IN VITRO AND IN VIVO BIOMECHANICAL INVESTIGATION OF THE CLINICAL PRACTICE OF DISC PROLAPSE PREVENTION AND REHABILITATION

By

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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

Underlying this thesis is the McKenzie school of thought, a physiotherapy approach that teaches clinicians to recommend particular exercises to their clients in an attempt to accelerate recovery/ prevent recurrence of disc prolapse. The recommendations are based on an untested clinical theory that movements opposite to those that cause disc prolapse can achieve reversal of disc prolapse. Little consideration has been given scientifically to the reversal of the failure process of the lumbar discs. Three in vitro and one in vivo study were designed to attain a greater understanding of both disc failure and the mechanics of its clinical treatment responses and thereby provide a foundation for evidence-based practice.

The first in vitro study in this thesis compared in vitro and in vivo herniated discs in an attempt to link the two and provide a more thorough understanding of the in vitro model proposed to test the mechanical theory underlying the McKenzie derangement approach. Ten C3/4 osteoligamentus porcine specimens were repeatedly flexed or flexed and side bent to result in posterior migration of the nucleus. Three of the 10 specimens had posterior migration of the nucleus. Statistically significant ($p < 0.01$) and clinically significant (>33%) disc height loss occurred in all 10 specimens. The results provide a sub-classification of in vitro herniated discs that is similar to the spectrum of herniated discs that occurs in vivo.

Continuing from the disc height loss sub-classification of post-herniated in vitro discs, the second in vitro study in this thesis pursues alternate methods of creating herniation with the goal of creating herniation without causing more than thirty three percent disc height loss of the specimens. Repeated flexion of porcine cervical specimens under a lower compression level (1kN) resulted in disc herniation but with loss of 50% of the pre-test disc height ($p < 0.001$). Re-hydrating specimens by injecting the disc after a period of failure testing with a barium sulphate nucleus mix ($n = 5$) or by placing the specimen in a saline bath for an extended period of time ($n = 4$) resulted in a significant increase of the disc height of the specimens. Further flexion testing of the specimens significantly reduced the disc height again. Intermittent saline injection of specimens ($n = 3$) during the failure procedure did not prevent or reduce the disc height loss that occurred in the absence of saline injections. Using higher compression levels (2 and 2.596kN, $n = 4$), failure testing under torque control ($n = 3$), non-physiologically starting the annular rupture ($n = 5$) and using hypolordotic thoracic porcine spines ($n = 9$) instead of porcine cervical spines were unsuccessful attempts at creating herniations. This study indicated that the in vitro model used in the first in vitro study displayed features from one end of the spectrum of damage seen clinically but was then the best-available. Combined these two studies provide a framework for interpretation of the results of the subsequent and third in vitro study in this thesis.

The focus of the third study is the mechanical investigation of the McKenzie clinical theory of the treatment response seen in vivo in prolapsed discs, which is that movements or positioning can alter the location of a displaced portion of nucleus in a prolapsed disc. This study is a proof of the principle on which this aspect of the McKenzie approach is based and provides, to the author's knowledge, the first scientific evidence supporting the theory that repeating movements opposite to those that caused posterior migration of the nucleus can centralize the prolapsed material. The results indicate that the McKenzie approach works on some prolapsed discs and not on others. Consideration of the changes in disc height of the specimens during the testing procedures offers some understanding of the varied success of this approach and exposes a vast area of future research that will refine the clinical approach and mechanical understanding of this specific disc pathology.

The fourth study, an in vivo study, provides a first look at the kinematics and kinetics of the current in vivo application of this approach. Twenty asymptomatic subjects volunteered to participate in this study and performed frequently prescribed McKenzie exercises and a selection of activities of daily living during which a 3-SPACE Isotrak system measured their three dimensional lumbar kinemetics. One subject underwent a series of McKenzie exercises while electromyography and three-dimensional lumbar motion were measured. Mean peak extension of extension in standing and extension in lying exercises were within 3% (SD 22.33%) of each other. An additional 6.75% (SD 11.18%) of extension occurred when the extension in lying exercise was combined with a Grade 3 Maitland extension mobilization to L3, a passive physiotherapy technique that involves the therapist applying intermittent low amplitude oscillations to, in this case, the posterior aspect of the spinous process with the goal of subsequently increasing the range of active motion in the direction of the mobilization. The peak extension during the extension in lying exercise was increased after the mobilization relative to the pre-mobilization range. The mean peak right side bend in the right side glide exercise, normalized to the full right side bend range, was 61% (SD 17.4%). The L4-5 forces at the position of peak extension in extension in lying and extension in standing were 828.97N and 1368.86N respectively. The peak flexion ranges of the activities of daily living investigated match those previously used to create disc prolapse when applied at high repetitions and under moderate axial compression. The lumbar spine ranges achieved in commonly prescribed McKenzie rehabilitative and preventative exercises and those that occur in seemingly non-problematic activities of daily living were quantified. The results of this study will enhance clinical practice by providing quantitative evidence of the relative peak motion of the McKenzie exercises as well as highlighting seemingly benign activities of daily living that involve levels of flexion, side bend and rotation sufficient to cause disc damage and even prolapse.

The macroscopic goal of this thesis was to attain a greater understanding of the mechanics of both disc failure and its clinical treatment responses and thereby provide a foundation for evidence-based practice, a goal that was successfully achieved. This thesis ultimately challenged and increased our understanding of pathological discs while simultaneously adding information to assist clinical decision making. Several new contributions to the existing knowledge of lumbar spine biomechanics and clinical concepts of treating disc prolapse have been made.

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my parents and siblings for their
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LIST OF ABBREVIATIONS

ADL Activities of daily living

DDD Degenerative Disc Disease

DCRA Distortion Compensated Roentgen Analysis

EIL Extension in Lying

EIS Extension in Standing

ELSG Extension Left Side Glide

ICC Intraclass Correlation Coefficient

IDP Intradisal Pressure

IVD Intervertebral Disc

LBP Low Back Pain

MLBP Mechanical Low Back Pain

MRI Magnetic Resonance Imaging

OHR Overhead Reach

RSG Right Side Glide

CHAPTER 1

INTRODUCTION

The Motivating Factors That Instigated This Body Of Work

Macroscopically, this thesis has been motivated by the absence of a biomechanical understanding of a widely accepted clinical approach to disc herniation, namely the McKenzie approach. Disc herniation, a potentially painful entity first described clinically in the early part of the 20th Century, is a term that refers to the displacement of a portion of the nucleus into the surrounding annular layers of the lumbar intervertebral discs (IVD) (Mixter and Barr 1934, Williams 1932, Crock 1986). Numerous loading mechanisms have been proposed to create herniation (Callaghan and McGill 2001, Aultman et al 2005, Andersson 1981, Brinckmann 1986, Kelsey et al 1984, Gordon et al 1991, Lu et al 1996a). The models successful in causing posterior displacement of a portion of the nucleus are those involving axial compression and repeated flexion (or flexion combined with side bend or rotation). Little consideration has been given scientifically to the reversal of the failure process at a mechanical level. Clinically, however, repeated extension is thought to reduce the symptoms and signs of disc herniation (McKenzie 2002). The McKenzie approach supposes that a displaced portion of nucleus in a damaged disc can be moved back towards the center of the disc using movements and positions specific to the direction of the displacement, as long as the outer annulus is intact and the nucleus is hydrostatic. Reversal or reduction in the symptoms and signs of disc herniation is the daily work of many clinicians. The experience gained by such work prompted the development of this particular systematic clinical approach, the McKenzie method to mechanical low back pain (MLBP). The McKenzie method is an algorithmic approach (see next section) to the assessment and subsequent treatment of a disc herniation or disc derangement, as it is referred to in the McKenzie classification system of MLBP. The pain, motion and functional response to repeated test movements and positions are integrated to identify the direction and extent of the mechanical forces required to reduce the symptoms. Classically in the derangement syndrome the distal symptoms are reduced first followed by those closer to the spine. This is a phenomenon called 'centralization', the hallmark of the McKenzie approach to the derangement syndrome. The centralization phenomenon has been shown to have clinical merit (Alexander et al 1998, Donelson et al 1990). Alexander et al (1998) was able to predict in 91% of cases using this repeated movement assessment technique those that would respond to non-surgical treatment of disc prolapse while Donelson et al (1990) reported that clients, who could not achieve centralization of symptoms as a result of repeated movements, did not respond well to conservative therapy and generally had a poor treatment outcome. Underlying this algorithm, and underlying this thesis, is the untested clinical theory that movement/ positioning of the spine

influence the position of a displaced portion of the nucleus in particular pathological discs. This theoretical mechanism and patho-anatomical explanation for centralization has not been scientifically substantiated. This thesis considers the possibility that a mechanical mechanism underlines the reported clinical observations and this aspect of the McKenzie method. Specific advice to a client from a therapist is based on acknowledgement of a recognized pattern of symptoms and signs. Best clinical practice appears to result from a knowledge and understanding of the foundation of the pattern combined with clinical skill. In the absence of a thorough understanding of the mode of effect (mechanical, sensory, psychological) of clinical treatment clinicians may have sub-optimal effects and efficiency. The benefits of such investigation lie in its potential to enhance the clinical treatment of damaged discs.

OVERVIEW OF THIS THESIS

This thesis is centered on the investigation of the mechanical foundation of a particular type of physiotherapy assessment and treatment, the McKenzie derangement program. The investigation of any component of clinical practice in a laboratory setting, especially a tissue laboratory, raises an array of challenging issues. Conceptually these issues can be appreciated by recognizing that physiotherapy practice is an 'outside-in' approach, viewing the in vivo spine from the outside and hypothesizing about the related spine mechanics, patho-anatomy and patho-physiology, while in vitro laboratory work is an 'inside-out' approach, viewing the in vitro spine segments and making deductions about the clinical presentation that the given mechanics may produce. Both approaches have merit but also have limitations. Bridging the two approaches is paramount to continued development of the evidence-based practice model.

The first of the four studies in this series was prompted by the clinical knowledge that the McKenzie derangement approach is not indicated in every damaged disc, that not every disc responds positively to this sppraoch. Every effort had to be made to ensure that the in vitro model proposed for this work was appropriate to the question and would allow conclusive interpretation of whether movement/ positioning of the spine influences the position of a displaced portion of the nucleus in particular pathological discs. Evidence-based practice can only be achieved through practice-based evidence; in essence, clinical approaches need to be scientifically investigated using models that are representative of the clinical use of the approach (Downing and Hunter 2003). This first study compares in vitro and in vivo herniated discs in an attempt to link the two and provide a more thorough understanding of the in vitro model proposed to test the mechanical theory underlying the McKenzie derangement approach.

This first study highlighted an area of discrepancy between the post-herniated in vitro porcine discs and the in vivo discs considered most appropriate to this work. The second study describes various attempts at paralleling the porcine in vitro model to the targeted in vivo discs. This study aims to bridge the discrepancy in the disc height of the in vitro and the targeted in vivo herniated discs.

Combined the first two studies provide a greater understanding of the post-herniated porcine disc and a framework for interpretation of the results of research using this post-herniated porcine in vitro model, more specifically for the third in vitro study in this series. The focus of the third study is the mechanical investigation of the McKenzie clinical theory of the treatment response seen in vivo in herniated discs. Specifically, it is theorized that a displaced portion of nucleus in a damaged disc can be moved back towards the center of the disc using movements and positions. This study is a proof of the principle on which this aspect of the McKenzie approach is based.

The McKenzie approach to the treatment and prevention of MLBP is a two-tiered approach that involves identifying the appropriate exercises to be performed by the client as well as the specific motions, postures and loads to be avoided in order to accelerate recovery and reduce the risk of recurrence of the MLBP. In general, for a posterior or posterolateral disc prolapse the recommended exercises involve extension movements while flexion movements are those to be avoided. Following scientific investigation of the conceptual model of the McKenzie derangement approach is an in vivo study that quantifies the mechanics of a number of McKenzie exercises and a number of activities of daily living (ADL) which are seemingly benign but may replicate the injury mechanism.

The hypotheses of each study are outlined after a review of the pertinent literature. The following literature review will begin by addressing relevant aspects of clinical practice, then transition into issues relating to diagnostic imaging, an area that is common to both clinical and laboratory work, and finish with issues concerning in vitro research.

BACKGROUND INFORMATION

Clinical Practice – looking from the "outside in".

MLBP, distinguished from low back pain LBP of visceral or psychosomatic origin, has been reported as one of the most commonly treated disorders in outpatient physiotherapy practices and as a condition that 60- 80% of individuals will experience at some time in their life (Jette et al 1994). Similar to other areas of medicine, classification of MLBP is regarded as fundamental to successful clinical practice (Brennan et al 2006, McKenzie & May 2003, Fritz et al 2003, Riddle 1998, Kilby et al 1990, Jette 1989, Sahrmann 1988, Stevens and McKenzie 1988, Deyo 1986), although a universally accepted classification system for MLBP does not exist. Physiotherapists traditionally

have been dependant on the patho-anatomical model of classification. Numerous structures have been implicated at one time or another as being a possible source of MLBP. Bogduk (1997) postulated that for a structure to the source of LBP the structure should have a nerve supply (DeLeo 2006, Bogduk 1983), be capable of causing pain similar to that seen clinically, should be susceptible to disease or injury and should have been shown to be a source of pain in patients. The vertebrae (Groen et al 1990, Hirsch 1963), lumbar muscles (Bogduk et al 1982, Bogduk 1980), the thoracolumbar fascia (Yahia et al 1992, Stillwell 1957, Konno et al 1994), dura mater (Edgar and Nundy 1964, Groen et al 1990, Smyth and Wright 1959), epidural plexus (Groen et al 1990), the interspinous ligaments (Bogduk et al 1982, Jensen et al 1994, Hockaday 1967), zygapophyseal joints (Hirsch et al 1952, McCall et al 1979, Fairbank et al 1981) and the discs (Bogduk 1990, Bogduk et al 1982, Jackson et al 1966, Yoshizawa 1980) comply to some degree with the requirements of the postulation. The most common diagnoses of MLBP using the patho-anatomical model of classification are disc lesions, zygapophyseal syndrome, and instability due to pars interarticularis defects (Bogduk et al 1995, Delitto et al 1995, McKenzie and May 2003). Over the last three decades there has been a gradual shift away from the patho-anatomical classification model and a move towards classification according to movement dysfunction and it's relation to pain behavior (McKenzie 1981, McKenzie & May 2003, O Sullivan et al 2000, Sahrmann 2002 and Delitto et al 1995). The McKenzie classification system is considered one of the most widely accepted physiotherapy approaches in diagnosis and management of MLBP (Varamini and Jam 2005). It is characterized by classifying MLBP as one of three distinct syndromes; (1) Postural (2) Dysfunction and (3) Derangement. These syndromes are identified by following a specific algorithm, the components of which include a review of the client's previous medical history, the client's current symptom behavior, the current spinal postures/ deformities, the quality and quantity of spinal movement, and, the signature of the McKenzie Approach, the response of the symptoms to repeated/ sustained test movements. In essence, the symptom behavior components of the McKenzie algorithm point the physiotherapist in the direction of a particular syndrome and the movement testing confirms/ changes the suspected syndrome (Table 1.1). During the movement and position testing the clinician considers the quantity and relative directional loss of range of spinal motion, the quality of curve reversal during movement, the presence of deviation from the mid-sagittal movement pathway during flexion and extension and the symptom response during and after movement. Although the word 'syndrome' implies the cause of the symptoms and signs is unknown and the McKenzie model is not a patho-anatomical model, the easiest way to learn and practice the McKenzie approach is to grasp the proposed patho-anatomical distinction between the three syndromes. The symptoms due to the

