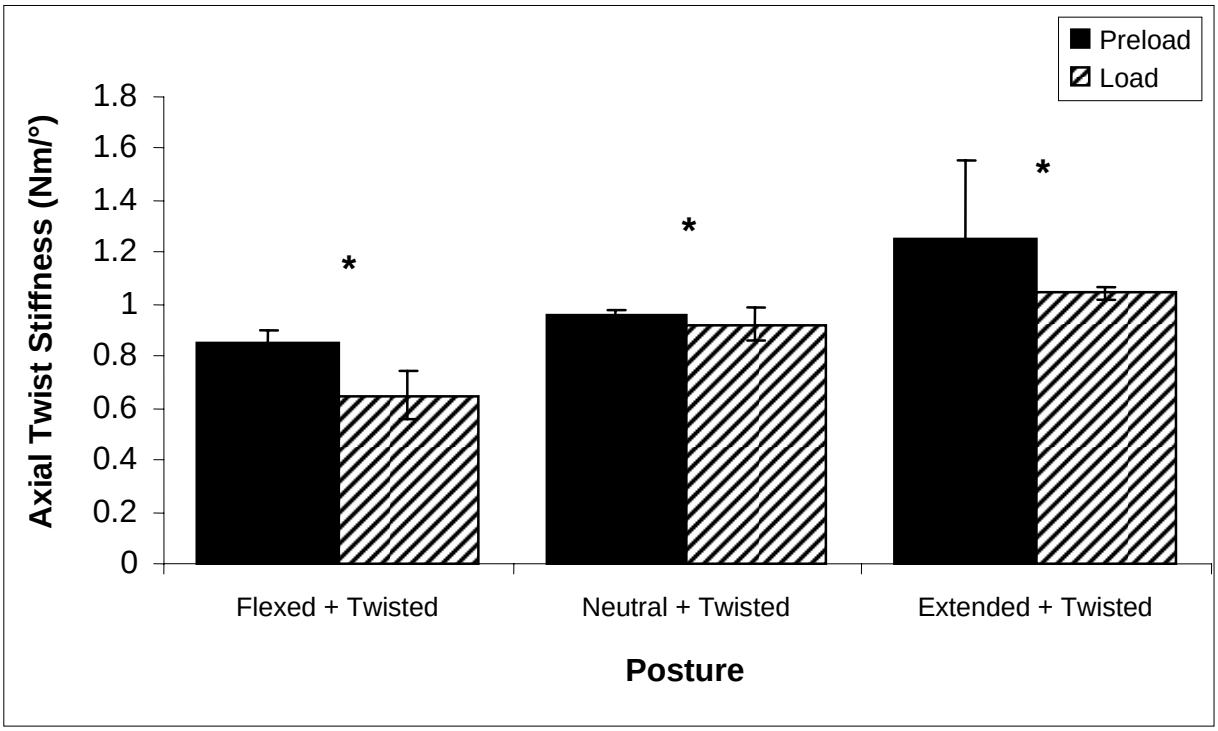

FIGURE 4.6 THE AVERAGE AXIAL TWIST STIFFNESS FOR EACH OF THE TWISTED POSTURES FOR EACH LOAD CONDITION. SIGNIFICANT DIFFERENCES WERE FOUND FOR POSTURE AND ARE DENOTED BY ASTERISKS (*).

This postural dependent joint function alters the moment resisting capability of the facets and will alter the load distribution between the facets and the other passive structures (i.e. intervertebral disc) in coupled postures. The postural effect was even more pronounced when the specimens were first exposed to repetitive loading. The changes in spine mechanics and resulting loading due to axial twist motion and/or moment and loading history may be key to understanding the formation of low back injuries, and eventually spinal instability. This in-vitro work provides the possible underlying spine mechanics that caused the findings of recent in-vivo investigations. Drake and Callaghan (2008a– Chapter #2) found larger

axial twist angles can be achieved when coupled with forward flexion, and Fazey et al. (2006) found the addition of axial rotation to flexion/extension postures caused asymmetrical loading of the intervertebral disc. An investigation on the annular strains and nuclear pressure predicted that the risk of disc damage increased during load combinations of axial rotation coupled with flexion (Schmidt et al., 2007). In the current study it was shown that when the trunk is flexed the facet joints partially disengage resulting in an increased the inter-facet distance. So if the rotation occurs prior to the contact of the facet articular surfaces, as reported from reconstructed animations (Haberl et al., 2004) in flexion there is a reduction in the facets moment and posture resisting capability. Thus, the load resisted by other tissues such as the intervertebral disc and surrounding musculature would increase. When the spine is extended it is clear that the reduced motion would support the opposite effects.

Support for the postural mechanism hypothesis suggested by Drake and Callaghan (2008a– Chapter #2) has been previously reported. Briefly, it has been found that axial rotation caused an asymmetry in back muscle activation patterns, and possibly reduced spinal stability (van Dieën, 1996). The extra motion acquired in non-neutral postures suggests that participants may require higher muscle activation to control spine stability, and other passive structures (such as the intervertebral disc) may be required to resist the portion of the load that the facets are unable to carry until engaged in non-neutral flexion-extension postures. Pearcy (1993) found that when the spine is flexed in-vivo the facet joints permit greater twist motion, but postural muscle action in flexed postures restricts active twist motion. In addition to the increased muscle activation that may be required to assume the non-neutral postures, higher muscle co-activation to maintain spine stability can lead to further increases in spine

compression due to muscle activation (Axler and McGill, 1997; Drake et al., 2006; Granata et al., 2005; Marras and Granata, 1995). There was an increase in the axial twist angle found for specimens that were repetitively loaded, suggesting with injury or diurnal height loss even further levels of activation may be required given the larger resulting motion. The higher muscular demands could lead to increased rates of fatigue, and possibly increases in motor control errors during movement which may leave the passive structures vulnerable to loads beyond their capacities. Axial twist loading has been shown to fracture the facets and accelerate annular injury formation when added to repetitive flexion-extension motions to create combined loading postures (Drake et al, 2005). Lastly, epidemiological reports have identified a higher risk of low back injury with jobs incorporating coupled postures involving twist motion (Kelsey et al., 1984; Manning et al., 1984; Marras et al., 1993) support this suggested postural mechanism leading to injury.

Due to the high cost and limited access associated with the use of a magnetic resonance scanner, the number of specimens was limited in this study. The use of an animal model provides a homogeneous specimen population and control over confounding variables such as age, diet, weight, level of activity, and disc degeneration, but limits the direct applicability to of the results to humans. Although the appropriateness of using porcine cervical spine segments as a model for human lumbar spine segments has been shown (Oxland et al., 1991; Yingling et al., 1999) anatomical, geometrical, and biomechanical, differences between the species may exist. A full justification of the use of a cervical porcine model has been previously documented (Callaghan and McGill, 2001; Drake et al., 2005; Tampier et al., 2007; Oxland et al., 1991; Yingling et al, 1999). In this study the spine mechanics were quantified from static positions that may be more representative of end range

postures which may not be where the injuries are caused during in-vivo coupled motions. Further research using dynamic imaging techniques on humans is required to confirm the results of this study.

To the knowledge of the researchers, the effect of posture and loading history on facet joint articulation has not been previously documented. As stated previously, this inter-facet distance may be a primary factor in explaining the increased risk of injury associated with axial twist motion and moments when combined with flexed postures as the relative effectiveness of the facet joints in resisting rotation and the resulting disc loading are altered. If the in-vivo lumbar spine axial twist angles reported by Drake and Callaghan (2008a– Chapter #2) were decomposed to a single spinal segment, the mean angles of twist would have been approximately 6.6° for neutral-twist, 7.3° for flexed-twist, and 5.0° for extended-twist postures. These values are similar to the angles found in this study for the same type of postures (Figure 4.6). From reconstructed animations (Haberl et al., 2004), rotation was shown to occur prior to the contact of the facet surfaces. The resulting position of the facet joint measured from the MRI images is similar to the calculated findings from a finite element modelling study in that the range of twist motion increased in the postures where an increased distance between the articulating surfaces of the facets was measured (Shirazi-Adl, 1994). The stiffness range of the specimens in this study were lower than those reported by researchers using human lumbar FSUs (Farfan et al., 1970; White et al., 1990), but is within the ranges reported for in-vivo lumbar spine (Drake and Callaghan, 2008a– Chapter #2; McGill et al., 1994).

This investigation of the effects of posture and loading on inter-facet spacing when combined with in-vivo findings suggests that axial twist moment/motion is likely identified

as a risk factor for low back injury due to the changes in the load distribution between the facets and disc/musculature that result from coupled postures. Although the load distribution was not directly quantified in this study, the change in load distribution can be inferred from the change in the spine mechanics. The changes in load distributions that have been shown by Schmidt et al. (2007) in the intervertebral disc during coupled motion loading are likely due to an altered inter-facet spacing. The FSUs that were damaged during the repetitive loading achieved larger axial twist angles for the same moment applied, and had larger inter-facet distances. This study supports the use of increased axial rotational motion clinically to indicate instability (Haughton et al., 2002; Ochia et al., 2006; Vitzthum et al., 2000) as long as a consistent flexion-extension posture is selected for comparison. Both posture and loading history were shown to affect the inter-facet spacing, and thereby the mechanics of the spine when axial twist motion/moment was coupled with various flexion-extension postures.

4.7 Conclusion

The findings from this study support the notion that coupled postures contribute to alterations in spinal kinematics, and by corollary the tissues that resist rotational exposures. These results suggest that the controversy regarding the structure primarily responsible for resisting rotation may be context dependent. When the spine is flexed, there is an increased distance between the facets, and so other passive structures (such as the intervertebral disc) would be responsible for resisting rotation until the facets contact. However, in neutral or extended spine postures, the relatively reduced facet spacing would indicate that the facet joints are the primary structure that is resisting rotation. Further, the inter-facet spacing may account for the passive rotational differences quantified in-vivo for combined postures

reported by Drake and Callaghan (2008a– Chapter #2). This study has highlighted the effect that coupled postures can have on the amount of rotation possible in the lumbar spine, and likewise the inter-facet spacing, which will be valuable in clinical settings.

4.8 Limitations

Due to the high cost and limited access associated with the use of a magnetic resonance scanner, the number of specimens was limited in this study. Further research using dynamic imaging techniques on humans in upright postures is required to confirm the transferability of the results of this study to human responses in activities of daily living.

4.9 Contributions

The findings from this study support the notion that coupled postures contribute to alterations in spinal kinematics, and by corollary, the tissues that resist rotational exposures. These results suggest that the controversy regarding the structure primarily responsible for resisting rotation may be context dependent. When the spine is flexed, there is an increased distance between the facets, and so other passive structures (such as the intervertebral disc) would be responsible for resisting rotation until the facets contact. However, in neutral or extended spine postures, the relatively reduced facet spacing would indicate that the facet joints are the primary structure that is resisting rotation. Further, the inter-facet spacing may account for the passive rotational differences quantified in-vivo for combined postures reported in Chapter #2 (Drake and Callaghan, 2008a– Chapter #2). This study has highlighted the effect that coupled postures can have on the amount of rotation possible in

the lumbar spine, and likewise the inter-facet spacing, which will be valuable in clinical settings. This investigation yielded a manuscript which has been accepted to the journal Spine.

CHAPTER 5

STUDY #3: THE EFFECTS OF PHYSIOLOGICAL AXIAL TWIST ROTATION RATES ON THE SPINE'S ACUTE FAILURE LIMIT USING A PORCINE MODEL.

Journal Article: Submitted, Journal of Biomechanics.

5.1 Introduction

The failure point due to axial twist moments in porcine functional spinal units needs to be quantified. Without failure limits and known injury mechanisms that result from a one-time exposure to twist loading, it is difficult to evaluate possible causes or risk factors of injury. Identifying failure limits would provide context for previously reported experimental results. Also, the acute maximum tolerances are required to determine appropriate exposures in sub-maximal, or repetitive loading scenarios. Yingling et al. (1997) have shown the rate of loading affects the ultimate load and mode of failure in porcine FSUs in acute compressive failure testing. These authors reported the displacement at failure decreased with increasing load rate, and resulted in a change in the observed injury patterns. These authors also stated that to improve the understanding of spinal function, to design appropriate prevention/rehabilitation programs, and to create valid spine models, the modulating factors of the mechanical characteristics of the spine need to be investigated and understood.

The lumbar spine is routinely exposed to twist moments and motions in common work tasks and daily activities. Yet there is a lack of information on the injury mechanisms resulting from acute twist exposures. Considering the anatomical structure of the lumbar spine, it can be postulated that the facet joints function to provide the primary resistance to any applied twist moments. Damage to the facet joints from twist load exposure has been

reported to have the potential to cause low back pain (Schwarzer et al., 1994, Schwarzer et al., 1995b), with the prevalence of painful facets as high as 15% in a working population (Schwarzer et al., 1994). The failure due to applied acute axial twist moment has been investigated previously, but resulted in conflicting conclusions regarding the primary load carrying structure either the facet joints (Adams and Hutton, 1981) or intervertebral disc (Farfan, 1969, Farfan et al., 1970). The methods used to generate the axial twist failure in these studies did not employ smoothly ramped loading from rest to failure with constant and controlled magnitudes of compression and applied twist loading rates. The deficiencies in knowledge on acute axial twist injury mechanisms motivated the current investigation of acute axial twist failure in neutral flexion postures.

To improve the understanding of spinal function, to design injury prevention strategies, and to create valid spine models, the modulating factors of the mechanical characteristics of the spine need to be investigated and understood (Yingling et al., 1997). FSU are viscoelastic structures with a loading response that is dependent on the speed at which the load/rotation is applied (Hutton and Adams, 1982; Hutton et al., 1979; Yingling et al., 1997). Marras et al. (1998) showed that in-vivo axial twist velocities, of approximately 2°/s and 4°/s for an FSU, influences the amount of rotation and the ability to generate moments. The compressive failure load of the lumbar spine has been shown to increase as the loading rate increased (Hutton and Adams, 1982; Hutton et al., 1979). Likewise, Yingling et al. (1997) found the rate of compressive loading affected the failure tolerance magnitude and mode of failure in FSUs in acute in-vitro compressive failure testing across a large range of rates. These differences in mode of failure with loading rate are logical given the amount of energy bone stores during loading is dependent on rate (Nordin and Frankel, 2001).

Therefore, when the bone fails the stored energy is released which impacts the amount of damage sustained by the bone and the surrounding tissues (Nordin and Frankel, 2001). To our knowledge, no study has examined the effects of axial twist rotation rates on acute failure mechanics or injury mechanisms.

5.2 Purpose

To improve the understanding of spinal function, to provide context for the establishment of exposure safety limits, and to create valid spine models, the modulating factors of the mechanical characteristics of the spine needed to be investigated and understood (Yingling et al., 1997). Acute failure due to applied twist loading in the spine needed to be quantified using rates that span the physiological range in-vivo. The primary purpose of this study was to investigate how axial twist rotation rates mitigated acute failure of FSUs in a neutral flexion posture. But a secondary objective was to contribute to the knowledge of twist loading tolerance modification in-vivo.

5.3 Hypotheses

1. Increases in velocity of acute axial twist rotations will correspond to increases in the twist moment recorded at failure.

   Findings opposed Hypothesis #1.

2. Increases in acute axial twist rotation rate will result in increases in failure energy.

   Findings opposed Hypothesis #2.

3. Increased acute axial twist rotation rate will increase the damage incurred by the specimens.

Findings supported Hypothesis #3.

5.4 Methods

Specimen Preparation and Fixation

Thirty porcine cervical spines that were frozen immediately following death were thawed for 12 hours at 24°C and then were dissected to osteoligamentous functional spinal units (FSU), consisting of the C3 and C4 vertebrae with the intervening disc and ligaments. The sectioned ends of each FSU were examined for degeneration and all specimens met the Grade 1 criteria according to the scale proposed by Galante (1967). The endplate area of the exposed superior C3 and inferior C4 of the FSU were measured. The preparation and fixation methods used in this investigation were modified from previously published protocols (Aultman et al., 2004; Callaghan and McGill, 2001; Drake et al., 2005; Tampier et al., 2007). Briefly, fixation was achieved using seven lengths of 18 gauge wire, tightly looped around the laminae and anterior processes. To eliminate any relative motion between the specimen and the aluminum cups, a stainless steel screw was inserted perpendicularly to the endplate in C3 superiorly and C4 inferiorly, two screws were inserted into the anterior-superior aspect of the C3 and the anterior-inferior aspect of C4 in the transverse plane, and approximately one-third of each end of the specimen was embedded in dental plaster. Approximately 0.5cm^3 of a barium sulfate radio-opaque solution was mixed with blue dye (Coomassie Brilliant Blue G-mix: 0.25% dye, 2.5%MeOH, 97.25% distilled water) and injected into the nucleus prior

to testing. This solution has proved effective for permitting the documentation of nuclear material tracking using radiography (Aultman et al., 2004; Callaghan and McGill, 2001; Drake et al., 2005; Tampier et al., 2007). One layer of plastic-backed cloth soaked in saline was wrapped around the specimens followed by a layer of plastic wrap to prevent air drying during testing.

Loading Protocol

The preload and flexion-extension range of motion testing used in this study were based on previously reported work (Callaghan and McGill, 2001; Drake et al., 2005). A custom 3-axis dynamic system capable of applying servo-hydraulic compression (Model 8872, Instron Canada, Burlington, Ontario, Canada), and servo-electric flexion-extension moments (rotational motor, Kollmorgen/Danaher Motion AMK23D servo motor and S20260-VTS servo amplifier, Radford, Virginia, USA), and axial twist moments (linear motors, SCN6-050-150 Dyadic Systems Company, Electromate Industrial Sales Limited, Woodbridge, Ontario, Canada) was used (Figure 5.1). The specimens were preloaded with 300N of compression in a neutral posture for 15 minutes to counter any swelling that had occurred postmortem. The specimens were then subjected to 1500N of compression and the flexion-extension range of motion (RoM) was quantified by flexing-extending each specimen just beyond the neutral zone five times at a rate of 0.5°/s. During both the flexion and extension directions, the values just before the point where the moment-angle curve deviated from the linear section were used to characterize the flexion-extension stiffness of the specimens. These are similar to the boundary of the elastic zone as described by Panjabi et al. (1989a) and the linear region identified by Adams et al. (1980).

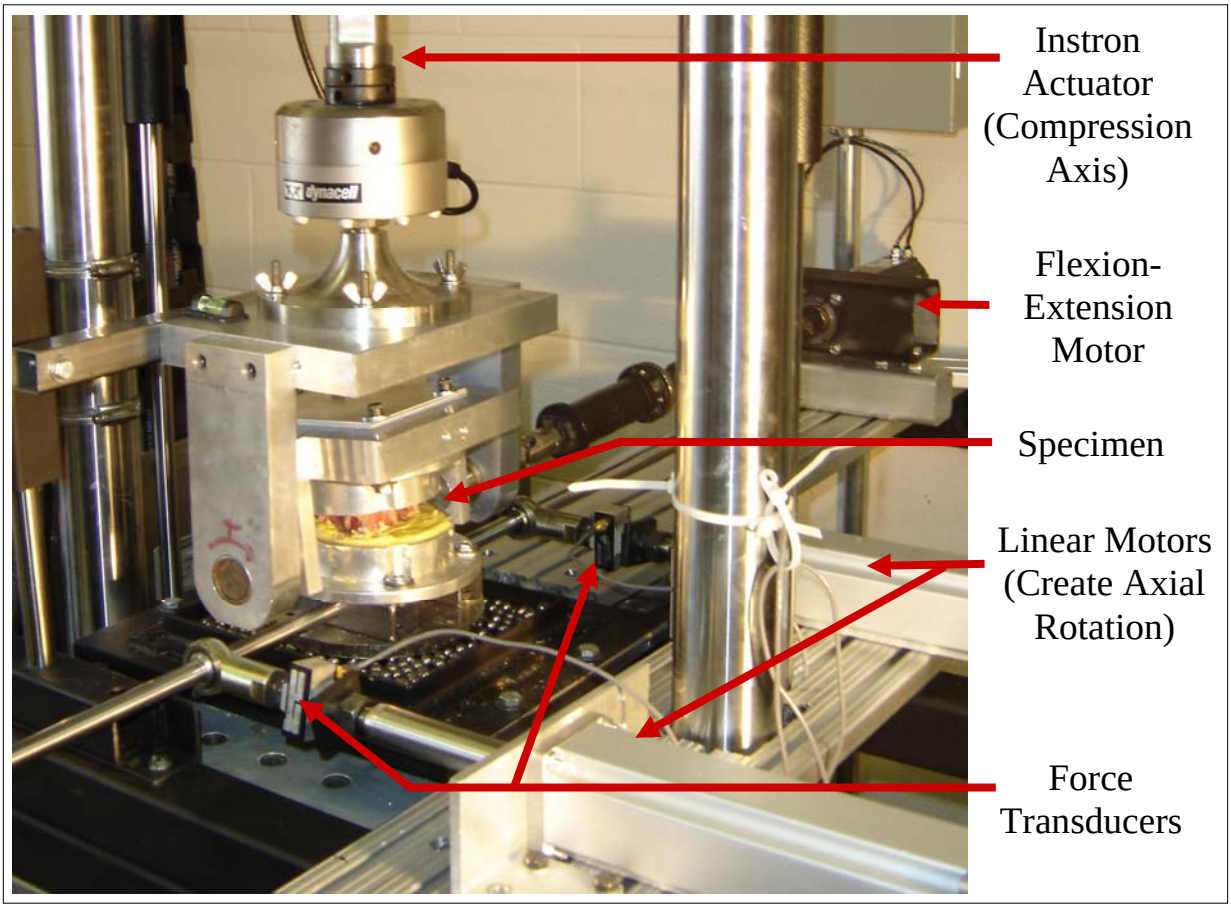

FIGURE 5.1 THE CUSTOM 3-AXIS DYNAMIC TESTING SYSTEM CAPABLE OF APPLYING COMPRESSION, FLEXION-EXTENSION, AND AXIAL TWIST LOADING. THE TWO LINEAR MOTORS ATTACHED VIA STEEL RODS MOVED IN OPPOSITE DIRECTIONS TO CREATE THE RESULTING AXIAL TWIST MOMENTS AND MOTIONS.

A flexion-extension RoM test was performed instead of an axial twist moment-angle to avoid causing additional damage to the specimens following the application of the axial twist rotation. Pilot worked showed that the flexion-extension RoM test did not cause additional damage to the specimens prior to or post-failure and so preserved the comparison of the failure characteristics with observed damage from the failure exposure.

The specimens were randomly assigned to an axial twist rotation rate of 2°/s, 6°/s, or 10°/s, and a failure direction (left or right). The twist rotation rates were selected from angular velocities reported for in-vivo lumbar spine activities. Reducing the whole lumbar spine rates to one FSU resulted in values of approximately 2°/s and 4°/s (Marras et al., 1998), 6°/s (McGill, 1992), 2°/s, 4°/s, 8°/s (Kumar et al., 2003), and 6°/s and 12°/s (McGill and Hoodless, 1990). The two linear motors worked in opposition to create axial twist motion in the specimens. The applied force was measured with two load cells (MLP-500-CO-C, A-Tech Instruments Limited, Scarborough, Ontario, Canada) located between the specimen and each motor. The axial twist reaction moment was calculated about the centre of the vertebral disc. The compressive load for all failure tests was 1500N, which is approximately 14% of the ultimate compressive load tolerance of C3/4 porcine cervical spine segments (Gunning et al. 2001, Parkinson et al., 2005). Following failure, the RoM test was repeated, and then the specimens were removed from the testing apparatus. The FSUs were X-rayed prior to, and following testing and both macroscopic and radiographic damage to the FSUs were recorded. The flexion–extension angles and moments, axial compressive force, axial deformation, and linear motor displacements and forces were A/D converted at a rate of 30 Hz.

Data Reduction and Statistical Analyses

The peak of the force-angle curve for the compression facet was used to identify the ultimate twist failure point (Figure 5.2). The twist failure moment and angle were calculated at this point using the force values generated by both motors creating the axial rotation (Figure 5.3). The failure energy was calculated using trapezoidal integration from the start of rotational loading to the ultimate twist failure point. The specimen pre- and post-failure heights were measured in the zero flexion-extension, zero twisted posture.

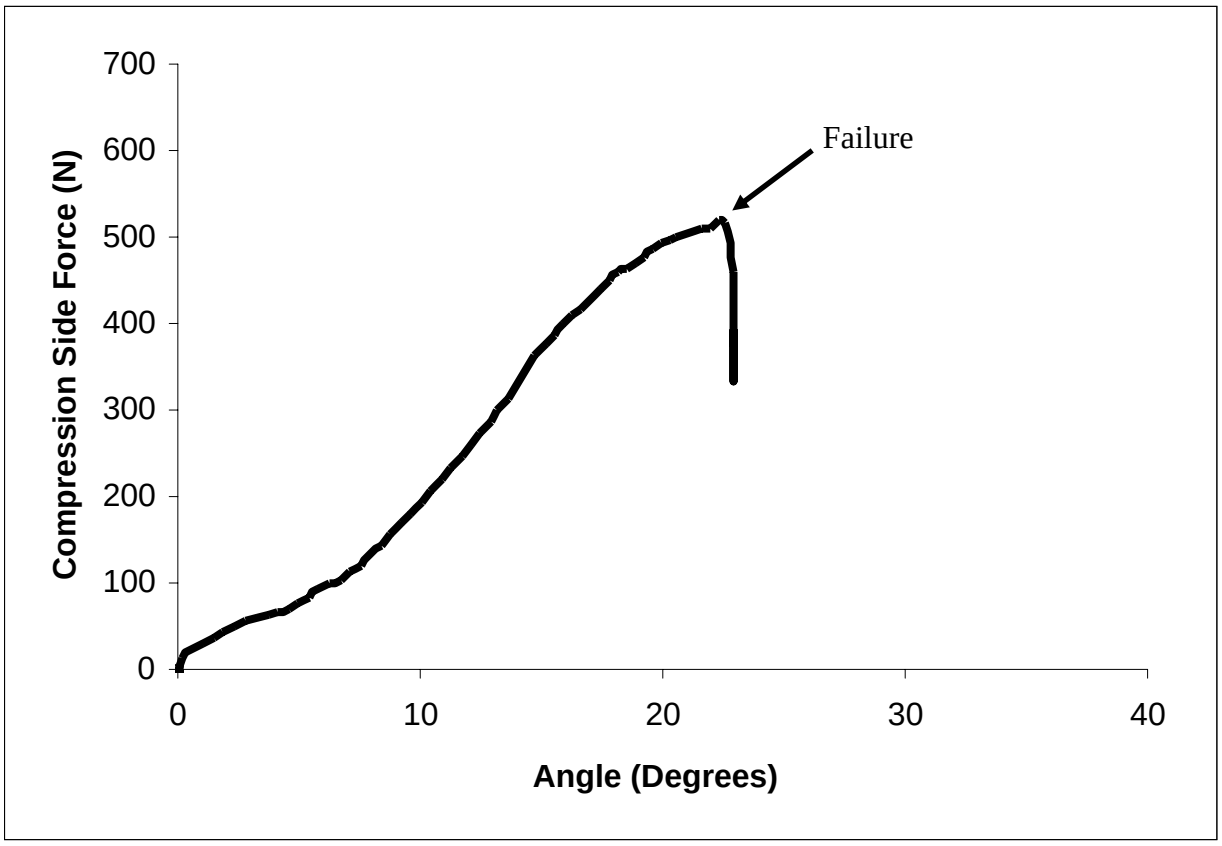

FIGURE 5.2 THE PEAK OF THE FORCE-ANGLE CURVE FOR THE COMPRESSION SIDE WAS USED TO IDENTIFY THE FAILURE POINT OF THE SPINE SEGMENTS.