Syndrome	Effects of Repeated Movements
Postural Syndrome	Pain is not reproduced Pain present when stationary Pain is not present during testing
Dysfunction Syndrome	Pain is produced at end-range movement only Stops shortly on release of end-range stress Fixed pain pattern during testing (same end-range pain) Will radiate slightly during testing of nerve root adherence Condition unchanged after testing (no better or worse) No rapid and lasting changes as a result of testing
Derangement Syndrome	Symptoms are produced or altered within movement range Painful arc may exist Variable pain pattern during testing Progressive decrease or increase during testing Centralization or peripheralization during testing Condition remains better or worse after testing Rapid and lasting changes as a result of testing

Table 1.1: Effects of repeated movement on the McKenzie syndrome. The distinct difference in symptom response to repeated movements provides clear classification of the mechanical low back pain. [From Van Wijmen (1994): The use of repeated movements in the McKenzie method of spinal examination. Chapter 42 In Grieve's Modern Manual Therapy, The Vertebral Column, 2nd Edition, Churchill Livingstone]

postural syndrome are thought to be due to sustained end-range stretch of pain-sensitive structures, while those of dysfunction are thought to occur due to stretching of sensitive, adaptively-shortened structures. Of most interest to this thesis, the symptoms of the derangement syndrome are patho-anatomically thought to be due to displacement of a portion of the nucleus into the annular layers. McKenzie's method of identification and treatment of the derangement syndrome stems from his clinical observation of abolition of many patients' most distal symptoms in response to repeated movement testing (Figure 1.1). This observation, later called the 'centralization phenomenon', is the hallmark of identifying and treating the derangement syndrome. McKenzie's (1981) patho-anatomical explanation of the rapid, often immediate, reduction in the client's distal symptoms after repeated movement in a particular direction is that it relates to a change in the location of the displaced portion of the nucleus. Similarly, according to McKenzie (1981) an increase in a client's distal symptoms or peripheralization of symptoms after repeated movement is suggestive of either an incorrect direction of treatment, causing further displacement of a portion of the nucleus, or the direction is correct but the movement is too rapid to allow reductive movement of the displaced nuclear portion. A competent annulus and a hydrostatic nucleus are considered critical to the success of this approach to disc derangement. This movement related mechanical theory involves fluid flow, hydrostatic pressure and stress gradients across the disc. To date no one is able to state categorically whether the conceptual model for the derangement syndrome is correct but the centralization phenomenon has been shown to have clinical merit. Using the centralization phenomenon cases that would respond successfully to non-surgical treatment of disc prolapse could be identified (Donelson et al 1990, Long 1995, Alexander et al 1998). In 1997, Donelson et al investigated the theory that centralization is dependent on a competent annulus in a study that compared the McKenzie classification of the symptom response to movement to discogram-determined annular competency. Ninety-one percent of those that centralized had an intact annulus, suggesting that indeed the success of the McKenzie approach to the derangement syndrome may be associated with a competent annulus. Evidence of a change in the location of the displaced portion of the nucleus is still lacking. This thesis takes the McKenzie Approach, a successful 'outside-in' approach, and considers it from a laboratory, inside-out, perspective. The plausibility of McKenzie's patho-anatomical explanation of the symptom response to repeated movement/ sustained positions in the derangement syndrome will be developed after a review of structure and function of healthy discs.

Lumbar Intervertebral Disc (IVD): Physiology and Mechanics of Healthy Lumbar Discs

A young healthy spinal IVD consists of a central semi-fluid mass of mucoid, the nucleus pulposus, which is surrounded by a peripheral fibrous ring, the annulus (Bogduk 1997). The three

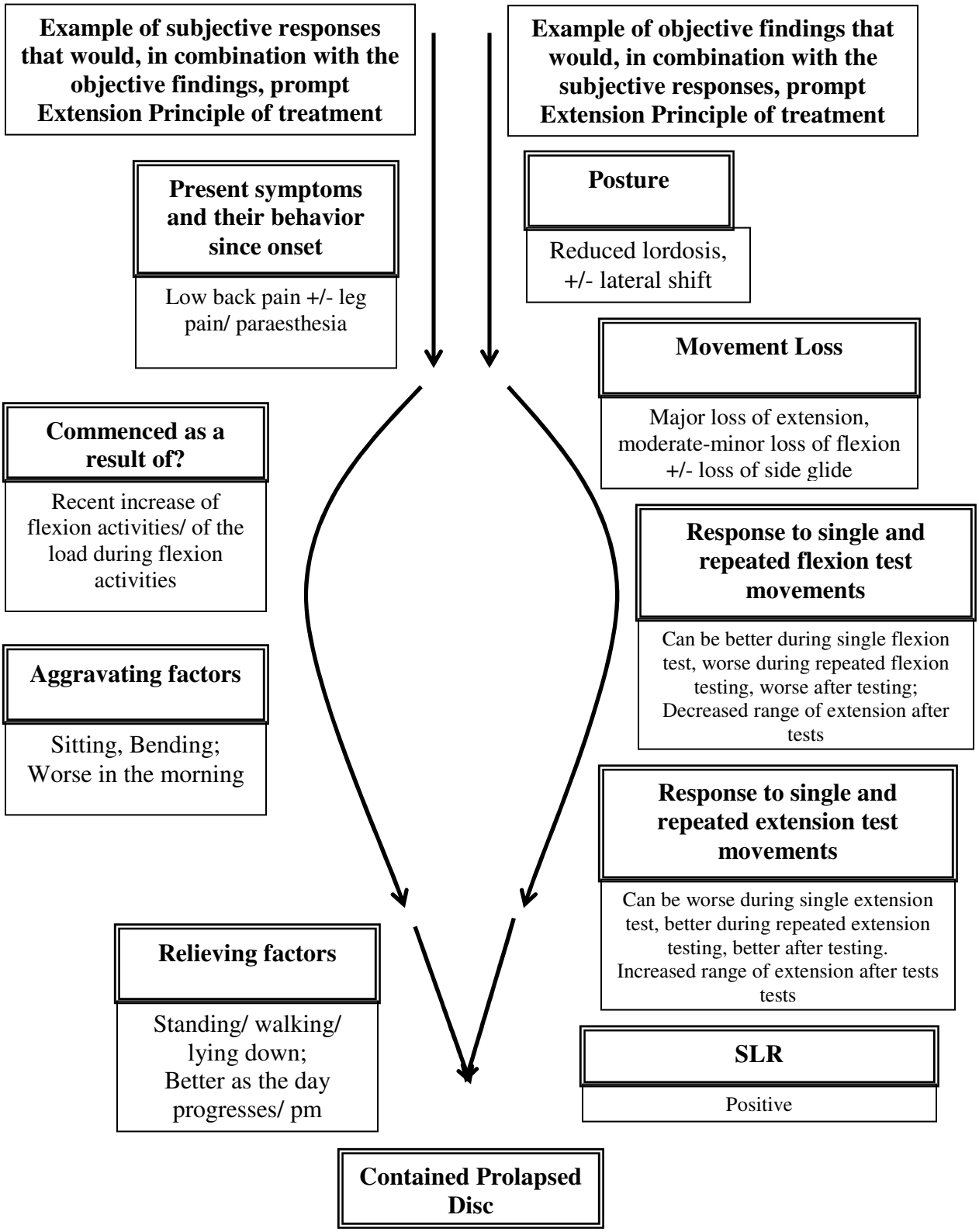

Figure 1.1: Selected components of the McKenzie Algorithm that combine to give a clinical impression of the derangement syndrome.

main constituents of the disc are water, collagen and proteoglycans, the proportions of which vary considerably within the disc (Ghosh 1990). The nucleus consists of approximately 70-90% water with proteoglycans being the next major component (Gower and Pedrini 1969, Naylor 1971, Taylor 1990). The proteoglycans, which consist mainly of glycoaminoglycans linked to proteins, are responsible for the water-binding capacity of the nucleus, which results in the fluid behavior of the nucleus (Urban and Maroudas 1980). Type II collagen fibers make up 15-20% of the dry weight of the nucleus (Bushell et al 1977) and the remainder of the nucleus consists of some elastin fibers and other non-collagenous proteins (Taylor and Little 1965). The proteoglycans and collagen fibrils account for the viscosity of the nucleus (Bogduk 1997). This nucleus is surrounded by fibrous concentric lamella, the fibers of each lamella are parallel and are orientated 30^0 - 70^0 to the vertical (Hickey and Hukins 1980, Bogduk 1997, Adams et al 2002), variable depending on the location of the fibers in the disc and on segment position. Consecutive lamellae run in opposite obliquity. 60-70% of the weight of the annulus is due to water (Gower and Pedrini 1969). It also consists of proteoglycans, type I and II collagen and a notable quantity of elastic fibers. The nucleus and the deeper layers of the annulus are distinct in childhood but less defined in early adulthood. The annulus is distinct from the nucleus peripherally. The lamellae are thick in the anterior and lateral portions of the annulus, but posteriorly they are thinner, approximately 50% of the thickness of the anterior and lateral annulus (McKenzie 1981, Cassidy et al 1989). Justified by the strong attachment of the annulus to the endplate and the weak attachment of the endplate to the vertebral bodies the vertebral endplate is often considered a part of the disc (Bogduk et al 1997). The endplate, a hyaline cartilage and fibro-cartilage mix, resembles and parallels the chemical structure of the nucleus and annulus. The endplate covers the nucleus in its entirety but fails to cover the entire extent of the annulus. The collagen fibers of the inner annulus enter the endplate and swing centrally within it. Peripherally the annular fibers insert directly into the vertebral body.

A disc separates two vertebral bodies allowing the upper vertebrae to tilt forwards without its lower edge coming into contact with the lower vertebral body. Given the disc's intervertebral position and function it has to be strong enough to sustain weight, be deformable enough to allow movement between the vertebrae and strong enough not to be injured on movement (Bogduk 1997). Does the disc meet these criteria? Proteoglycans endow the disc matrix with a high osmotic pressure and a low hydraulic permeability and hence constitute the compression-resisting component of the disc. Chrondrotin sulphate and keratin sulphate, both proteoglycans, contain negative charged acidic groups, the concentration of which controls the osmotic pressure of the disc (1-3 atmospheres). High osmotic pressure results in, and maintains under external load, hydration of the disc. Furthermore, the rate at which fluid can flow into or out of the disc under a pressure depends on the hydraulic

permeability of the matrix, which is directly related to pore size distribution, determined by proteoglycan concentration. When the proteoglycan concentration is low (i.e. the disc is well hydrated) then the pore size is large and the fluid flow from the tissue is fastest. As fluid is lost from the tissue the proteoglycan concentration increases and the rate of fluid loss slows. When the in vivo proteoglycan concentration of the disc is high the disc will swell if not simultaneously compressed. The proteoglycan concentration of the nucleus is greater than that of the annulus and it was thought that the nucleus was the compression-resisting component of the disc. However, Markolf and Morris (1974) demonstrated that a disc with the nucleus removed lost height but preserves properties of axial stiffness, creep and relaxation rates when under briefly applied loads. Subsequently, McNally and Adams (1992) showed that the hydrostatic pressures are equal across the nucleus and also across the annulus in non-degenerative discs. The presence of the nucleus maintains hydrostatic pressure on the annulus preventing delamination (i.e. separation of the lamallae) and maintains the integrity of the disc.

Compression of the nucleus is limited by the proteoglycan component and related water content of the nucleus but on axial loading the nucleus tends to change shape, bulging radially (outwards towards the annulus) and decreasing in height. The pressure exerted on the endplates by the nucleus serves to transmit part of the load from one vertebra to the next while the radial pressure on the annulus braces it and prevents the annulus from buckling. The annulus through its densely packed collagen lamellae resists this radial pressure allowing minimal radial expansion of the disc (Brinkmann 1986). The energy stored in the elastic type-II collagen fibers during the radial expansion is released on unloading and the deformation of the nucleus and annulus is reversed. Water is squeezed from the compressed disc, resulting in loss of disc height (Kraemer et al 1985, Urban and Maroudas 1980), which is recoverable in young healthy discs as water is imbibed under a lower level of compression (Kraemer et al 1985). Unlike that of axial loading, deformation of the disc secondary to rocking movements is not uniform across the annulus. Flexion and extension of the spine involve sagittal rotation and small amplitude translations (Pearcy and Tibrewel 1984, Twomey and Taylor 1983). On flexion, anterior sagittal rotation and anterior translation, the anterior annulus will be compressed and tend to buckle while the posterior annulus will be tensioned. The anterior nucleus will also be compressed while the compression on the posterior nucleus will be reduced thus allowing the nucleus to deform posteriorly. Brault et al (1997) and others (Lyndsay et al 2007, Fazey et al 2006, Schnebel et al 1988, Fennel et al 1996) have shown, using magnetic resonance imaging (MRI) pixel intensity changes, that the nucleus of the lumbar discs moves by approximately 1mm posteriorly in flexion of the spine segments and anterior on extension of the spine segments. In flexion, fluids and metabolites are also forced out of the anterior, more compressed parts of the discs

and squeezed back into the posterior parts (Adams and Hutton 1985, 1986). The in vivo nuclear pressure increases on flexion, due to the additional compressive loads applied to the discs by the increased ligament stiffness and the action of the back muscles, and decreases on extension, due to the altered disc-facet joint load-sharing. The increased intradiscal pressure (IDP) in flexion can be reduced by cutting the posterior ligaments of the spine (McNally and Adams 1992, Adams et al 1994). Shah et al (1978) showed increased tangential stress in the posterior annulus on flexion. The combination of posterior annular tension and increased posterior annular tangential stress in flexion is of significance knowing that the posterior part of the annulus is the weakest and that important neural structures, nerve root and spinal cord, lie in close proximity to the posterior annulus.

Like all biological tissues the disc has viscoelastic properties. The stress response of the disc depends on both the strain applied and the rate at which this strain is applied. A viscoelastic material has infinite material responses, the responses influenced by dynamic strain rate, static pre-load, temperature, and time effects such as creep and relaxation. Leahy and Hukins (2001) measured the viscoelastic properties of the nucleus pulposus of sheep under compression. A preload of 200N of compression was applied to each specimen and sinusoidal cyclic compression of amplitude 10μm was applied at eight frequencies in the range 0.1 – 10Hz at a temperature of 37^0 C. There was no clear dependence of the elastic or bulk moduli on frequency. However, the phase angle between the applied stress and resulting strain showed clear frequency dependence, passing through a minimum at 0.9 +/- 0.2Hz. The authors suggest this indicates that the viscous effects in the nucleus are minimized at a frequency close to 1Hz. Keller et al (1989) investigated the viscoelastic behavior of normal and surgically altered in vivo porcine lumbar segments under static compressive loads (of approximately 58.0 N). The load was applied for 8 minutes and a further 8 minutes allowed for recovery time. On average 85% of the disc height was regained within the 8 minutes of removal of the compressive load. Numerous disc models have been proposed to extend our understanding of the viscoelastic properties of the disc and the complexity of the task is highlighted by the array of finite element models designed to capture the characteristics of the disc. A number of mechanical models treat the intervertebral nucleus as an incompressible inviscid fluid. Belytschko et al (1974) used an axisymmetric linear viscoelastic model to investigate the behavior of the disc under axial load. The results suggested a need to include anisotrophy of the annulus to improve the accuracy of the model. Subsequently, Kulak et al (1976) modeled the annulus as an axisymmetric nonlinear orthotrophic material. Lin et al (1978) reverted to modeling the annulus as a linear orthotrophic material but considered it 3-dimensional rather than axisymmetric. Shirazi-Adl et al (1984) included material and geometric nonlinearities in their 3-dimensional finite element model investigating the strain response of the vertebral body, endplate and annulus under axial compression. The model was validated by

comparing the predictions with experimental results of axial displacement, disc bulge, end-plate bulge and IDP. The results indicate that for a normal disc with an incompressible nucleus, the most vulnerable elements under compressive load are the cancellous bone and the end-plate adjacent to the nucleus space, results reported by numerous authors on in vitro testing. Keller et al (1989) compared the predictions of a rheological model consisting of a spring element in series with a Kelvin model with in vivo experimental results and found excellent agreement between the two sets of results. The model produced an initial instantaneous deformation directly proportional to the initial applied load. The 3–parameter model predicted that, after its original instantaneous deformation, the intervertebral joint would continue to deform at a continuously decreasing rate until equilibrium. Lu et al (1996) constructed a 3-dimensional finite element model of the disc, all material properties were assumed to be linear and the viscoelastic properties were modeled as quasilinear. Their model showed that annular fissures commence at the inner fibers and that saturated discs fail sooner than dehydrated discs. Iatridis et al (1997) used an integral formulation of a linear viscoelastic constitutive model with a variable amplitude relaxation spectrum to model torsion shear behavior of the nucleus under transient and dynamic conditions. The experimental data of the same study indicated that the nucleus behaves as a fluid under static conditions, given by the equilibrium values of close to zero on a stress-relaxation test. However, on dynamic testing the nucleus behaved more 'solid-like', the stiffness of the nucleus increasing with increasing angular frequency. Argoubi and Shirazi-Adl (1996) investigated the non-linear three-dimensional poroelastic creep response of a lumbar motion segment under a constant axial compression (400, 1200 or 2000N) for a period of 2 hours. As time progressed axial displacement increased, pore pressure decreased, annulus bulk underwent larger compressive stress, fiber layers became slack and facets carried larger loads. However the authors note that the relative influence of fluid movement and inherent tissue viscoelasticity in the temporal response of a motion segment is not yet known. Riches et al (2002) compared experimental data with the results of a one-dimensional poroelastic model, which included permeability and osmotic pressure as functions of strain. They applied compression for 20 minutes to human cadavers (all ligaments, muscles, facet joints and posterior elements were removed), allowed recovery under 0N for 40mins and repeated the cycle 5 times. Their results showed that the disc expands when unloaded faster than it compresses under load. Bardet (1992) proposed a viscoelastic model to describe the dynamic response of saturated poroelastic materials that obey the Biot theory (1956) and reported that it is simpler to use than poroelastic models but yields similar results for a wide range of saturation levels and dynamic loadings. Simple and elaborate modeling approaches have been taken but many disc parameters remain unquantified hindering the accuracy of the models.

Mechanisms of Disc Failure

Early studies attempted to produce herniation with static loads in compression. Several investigators found end-plate fracture before any evidence of disc injury (Brown et al 1957). Farfan et al (1976) hypothesized rotation as a mechanism of disc prolapse, partial herniation, but not full herniation. Other investigators observed that flexion of 9^0 or greater led to supraspinous and interspinous ligament injury (Adams and Hutton 1980) or endplate fracture but not disc injury. Gordon et al (1991) produced disc ruptures (herniation) by combining rotation ($1\text{-}3^0$), flexion (7^0) and compression (1334 N) within physiological ranges. Ten of these discs failed through annular protrusions while four failed through nuclear extrusion through annular tears. This suggests that those that undergo increased total or segment flexion in their ADL are more at risk of disc herniations when combined with a given level of compression and rotation. More recently Callaghan and McGill (2001) have consistently been able to herniate porcine cervical discs by repeated full flexion under low levels of compression. Axial rotation of the lumbar spine involves torsion of the IVD and impaction of the facet joints (Bogduk 1997). During axial rotation all the fibers of the annulus that are inclined toward the direction of rotation will be strained. Based on the stress-strain curve of collagen the maximum range of rotation of a lumbar IV disc without injury is 3^0 (Hickey and Hukins 1980). To rotate beyond 3^0 the upper vertebrae must pivot on the impacted joint and this joint becomes the site of the new axis of rotation. Both the vertebral body and the opposite inferior articular process will then swing around this new axis. This will strain the disc and the capsule of the facet joint. Haher et al (1989) in an in vitro study looked at the contribution of the columns of the spine to torsional rigidity. In the intact lumbar spine the annulus was the most effective structure in resisting torsion. The compressed facet joint, lying in the vicinity of the axis of rotation, experienced significantly less torsional stress. This is contrary to the results of previous studies, which showed that the facet joints are responsible for rotational resistance in the lumbar spine (Adams and Hutton 1981). These differences may be due to the differences in the axis of rotation of the specimens used in the studies.

Endplate fractures and nuclear herniation seem to be two extremes of a continuous spectrum of mechanically driven internal disruption of the disc. The formation of distinct fissures in the annulus, radial and circumferential, has been previously reported (Adams and Hutton 1985, Thompson et al 1990, Brinckmann and Horst 1985, Brinckmann and Grootenboer 1991, Videman and Markku 2004) and defined as fissures that are perpendicular to the endplates and those that run along the circumference of the disc respectively. Also previously reported are rim lesions (Osti et al 1990) which are radial tears at the periphery of the annulus adjacent to the endplates. More recently Tampier et al (in press) identified the formation of small clefts in between the layers of the annulus

through which the nucleus pulposus was 'pumped' in porcine cervical spines tested under axial compression and repeated flexion (Callaghan and McGill 2001). Once a pocket of nucleus within a cleft acquired enough pressure a new cleft was formed in the weakest part of the layer and the cycle repeated. A portion of the nucleus was eventually displaced to the outer annulus as propagation of the cleft formation continued peripherally. Whether cleft formation is the only pathway through which a portion of the nucleus becomes displaced, a precursor to rupture or a totally separate process, rupture and cleft formation possibly occurring under different conditions will be the topic of future research.

Thus far the loading conditions (axial compression, flexion ± side flexion or rotation) that result in herniation and the pathway(s) of herniation have been discussed. Consideration of the underlying mechanics that lead to herniation is vital in advance of consideration of the proposed patho-anatomical McKenzie theory. Under pure axial compression the annulus and nucleus bulge radially, the outward pressure of the nucleus preventing inward buckling of the inner annulus. Peak stress occurs in the inner posterior annulus (Adams 1995, Adams et al 1980). Flexion increases compression of the anterior annulus while reducing compression/ causing tension of the posterior annulus (Shah 1980, Adams and Hutton 1982). Adams and Hutton (1982) reported that in full flexion a 50% increase in the posterior annular height and a 30% decrease in the height of the anterior annulus occur relative to their respective heights in erect standing. They also reported that the posterior annulus thins to 67% of its thickness in lordotic posture while the anterior annulus buckles inward from the additional compression during flexion. Flexion causes a large increase in the intradiscal pressure because of the increased ligament tension and contraction of the back muscles. There is increased hydraulic stress on the posterior annulus as fluid moves away from the high anterior compression. Adding axial rotation to the loading pattern will result in increased strain of the annular fibers inclined in the direction of the rotation (Bogduk 1997). Hickey and Hukins (1980) reported that the annular fibers can tolerate up to 3^0 of rotation before microscopic failure begins. In summary, the posterior annulus has to resist increased intradiscal pressure, increased hydraulic pressure of the nucleus and increased tension in response to the loading conditions of axial compression, flexion ± side flexion or rotation, previously identified as resulting in herniation. This combination of mechanical forces appears to be the recipe to pump a portion of the nucleus through clefts/ ruptures in the posterior/posterolateral annulus. Supporting the argument that a portion of the nucleus will be displaced in the direction of the annulus that is under maximum combined stress are the findings of Aultman et al (2005) (Appendix A), a study in which specimens that were repeatedly flexed where the flexion axis was moved 30^0 to the left of the sagittal plane developed right postero-lateral disc herniations, predictable in 15 out of 16 specimens. Specifically, the site of the nucleus breach was determined by the bending axis, a finding also reported by Tsantrizos et al 2007.

Extension is often the direction of movement associated with the McKenzie Approach. Donelson et al (1991) found extension movements were more likely to result in pain centralization than were flexion movements, a finding that may have contributed to the misconception that the McKenzie approach consists entirely of extension (Robinson 1994). Consider now the mechanics of extension, especially in the context of the patho-anatomical McKenzie theory. Can extension alter the position of the displaced portion of nucleus in the damaged annulus? If the combination of increased compression of the anterior aspect of the disc, increased intradiscal pressure and increased tension of the posterior annulus results in a stress gradient that displaces a portion of the nucleus in flexion - is there a corresponding stress gradient that can reverse the nuclear infiltration of the annulus? Extension involves a posterior sagittal rotation and a small posterior translation. Adams et al (2000) investigated the effects of backward bending on healthy lumbar IVD and found that 2^0 of extension increased the maximum compressive stress within the posterior annulus by an average of 16%, compared with the neutral posture, in degenerated and healthy IVD. Backward bending tended to reduce the compression in the anterior annulus and nucleus (to 0.5 MPa) and increase it (to 2.75 MPa) in the posterior annulus, creating a greater stress gradient across the disc than in a neutral position. In such cases the displaced nucleus would be under greater compression in the posterior annulus. However, in degenerate spines the results were more variable. In 7 of 19 degenerated specimens extension caused a reduction, of up to 40%, in the maximum compression in the posterior annulus while in the other degenerate discs the compression was increased by 43% relative to the neutral posture. Adams et al (2000), Pollintine et al (2004) and Rousseau et al (2006) suggested that the reduction in the compression in the posterior annulus was indicative of neural arch shielding which would occur when the disc has lost sufficient height and the axis of rotation has moved posterior of the annulus (Figure 1 2). This stress shielding may account for the positive clinical results attributed to extension. Based on this argument it is the decrease in compression of the posterior annulus due to a change in the center of rotation of extension that may allow the change in position of displaced nucleus and explain the success of the McKenzie approach.

The Role Of Diagnostic Imaging In Bridging The Inter-Disciplinary Gap

The jump from the internal disruption of the disc to the clinical pattern associated with these changes is still extensive. The use of diagnostic imaging to bridge this expanse has been disappointing. Extruded or sequestrated disc with nerve compression seen on MRI in a patient with acute radicular leg pain is considered a reasonable explanation for such pain. However, numerous

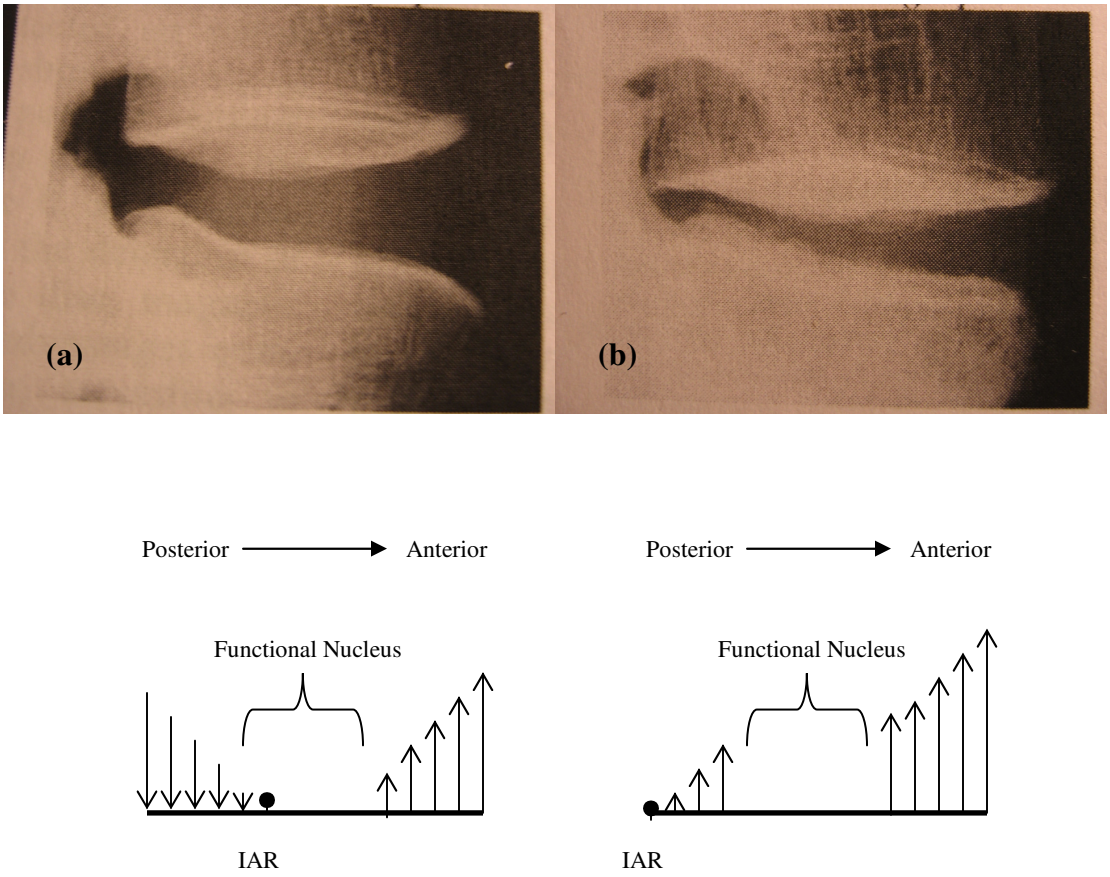

Figure 1.2: A schematic of the stress gradient across the disc during extension of (a) a hydrated healthy disc and (b) a dehydrated, degenerated disc. In a healthy hydrated disc the instanteous axis of rotation (IAR) of extension lies in the posterior aspect of the nucleus. As disc height decreases the contact of the articular surfaces of the facet joint increases moving the IAR more posteriorly. The anterior annulus and posterior annulus are both under tension as the neural arch shields the posterior annulus from compression. Adams et al (2000) suggests that this may account for the success of the McKenzie extension approach.