The average flexion–extension axis stiffness was calculated from the slope of the line connecting the maximum and minimum flexion-extension moment and angle values (the ends of the linear region) for each of the last three of the five repeats in the RoM test to provide the overall average flexion-extension stiffness for the specimen. Load–displacement curves from RoM tests have been shown to become consistent following two repeats (Dhillon et al., 2001).

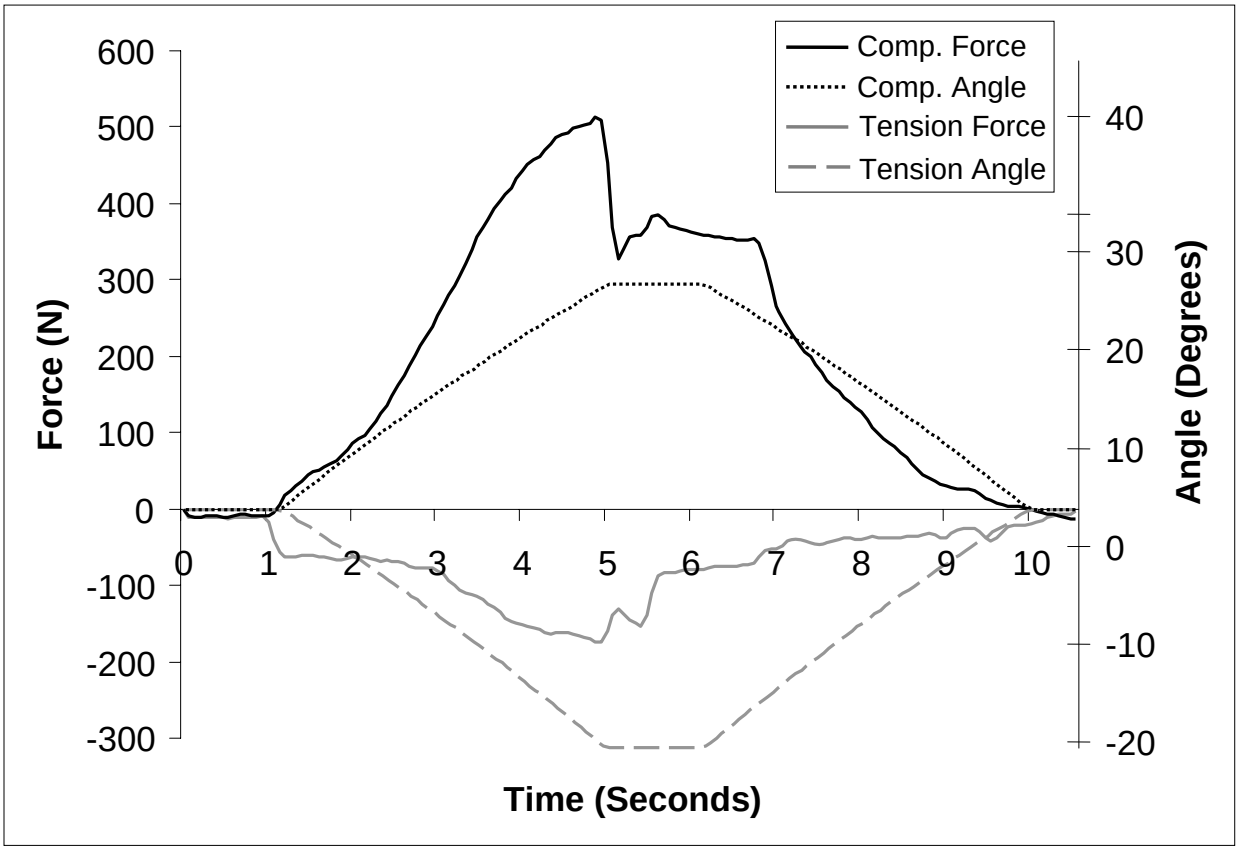

FIGURE 5.3 A REPRESENTATIVE CURVE DEPICTING THE RESULTANT ANGLE AND FORCE TRACING FOR THE FAILURE TRIALS. THE FORCE MEASURED ON THE COMPRESSION (COMP.) AND TENSION SIDES OF THE SPECIMEN AND THE RELATED ANGLE DATA ARE SHOWN.

The facet angles were measured from the post-failure sectioned X-ray using the distinct anatomical feature of the transverse processes to identify the coronal plane, and the bisection of the spinous process to identify the sagittal plane. The facets of the C3 vertebrae were measured since the C3 vertebra sustained no detectable damage. The facet angle was measured between a straight line connecting the edges of the facet and the coronal plane as illustrated in Figure 5.4 (Boden et al., 1996; Panjabi et al., 1993). The facet angle measures were performed three times by one observer from randomized and blinded images. Facet

tropism was calculated as the difference between the left and right facet angles, and the degree of tropism was classified using the scale proposed Boden et al. (1996). The macroscopic damage of the specimens observed visually and radiographically was recorded.

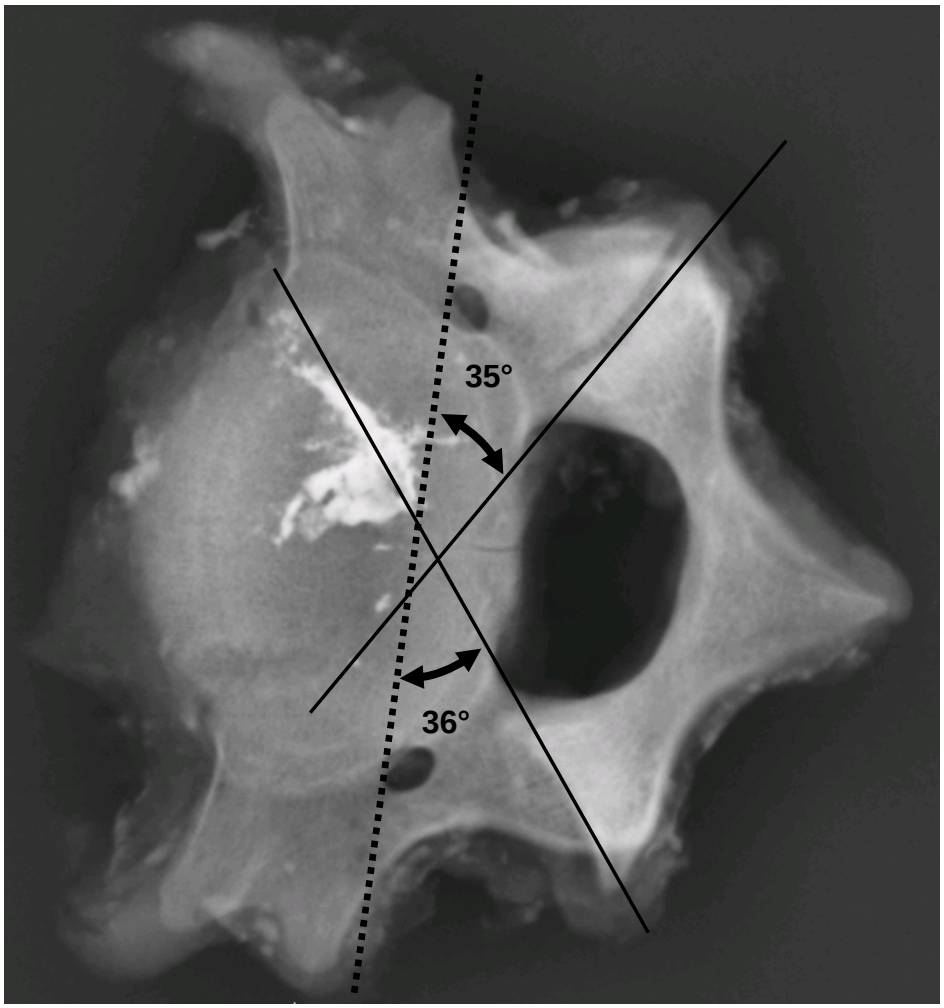

FIGURE 5.4 THE INDIVIDUAL FACET ANGLES WERE MEASURED FROM THE C3 X-RAY USING THE DISTINCT ANATOMICAL FEATURE OF THE TRANSVERSE PROCESSES TO IDENTIFY THE CORONAL PLANE, AND THE BISECTION OF THE SPINOUS PROCESS TO IDENTIFY THE SAGITTAL PLANE (REMOVED FOR CLARITY). THE FACET ANGLE WAS ASSIGNED BETWEEN A STRAIGHT LINE BETWEEN THE EDGES OF THE FACET AND THE CORONAL PLANE. THE FACET ANGLE DIFFERENCE (RIGHT-LEFT) WAS CALCULATED FROM THIS DATA.

The failure twist moment, angle, and energy, pre-post specimen height difference, endplate area, flexion-extension passive stiffness, and facet tropism were analysed using a two-way repeated measures ANOVA (rotation rate, direction of loading). A two-way repeated measures ANOVA (rotation rate, facet tropism) was also used to evaluate if facet tropism effected the failure moments and angles. The individual X-ray facet angles from the left and right side of the FSU were analysed using a three-way repeated measures ANOVA (rotation rate, direction of loading, observer repeat measure). A Student–Newman–Keuls post hoc test was used to test any significant main effects. A Least Square Means was used to test any significant interaction effects from the two-way ANOVAs (between rotation rate and direction of loading or facet tropism). No three-way interactions occurred. All statistical tests were performed at a 95% confidence level. Least square means approach was used to test any significant interactions.

5.5 Results

All of the specimens failed at the junction of the laminae with the superior articular facets of the C4 vertebra. The failure occurred in the facet joint that was compressed due to the applied axial twist rotation. In addition to the facet fractures, there was no observable disruption of the annular fibres of the intervertebral disc, and there was no movement of the nucleus pulposus observed upon visual inspection or on the X-rays in 90% specimens under the 2°/s twist rate (Figure 5.5). Sixty percent of specimens in the 6°/s condition exhibited a facet fracture only, and the remaining 40% had facet fractures with endplate damage without any nucleus pulposus inclusion into the vertebral body. Failure to the facet, endplate, annulus, and inclusion of the nucleus into the vertebral body characterized the 10°/s rotation

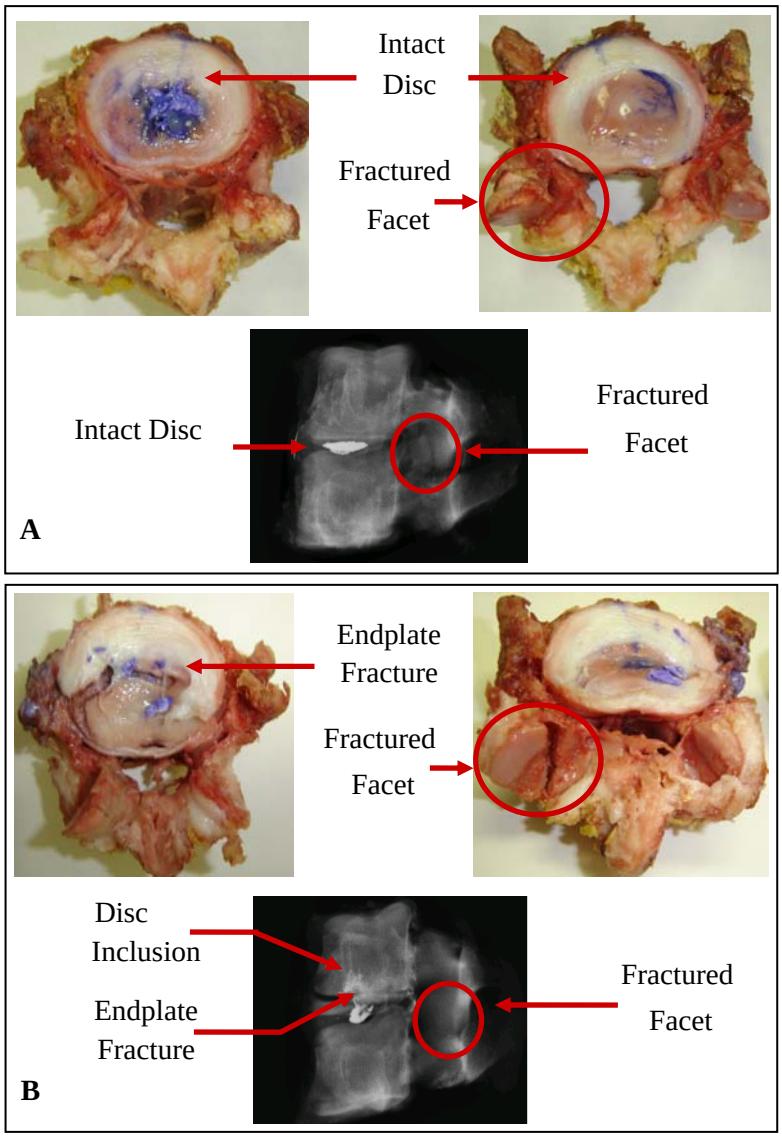

FIGURE 5.5 TYPICAL FAILURE RESPONSES FOR THE LOW AND HIGH PHYSIOLOGIC AXIAL TWIST ROTATION RATES APPLIED IN THIS STUDY: (A) 2°/S, FAILURE TO THE FACET ONLY. THE ANNULUS AND NUCLEUS ARE INTACT, (B) 10°/S, FAILURE TO THE FACET, ENDPLATE, ANNULUS, AND INCLUSION OF NUCLEUS INTO THE VERTEBRAL BODY.

rate in 80% of the specimens. The other 20% of the 10°/s loading group had similar damage without nuclear inclusion. There were no annular fibre or nucleus pulposus injuries observed in the absence of endplate failures characterized by cracks and or separation from the vertebral body. There were no interactions between ($P = 0.436$) or main effects ($P > 0.210$) of rotation rate and direction of loading on the endplate area (7.7 ±0.7 cm^2).