disc abnormalities have been identified on MRI of asymptomatic patients, hindering the interpretation of the findings in a clinical setting (Boden et al 1990, Jensen et al 1994, Boos et al 1995). Qualitative and quantitative classification of MRI findings have been investigated to determine the ability of the finding to distinguish between levels of degenerative disc disease (DDD) and to determine the correlation between MRI findings and the morphological and biochemical changes associated with DDD. Benneker et al (2005) found that signal intensity, Modic changes, disc extension beyond the interspace, nucleus pulposus shape and endplate integrity all correlated significantly with morphological grade. The signal intensity also correlated significantly with water and proteoglycan content. However, the link between morphological and biochemical changes of the IVD and symptoms and clinical signs has not been established. The symptomatic and structural results of discograms have been compared with MRI findings to gain further insight into clinically relevant MRI findings. Weishaulpt et al (2001) found DDD findings on MRI had a high negative predictive value in terms of pain prediction and low specificity while high-intensity zones had a high specificity but a low positive predictive value in terms of pain prediction. Moderate to severe endplate abnormalities had 100% positive predictive value and 100% specificity. Unfortunately, there is no clear diagnostic imaging system that definitively links the findings of diagnostic imaging to a clinical presentation. However, as will be discussed in chapter 2, the literature provides many indications of the mechanical relevance of the findings of diagnostic imaging and this enables preliminary interpretation of their clinical relevance.

In Vitro Testing

Animal Model

Animal models are a necessary part of the quest for a greater understanding of in vivo biomechanics. These models are controllable, reproducible and cost-effective, unlike specimens from human cadavers where age, exercise level, diet, environment, personal factors and genes add variability to a sample. The limitations of in vitro studies include the inability to consider the roles the muscles may play in vivo, the lack of in vivo recovery of viscoelastic tissues and the in vivo potential to heal. The knowledge and understanding gained by the in vitro studies is often incorporated into in vivo work which captures the deficits of the in vitro work (An and Masuda 2006). The in vitro lack of recovery times that are equivalent to in vivo is problematic but knowing the biomechanical implications, for example disc height loss, of such limitations prevents incorrect interpretation of the results. The number of repetitions of motion applied to the specimens in these studies would occur in a two week period in vivo (Brinckmann et al 1988) and precede collagen repair and proteoglycan turnover in vivo (Adams and Hutton 1982, Urban et al 2004). Numerous

animal models have been used to replicate the human lumbar spine including sand rats (Silberberg et al 1979, Alder et al 1983, Moskowitz et al 1990 , Ziv et al 1992), mice and hamsters (Berry 1961, Silberberg and Gerristen 1976), rabbits (Sullivan et al 1971, Kroeber et al 2002) multiple canine breeds (Goggin et al 1970, Ghosh et al 1976, Cole et al 1986), pigs (Kappa et al 1994, Pfeiffer et al 1994, Callahgan and McGill 2001, Holm et al 2004, Cinotti et al 2005), sheep (Lappalainen et al 2002, Leahy and Hukins 2001) and cattle (Simunic et al 2001). The smaller animals are suitable for biological studies while the large species are more desirable than the smaller species for biomechanical investigation applicable to human spines (Singh et al 2005, Adams 1995, An and Masuda 2006). The in vitro porcine cervical model used in the current research has been shown to be anatomically, geometrically, functionally (Yingling et al 1999, Oxland et al 1991) and microscopically (Tampier et al 2006) similar to human lumbar spines. Yingling et al 1999 showed that the porcine specimens have smaller endplate depth and width (approximately 2/3 of human lumbar spines), smaller spinal canal width (approximately 18mm in comparison to 22mm in human lumbar spine), equivalent facet orientation (45^0 to the sagittal plane) and displayed similar trends in shear stiffness and compressive tolerance to human specimens. Microscopically, porcine cervical discs are similar to human lumbar discs in the number of, width of and angle of the annular layers as well as the type of cells and collagen in the disc (Tampier et al (in press)). Based on this the porcine disc was considered a good model for the investigations proposed, was readily available and provided a reproducible model.

Disc Hydration

In vivo hydration of the disc is determined by the equilibrium between osmotic pressure and the compression on the disc. As the osmotic pressure cannot adequately counter the cumulative compression applied over the course of a day the fluid volume of the disc decreases by 8-20% per day (Botsford et al 1994). Reilly et al (1984) reported a 19mm loss in sitting height over the course of a day (equivalent to 1.5mm per disc (Adams et al 1987)), 54% of the loss occurred in the first 30 minutes after rising. The hydration level of an IVD directly impacts its flow-independent and flow-dependent viscoelastic properties (van der Veen et al 2005, Race et al 2000, Costi et al 2002, Adams et al 1990, Costi et al 2002). It changes the molecular bonds, the saturation and bulk modulus, the porosity and the permeability of the disc (Iatridis et al 1997). Given the change in the viscoelastic behavior of a saturated disc the hydration level of the disc is an important variable when considering injury mechanisms. It has been shown previously to accelerate and alter the injury process. Lu et al (1996) constructed a 3-dimensional finite element model of the disc, described above. Their model showed that saturated discs fail sooner than dehydrated discs when loaded in compression, flexion

and side-flexion. This finding paralleled the findings of Gunning et al (2001) in an in vitro study in which they investigated the failure tolerance of specimens with one of three hydration levels – the super hydrated discs were the first to fail. A compressive force equal to body weight can expel 10-15% of the water from cadaveric discs in 4 hours (Adams et al 1987). Given the significance of the hydration level of the disc some consideration has been given to the optimal method of maintaining hydration during in vitro testing – enclosing the specimen in a humidity/solution chamber (Brinckmann et al 1988, Adams and Hutton 1985) or wrapping the specimen in saline soaked gauze and/or wrapping the specimen in plastic (Gordon et al 1991, Adams et al 2000, Callaghan and McGill 2001) are the more commonly used methods. Given that the osmotic pressure of the lumbar spine has been reported as ranging from 0.15 to 0.2MPa and that hydrostatic pressure ranges from 0.6 to1MPa when measured under normal daily loading, it appears the best method to rehydrate the disc, although not always practical, is unloading the disc during testing. Andersson and Schultz (1979) measured the mechanical properties of lumbar motion segments both before and after saline injection and found that those segments that retained the saline had reduced mean motion, especially in flexion and lateral bending. The volume of saline injected was not reported but the volume was sufficient to raise the intradiscal pressure to 3-6 times the non-injected value. A compression level of 400N was used for all loading tests. As will be described in subsequent chapters, dehydration of the specimens and the associated disc height loss appears to be a limitation of this in vitro methodology. The biomechanical implications of such changes are discussed from mechanical and clinical perspectives.

Effects of Freezing Specimens

The freeze-thaw process that is necessary to the storage of in vitro specimens also affects hydration of the disc. Leahy and Hukins (2001) found that the elastic modulus of the nucleus pulposus of sheep IVD was greater in specimens that had been frozen prior to testing while Bass et al (1997) reported greater permeability in frozen specimens. The water content was significantly higher in fresh specimen but no significant correlation between the elastic modulus, loss modulus, phase angle and water content was found. Dhillion et al (2001) showed that freezing has an insignificant effect on the elastic and time-dependent properties of human IVDs. They tested 20 human motion segments, 50% of which were tested, frozen for 3 weeks and retested while the other 50% were frozen for 3 weeks and then tested. Each specimen was subjected to 5 cycles of compressive creep under 1MPa for 20 minutes, followed by a 40-minute recovery under no load. Similarly, Smeathers and Joanes (1988) reported that the ice crystal growth during the freeze-thaw cycle will slightly disrupt the organization of the IV disc so rendering it less stiff and less resilient as observed, but these changes were very small relative to experimental errors and specimen variation. Separate to freezing,

unloading the spine after death results in swelling of the hydrophilic discs. This has lead to the practice of applying an axial compression preload to standardize the hydration level of the specimen at the start of in vitro testing (Adams 1995, Callaghan and McGill 2001, Gordon et al 1991).

In summary, the McKenzie approach is a widely accepted classification system in clinical practice that has been shown to have clinical merit but its underlying conceptual model remains largely unsubstantiated. Investigation of the mechanics of the conceptual model in an in vitro laboratory required thorough consideration of the proposed post-herniated porcine cervical disc in the absence of a definitive understanding of the best replica of the in vivo herniated disc. The awareness and compromise of the limitations of the in vitro work are highlighted. Knowing the existing understanding and knowledge of relevant aspects of clinical practice, healthy and pathological disc mechanics, diagnostic imaging and in vitro testing provides a context for the series of studies in this thesis, which are as follows:

Study 1 (Chapter 2):

Creating Disc 'Herniation' In Vitro: Is There In Vivo - In Vitro Disc Height Equivalency?

This study compared in vitro and in vivo herniated discs in an attempt to link the two and provide a more thorough understanding of the in vitro model proposed to test the mechanical theory underlying the McKenzie derangement approach. Wilke et al (2006) reported a high level of agreement (KAPPA 0.862) between disc height loss and mild, moderate and severe morphological classifications of disc degeneration; 1-33% of disc height loss matching mild degeneration, 33% - 66% loss indicative of moderate degeneration and disc height loss of >66% identifying severely degenerate discs. This classification system was used to categorize the disc height loss that occurred in these specimens after the in vitro testing.

H: A modified version of the in vitro methodology of Callaghan and McGill (2001) will produce disc herniations in porcine cervical spines without concurrently causing moderate to severe disc height loss (>33% of pre-test disc height).

Study 2 (Chapter 3):

Attempts To Create Disc Herniations; Successes And Failures

The second study describes various attempts at paralleling the porcine in vitro model to the targeted in vivo discs. This study aimed to bridge the discrepancy in the disc height of the in vitro and the targeted in vivo herniated discs. Axial compression of 1472N (study 1) resulted in moderate levels of disc height loss. Higher and lower levels of axial compression, both shown by other researches to

result in disc herniation, were investigated and the disc height of the post-tested porcine cervical specimens quantified. Torque control of the test movements, rather than position control, had been shown to be less damaging to the discs (Callaghan and McGill 2001) and was considered another possible means of preserving disc height while creating herniations. Alternative means of re-hydrating the discs was also considered. The additional artificial hydration methods attempted in this study included frequent saline injection during testing, injection of the radio-opaque mix during testing (Adams and Hutton 1985), immerging the specimen in a saline bath for a prolonged period of time (Gunning et al 2001) and administering saline to the specimen via an in situ IV line. Three key alternative methods of creating herniation were also worthy of consideration, ruptured the annulus using a scalpel prior to loading the specimens. Artificially starting the herniation process by rupturing the annulus (Brinckmann 1986, Keller et al 1990, Simunic et al 2001); injecting pressured air into the disc (Oliphant et al 2006) and using the relatively hypo-lordotic porcine thoracic segments (Farcy et al 1997, Keegan 1953, LaGrone 1988) were modifications of the methodology of study 1 that were attempted to preserve the disc height of the specimens while concomitantly herniating them.

H: The hypotheses of this study were that (1) modifying the loading pattern that had previously been used to cause herniation, (2) rehydrating the discs during testing and/or (3) using a different model, would create disc herniation without causing more than 33% of disc height loss.

Chapter 4:
The McKenzie Approach To Disc Prolapse – Evidence Of A Mechanical Foundation?

The focus of the third study is the mechanical investigation of the McKenzie clinical theory of the treatment response seen in vivo in herniated discs. Specifically, it is theorized that a displaced portion of nucleus in a damaged disc can be moved back towards the center of the disc using movements and positions. This study is a proof of the principle on which this aspect of the McKenzie approach is based.

H: Repeated motion, opposite to the motion that caused the disc prolapse, will reverse the position of the displaced portion of nucleus.

H: Discs that do not respond to reversal testing will have extensive circumferential annular tears or will have a herniation with extrusion of nuclear material

H: The specimens that prolapse will have been dissected from hypolordotic spines.

Chapter 5:

McKenzie Extension Exercises – Quantified And Considered

The McKenzie approach to the treatment and prevention of (MLBP) is a two-tiered approach that involves identifying the appropriate exercises to be performed by the client as well as the specific motions, postures and loads to be avoided in order to accelerate recovery and reduce the risk of recurrence of the MLBP. In general, for a posterior or posterolateral disc prolapse the recommended exercises involve extension movements while flexion movements are those to be avoided.

H: The extension of EIL would be greater than that of EIS

H: The range of extension would be increased during and after extension mobilization

H: The peak flexion, ± side bend and rotation, ranges of the ADL investigated match those previously used to create disc prolapse.

CHAPTER 2

CREATING DISC 'HERNIATION' IN VITRO: IS THERE IN VIVO - IN VITRO DISC HEIGHT EQUIVALENCY?

ABSTRACT

Background; Numerous loading mechanisms have been proposed to create disc herniation in vitro, a clinical entity with which a portion of the nucleus is displaced into the annular layers of the intervertebral disc (IVD). An intra- and inter-disciplinary lack of consensus on the terminology and morphology associated with herniation exists. Consideration of the equivalency of the disc damage created by an in vitro porcine model to that seen in vivo is vital to research investigating in vivo treatment effects.

Methodology; The C3/4 segments of 10 porcine cervical spines were dissected from porcine spines and potted in ultra high-density polyethylene cups for testing. In order to track the position of the nucleus radiologically 0.55ml of a radio-opaque mixture ($BaSO_4$, Coomasie blue dye, distilled water, the C2/3 nucleus) was injected into the C3/4 IVD through the anterior annulus using a 20 gauge needle. Anterior and lateral view X-ray images were taken prior to testing to establish that the disc was healthy and that the nucleus was contained within the annulus. The specimens were wrapped in a layer of saline (0.9% NaCl) soaked plastic-backed material and a layer of polythene film to prevent dehydration and placed in a servohydraulic dynamic testing machine. Each specimen was preloaded (260N for 879s) to reverse the effects of freezing, following which the potted specimens were removed from the testing machine and anterior and lateral view X-ray images were taken in a standard frontal and transverse plane position prior to further testing (the pre-test x-ray images). The failure test involved repeatedly flexing or flexing and side flexing the specimens under 1.472kN of axial compression at a rate of 45^0/s and a frequency of 1Hz. The x-ray procedure was repeated intermittently (at least every 30 minutes) during the failure test and again after the 90 minutes of testing (post-test x-ray images). The pre- and post-test disc height was measured from the lateral x-ray images by two observers. The stiffness of the specimens was calculated from the torque-angular deformation curves during the failure testing.

Results; High inter-rater reliability (intraclass correlation = 0.90) was found for disc height measurement of the specimens before and after testing. Three of the 10 specimens had posterior

migration of the nucleus. Clinically significant (>33%) disc height loss occurred. The stiffness of specimens increased significantly ($p < 0.01$) during the herniation procedure.

Conclusion; The methodology of repeatedly flexing while axially compressing porcine cervical specimens previously used to create herniation was associated with simultaneous moderate disc height loss of the specimen. Similar levels of disc height loss occurred whether the specimens herniated or not. This level of disc height loss has previously been shown to be associated with moderate degenerative changes, less movement-related changes in intradiscal pressure and an altered load distribution across the spinal segment. Efforts to modify the methodology and reduce the disc height loss are warranted when the mechanical changes associated with such disc height loss could potentially impact the results of specific research.

INTRODUCTION

Numerous loading mechanisms have been proposed to create disc herniation (Callaghan and McGill 2001, Aultman et al 2005, Andersson 1981, Brinckmann 1986, Lappalainen et al 2002, Freeman et al 2003, Cinotti et al 2005, Kelsey et al 1984, Gordon et al 1991, Lu et al 1996). Early studies attempted to produce herniation in the lumbar discs with static loads in compression. Several investigators found end-plate fracture before any evidence of disc injury (Brown et al 1957, Brinckmann et al 1988, Wilder et al 1988). Brinckmann (1986) applied 1000N of compression to the lumbar discs of human cadavers when held in a neutral position, the annulus of which had been internally divided except for the outer 1mm. Despite the weakened annulus, disc extrusion at the site of the annular division was never found. Adams and Hutton (1985) subjected the lumbar motion segments of cadavers to fatigue loading in cyclic compression and static flexion and successfully prolapsed 6 of the 29 discs tested. More recently, researchers investigated the potential to herniate discs under compression and repeated single or multiple plane motion (Gordon et al 1991, Callaghan and McGill 2001, Aultman et al 2005). Callaghan and McGill (2001) created herniation in porcine cervical discs by applying repeated flexion/extension under low-moderate levels of compression. Gordon et al (1991) produced disc ruptures with 100% consistency by adding rotation to flexion and compression. Ten of these discs failed through annular protrusions while four failed by nuclear extrusion through annular tears. Likewise, Aultman et al (2005) successfully produced herniation in 15 out of 16 discs with repeated combined flexion and side flexion under moderate levels of compression. Although high repetitions of motion are required to cause disc rupture, these in vitro studies have largely attempted to parallel in vivo physiological loading to gain an understanding of the in vivo cause of herniations. Many others have opted to create herniation non-physiologically (surgical stab models, endplate disruption models, computer-based models) with the goal of investigating the factors that modulate the incidence of herniation or the biomechanical factors impacted by herniation (Lappalainen et al 2002, Freeman et al 2003, Cinotti et al 2005, Simunic et al 2001, Belytschko et al 1974). For example, Simunic et al (2001) investigated the impact the hydration level of the disc had on their ability to cause nuclear extrusion through a full division of the annular wall created using a scalpel in bovine caudal discs. The discs, loaded in compression and flexion under various hydration levels, failed more readily when more hydrated, echoing the findings of Gunning et al (2001). Keller et al (1990) also ruptured the annulus of discs, first with a scalpel and then a coring tool, to investigate the changes in the creep-recovery response of the damaged discs. Cinotti et al (2005) found that disc bulges occurred in many of the discs upon which they had inflicted endplate injury in in-vivo porcine discs, a study designed to investigate the histological and biochemical degenerative effects of endplate injury. A number of computer models have been

designed to facilitate investigation of disc rupture (Shirazi–Adl 1989, Belytschko et al 1974). Belytschko et al (1974) used a finite element model to consider the impact of radial annular tears on the behavior of the (IVD) and found lower intradiscal pressure and greater compression and bulging with annular tears. In summary, this literature describes a number of the many physiological and non-physiological methods of creating disc damage in human, animal and computer models. Central to the above studies is displacement of a portion of the nucleus into the annular layers, however, the terminology and the definition of the terminology used to describe this IVD damage, the scale classifying the damage, the extent of the damage created and the characteristics of disc degeneration that co-exist with specific damage are not necessarily equivalent. Consideration of the equivalency of the disc damage created by these various methods and models to that seen in vivo is vital to research investigating in vivo treatment effects. This study compares in vitro and in vivo herniated discs in an attempt to link the two and provide a more thorough understanding of the post-herniated porcine cervical in vitro model.

Terminology relating to and grading of disc herniation

"Protrusion, extrusion….confusion", the title of a 1993 editorial letter (Swartz 1993) that emphasized the intra- and inter-disciplinary lack of consensus on the meaning of the terms herniation, protrusion, sequestration, bulging and extrusion. The lack of consensus extends to whether or not these terms are morphologically and/or clinically distinct enough to be of value. Despite a number of studies designed to categorically define these terms, universally accepted definitions are not available (Milette et al 1999, Yussen and Swartz 1993). Even more obscure is an understanding of the clinical significance of these morphological terms used in diagnostic imaging reports and in academia. The details of the confusion will be discussed in a subsequent paper but for the purpose of this paper suffice it to highlight the inconsistency and to provide a work-in-progress definition of the term herniation, being the more commonly used of these terms in research, as the posterior displacement of a portion of the nucleus through the annular layers to at least the outer 1/3 of the annulus and no further than the outer border of the annulus. In addition to the confusion posed by disc damage terminology, the scales classifying the damage only exacerbate the problem. Many of the scales used in research to classify disc herniation describe a spectrum of the characteristics of degenerative disc disease (DDD) with herniation embedded in the scale as one these characteristics. Kettler and Wilke (2006) searched the literature and reviewed the existing grading systems for lumbar disc degeneration. They found 22 different grading systems (Table 2.1). The design of these grading systems vary considerably in terms of the starting point of the scale, the number of grades in the scale, the nomenclature of the grades, the grade that describes herniation versus other DDD

Classification of Grading System	Grading System
Macroscopic Anatomy	Nachemson 1960
	Galante 1967
	Thompson et al 1990
	Ziv et al 1993
	Adams et al 1996
Histology	Gunzburg et al 1992
	Berlemann et al 1998
	Boos et al 2002
Plain Radiography	Kellgren and Lawrence 1952
	Kellgren et al 1963
	Gordon 1991
	Mimura et al 1994
	Lane et al 1993
	Madan et al 2003
Magnetic Resonance Imaging	Scheiderman et al 1987
	Butler et al 1990
	Tertti et al 1991
	Gunzburg et al 1992
	Pfirrmann et al 2001
Discography	Adams et al 1996
	Schneiderman et al 1987
	Gunzburg et al 1992

Table 2.1. The classification systems of DDD reported by Kettler and Wilke (2006)

characteristics, the factors combined at each grade as well as the appropriateness of the scale to interdisciplinary (in vitro and in vivo) use. For example, herniation is described as Grade 3 of the 5-point (grades 1-5) macroscopic anatomy scale of Thompson et al (1990). This grade is associated with early osteophytes and focal defects of the endplate cartilage while a mention of disc height is not made. Grade 4 in Pfirrmann et al's (2001) magnetic resonance imaging (MRI) scale has normal to moderately decreased disc space associated with the changes in the annulus and nucleus but osteophytes and changes of the endplate cartilage are not described. Meanwhile, Mimura et al (1994) in their radiological scale, include 5-8 osteophytes of < 3mm and disc height loss of up to 50% in their definition of grade 2 DDD (grades 0-4). While Lane et al (1993) in their radiography scale define grade 1 DDD as having 'small' osteophytes with definite but mild narrowing of the joint space (disc height). While the sensitivity of the imaging method may account for some of the variation in the grading system (Cihangiroglu et al 2004, Auerbach et al 2006, Birney et al 1992, Boden 1996), the differences in these morphological, macroscopic anatomical and radiological DDD and herniation grading systems make it very difficult to compare and contrast the disc damage created. Consideration of how herniation and disc degeneration are inter-related and whether or not disc herniation exists independent of DDD is a first step to understanding our ability to identify the disc damage that is most appropriate to this body of work.

Degenerate discs versus herniated discs

Adams and Roughley (2006) recently defined disc degeneration as an aberrant, cell-mediated response to progressive structural failure, which when painful is called DDD. DDD is characterized by progressive fibrosis with loss of distinction between the annulus and the nucleus, as well as loss of organization of the annular lamellae and thinning of the cartilaginous endplates (Jensen et al 1994, Roberts et al 2006, Giles and Kaveri 1991). The relative composition of the proteoglycans is altered, which impedes the ability of the IVD to withstand force without releasing water (Brown 1971). Radiologically, the cardinal features of disc degeneration are said to be disc space narrowing and osteophyte formation (Lawrence 1969). Losses of disc height and signal intensity have also been identified as characteristic of late disc degeneration as seen on MRI (Schiebler et al 1991). Benneker et al (2005) found that disc height loss, as determined from plain x-ray film, was significantly correlated with the morphological grade of DDD. Frobin et al (2001) in their MRI–radiological study concluded that the early process of disc degeneration precedes the process of disc height loss but later stages of degeneration are correlated with disc height loss, findings similar to that of Berlemann et al (1998). Wilke et al (2006) reported a high level of agreement (KAPPA 0.862) between disc height loss and mild, moderate and severe morphological classifications of disc degeneration; 1-33% of disc

height loss matching mild degeneration, 33% - 66% loss indicative of moderate degeneration and disc height loss of >66% identifying severely degenerate discs. In addition to the cellular and hydration changes of disc degeneration, disc herniation is a particular form of structural damage that is also associated with degeneration (Osti et al 1992, Jensen et al 1994) but two schools of thought concerning the etiology of herniation exist, one being degeneration precedes herniation while the other claims that herniation precipitates degeneration (Stokes and Iatridis 2004). In the case of the former, it is thought that accelerated and premature biochemical changes in the disc, possibly genetically driven, reduce the ability of the disc to withstand load leaving it vulnerable to eventual herniation (Roughley 2004, Hendry 1958, Battie et al 1995, Sambrook 1999). This pathway is unlikely to target only a single level. In contrast, the latter case is more likely seen at a specific level. In this case repeated mechanical loading of or trauma to the disc causes the disc to herniate, which then interrupts the nutritional pathways and proteoglycan synthesis in the disc precipitating a cascade of non-reversible cell-mediated responses leading to further disruption (Grunhagen et al 2006, Chen et al 2006, Battie 2004, Anderson et al 2002, An et al 2004, Bibby and Urban 2004, Bibby et al 2005, Buckwalter 1995, Adams and Dolan 1995, Lipton and Muir 1981). It is conceivable that both pathways can occur. Holodny et al (2000) distinguish between herniations in 'normal-appearing' discs and those in degenerate discs in a study that considers the cause-and-effect relationship between the volume of herniated material and the resulting disc height loss. Eighty-four percent of the 51 herniations discs considered occurred in discs that were at most mildly degenerate. The level of degeneration was classified according to the MRI based system of Yu et al (1989), which includes a five level evaluation of signal intensity and disc height. A number of herniation case studies described in the literature indicate normal radiographic images and thus the absence of the signature DDD characteristics of disc height loss and osteophyte formation. Unfortunately, in larger in vivo herniation studies and in many health care systems MRI and discography of in vivo herniations are not performed for at least 3 months after the initial onset of symptoms, allowing adequate time for cell-mediated degeneration of the disc to occur subsequent to the initial herniation (Sobajima et al 2004). Given the timeline, it is difficult to determine from MRI which is the chicken and which is the egg. A number of studies have shown that hydrated discs are herniate faster than dehydrated discs (Lu et al 1996, Gunning et al 2001), a finding that is suggestive of a separate mechanism of failure than that related to herniation in degenerative discs. Taylor and Twomey (1985) state that true nuclear herniation is a condition of young people while disc degeneration is a condition seen in elderly discs with which significant disc height loss is associated. Decreased disc height has been shown to increase the facet joint forces, reduce the movement-related change in intradiscal pressure and result in a portion of the axial compressive force being transmitted though the neural arch

(Dunlop et al 1984, Adams et al 1988, Adams and Hutton 1980, Adams et al 1994, Adams et al 2000, Rohlmann et al 2005). DDD and the associated disc height loss can result in spinal canal or intervertebral foramina stenosis, conditions that are clinically different from a herniated disc. Young in vivo herniations differ in presentation and prognosis from discs that have marked degeneration, even if herniation coexists (Adams and Hutton 1985). This clinical distinction seems to be camouflaged by the DDD scales used to classify herniation in research.

What is clear from the literature is that a spectrum of severity of disc degeneration exists (Wilke et al 2006, Frobin et al 2001, Schiebler et al 1991, Benneker et al 2005, Jensen et al 1994, Lawrence 1969) that herniations have been associated with the spectrum of degenerate discs (mild-severe) (Hendry 1958, Lipton and Muir 1981, Battie et al 1995, Adams and Dolan 1995, Sambrook et al 1999, Holodny et al 2000), and that disc height loss is a reasonable indicator of the severity of degeneration in a disc (Frobin et al 2001, Schiebler et al 1991, Benneker et al 2005, Wilke et al 2006). In degenerate discs, greater disc height loss is associated with a more fibrosed nucleus (Adams and Roughley 2006, Jensen et al 1994), less movement-related changes in intradiscal pressure (Adams et al 1994, Adams et al 2000, Rohlmann et al 2005) and a change in the load distribution of the spinal segments (Dunlop et al 1984, Adams et al 1988, Adams and Hutton 1980, Adams et al 1994, Adams et al 2000, Rohlmann et al 2005). Given that disc height loss has been used to distinguish between different levels of DDD it was considered reasonable to use disc height loss to identify the in vitro disc herniations that are most appropriate to investigation of a clinical entity to which hydration, a hydrostatic nucleus, intradiscal pressure and inter-segmental load distribution are important factors. A review of normal disc height, the disc height loss associated with DDD as well as a review of techniques to measure disc height, are paramount to distinguishing between herniated discs that coexist with mechanical characteristics of mild versus moderate – severe degeneration.