The pre-post failure specimen height difference for the 10°/s rotation rate (3.1 ±0.7mm) was significantly different from the 2°/s and 6°/s twist rates (2.3 ±0.7mm) which were not significantly different. The failure energy was not modified by rotation rate ($P = 0.109$), and was 1438.8 ±461.1Nm° across the three rotation rates. There was no interaction between direction of loading (left or right) and rotation rate ($P > 0.450$), and no main effect of direction of loading ($P > 0.236$) for either the failure energy or the pre-post failure specimen height.

The specimens had greater flexion-extension passive stiffness following the failure of the facet joint compared to the RoM test prior to failure for the 6°/s and 10°/s groups (Figure 5.6, $P < 0.038$). Representative flexion-extension stiffness curves for the 2°/s and 10°/s twist rates in Figure 5.7 illustrate the difference between the RoM tests prior to and following failure testing. The 10°/s twist rate post failure flexion-extension passive stiffness values were 1.62 ±0.61Nm/° which was greater than both the post failure 2°/s and 6°/s twist rates ($P < 0.0001$), which were not significantly different (0.97 ±0.12Nm/°, $P = 0.160$). There were no differences in the initial flexion-extension stiffness values for the three test groups (0.78 ±0.07Nm/°, $P > 0.223$). There was no effect of rotation rate or direction of loading (left or right) for the peak twist failure moment ($P > 0.15$), twist failure angle ($P > 0.110$), facet tropism ($P > 0.360$), individual X-ray facet angles ($P > 0.170$), and endplate area ($P > 0.914$).

The similarity of the failure moment-angle curves observed from the three rotation rates are shown in Figure 5.8.

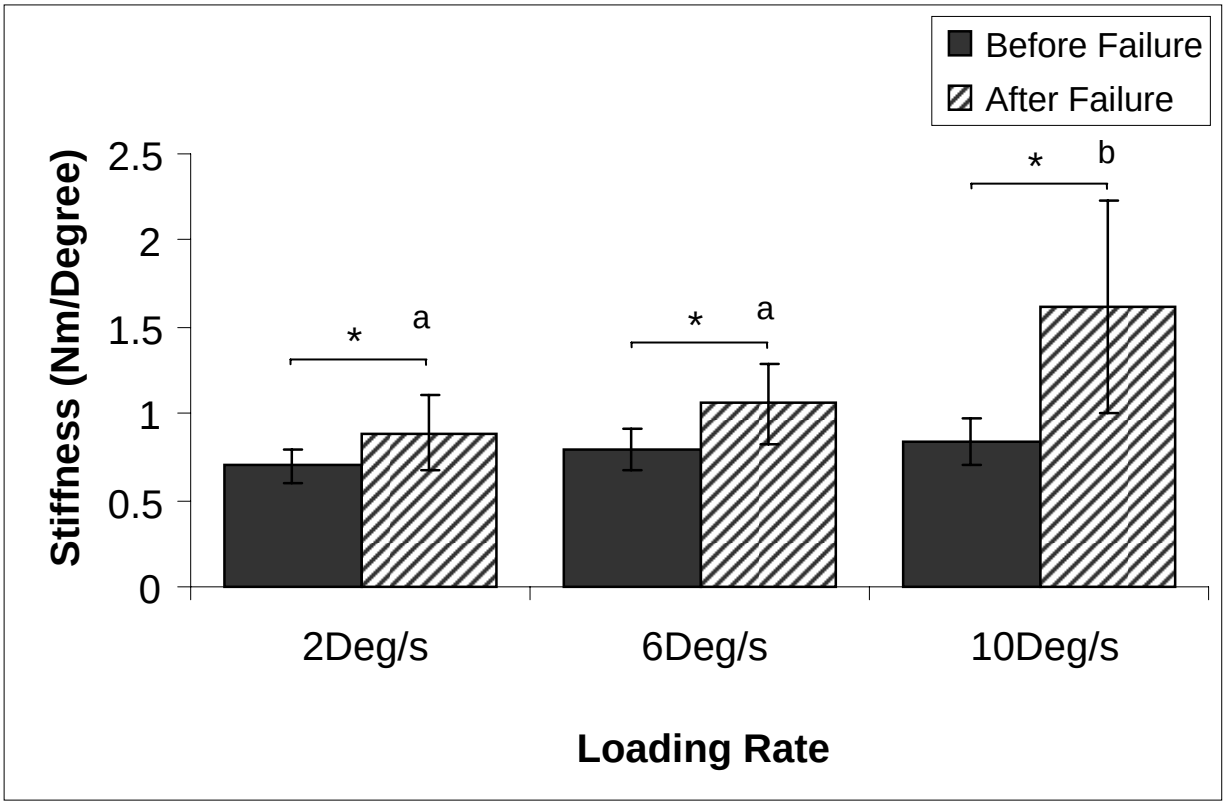

FIGURE 5.6 THE FLEXION-EXTENSION STIFFNESS VALUES BEFORE AND AFTER ACUTE FAILURE, WERE SIGNIFICANTLY DIFFERENT FOR EACH ROTATION RATE AS INDICATED BY THE ASTERISKS (*). THE DIFFERENCES AFTER LOADING ARE INDICATED WITH DIFFERENT LETTERS. THERE WERE NO DIFFERENCES IN THE PRIOR LOADING ACROSS RATES.

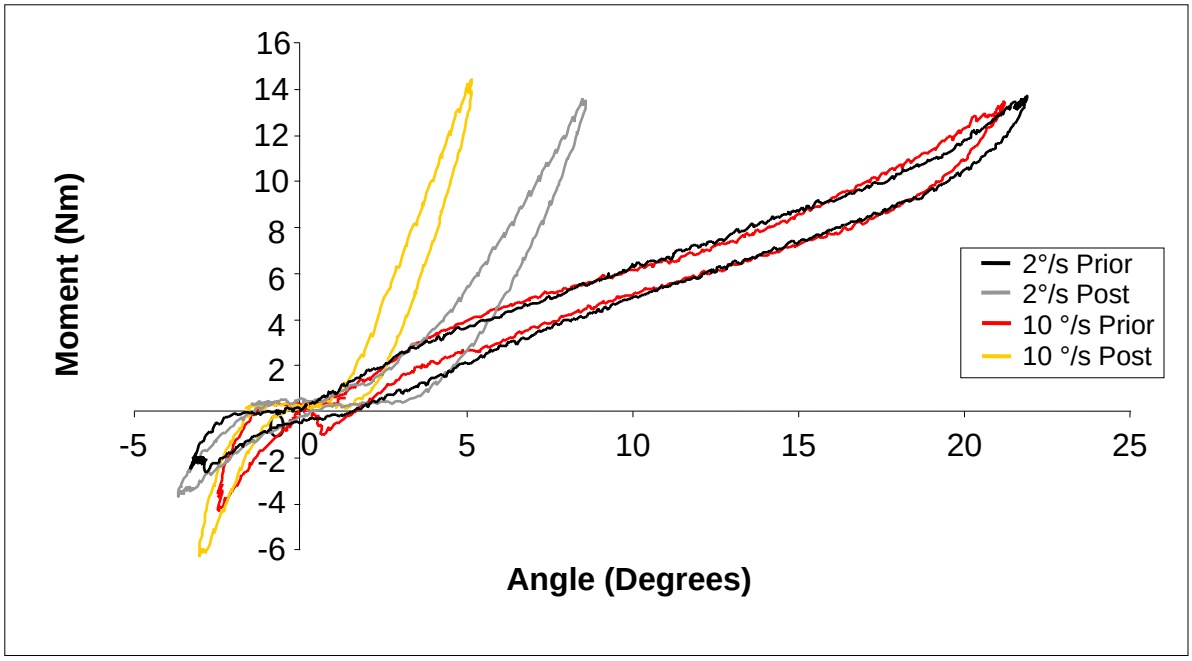

FIGURE 5.7 REPRESENTATIVE CURVES FOR THE FLEXION-EXTENSION RANGE OF MOTION TESTING MOMENT-ANGLE RELATIONSHIP FOR THE 2°/S AND 10°/S ROTATION RATES, BOTH PRIOR TO (BLACK/GREY)AND FOLLOWING (RED/YELLOW) THE ACUTE FAILURE.

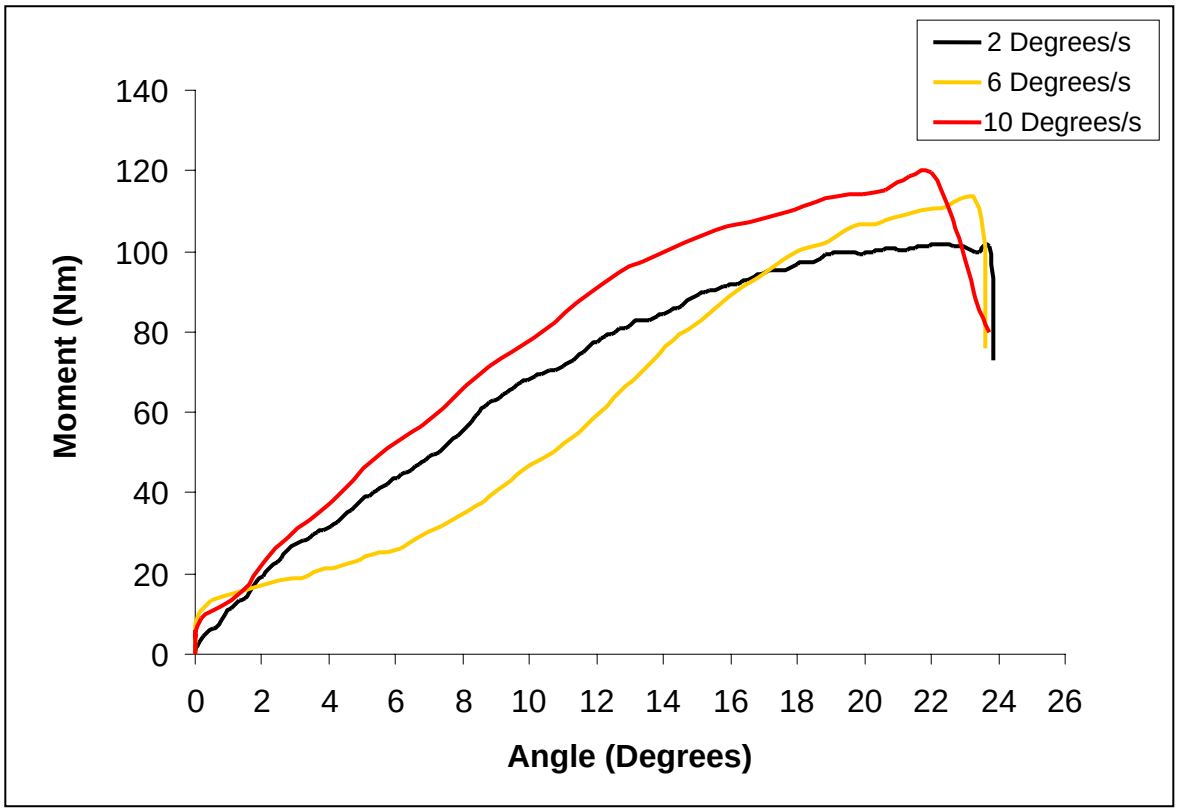

FIGURE 5.8 THE MOMENT-ANGLE RELATIONSHIP FOR A TYPICAL SPECIMEN RESPONSE TO LOADING FROM EACH OF THE THREE ROTATIONAL RATES TO FAILURE IS DEPICTED.

The intra-observer correlation coefficient for the three individual facet angle measurements from the radiographs was >0.94 between each subsequent observation. The average facet angle was 42.5 ±4.3°. The facet tropism measured was 0.3 ±2.5°, ranging from 0° to 5.7° across specimens. The specimens are classified as having "no tropism" since all the measures were less than 6° (mild tropism 6-10°; Boden et al., 1996). There was no effect of facet tropism on the twist failure angle (P = 0.619) or twist failure moment (P = 0.130)

values obtained. The specimens failed at an average of 104.8 ±13.9 Nm of axial twist moment and at an average twist angle of 20.6 ±3.7° across the three twist rotation rates.

5.6 Discussion

The facet joint failed in all trials, regardless of the loading rate applied, suggesting this structure is primarily responsible for resisting axial twist moments in a neutral posture. Despite the absence of influence of the rotational rate on the failure values, there was increased in damage as rate increased. The passive flexion-extension stiffness increased and the specimen height decreased post-failure in the 10°/s rotation rate compared to the lower rotation rates. Unlike the compressive literature, there was no observed effect of twist rotation rate on the failure moment or angle recorded. Yingling et al. (1997) applied five compressive loading rates between 100N/s and 16,000N/s to porcine FSUs, and found the compressive load at failure of the specimens increased between the100N/s and 1000N/s loading rates, while the higher rates had no additional effects. However, the observation of escalating damage of the FSUs with increasing load rate was similar between the acute rotational and compressive loading exposures. If higher rotation rates had been applied, perhaps a similar response to the compressive loading would have been observed. Since the main application of the data was to improve the understanding of tolerance modification of spine failure mechanics and injury mechanisms, the use of physiological rotation rates were critical.

The focus of this study was to investigate whether axial twist rotation rate modified acute twist failure values and mechanisms in a spine joint. The main limitation of this work is the use of animal specimens and the associated lack in the range of degrees of degeneration

(in-vitro versus in-vivo). The porcine specimens are representative of adolescent human spines with Grade 1 degeneration (Galante, 1967), which does not reflect the workforce population. The studies that have investigated acute axial twist loading used human cadaveric FSUs that ranged in age from 18 to 86 years, which introduces large variability in the data. Human lumbar segments would have ideally been used in this study, but suitable human specimens from a similar sample group are difficult to obtain (Alini et al., 2008). Porcine cervical and human lumbar spine segments have functional, geometrical, and anatomical similarities making porcine specimens a reasonable model for human tissues (Oxland et al., 1991; Yingling et al., 1999) while also controlling for age, weight, diet, and physical activity.

Although there is evidence that suggests there is a causal link between facet tropism and the development of disc degeneration/herniation (Karacan et al., 2004), the majority of the research has reported no such association (Ahmed et al., 1990; Boden et al., 1996; Grogan et al, 1997; Karacan et al, 2004; Lee et al, 2006; Masharawi et al., 2004). To the author's knowledge, the relationship between of facet tropism and acute twist loading exposure had not been examined, however there was no tropism in the specimens in this study. Therefore, the relationship between facet tropism and acute twist loading failure variables remains to be determined.

The range of twist rotation at failure from this study fall into the range reported for cadaver specimens in axial twist failure testing of approximately 1° to 22.6° (Adams and Hutton, 1981; Farfan et al., 1970; White and Panjabi, 1990). The differing twist failure angles were obtained from similar aged cadaveric specimens; 27-86 years in Farfan et al. (1970) and 18-77 years in Adams and Hutton (1981). Farfan (1969) reported that intact FSUs, under approximately 427N of compression, failed with 32.7Nm (range 26.0-39.5Nm)

of applied twist moment. Adams and Hutton (1981) reported intact FSUs under approximately 459.4N of compression failed at 18.04Nm (range 12.7-23.4Nm) of applied moment. Based on this data and an assumption of a linear relationship between compression applied and rotational failure moment of FSUs, the porcine specimens in this study would likely be within the range of human FSUs, if the human specimens were also loaded with 1500N of compression during failure testing. From the stepwise application of applied twist moments, via a rotating plate driven by axial compression at a rate of 333N/s, Adams and Hutton (1981) recorded 1.2° average twist failure angle. The axial twist rotation at failure reported by Farfan et al. (1970) for human FSUs was 22.6° which is similar to the 20.6° peak twist failure angle found for the porcine specimens in this study.

Farfan (1969) and Farfan et al. (1970) postulated that annular tears are more likely the result of axial twist than compression loading since this type of IVD damage was found without injury to the bony structures. Whereas Adams and Hutton (1981) concluded the compression facet was the structure primarily responsible for the resistance of applied twist moment and twist motion, with the disc playing a minor role. It has been shown that the IVD can be damaged in the absence of facet failure, but required highly repetitive axial twist rotational loading combined with a static flexed posture (Drake and Callaghan, 2008c– Chapter #6). Further, the highest flexion-extension stiffness following acute rotational failure was 1.62 ±0.61 Nm/°. This is less than the flexion-extension stiffness, approximately 3Nm/°, reported for a repetitive loading protocol known to damage the IVD (herniation) and not the facet joints (Callaghan and McGill, 2001; Drake et al., 2005). Therefore, from the current findings, it appears that in acute axial twist rotation in a neutral flexion posture that the compression facet resists the majority of the moment applied, with the IVD becoming

increasingly involved at the higher twist rates. To assign specific load carriage percentages for acute rotational failure to the facets, ligaments, facet capsule, and IVD, a partitioning study needs to be completed using a similar approach to the current study (controlled compression, rotation rate, and specimen population).

This study has contributed knowledge on the effects of axial twist rotation rates on one-time failure mechanics and the associated injury mechanisms. The generated information can offer context to existing and future submaximal acute and repetitive twist loading literature. The data may be used to improve understanding of spine function and tolerance modification which is necessary to eventually design injury prevention strategies, and create valid spine models. Perhaps the most important outcome of this work was the clarification that the facet joint offers the primary resistance to applied twist moments in a neutral flexion posture.

5.7 Conclusion

There was an effect of increasing the physiological axial twist rotation rate on the specimen damage sustained, and the specimen height and passive flexion-extension stiffness response post-failure. The ultimate twist moment, angle, and energy at failure were not influence by changes in physiological twist rate. This work will be useful in contributing to understanding the mechanical characteristics and injury mechanisms of the spine when exposed to twist loading, and may contribute to tolerance modification knowledge.

5.8 Limitations

The focus of this study was to investigate whether acute axial twist rotation rates modify acute axial rotation failure mechanisms in a spine joint. The acute twist rotation rates selected in this study span the physiologic range reported to occur in-vivo (approximately <10°/s). Although larger acute twist rotation rates could have been examined, they are not representative of the non-accidental axial twist loading exposures seen in industry. The primary objective for investigating acute twist rotation rates was to elucidate the associated twist loading failure injury mechanism.

5.9 Contributions

This study contributes to the understanding of the acute twist loading failure injury mechanisms and provides failure limits for axial twist moment and angle. This study helps to provide context for existing porcine twist loading studies and a foundation for future work on maximal and submaximal twist loading exposures in using acute and repetitive approaches. This knowledge could contribute to establishing twisting loading protocols for human specimens, which may eventually help provide safe exposure limits and safety guidelines in industry. The following manuscript has been prepared on this work and submitted to the Journal of Biomechanics for publication.

CHAPTER 6

STUDY #4: INTERVERTEBRAL NEURAL FORAMINA DEFORMATION DUE TOTWO TYPES OF REPETITIVE COMBINED LOADING

Journal Article: Submitted, Clinical Biomechanics.

6.1 Introduction

Several factors are known to elicit pain from neural tissues in the spine. The most direct method is through neural tissue compression. Compression was confirmed as a pain generating mechanism through the use of an in-vivo rat model (Hubbard et al., 2008; Winkelstein et al., 2002 and 2004). It has been shown that possible causes for compression of the spinal cord and/or nerve roots include herniation through physical compression from extruded nucleus pulposus material, vertebral body height loss, and IVD height loss (Nuckley et al., 2002). Two causes of decreased disc height and vertebral height are water loss (Masuoka et al., 2007) and herniation (Callaghan and McGill, 2001). Repetitive combined loading has been observed to alter loading mechanics, and cause herniations in-vitro (Callaghan and McGill, 2001; Drake et al., 2005). It has been established that non-neutral postures combined with repeated loading are required to cause herniation (Adams and Hutton, 1985; Callaghan and McGill, 2001; Drake et al., 2005; Gordon et al., 1991). While Au et al. (2001) hypothesized that more water is lost during axial twist motions versus other types of movements, the role of axial twist in spine damage remains controversial (Adams and Hutton, 1981; Farfan et al., 1970). Despite these findings, the effect of repetitive combined loading on the integrity of the IVF has not been investigated to the author's knowledge.

In addition to the mechanical mechanism of pain generation (i.e. compression), herniation poses another problem as the outer layers of the annulus are innervated by a network of nociceptors (pain fibres) and mechanoreceptors (Bogduk et al., 1981). So in the absence of complete nuclear extrusion when the nucleus pulposus is still contained in the annular layers, there is the potential for pain generation. Further, nucleus pulposus material has been shown to alter the structure and function of the nerve roots in the absence of mechanical deformation (i.e. compression) by Cornefjord, et al. (1996) and Kayama et al. (1998). The nucleus pulposus is a noxious stimulus that can damage the structure and function of nerve tissue causing pain on contact (Chen et al., 2003; Olmarker et al., 1993; 1995; 1996). The contribution of IVD damage to the development of constricted neural spaces has not been explored.

The size of the neural spaces in the spine are dependent on posture (Harrison et al., 1999, Panjabi et al., 1983), but the effects of posture and load exposure on the nerve roots have not been addressed for the lumbar region. Accordingly, Norwicki et al. (1990) reported no statistical differences in IVF diameter or cross sectional area after loading human cadaveric functional spinal segments (ranging from T12 to S1) with approximately 3000N of axial compression in a neutral posture. Coupled postures (i.e. flexion and axial twist) have been shown to alter range of motion and passive rotational stiffness in-vivo (Drake and Callaghan, 2008a– Chapter #2). Whether pain could be generated by repetitive complex loading by reducing the opening size of the IVF is not known. Since complex loading is typical for physiologic exposures experienced by the spine in-vivo, the effects of this type of loading require attention.

6.2 Purpose

Several structures have been identified in the literature as sources for low back pain. Whether enough compression of the IVF can be elicited to cause pain from two different types of repetitive combined loading is not known. Since combined loading is how the spine is routinely loaded in-vivo, the effects of these type of loading scenarios requires attention. Therefore, the objectives of this study were two-fold: to measure the occlusion of the intervertebral foramina during dynamic loading known to generate intervertebral joint injuries. The outcome measures will be compared to levels of mechanical compression that have been reported to elicit pain behaviours in animals (Hubbard et al., 2008; Winkelstein et al., 2004) to determine whether combined loading is a viable IVF pain generating pathway.

6.3 Hypotheses

1. The neural spaces will be occluded greater when loaded with repetitive axial twist loading.

   Findings opposed Hypothesis #1.

2. A decrease in disc height will accompany an increase in IVF occlusion.

   Findings opposed Hypothesis #2.

3. A decrease in disc height will be a result for specimens with complete herniations of the IVD.

   Findings opposed Hypothesis #2.

6.4 Methods

Specimen Preparation and Fixation

Sixteen porcine cervical spine motion segments (C5/6) were obtained from a common source to control for diet, age, activity level, and weight. Although the appropriateness of using porcine cervical spine segments as a model for human lumbar spine segments has been shown (anatomical, geometrical, and biomechanical) by Oxland et al. (1991) and Yingling et al. (1999), differences between species may exist (Alini et al., 2008). A full justification of the use of a cervical porcine model has been previously documented (Aultman et al., 2004; Callaghan and McGill, 2001; Drake et al., 2005; Drake et al., 2008b– Chapter #5; Tampier et al., 2007). The spines were frozen immediately following death, but thawed for 12 hours at 24°C prior to dissection to osteoligamentous segments consisting of two vertebrae and the intervening ligaments and disc (functional spine unit, FSU). The exposed ends of each FSU were examined and graded for degeneration according to the scale proposed by Galante (1967), and the dimensions measured to provide a measure of endplate area. All FSUs met the Grade 1 criteria. The preparation and fixation method used in this investigation have been previously reported elsewhere (Drake and Callaghan, 2008b– Chapter #5). Briefly, seven lengths of 18-gauge wire were looped around the laminae and anterior processes, stainless steel screws were inserted approximately 0.5cm into the superior and inferior vertebral bodies perpendicular to the endplate. Two screws were inserted anteriorly into the vertebral body of the superior and inferior ends of the FSU in the transverse plane, and these screws and the free ends of the specimens were embedded in dental plaster to fix the FSUs to aluminum cups. Approximately $0.5cm^3$ of a barium sulphate radio-opaque solution was mixed with blue dye (Coomassie Brilliant Blue G-mix: 0.25% dye, 2.5%MeOH, 97.25%

distilled water) was injected into the nucleus prior to testing. This solution has proved effective for permitting the documentation of nuclear material tracking using radiography (Aultman et al., 2004; Callaghan and McGill, 2001; Drake et al., 2005; Drake and Callaghan, 2008b– Chapter #5; Tampier et al., 2007). One layer of plastic-backed cloth soaked in saline was wrapped around the specimens followed by a layer of plastic wrap to prevent air drying during testing.

Loading Protocols

The FSUs were loaded with 1500N of constant compression combined with either (1) repetitive flexion-extension motions (15.5 ±1.4° flexion,-4.75 ±1.6° extension) or (2) static flexion (16.4 ±2.1°) and repetitive left axial twist motion (5.2°). The repetitive axial twist angle was approximately 25% of the twist angle reported at failure in acute loading studies (Drake and Callaghan, 2008b– Chapter #5; Farfan et al., 1970). A custom 3-axis dynamic system capable of applying servo-hydraulic compression (Model 8872, Instron Canada, Burlington, Ontario, Canada), and servo-electric flexion-extension moments (rotational motor, Kollmorgen/Danaher Motion AMK23D servo motor and S20260-VTS servo amplifier, Radford, Virginia, USA), and axial twist moments (linear motors, SCN6-050-150 Dyadic Systems Company, Electromate Industrial Sales Limited, Woodbridge, Ontario, Canada) was used. For the axial twist loading the linear motors worked in opposition to apply axial rotation to the specimens under displacement control (Figure 5.1). The force was measured with two load cells (MLP-500-CO-C, A-Tech Instruments Limited, Scarborough, Ontario, Canada) located between the specimen and each linear motor. The axial twist reaction moment was calculated about the centre of the vertebral disc.

During specimen preloading at 300N of compression for 15 minutes, the custom system found the neutral flexion/extension posture as described by Callaghan and McGill (2001). Prior to loading and following blocks of 1000 loading cycles, the specimens were subjected to flexion-extension range of motion testing and planar radiography to document the IVD condition. Under 1500N of compression, the range of motion tests flexed and extended the specimens just beyond the neutral zone five times at a rate of 0.5°/s (Callaghan and McGill, 2001; Drake et al., 2005). The values just before the point where the moment versus angular position curve deviated from the linear section during both the flexion and extension directions were used as the targets in the subsequent testing. These are similar to the boundary of the elastic zone as described by Panjabi et al. (1989) and the linear region identified by Adams et al. (1980). These flexion and extension limits were used as the position controlled targets in the group exposed to the repetitive flexion-extension motions and as the flexion target for the group exposed to the static flexed position and the repetitive axial twist motion. The specimens were then loaded with 1500N of compression in blocks of 1000 cycles of flexion-extension motions or axial twist motions. Testing was terminated when herniation was determined radiographically or after 10,000 loading cycles had been applied. The flexion-extension angular position and moment, axial compression, axial deformation, linear motor positions, and force transducers were sampled at a rate of 30Hz.

Pressure Measurement

There have been two types of instruments designed to measure neural spaces. The method used by Pintar et al. (1996) was constructed to simulate the effects of mid-cervical burst fracture formation. The measurement component of the artificial spinal cord was constructed using a seven sensor array (spaced 18mm apart to match with each cervical

vertebrae) comprised of $1cm^2$, 110μm thick piezo-electric material plated with nickel-aluminum. The frequency range was reported as approximately 0 to 1GHz, with a dynamic range of 0.0007 to 7 x $10^{10}N/m^2$. The sensing strip was water-sealed using tape and each sensor was individually amplified. To mimic the spinal cord a collagen encased mixture, that was further encased in Plexiglas® tube, was used that had one location available to apply loads and to measure the loading responses. The artificial spinal cord was arranged posterior to the sensors which were adjacent to the anterior surface of the spinal canal, with positioning of the sensors confirmed radiographically. The system was validated through comparison studies using a cat model. The use of a system based on this design would not be appropriate for the given investigation since measurements are only possible where sections of Plexiglas® are removed and the loads are properly aligned with the sensors. More importantly, this method is not applicable to the IVF.