Disc Height

The height of the IVD is influenced by several factors. Reversible height changes of approximately 8-20% (Botsford et al 1994, Adams et al 1987) have been reported to occur diurnally due to the dynamic balance between the compressive forces on the disc and the disc's osmotic pressure. Proteoglycans endow the disc matrix with a high osmotic pressure and a low hydraulic permeability and hence constitute the compression-resisting component of the disc. Chrondrotin sulphate and keratin sulphate, both proteoglycans, contain negatively charged acidic groups, the concentration of which controls the osmotic pressure of the disc. High osmotic pressure results in, and maintains under external load, hydration of the disc. Furthermore, the rate at which fluid can flow into or out of the disc under a pressure driven force depends on the hydraulic permeability of the

matrix which is directly related to pore size distribution, determined by proteoglycan concentration. When the proteoglycan concentration is low (i.e. the disc is well hydrated) then the pore size is large and the fluid flow from the tissue is fastest. As fluid is lost from the tissue the proteoglycan concentration increases and the rate of fluid loss slows. The in vivo proteoglycan concentration of the disc is high thus without compression (caused by body weight, ligaments and muscles) the disc will swell. The relative composition of the proteoglycans is altered in degenerate discs, which impedes the ability of the IVD to withstand force without releasing water (Brown 1971). Farfan (1973) defined disc height as the mean of the distance between ventral corners and the dorsal corners of the superior and inferior vertebrae and expressed disc height in units of disc depth to allow for radiographic distortion (Figure 2.1). Using this measurement technique, Tibrewal and Pearcy (1984) compared the disc height of the L4/5 and L5/S1 discs of healthy subjects to those of subjects that had prolapsed discs. The only clinical information provided about the subjects with prolapsed discs was that they all subsequently underwent discectomy, following which disc height was also reported. Statistically significant decreases in disc height were found after the discectomy in comparison to controls. Non-significant differences of 0.5-2mm (<15%) were found between the discs of the healthy controls and those of subjects with prolapsed discs pre-surgery. Tibrewal and Pearcy (1984), using Farfan's (1973) method to measure disc height, reported an intra-observer error of a maximum difference of 0.7mm and inter-observer error of a maximum mean difference between observers of 0.75mm. Based on Farfan's 1973 definition of disc height, Frobin et al (1997) used a technique called distortion-compensated roentgen analysis (DCRA) to measure disc height (Figure 2.2). In the DCRA technique the disc height is defined as the sum of the perpendicular distances from the ventral corner of the cranial vertebra and the ventral corner of the caudal vertebra to the bisectrex of the disc (Frobin et al 1997, Frobin 2001). This disc height is normalized to the mean depth of the cranial vertebra and in healthy subjects, mean values of this quotient range from 0.23 – 0.42 (Frobin et al 2001). The benefits of this technique are that the ventral landmarks are imaged virtually free from distortion, the disc height measurement is not influenced by sagittal plane displacement and small deviations in the rotation and lateral tilt of the spine when x-rayed don't alter the disc height measurement. The relative error of disc height as measured by the DCRA method was calculated to be 4.93%. Frobin et al (2001) reported an inter-observer KAPPA index of 0.82, considered to be a high level of agreement by Landis and Koch (1977). Frobin et al (1997) compiled a normative disc height database of human lumbar spines based on 892 lateral radiographic views. Segments with altered spine structure (due to osteoporosis, ankylosing spondylitis, scoliosis, Scheuermann's disease, metastatic disease, previous spinal surgery) were excluded. The subject pool (16-57 years old) was further amended to include only measurements of non-pathological discs (Excluding for example irregularities of endplates,

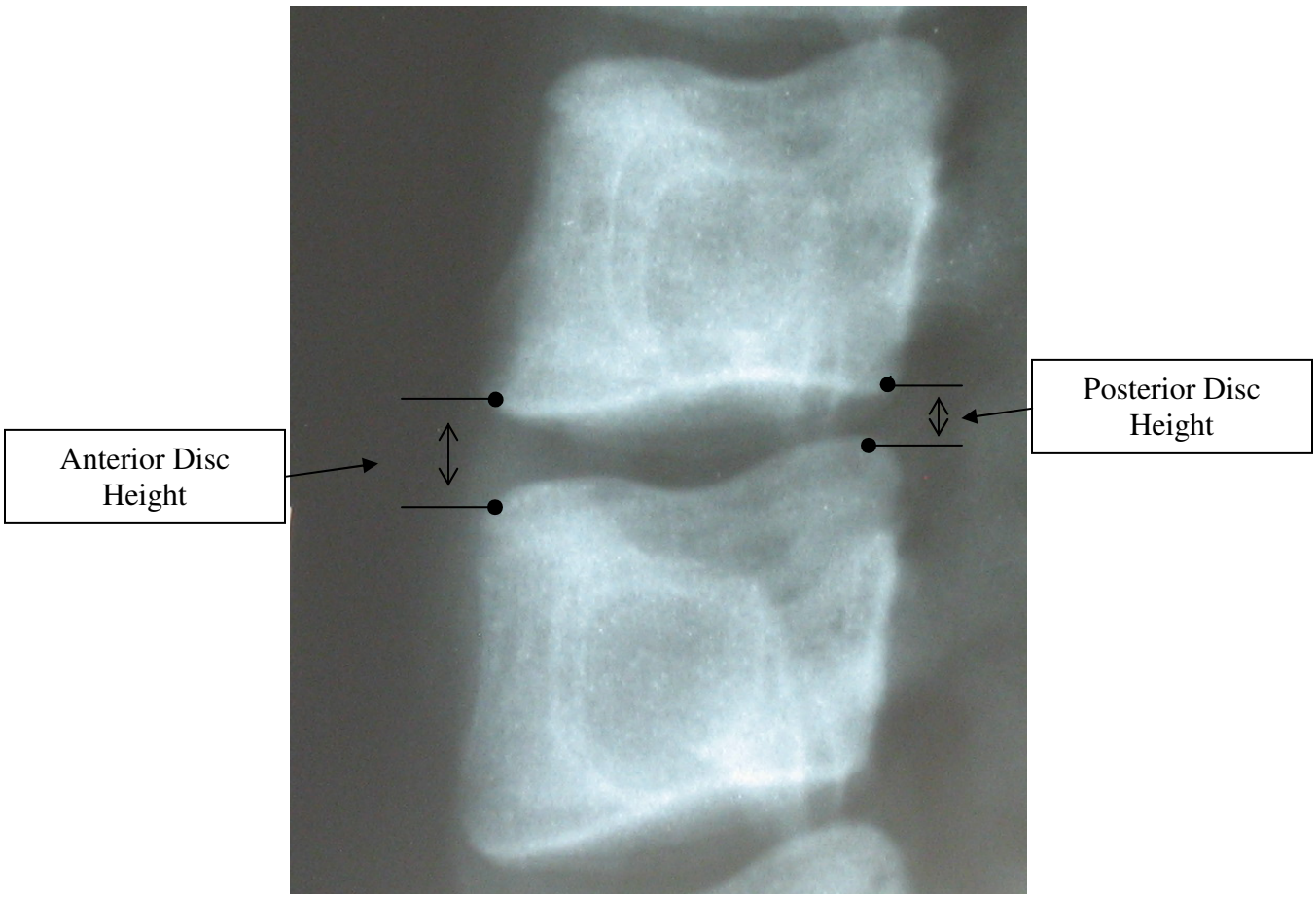

Figure 2.1: Farfan et al (1973) defined disc height as the mean of the distance between ventral edges and the dorsal edges of the superior and inferior vertebrae. The edges of the vertebral bodies are identified as the points that are furthest from the center of the vertebral body. Disc height was expressed in units of disc depth to allow for radiographic distortion.

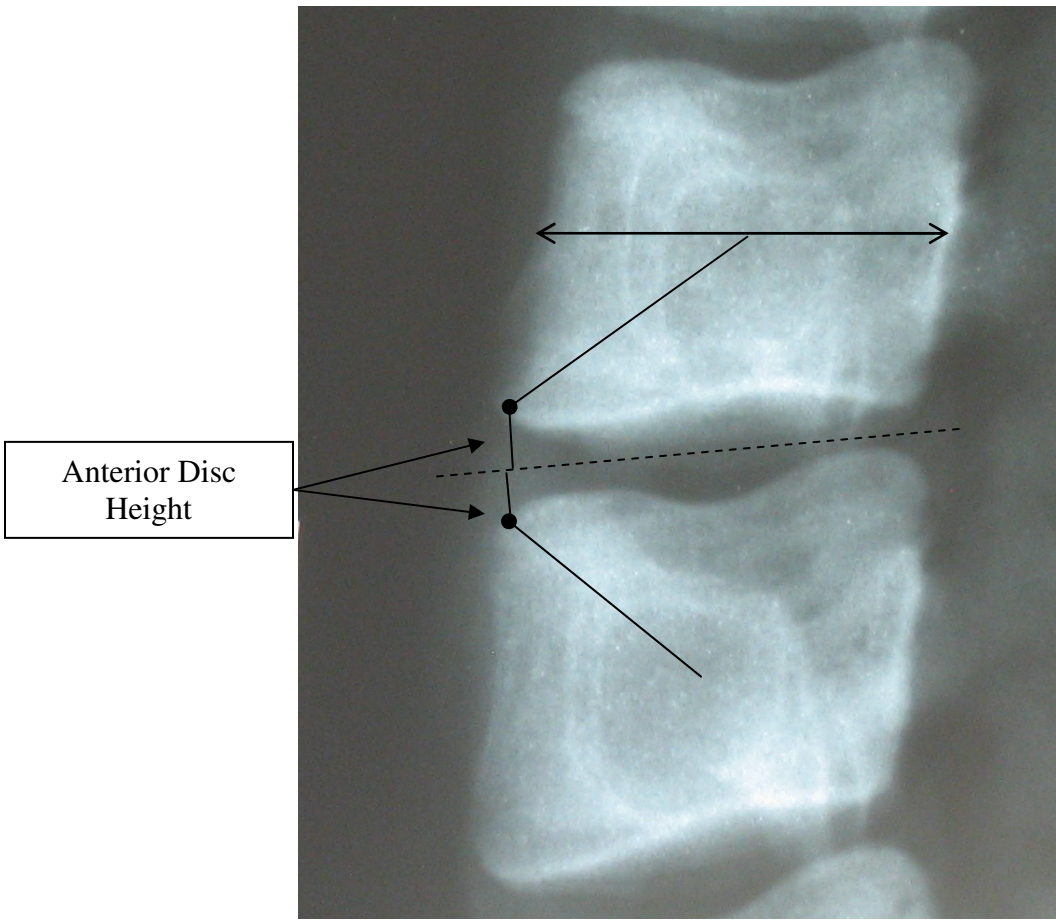

Figure 2.2: Frobin et al (1997) measured the anterior disc height as the sum of the perpendicular distances from the anterior edges of the superior and inferior vertebral bodies (anterior black dots) to the midplane of the disc (dashed line) and normalized it to the width of the superior vertebral body (solid arrowed horizontal black line). The anterior edges of the vertebral bodies are identified as the points that are furthest from the center of the vertebral body.

sagittal plane displacement and decreased disc height). The scatter of disc height in this subject pool was small (<12%), suggesting that in the absence of pathology there is not an age-related decrease in disc height. The disc height depended on gender (males >females). Disc height loss due to pathology was considered by Wilke et al (2006) (Figure 2.3). Anterior and posterior disc height was measured in a manner similar to Frobin et al (1997, 2001) and expressed as a percentage of normal values (Frobin et al 1997). Wilke et al (2006) reported a high level of agreement (KAPPA 0.862) between disc height loss measured in this way and the degree of degeneration seen on MRI, as determined by two experienced observers. In this classification system discs with height loss of 1- 33% are classified as mildly degenerate, those with 33% - 66% loss are considered moderately degenerate, while those with disc height loss of >66% are considered severely degenerate (Wilke et al 2006).

In summary, in addition to diurnal disc height changes of 8-20% (Adams et al 1987, Botsford et al 1994) pathological disc height loss can occur due to loss of the proteoglycan water-binding molecules (Brown 1971, Bogduk 1997). A number of different disc height measurement schemes have been reported in the literature (Farfan et al 1973, Frobin et al 1997, Wilke et al 2006) and the severity of the disc height loss associated with different levels of degeneration has been defined (Wilke et al 2006, Mimura et al 1994).