The method described by Raynak et al. (1998) is capable of dynamically measuring occlusions in the spinal canal as well as intervertebral foramina, and was designed to measure neural spaces in a multi-motion cervical spine segment. Two types of systems based on Ohm's Law were developed: a spinal canal occlusion transducer (SCOT) and an intervertebral foramina occlusion transducer (IFOT) are shown in Figure 6.1. The differences between these systems are that the IFOT is smaller and has only one active sensing region. Compliant oversized tubing was used (15.9mm outer diameter x 12.7mm inner diameter) so that the system would deform in conjunction with any occlusion. The tube was filled with 0.9% saline to create an isolated resistive element with less than 3% variation over a temperature range of 23-27°C. A constant 2.0KHz amplitude current of 20μAmp was introduced into the tube via 2mm electrodes fixed to the inside wall near the tube end and

traveling the length of the tube to a ground at the opposite end. Through small holes in the tube, eight sensing electrodes (20mm apart) monitored the voltages at various points along the tube. When taken pair-wise the sensors created seven independent sensing regions. Any deformation of the tube due to an occlusion (thereby changing the resistance) produced a proportional change in the differential voltage measured. The systems were tested using comparisons of output to known degrees of tube deformation. The authors mentioned that the collection of calibration trials may be required to obtain accurate results from these systems.

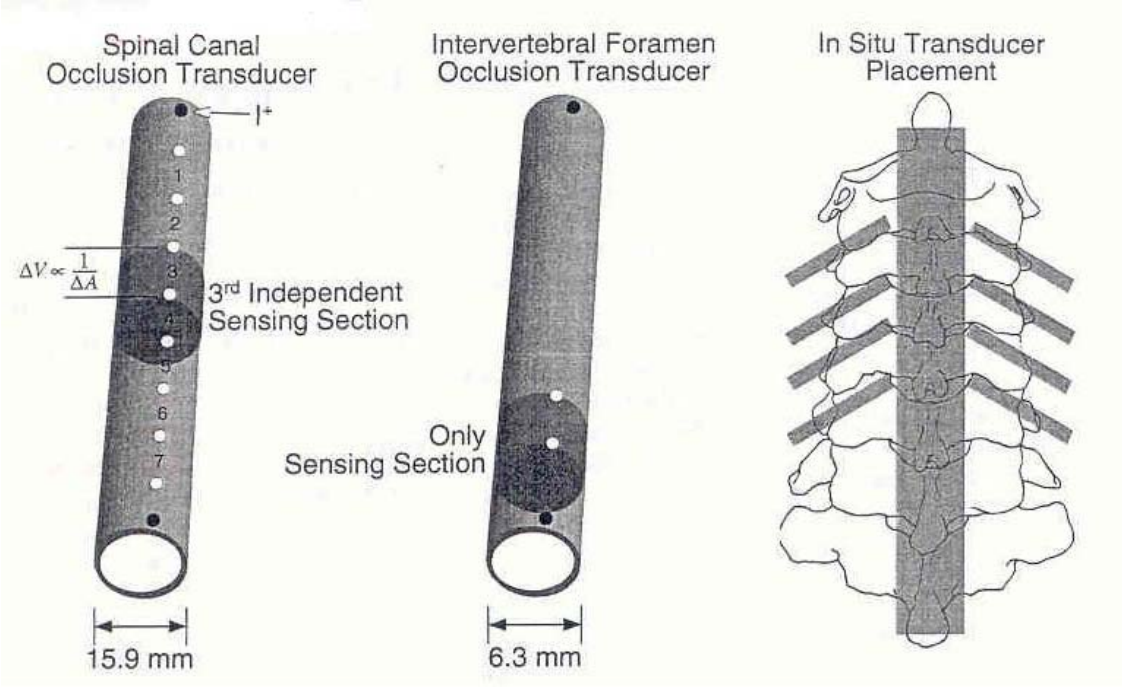

FIGURE 6.1 TWO TYPES OF MEASURING SYSTEMS BASED ON OHM'S LAW WERE DEVELOPED TO MEASURE NEURAL SPACES DYNAMICALLY. THE SPINAL CANAL OCCLUSION TRANSDUCER (SCOT) IS ON THE LEFT, AND THE INTERVERTEBRAL FORAMINA OCCLUSION TRANSDUCER (IFOT) IS ON THE RIGHT. (FROM RAYNAK, G.C., NUCKLEY, D.J., TENCER, A.F., CHING, R.P., 1998. TRANSDUCERS FOR DYNAMIC MEASUREMENT OF SPINE NEURAL-SPACE OCCLUSIONS. JOURNAL OF BIOMEDICAL ENGINEERING. 120, 787-791.)

Pressure was measured in the IVF of both the left and right side of the specimens using a 5cm sensing length of 1.58 mm thick walled flexible plastic tubing (Tygon® R-3603 Laboratory Tubing, Durometer Hardness 55 Shore A, Saint-Gobain Performance Plastics, Aurora, Ohio, USA) with a 4.76mm outer diameter. The sensing ends were sealed using a layer of ethyl-2-cyanoacrylate and silicon. The sensing tubes were connected to a 0-13790Pa (0-2 PSI) dual pressure monitoring system (Honeywell ASCX01DN, Morristown, New Jersey, USA) via a 25cm length of tube with an outer diameter of 3.18mm. The tubes, which were approximately the size of the nerve roots (Figure 6.2), were inserted anterior-laterally through the IVF and terminated in the spinal canal (Figure 6.3) after the nerve roots and spinal cord had been removed.

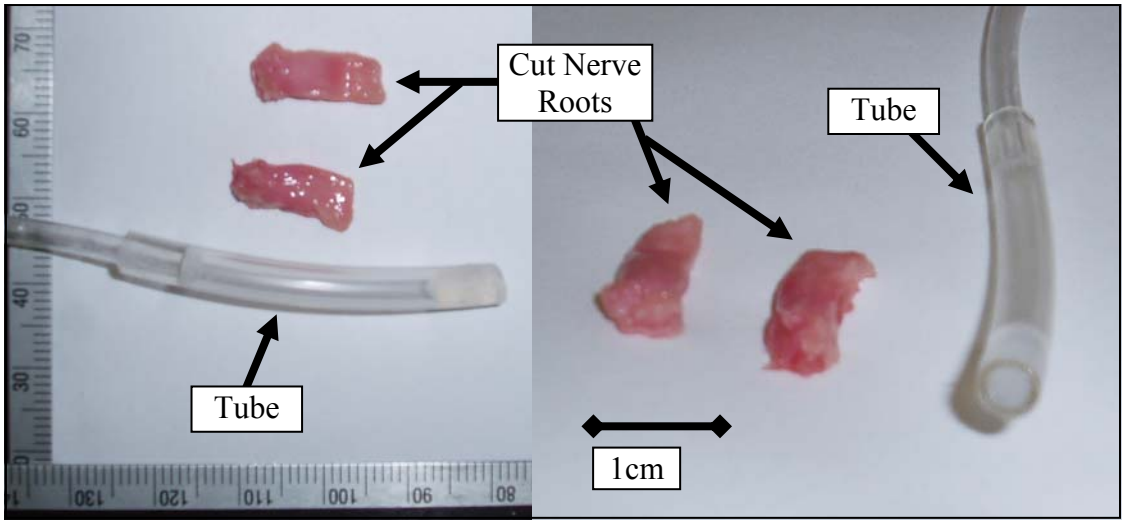

FIGURE 6.2 THE TUBES USED FOR MEASURING THE PRESSURE IN THE INTERVERTEBRAL FORAMINA WERE APPROXIMATELY THE SAME SIZE AS THE NERVE ROOT THAT WAS REMOVED PRIOR TO TUBE INSERTION.

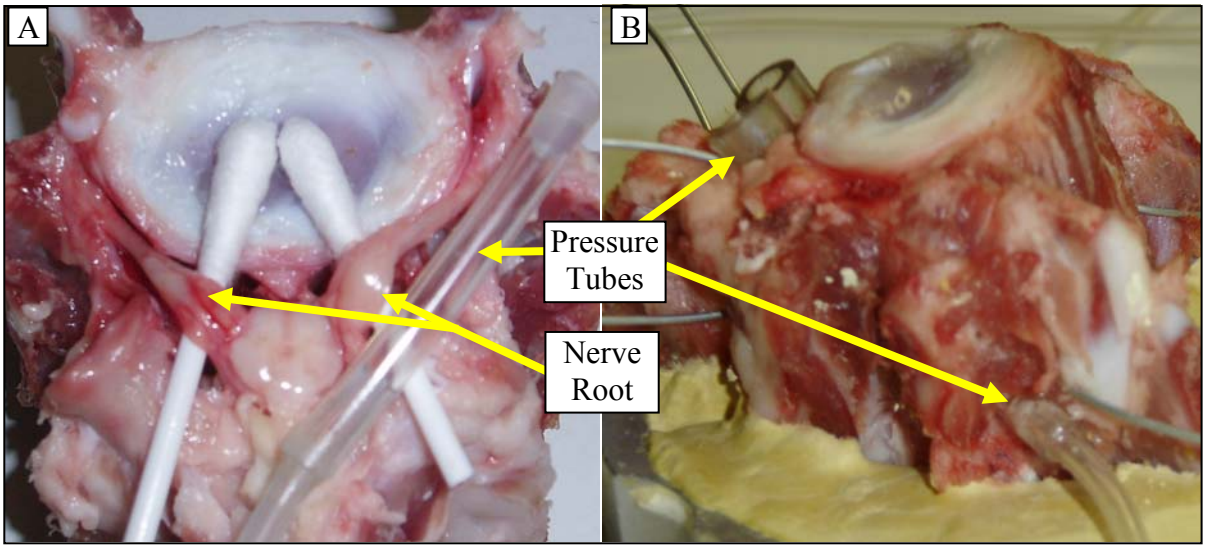

FIGURE 6.3 THE NERVE ROOTS IN THE INTERVERTEBRAL FORAMINA WERE REMOVED AND REPLACED WITH PLASTIC TUBING BILATERALLY (A). THE TUBES RAN THROUGH THE IVF TO THE SPINAL CANAL (B).

The pressure change in the tubes was related to tube deformation using calibration trials prior to and following testing. Approximately 1/3 of a 1cm ball bearing was embedded in a custom plate that was attached to the flexion-extension rotational carriage of the 3-axis testing system. With the carriage in the absolute zero position and the tube taped flat to an aluminum block, the compressive actuator was lowered until the exposed portion of the ball bearing just contacted the tube surface in the approximate centre of the tube. This is approximately the same location that would be in the IVF. The actuator was then lowered to at least five different depths (not exceeding 2.5 mm). The relationship between the deformation of the tube and the pressure output was found using the actuator vertical displacement. A representative pressure-axial deformation curve of a typical calibration trial is presented in Figure 6.4. These calibration trials also enabled the integrity of the tube to be

examined. The outcome measures were compared to known levels of mechanical neural compression, which have been shown to cause pain, to assess viable pain generating mechanisms associated with IVD injury.

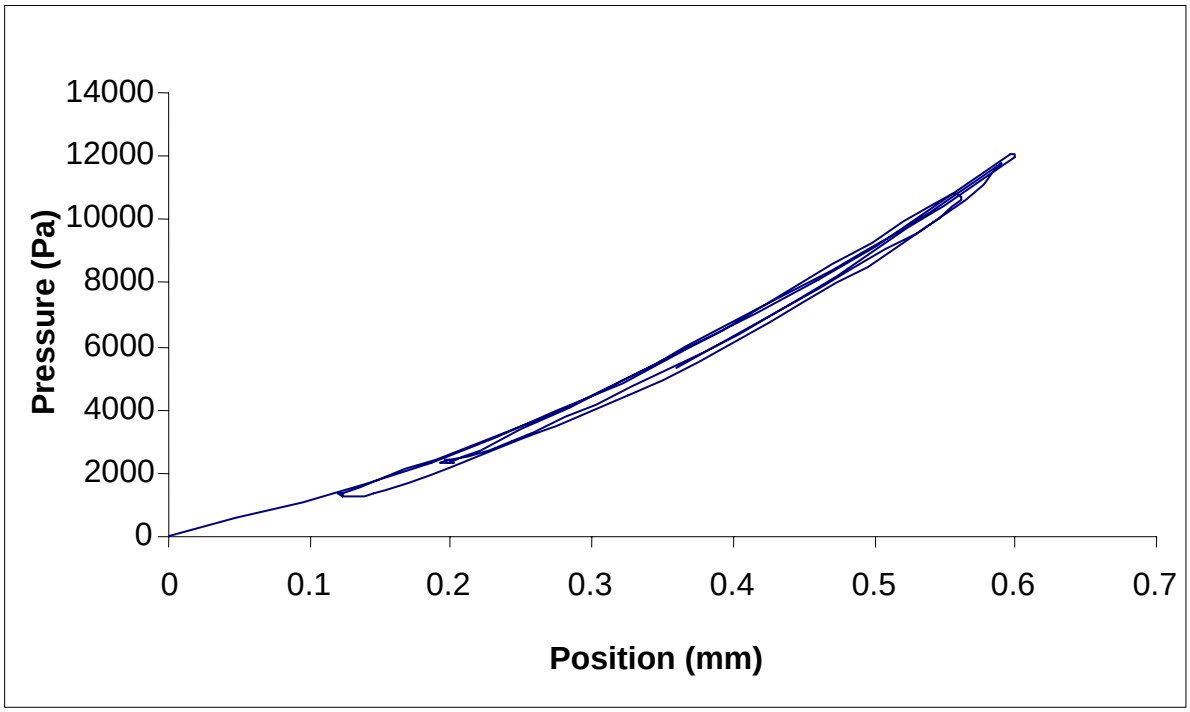

FIGURE 6.4 A REPRESENTATIVE PRESSURE-AXIAL DEFORMATION CURVE OF A TUBE CALIBRATION TRIAL.