Summary of Introduction

Science has produced a number of computer and in vitro models that facilitate a greater understanding of the loading that causes a disc to herniate (Callaghan and McGill 2001, Aultman et al 2005, Andersson 1981, Brinckmann 1986, Lappalainen et al 2002, Freeman et al 2003, Cinotti et al 2005, Kelsey et al 1984, Gordon et al 1991, Lu et al 1996) but differences in terminology hinder a thorough understanding of the specific and coexisting damage created (Nachemson 1960, Galante 1967, Thompson et al 1990, Ziv et al 1993, Adams et al 1996, Gunzburg et al 1992, Berlemann et al 1998, Boos et al 2002, Kellgren and Lawrence 1952, Kellgren et al 1963, Gordon 1991, Mimura 1994, Lane et al 1993, Madan et al 2003, Scheiderman et al 1987, Butler et al 1990, Tertti et al 1991, Gunzburg et al 1992, Pfirrmann et al 2001, Adams et al 1996). The term herniation is used in academia within the framework of degenerate disc disease, of which a spectrum of severity exists, (Frobin et al 2001, Schiebler et al 1991, Benneker et al 2005, Jensen et al 1994, Lawrence 1969) while clinically in a young otherwise healthy disc the term herniation is associated with a pattern of symptoms, signs and movement behavior that differs from that of moderate-severe DDD herniation (Stokes and Iatridis 2004, Holodyn et al 2000, Adams and Dolan 1995, Taylor and Twomey 1985, Adams and Hutton 1985, Lipton and Muir 1981). Disc height loss has been shown to be an indicator of the severity of degeneration in a disc (Frobin et al 2001, Schiebler et al 1991, Benneker et al 2005,

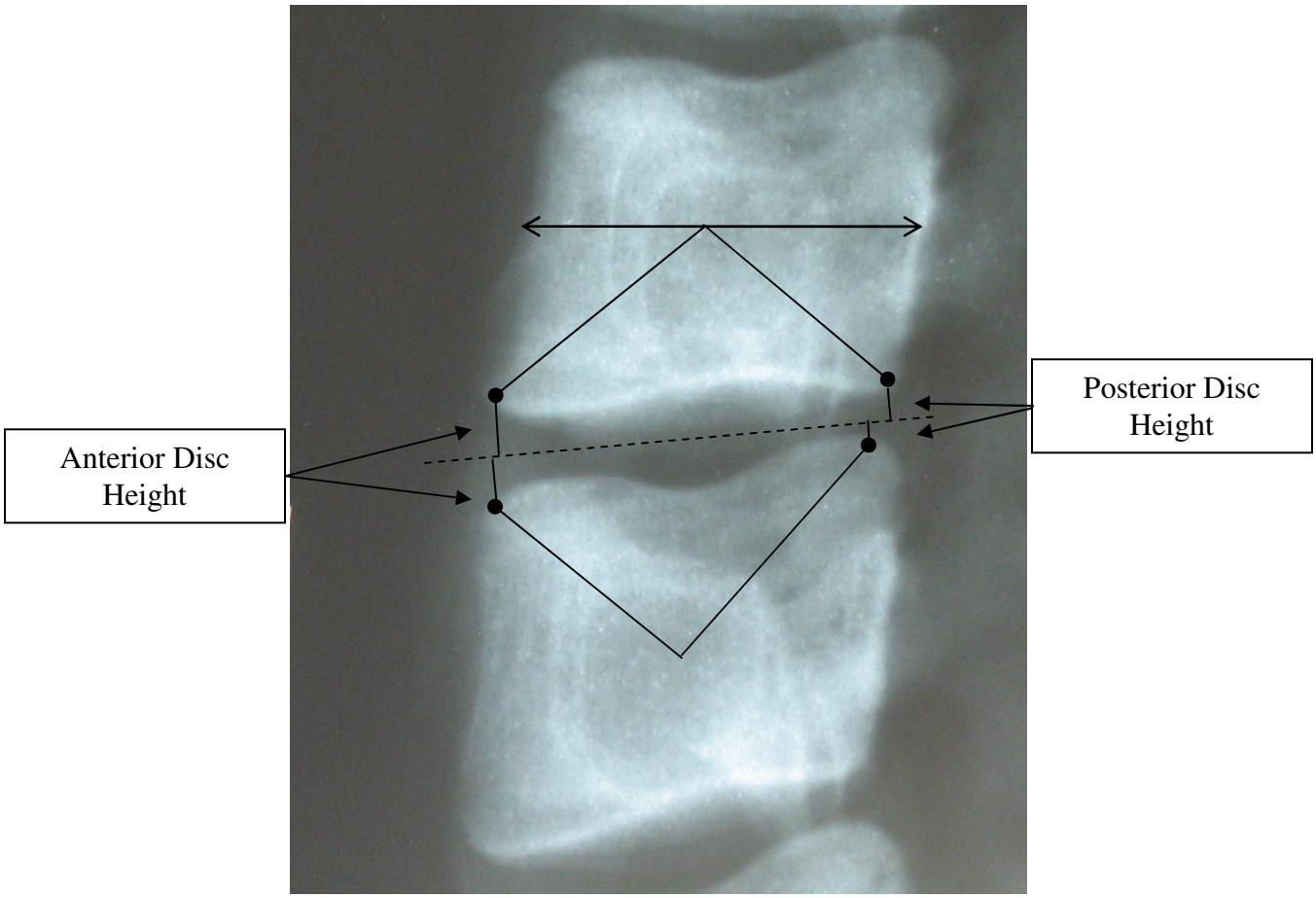

Figure 2.3: Wilke et al (2006) calculated anterior disc height as the sum of the perpendicular distances from the anterior edges of the superior and inferior vertebral bodies (anterior black dots) to the midplane of the disc (dashed line). The edges of the vertebral bodies were identified as the points that were furthest from the center of the vertebral body. Posterior disc height was calculated as the sum of the distances from the posterior edges of the superior and inferior vertebral bodies (posterior black dots) to the midplane of the disc. Both in Wilke et al (2006) and in the current study the disc height was measured directly from the lateral x-ray images by each observer. Wilke et al (2006) expressed disc height as a percentage of the normal values reported by Frobin et al (1997). In the current study disc height loss was expressed as a percentage of the pre-test disc height (post-preload) and the anterior and posterior disc height loss were averaged to give one figure for disc height loss.

Wilke et al 2006). Greater disc height loss is associated with a more fibrosed nucleus (Adams and Roughley 2006, Jensen et al 1994), less movement-related changes in intradiscal pressure (Adams et al 1994, Adams et al 2000, Rohlmann et al 2005) and an altered load distribution across the spinal segment (Dunlop et al 1984, Adams et al 1988, Adams and Hutton 1980, Adams et al 1994, Adams et al 2000, Rohlmann et al 2005). While challenging amid the terminology confusion and different inter-disciplinary perspectives, it is pertinent to strive to understand the proposed in vitro herniated disc model in both a clinical and scientific context. Does this model display mechanical characteristics of herniation and mild degeneration or those of herniation and moderate-severe degeneration? Given that disc height loss has been used to distinguish between different levels of DDD it was considered reasonable to use disc height loss to make this distinction. A number of disc height measurement techniques have been documented (Farfan et al 1973, Frobin et al 1997, Wilke et al 2006) and are compared in this study.

Objectives and purpose of this study

The objective of this study was to assess whether the proposed in vitro post-herniated porcine model is representative of a herniated but at most mildly degenerate disc. The importance of understanding the disc height loss associated with the post-herniated porcine cervical specimen relates to the altered disc mechanics associated with disc height loss. The classification of mild degeneration was based on disc height loss of 1-33%, as per Wilke et al (2006). The purpose of understanding the in vitro post-herniated porcine model is to facilitate future investigation of clinical treatment approaches and to ensure that the interpretation of such research is precise given the clinical question being investigated and the model utilized. The hypothesis of this study was that a modified version of the methodology of Callaghan and McGill (2001) would create, in porcine specimens, herniations associated with at most 33% disc height loss (Wilke et al 2006).

METHODOLOGY

Creating disc herniation

The C3-6 segments of 10 porcine cervical spines were dissected from porcine spines that had been bagged and frozen immediately post-mortem and subsequently thawed at room temperature for 12-15 hours prior to dissection. A lateral x-ray image (Mercury Modular X-ray, 007 mas, 100ma, 54 kvp) of the C3-6 intact segments was taken before the C3/4 osteoligamentus specimens were dissected. The method used to create disc herniation is based on that of Callaghan and McGill (2001). The C2/3 and C4/5 (IVDs) were examined for degeneration and all specimens were classified as Grade 1 on

Galante's scale of disc degeneration. For the purposes of testing, the specimens were potted (wired using 18 gauge steel wires and cemented with non-exothermic dental stone (Denstone ®, Miles Inc., South Bend, IN, USA)) in ultra high-density polyethylene cups. Wood screws were inserted, to a maximum depth of 1 cm, into both the top cup and center of the C3 vertebral body and the bottom cup and center of the C4 vertebral body to ensure bonding. In order to track the position of the nucleus radiologically a total of 0.55ml of a radio-opaque mixture ($BaSO_4$ mixture) was injected into the C3/4 IVD through the anterior annulus using a 20 gauge needle. The mixture included a solution of barium sulphate, Blue dye (consisting of 250mg Coomassie brilliant blue, 97.25 ml of distilled H_20 and 2.5 ml of methanol) and distilled water, in a 2:1:2 ratio, with which the C2/3 nucleus was mixed. The C2/3 nucleus was the harvested nucleus of the adjacent segment which was mixed with the radio-opaque solution to eliminate the possibility that the less viscous $BaSO_4$ mixture could herniate through annular clefts/ ruptures that the more viscous nucleus would not. The needle aperture was sealed with superglue after the injection. A layer of saline (0.9% NaCl) soaked plastic-backed material and a layer of polythene film were wrapped around the specimens to rehydrate them. The specimen was placed in a servohydraulic dynamic testing jig (model 8511, Instron Canada, Burlington, Ont., Canada), which had been modified to apply both axial compression and single plane pure moments simultaneously (Callaghan and McGill 2001). An unconstrained testing apparatus was utilized so the cup containing the C3 segment was fixed in position, the center of the testing plate in line with the geometric center of the C2/3 disc, while the lower cup, containing the C4 segment was free to translate and rotate on a low friction surface (Figure 2.4). Each specimen was preloaded (260N for 879s) to reverse the effects of freezing during which time the Instron found a position of zero torque for the specimen. Following preload the potted specimens were removed from the jig and anterior and lateral view X-ray images were taken prior to further testing (the pre-test x-ray images). Moulds supporting the upper and lower cups were made to maintain a standard frontal and transverse plane position of the specimen during the x-ray process. As earlier pilot work had shown that the position of the nucleus was not altered whether the specimen was x-rayed in a pre-determined sagittal plane position or as determined by the stiffness of the specimen after testing it was decided not to fix the sagittal plane position of the specimens for x-ray. The argument being that clamping the specimens into a pre-determined position would have undesirably varied the compression of the specimens at the time of x-ray and additionally made it more difficult to maintain the standard frontal plane position of the specimens. In order to identify and standardize the maximum and minimum angles to be used for dynamic failure testing the torque-angular deformation relationship of each specimen was recorded (Figure 2.5). To do this each specimen was axially compressed (1472N) and rotated 5 times, in either pure flexion or combined flexion and side flexion (depending on the

direction of the test motion), at a rate of 0.5^0/s during which the torque-angular deformation curve was plotted. The angle at which the stiffness of the torque – angular deformation curve of the specimen increased was the maximum angle used in the dynamic testing and an angle 10^0 less than the maximum was the minimum angle used. The first 7 specimens were repeatedly flexed under axial compression of 1472N at a rate of 45^0/s and a frequency of 1Hz for 90 minutes. In an attempt to accelerate the herniation of the disc with the possibility of less disc height loss the direction of the test motion was changed to combined flexion and side flexion for the remaining 3 specimens. These 3 specimens were also tested under axial compression of 1472N at a rate of 45^0/s and a frequency of 1Hz for 90 minutes. The x-ray procedure was repeated intermittently (at least every 30 minutes) during the failure test and again after the 90 minutes of testing (post-test x-ray images).

DATA ANALYSIS

The change in the position of the nucleus visible on the lateral x-rays was grossly classified according to the discographic scale of Adams and Hutton (1986). In order to allow for comparison with the existing disc height literature disc height was measured and reported in absolute terms and then normalized to the depth of the vertebral body as per Farfan (1973), according to the DCRA method as per Frobin et al (1997) and as a percentage as per Wilke (2006). The measurement methods of Farfan (1973), Frobin et al (1997) and Wilke et al (2006) are discussed above. The specimen disc height was measured directly from the pre-test and post-test x-ray images were measured according to Farfan et al (1973), Frobin et al (1997) and Wilke et al (2006) by the investigator on two occasions (6 months apart) and also by an independent physiatrist who is experienced in reviewing radiological images (twice on the same occasion). A two-way random absolute agreement model intraclass correlation coefficient (ICC) was used to assess intra- and inter-rater reliability of disc height measurement. Repeated measures ANOVAs were also used to compare the absolute (prior to normalization) disc height results measured according to Farfan et al (1973), Frobin et al (1997) and Wilke et al (2006). The average of the disc height measurements of both observers were used for further analysis. Paired t-tests were used to test if significant changes ($p<0.01$) in disc height and stiffness occurred after the failure procedure. Repeated measures ANOVAs were used to test if significant between- and within-group (herniated or not, direction of testing) differences occurred in pre- and post-test disc height and stiffness. The stiffness of the specimens was recorded as the slope of the line joining the minimum to maximum angles of the repeated motion on the torque-deformation curve. The mean stiffness over the first 10 cycles of the dynamic test was considered the pre-test stiffness and was compared to that of the last 10 cycles, the

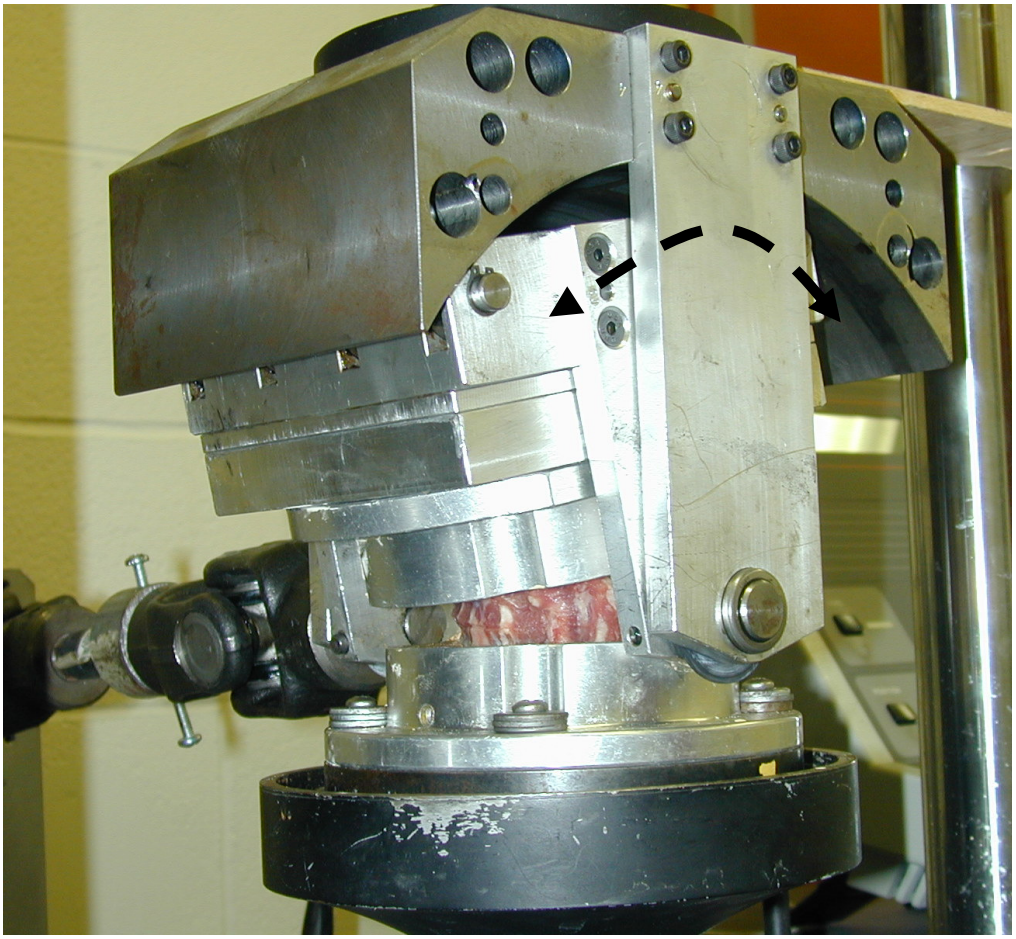

Figure 2.4: A photograph of a specimen, wired and cemented in cups, positioned in the servohydraulic jig. The plate rotated, indicated by the black arrow, causing the determined motion. The specimen was wrapped in saline soaked material during the testing.