Post-Loading Protocols and Statistical Analyses

Following loading, the specimens were removed from the aluminum cups and X-rays were taken of the intact and sectioned FSU (through the disc). The specimens were photographed and inspected for fractures and displacement of the nucleus pulposus. Damage to the cartilage on the articular surfaces of the facets could not be assigned to the loading

since the damage could have been incurred during the post-testing dissection procedures. The X-rays were examined on a cycle by cycle basis, with the initiation, propagation, and posterior accumulation of nuclear material recorded. Both nuclear extrusion and annular bulge were described as herniation since these cannot always be distinguished radiographically (Adams and Hutton, 1982).

The resultant structural failure of the disc and vertebrae were compared to the measured pre-post tube pressure changes and tube deformation, flexion-extension stiffness, and specimen vertical height. The endplate area, flexion-extension limits obtained from the first range of motion test that preceded repetitive testing, and the total cycles applied at loading termination were also compared. One way ANOVAs were performed on all of the measures to compare the two repetitive loading types.

6.5 Results

There was significantly different pressure (pre-post difference) in the IVF of specimens that were repetitively flexed-extended ($P = 0.028$) compared to those that were repetitively twisted (Figure 6.5). The repetitive flexion-extension loading caused a pressure of 6015.5 ±3530.2Pa in the foramina whereas the repetitive axial twist loading caused an IVF pressure of 2792.1 ±1195.6Pa). The calculated tube deformation was 0.29 ±0.18mm and 0.14 ±0.06mm for the flexion-extension and twist loading respectively. The documented failures were different between the repetitively loaded groups. All of the flexed-extended specimens herniated after an average of 5750 ±1065 cycles of loading. The repetitively axial twisted group sustained 9750 ±463 cycles of loading, with two (25%) specimens having no observable damage, one (12.5%) having a facet fracture with no IVD damage, and five

(62.5%) having incomplete herniations. There were no significant differences in the vertical height loss or percent difference flexion-extension stiffness between the groups ($P > 0.199$). The height loss was 3.50 ±0.88mm, and the pre-post percent difference in flexion-extension stiffness (normalized to endplate area) was 54.9 ±23.4%. The endplate area was not statistically different for the two groups ($P = 0.473$), so the overall average endplate area across the 16 FSUs was 7.8 ±0.4cm^2.

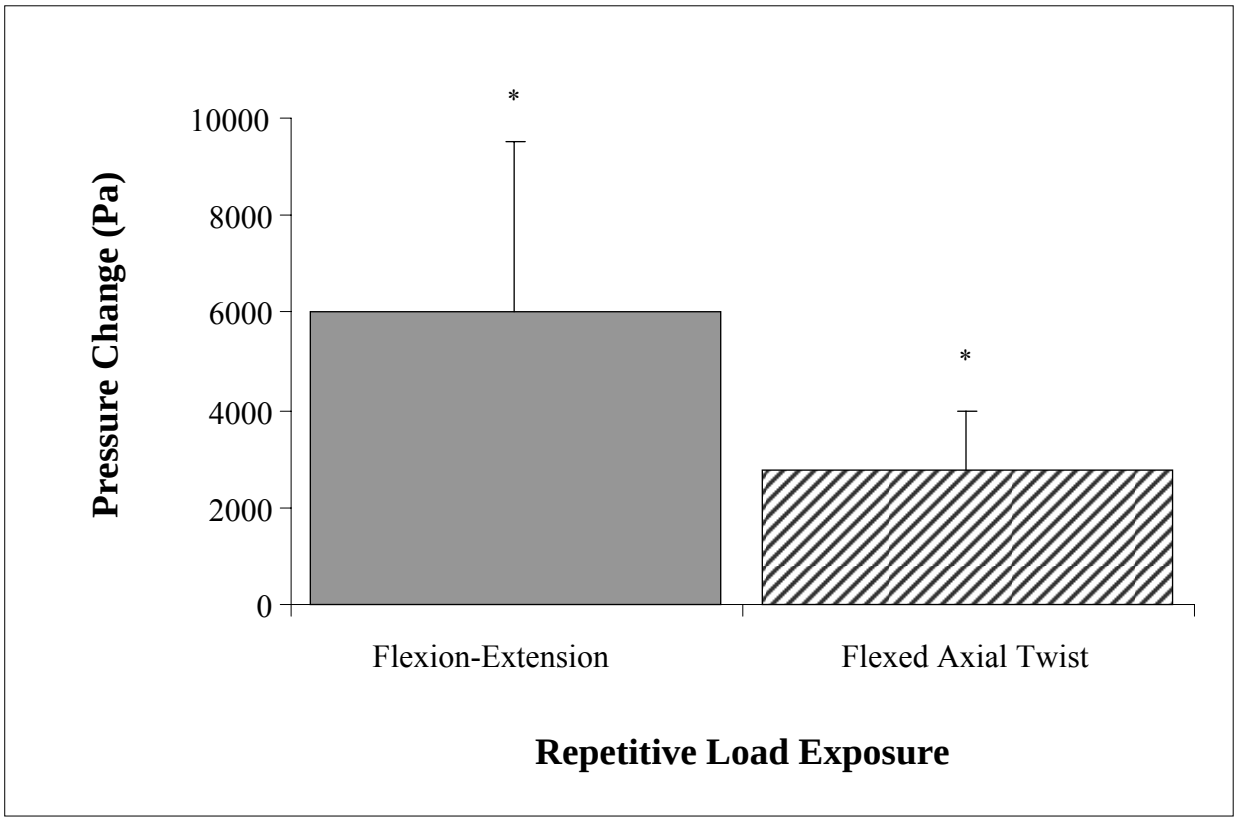

FIGURE 6.5 PRE-POST PRESSURE CHANGES MEASURED IN THE INTERVERTEBRAL FORAMINA UNDER TWO TYPES OF REPETITIVE LOADING: FLEXION-EXTENSION MOTIONS, AND LEFT AXIAL TWIST MOTION COMBINED WITH STATIC FLEXION WERE STATISTICALLY DIFFERENT AS DENOTED BY THE ASTERISKS (*).

6.6 Discussion

Decreased IVF height due to repetitive flexion-extension motions appears to be a viable pain generating pathway that does not directly correspond to simply a loss of specimen or disc height. The repetitive axial twist loading in a static flexed posture may also be a possible pain generating pathway, however additional loading cycles may be required to achieve the same IVF deformation as the flexion-extension repetitive loading exposure. Considering that static flexed axial twist repetitive loading predominantly caused damage to the intervertebral disc (62.5% of FSUs) suggests that twist loading needs to be combined with non-neutral flexion posture to cause IVD damage. These findings resolve the controversy of whether axial twist exposures can damage the IVD in absence of facet damage, and support previous studies that have identified axial twist loading as a possible modulator of injury in combined postures (Drake et al., 2005; Drake and Callaghan, 2008a–Chapter #2; Pearcy and Hindle, 1991; Schmidt et al., 2007).

The nerve root compression force exposures reported in Hubbard et al. (2008) required to initiate (26.29mN) and sustain (38.16mN) pain behaviours in rats were converted to a force/volume value so the outcomes of the current study could be contextualized. Since the average size of the nerve root diameter was not published in Hubbard et al.'s work (2008) the nerve root diameter (1.25mm) was obtained from direct measurement of the C7 nerve roots in a healthy male Winstar rat (300g, Charles River Laboratories, St. Constant, Quebec, Canada). Hubbard et al. (2008) used 0.7mm wide compression platens. In this study, the mean pressures for the repetitive flexion-extension and axial twist loading groups respectively were 44% and 20% of the pressure threshold reported to initially elicit pain behaviours in rats, and were 30% and 14% of the pressure required to elicit the sustained

pain response seven days after exposure to the 15 minute duration nerve root compression (Hubbard et al., 2008). The flexion-extension and axial twist loading group values ranged as high as 70% and 29% of the values required to initiate pain, and 48% and 20% of the sustained pain values respectively. Based on the data obtained, if the exposure duration to repetitive axial twist was increased, it is likely that partial herniations would be completed and higher IVF values would have been recorded. It appears that damage to the disc is a greater threat to IVF integrity than simply decreases in specimen height.

A limitation of the current study was in the lack of the ability to partition the specimen height loss between the vertebrae and the disc. It should be noted that loading type may effect which structures in the specimen lose height. Comparing the damage sustained per number of loading cycles between the repetitive flexion-extension and axial twist motions, it is likely that the loading type is tissue- and rate-dependent. Given the dominance of herniation in the specimens that were repetitively flexed-extended, it is possible that the majority of the height loss was from the disc. It is unknown if the mechanism for height loss is the same for the exposure to repetitive flexion-extension moments and twist moments. To help determine the mechanisms of the decreased IVF height observed in this study, future research should monitor the changes in disc height and specimen height throughout both types of repetitive loading presented. This information may be useful to consider in the diagnosis and treatment of nerve root compression.

Panjabi et al. (1983) tested human cadaveric FSUs in three dimensions and measured the intervertebral foramina, revealing that both posture and degree of disc degeneration alter neural space area. These researchers found that the size of the IVF was 185mm^2 and 108mm^2 for non-degenerated and degenerated specimens respectively, and so disc degeneration may

play a role in the compression of nerve roots during physiologic ranges of motion (Panjabi et al., 1983). This finding is supported by the increased pressure found for the flexed-extended group where 100% of the specimens herniated. Nuckley et al. (2002) flexed the spine in 1Nm increments up to 4Nm for flexion/extension and lateral bending, and up to 3Nm for axial twist, holding each increment for three seconds in cervical spinal segments, while tracking any deformations in the IVF and spinal canal using custom occlusion transducers and an Optotrak motion analysis system. For the lower cervical spine, Nuckley et al. (2002) found the IVF was significantly changed in rotation (5-10%) and combined simulated physiological motions of bending with extension (19-20%). Likewise, in the current findings, the repetitive axial twist exposure had smaller increases in IVF pressure when compared to the repetitive flexion-extension loading. To the knowledge of the authors, this study was the first to measure the IVF during repeated loading protocols, and during the development of known functional spine unit injuries.

It is important to address any loading that can cause herniations since contact of extruded nucleus pulposus material can alter the structure and function of nerve roots in the absence of mechanical deformation (Cornefjord, et al., 1996; Kayama et al., 1998). Cornefjord et al. (1996) postulated that this reaction occurs due to a neurotoxic, vascular, inflammatory, or immunologic type of pathway. The effects observed include decreased nerve conduction velocity (Otani et al., 1997), increases in vascular permeability, and membrane structural changes (Kayama et al., 1998). Also, the outer layers of the annulus are innervated by a network of nociceptors (pain fibres) and mechanoreceptors (Bogduk et al., 1981). Therefore, while the finding that repetitive loading exposures cause IVF mechanical deformation and can potentially elicit pain through nerve root compression, it is important to

consider that this same loading exposure could also trigger pain responses through non-mechanical pathways.

This investigation of the repetitive loading effects on IVF space suggests that repetitive flexion-extension loading exposures are capable of increasing the risk for nerve root compression and the development of pain through two pathways: mechanically and physiologically. Although exposure to repetitive axial twist caused an increase in the IVF pressure, a larger number of repetitions would be required to equal the outcomes from the flexion-extension exposure. In part this may be due to the magnitude of the axial twist angle selected for this study. Whether the increased pressure within the IVF is due entirely to the damage sustained by the intervertebral disc during herniation or it is dependent on vertebral height loss could not be directly addressed.

6.7 Conclusion

Repetitive loading of dynamic flexion-extension motions, appears to be a viable pain generating pathway in absence of distinguishing height loss. Fewer loading cycles are required to cause herniations with flexion-extension loading as compared to the static flexed axial twist repetitive loading. This information may be useful to consider when determining treatment and rehabilitation to address nerve root compression.

6.8 Limitations

The precise location about the circumference of the tube where the compression was occurring was not identifiable, even though the use of the pressure transducers provided a method to quantify IVF deformation during cumulative or repetitive loading injuries. Also,

the insertion of the tubing may have altered the biomechanics of the FSUs, although this is unlikely since the tube walls were only 1.58mm thick. Further, in this study the spine mechanics were quantified from static positions that may be more representative of end range postures which may not be where the injuries are caused during in-vivo coupled motions.

It is possible the plastic tubing used in this experiment may have been affected by temperature. Precautions to avoid such effects were to only test specimens that had been thawed for 12 hours at 24°C so the entire specimen was at room temperature prior to testing. The durometer response of 55 Shore A was reported from using the American Society for Testing & Materials method at 23°C. The tubes are reported to have a maximum recommended operating temperature of 73°C (www.tygon.com). The tubes may have also had an increased hysteresis effect over the course of the testing. However, data was only used from tests that had pre-post calibration trials where the tubes responded to loading and unloading similarly, like the representative curve shown in Figure 6.4.

The last limitation with this study is the use of the tube deformation as a direct surrogate for nerve deformation. There may be differences in the compliance of the tubes compared to the compliance of nerve roots. Although there is information on the outcomes of exposure to various levels of compression and load duration (Hubbard et al., 2008; White and Panjabi, 1990), compliance or stiffness data for nerve roots have not been reported to the knowledge of the authors. The stiffness of the tubes used was on the order of 0.21N/mm, which would have had little resistance to the applied compression load of 1500N. The maximal tube deformations were 0.4mm or 3.5mm of specimen height loss indicating that at most 0.08N or 0.74N of force was resisted by the tubes when peak pressures were measured. Peripheral nerves have been shown to withstand 1.6×10^6Pa of pressure at failure (White and

Panjabi, 1990) which is well below the maximum pressure of 6 x 10^3Pa measured. However, without information of the size of the nerve when in the IVF it is not possible to determine the stiffness of the nerve compared to the tube. If an assumption on size was made to equal the tubes tested of 4.67 mm diameter than the average stiffness for the nerve would be approximately 21N/mm.

6.9 Contributions

This investigation of the effects of repetitive loading on IVF space suggests that repetitive flexion-extension loading exposures are capable of increasing the risk for nerve root compression and the development of pain through two pathways: mechanically and physiologically. Whether the increased pressure within the IVF is due entirely to the damage sustained by the intervertebral disc during herniation or it is dependent on vertebral height loss could not be directly addressed. This work has been submitted to Clinical Biomechanics for publication.

CHAPTER 7

SUMMARY

7.1 Overview

There is a lack of consensus from the limited work that has examined the role of axial twist moments and motions in the development of spine injuries and the generation of low back pain. Taken in concert with the large body of epidemiologic data identifying axial twist moments/motion as risk factors for low back disorders and or pain, this demonstrated the need for this series of thesis studies.

7.2 Common Limitations to All Thesis Studies

The focus of the in-vitro testing in this thesis was on injury mechanisms to the IVD, namely annular destruction and nuclear prolapse/herniation resulting from combined loading exposures. The common limitation across the four studies is the use of populations that lack a range in the degrees of IVD degeneration as a result of the age of the populations. Adams et al. (1982) has stated that there is close association between age and disc degeneration. Drake and Callaghan (2008a– Chapter #2) was an in-vivo investigation that used human participants aged 18-27 years. The animal specimens in the in-vitro experiments, Drake et al. (2008– Chapter #4) and Drake and Callaghan (2008b– Chapter #5; 2008c– Chapter #6), were raised for human consumption and obtained from a meat processing plant supplier.

To compare the amount and quality of degeneration across different populations requires a hierarchical type system. Galante (1967) proposed four grades of disc degeneration

for the spine from macroscopic evaluations. The four grades describing the degree of disc degeneration are (Galante, 1967, page 30):

> *"Grade 1. Normal discs. Annulus free from ruptures and shiny white; shiny white gelatinous nucleus.*
> *Grade 2. The appearance is normal but the nucleus exhibits a more fibrous structure. A clear boundary is present between annulus and nucleus.*
> *Grade 3. Isolated fissures in the annulus. The nucleus is dry and occasionally discoloured. The boundary between the nucleus and annulus is no longer distinct.*
> *Grade 4. Severe changes. Ruptures and sequestrate in both annulus and nucleus. Marginal osteophytes often found."*

The porcine specimens used are representative of adolescent human spines and were classified as having Grade 1 degeneration (Galante, 1967), which does not reflect the majority of the working population. Disc degeneration has been reported to first appear at the ages of 20 in men and 30 in women (Miller et al., 1988). Most people who are between the ages of 30 and 50 will have moderately degenerated discs or discs classified as Grades 2 or 3 (Adams and Hutton, 1982). Research has shown this middle aged population has the highest occurrence of in-vivo IVD disc herniations (Adams and Hutton, 1982; Kraemer, 1995), whereas severely degenerated Grade 4 disc do not herniate. Disc degeneration appears to be a natural process occurring in all human spines, increasing in severity with advancing age (Kraemer, 1995). It has been established that Grade 1 discs herniate less readily than the moderately degenerated group (Adams and Hutton, 1982). However, Adams et al. (1982) reported even human Grade 1 discs can herniate, so although disc degeneration is important to consider, it is not crucial in determining whether a disc will ultimately herniate. In-vitro studies have used cadaver specimens ranging anywhere from age 0 to 96 years of age (Adams and Hutton, 1981, 1982; Farfan et al., 1970; Miller et al., 1988; Nachemson, 1960).

Other contributions to degeneration, apart from age, cannot be determined or controlled for when cadaveric specimens are used. These other contributors include cause of death, diet, lifestyle, activity level, occupation, and general health. In addition to the availability of and access to animal model specimens, the benefit of using such a model is the ability to control for these potentially confounding factors. So while it is important to remember that animal tissues are only a model of human responses, the ability to control the testing population can be highly advantageous.

The relationship between the disc degeneration and loading exposures is not clear. Several authors suggest that if a loading exposure causes damage to the disc the onset of degeneration will be accelerated (Adams and Hutton, 1981; Farfan 1969; Farfan et al., 1970; White and Panjabi, 1990). The problem with accelerated development of disc degeneration is the possibility of developing restrictions in motion prematurely due to the potential for altered mechanics of the disc. However, Nachemson et al. (1979) found that even in severely degenerated IVDs the disc height was the same as their healthy average, and age (and so degree of degeneration) was not generally related to the mechanical behaviour of the FSU. Although there is a lack of consensus regarding the effects of the degree of disc degeneration on the biomechanical behaviours and the injury mechanisms of the IVD, the effects of degeneration were not incorporated into this thesis. Although participants from different age cohorts could have been recruited for Drake and Callaghan (2008a – Chapter #2), the equipment required to perform the passive RoM testing presented challenges that would not have been tolerated by older persons. However, applying the knowledge generated from investigating the biomechanics of the axial twist moment-angle relationship may give insight into the natural and accelerated disc degeneration processes.

An animal model that replicates the responses of human tissue exactly does not exist (Alini et al., 2008). As long as the model selected is appropriate to address the research questions posed, the literature supports the use of animal models. Mechanistic research, which was performed in this thesis, is especially suited for animal model use (Alini et al., 2008). It is unrealistic for every in-vivo study to collect data from every decade of life. Although the participants used in this thesis work were at the low end of the age range of the population to most likely have moderate levels of disc degeneration, age and gender were controlled and so did not confound outcome measures for this research. Future studies should revisit and modify the methodologies so the effects of age and gender on coupled postures and combined loading can be examined. Therefore, in this thesis cervical porcine spine segments (C3/4, C5/6) and male university aged participants were used.

Schmidt et al. (2008) have modelled the response of the L4/5 joint to 1.5Nm, 4.5Nm, and 7.5Nm of flexion-extension, lateral bend, and twist moments. With 500N of compression, they found that the centre of rotation migrates posteriorly towards the compression facet joint with the application of the 7.5Nm of twist moment, but remains centrally located in the disc for a twist moment of 1.5Nm (Schmidt et al., 2008). Interestingly, these authors found that with the same loading magnitudes the facets become unloaded for applied flexion moments. The implications of a shift in the centre of rotation are that the distribution of the shear stresses also shifts (Nordin and Frankel, 2001) which could alter the load distribution between the disc and facet. Also, Haberl et al. (2004) hypothesized that when the facets contact, the twist moment is translated to a coupled moment of flexion-extension and lateral bend. The hypothesized load redistribution may explain the different damage results observed in response to the loading exposures in the current work.

Combining the results of the four studies in this thesis together the theoretical distribution of loading in a flexed posture appears to be primarily the disc and capsular ligaments for approximately the first 3° (Drake et al., 2008– Chapter #4), and an additional 2° would likely be gained once the articular surfaces of the facets contact due to cartilage compression. A further 15° of rotation was present until complete failure occurred at 20° (Drake and Callaghan, 2008b– Chapter #5). The viscoelastic nature of bone will allow the articular process to bend prior to failing. This permits additional twist motion at the facet joint, but the bone acquires microfailure during the process. If the loading continues to increase, the microfailures will accumulate and will eventually lead to failure. This rationale may explain the increases in rotation angle and decreases in rotational stiffness found in Drake and Callaghan (2008a– Chapters #2) and Drake et al. (2008– Chapter #4). In Drake et al. (2008– Chapter #4), with no compression load applied during the imaging component of the study, the possibility for sliding of the facet joints may also contribute to the observed motion. Due to the very large twist moment applied in Drake and Callaghan (2008b– Chapter #5) under a compressive load of 1500N, Schmidt et al. (2008) would likely predict that primarily the facets would be loaded, which is corroborated by all of the specimens incurring fractures to the facet joints. Despite the support for this theoretical loading-failure pathway, quantification of the load distribution and centre of rotation should be performed to verify the model predictions and hypotheses of the mechanism of injury presented.

7.3 Impact

The specific contributions made by each study have been provided within the respective study Chapter. The impact of this work can be determined from the development

of useful knowledge, both new knowledge and the expansion of existing knowledge as generated from each study.

Knowledge Developed

Global Question #1

Does the passive contribution of the facet joints change when resisting twist moments over a large range of flexion postures? How do coupled postures affect facet joint mechanics?

With increasingly flexed postures the following three observations were made: a decreased rotational stiffness in-vivo (Drake and Callaghan, 2008a– Chapter #2), increased in-vivo (Drake and Callaghan, 2008a– Chapter #2) and in-vitro rotation angles (Drake et al., 2008– Chapter #4), and increased in inter-facet spacing in-vitro (Drake et al., 2008– Chapter #4). Coupled postures alter the initial state of facet articulation by increasing or decreasing the inter-facet spacing (Drake et al., 2008– Chapter #4). In a neutral posture the facet joints likely resisted the majority of any applied twist moment, given the injury outcomes (Drake and Callaghan, 2008b– Chapters #5; 2008c– Chapter # 6). The rotational angle was greater in flexed postures (increased inter-facet spacing) than extended postures (decreased inter-facet spacing) for the same applied twist moment (Drake and Callaghan (2008a– Chapter #2; Drake et al., 2008– Chapter #4). These findings support the postulate that there is a postural mediated mechanism that governs the load distribution between the facet joints and other

structures of the spine (i.e. IVD, ligaments). However, the magnitude of change in load distribution remains to be quantified.

This is supported by the development of partial herniations in specimens that were exposed to 10,000 cycles of 5.2° rotational loading while in a static flexed posture (Drake and Callaghan, 2008c– Chapter #6), as compared to the failure patterns achieved after 10,000 cycles of 1.5° rotational loading while in a neutral posture (Liu et al., 1985). Liu et al. (1985) reported failure to occur in the endplates, facets, laminae and capsular ligaments, but not the disc. If the load distribution was unchanged, the higher rotational displacement should have caused the specimen to fail in less cycles of loading, and the location of failure should not have changed.

Global Question #2

Does the velocity of axial twist rotation affect injury outcomes, and twist angle or moment at failure?

Only one study in this thesis examined the response of the spine to different twist rotation rates (Drake and Callaghan, 2008b– Chapter #5). Controlling for the compression exposure and the velocity of rotations made the exposures repeatable, but limited the applicability of the findings. From Farfan (1969) and Adams and Hutton (1981) it is difficult to determine how much of the load resisted was due to the applied twist moment or compressive load since both varied (Table 3.1 and 3.2). This research was primarily a mechanistic investigation of load rate, but the outcomes may contribute to the establishment

of human tolerance limits for twist loading exposure. The three physiologic loading rates were not shown to affect the twist failure moment or angle in neutral flexion/extension postures.

Global Question #3

Is it possible for combined loading to have the ability to cause enough FSU deformation to generate pain?

The disc has relatively low innervation in comparison to the richly innervated facet capsule and vertebra (Cavanaugh et al., 1995; Cavanaugh et al., 1996). Consider the discussion above regarding the changes in load distribution caused by combined loading that includes twist moments, and the accompanying shift of injury location from primarily the bony elements of the spine to the disc. The initial response to this question may be no since the damage was moved to the less innervated structure. Although the disc is relatively un-innervated, the outer layers of the annulus are innervated by a network of nociceptors (pain fibres) and mechanoreceptors (Bogduk et al., 1981; Cavanaugh et al., 1995). Pain would be directly generated as the nucleus pulposus disrupted the annular fibres in the process of herniation. If the nucleus was extruded into the spinal canal, then pain could also be generated through direct compression of the spinal cord or nerve roots. Additionally, the nucleus pulposus has been shown (Cornefjord, et al., 1996; Kayama et al., 1998) to alter the structure and function of the nerve roots in the absence of mechanical deformation (i.e.

compression) or injury (i.e. herniation). So the nucleus pulposus is a noxious chemical stimulus that elicits pain from nerve tissue.

Compression of the nerve roots through a decrease in IVF space (acquired IVF stenosis) is documented to be caused only through decreases in disc height. Decreased IVF height due to repetitive flexion-extension motions appears to be a viable pain generating pathway that may not directly correspond to simply a loss of specimen or disc height (Drake and Callaghan, 2008c– Chapter #6). This is new evidence for combined loading to generate pain through FSU deformation. The objective of many traditional treatments for acquired nerve root compression (stenosis), are centred on restoring lost disc height (Borenstein, et al., 2004). The reasoning presented is that an increase of disc height will alleviate the source of the nerve root compression (Borenstein, et al., 2004). Unfortunately, nerve root compression caused by repetitive flexion-extension motions type may not be alleviated through the traditional disc height increasing strategies. Currently, does not directly appear in the classification list for lumbar spinal stenosis (Borenstein, et al., 2004). It is important to remember, that before any changes are made to diagnostic protocols, the limiting factor of the current study, an inability in partitioning specimen and disc height loss, needs to be addressed, and human specimens and people examined.

7.4 Global Conclusions

This collection of studies was focused on determining whether altered load distribution in the spine, specifically the IVD and facets, in response to applied axial twist moments (when added in combination with one and two axes of additional loading) was

occurring, and to examine how these modes of loading can contribute and/or alter the development of injury and pain.

7.5 Future Research Directions

Important implications for clinicians, researchers, and ergonomists have been generated from the studies in this thesis. Building on the knowledge developed in this thesis, new research questions can be formed. These important next-step research questions include: What is the magnitude of the load distribution shift occurring between the facet and disc for different loading exposures (acute/repetitive; single/multi-axis) (Drake and Callaghan, 2008a–Chapter #2; 2008c– Chapter #6; Drake et al., 2008– Chapter #4)? Does a critical angle of flexion exist that is required to cause this shift to occur (Drake and Callaghan, 2008a– Chapter #2; 2008c– Chapter #6; Drake et al., 2008– Chapter #4)? How does flexion-extension posture modulate acute twist moment tolerance and failure outcomes (Drake and Callaghan, 2008b– Chapter #5)? How is the specimen height loss partitioned between vertebra and the IVD (Drake and Callaghan, 2008c– Chapter #6)?

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