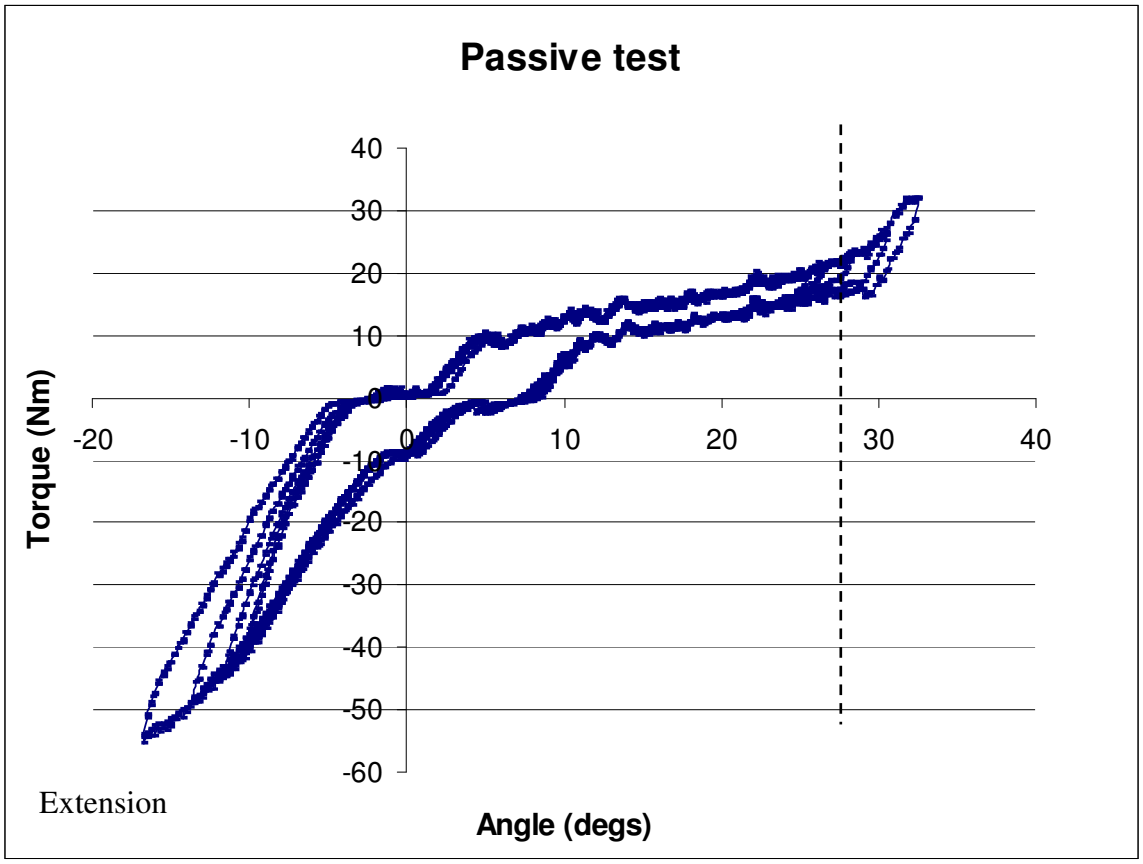

Figure 2.5: In order to determine the range of cyclic motion used to create disc herniation the specimens were rotated in flexion or flexion and side bend under 1472N of axial compression. The torque and angular deformation during these cycles was graphed and the angle at which the stiffness of the specimen changed was identified, indicated by the dashed line. This angle was the maximum angle used in the testing procedure. The minimum angle used was 10^0 less than this angle.

final stiffness. A two-way Pearson's correlation was used to analyze the relationship between disc height and stiffness.

RESULTS

All 10 specimens tested initially had a healthy disc with the nucleus contained within the inner walls of the annulus. Three of the 10 discs tested had posterior migration of the nucleus after the herniation procedure, while 1 of the 10 herniated anteriorly. As the specimen that herniated anteriorly was not notably different from the other specimens in size, stiffness or shape it is assumed that the needle aperture was inadequately sealed and this specimen was not included in the analysis. The posteriorly herniated discs were all grade 4 on the discographic scale of Adams et al (1986) described as a degenerated disc with a radial fissure leading to the outer edge of the annulus.

High inter-rater reliability (ICC = 0.90) was found for disc height measurement of the specimens, measured according to Farfan et al (1973), Frobin et al (1997) and (Wilke et al 2006). Table 2.2 shows the mean (and one standard deviation) pre-test and post-test disc height measurements of the specimens in this study and are compared to corresponding disc height data from the literature. The absolute pre-test anterior and posterior porcine disc height measurements are smaller than the disc height of healthy human L4/5 and L5/S1 segments reported by Tibrewal and Pearcy (1984), both measured according to Farfan (1973). However, when the disc height is normalized to the depth of the vertebral body, the pre-test disc height measurements of these specimens are within the normal disc height range of healthy human in vivo discs (Frobin et al 1997). This suggests that the vertebral body and disc height are similarly proportioned in human lumbar and porcine cervical spines.

Statistically significant ($p<0.001$) disc height loss (Wilke et al 2006) occurred during the dynamic failure testing relative to that of the pre-test disc height. The mean disc height loss following testing was 57.05% (SD 11.19%), which is comparable to the disc height loss of moderate disc degeneration (Wilke et al 2006). No significant differences ($p = 0.87$) in the mean disc height loss of specimens that posteriorly herniated (60.14 SD 7.50%) and those that did not herniate (55.55% SD 13.02%) were found. Similarly, no significant difference ($p= 0.35$) in disc height loss was found between specimens that were tested in flexion versus those that were tested in combined flexion and side flexion under the same axial load. The mean stiffness of the segments had increased significantly ($p < 0.001$) from 1.25 (SD 0.22) to 2.11 Nm/degree (SD 0.46) by the end of the test. The change in stiffness did not correlate with the change in disc height.

	Current study			Tibrewal et al (1985)		
	Mean Disc Height (mm)					
	Mean Anterior (SD)	Mean Posterior (SD)	Mean	Anterior (Range)	Posterior (Range)	Mean
	Healthy specimen					
Porcine C3/4	6.25	3.53	4.98			
Human L1-2				8 (6-11)	4 (2-6)	6
Human L2-3				11 (8-13)	4.5 (2-6)	7.75
Human L3-4				12.5 (11-15)	4.5 (3-6)	8.5
Human L4-5				14 (11-16)	5.5 (3-8)	9.75
Human L5-S1				13 (9-16)	4.5 (3-6)	8.75
	Herniated specimen					
Porcine C3/4	2.92	1.63	2.27			
Human L4-5				11	3.5	7.25
Human L5-S1				11	4	7.5

	Current study	Frobin et al (1997)
	Mean disc height (SD)	
	Healthy specimen	
Porcine	0.28 (0.04)	
Human L1-2		0.27 (0.04)
Human L2-3		0.32 (0.04)
Human L3-4		0.37 (0.04)
Human L4-5		0.41 (0.05)
Human L5-S1		0.40 (0.05)

	Current study	Wilke et al (2006)
	*Mean (SD) Disc Height loss (%)	DDD Equivalency
Porcine Post Testing (All specimens)	57.08 (11.19)	Moderate DDD
Porcine Post Herniation	60.14 (7.50)	Moderate DDD

Table 2.2: The disc height measurements of the specimens in this study are shown and compared to those in the literature. The disc height compared with those of Tibrewal et al (1985) was measured according to Farfan et al (1973). *Statistically significant disc height loss occurred. These levels of disc height loss have previously been associated with moderate degenerative disc disease.

DISCUSSION

This study was designed to assess the ability of an in vitro methodology to produce disc herniations in porcine cervical spines without concurrently causing moderate (33-66%) to severe (>66%) disc height loss. The specimens that herniated, and those that did not, simultaneously lost >50% of their pre-test disc height. Only one specimen lost less than 50% (35%) of the pre-test disc height. It appears that although this methodology can successfully create disc herniations in porcine cervical specimens significant disc height loss occurs concurrently. It remains a valuable methodology and model for the investigation of issues relating to human disc failure, however, the impact of the more than 50% disc height loss on subsequent investigation of the herniated disc may be an important consideration.

The limitations of this study include the use of an animal model and the small sample size, divided between two test groups. Animal models are a necessary part of the quest for a greater understanding of in vivo biomechanics. These models are controllable, reproducible and cost-effective, unlike specimens from human cadavers where age, exercise level, diet, environment, personal factors and genes add variability to a sample. More specifically, the porcine cervical spine model has been shown to be anatomically, geometrically, functionally (Yingling et al 1999, Oxland et al 1991) and microscopically (Tampier et al, in press) similar to human lumbar spines and was not only a reasonable surrogate for human spines but also provided a reproducible model. The use of an in vitro model to replicate an in vivo phenomenon hinders the interpretation of such work in an in vivo context. Consideration of the impact of freezing spines and of eliminating the usual in vivo cellular changes of the disc are warranted. Dhillon et al (2001) and Callaghan and McGill (1995) have shown that freezing does not change the time-dependant behavior nor elastic properties of the frozen specimens and thus freezing is not considered to significantly alter the results obtained from this in vitro model. The number of repetitions of motion applied to the specimens in this study would occur in a two week period in vivo (Brinckmann et al 1988). Collagen repair and proteoglycan turnover would not occur in such a short timeframe in vivo (Adams and Hutton 1982, Urban et al 2004). The specimen pool of 10 was justified by the fact that using an animal model from a population that was homogenous in age, exercise, diet, and breed reduced individual variability of the specimens. Furthermore, the behavior of the specimen in the sample was similar, irrespective of the direction of the test motion used, reducing the need to increase the sample size.

While a number of studies have created disc herniations in vitro the coexisting disc height loss has not always been reported. Adams and Hutton (1985) caused posterior migration of the nucleus in 6 out of 29 human cadaveric lumbar specimens by applying increasing cyclic compression (sin-wave,

0.3Hz, final compression mean = 3451.67N) to progressively flexed specimens (mean maximum flexion angle = 14^0). Disc height pre- or post- testing was not reported in this study. Contrary to findings of the current study, Gordon et al (1991), in a study designed to create disc herniations in human cadaveric lumbar specimens, reported that all 14 of the specimens tested had 'at most minimal narrowing' prior to and after the failure testing. Callaghan and McGill (2001) focused on creating, and successfully created, disc herniation in vitro by loading porcine cervical specimens in repeated flexion and low-moderate levels of compression. Axial creep of 8-11mm was reported in Callaghan and McGill (2001) and a trend towards increased axial creep with increased compressive load was found. The current study largely followed the methodology of Callaghan and McGill (2001) and similarly created disc herniations under a moderate level of axial compression and repeated flexion or combined flexion and side flexion. The appropriateness of using a porcine cervical model to represent human lumbar discs was supported by Callaghan and McGill (2001) by comparing the anatomical, geometrical and functional differences and similarities between human lumbar and porcine cervical spines (Yingling et al 1997). The in vitro post-herniated cervical porcine discs match in vivo human herniated discs in that a portion of the nucleus is displaced posteriorly through a damaged annulus. However, the coexisting disc height loss of the in vitro specimen needed to be considered and quantified. Herniation has been associated with various levels of degeneration but the clinical presentation of such herniations differ (Stokes and Iatridis 2004, Holodyn et al 2000, Adams and Dolan 1995, Taylor and Twomey 1985, Adams and Hutton 1985, Lipson and Muir 1981). Disc height loss has been earmarked as a characteristic that distinguishes between mild, moderate and severe levels of disc degeneration (Frobin et al 2001, Schiebler et al 1991, Benneker et al 2005, Wilke et al 2006) and with which important mechanical changes have been associated including less movement-related changes in intradiscal pressure (Adams et al 1994, Adams et al 2000, Rohlmann et al 2005) and an altered load distribution across the spinal segment (Dunlop et al 1984, Adams et al 1988, Adams and Hutton 1980, Adams et al 1994, Adams et al 2000, Rohlmann et al 2005). The current methodology produced herniated discs that had disc height loss equivalent to moderately degenerated discs (Wilke et al 2006, Mimura et al 1994). Wilke et al (2006) reported the agreement between height loss and their macroscopic (MRI) classification to be 0.862 (KAPPA value). The cellular change that disc height loss is attributed to is a depletion of the hydrophilic proteoglycan component of the disc (Osti et al 1990, Melrose et al 1992, Kaapa et al 1994), a depletion that occurs with aging (Roughley 2004) and at an accelerated rate with degeneration. In vivo, the degeneration can be part of a disease process, occurring at multiple levels, or due to an injury at a particular level resulting in progressive cellular changes (Stokes and Iatridis 2004). Associated with these cellular changes is increasing fibrosis of the nucleus. A fibrotic nucleus looses its hydrostatic properties,

hindering its ability to flow and thus migrate into a damaged annulus. In other words the cellular changes associated with disc height loss impede the ability of the disc to herniate. Interestingly, the disc height loss associated with in vitro testing cannot be attributed to these cellular changes and is more likely related to the prolonged continuous axial compression and accelerated loading of the methodology. The dehydration and disc height loss caused by repeated motion in the specimens created potentially problematic levels of disc height loss. As discussed earlier in this paper, the changes in mechanics imposed by disc height loss are of utmost importance to this body of work. Disc height loss of 20, 40 and 60% of normal was included in a finite element model to represent mild, moderate and severe degeneration in a study designed to analyze the influence of disc degeneration on the mechanical properties of the L3/4 motion segment (Rohlmann et al 2005). The key results of the analysis were; (1) The range of flexion, extension and lateral bend and coupled motion decreased for moderately and severely degenerate discs; (2) The change of intradiscal pressure (IDP) in the nucleus on flexion, extension, lateral bend and rotation is lower in degenerate discs than in healthy discs; (3) The pressure is direction dependent and the nucleus is no longer hydrostatic in degenerate discs; (4) The facet joint forces are higher in degenerate discs than in healthy discs. Dunlop et al (1984) also showed in an in vitro cadaver study that decreasing disc height by 1 and 4 mm significantly increased the intra-articular facet pressure. In addition, a portion of the axial compressive force is transmitted to the neural arch when the disc looses height (Dunlop et al 1984, Adams et al 1988, Adams and Hutton 1980, Adams et al 2000, Adams et al 1994). The disc height loss found in this study falls within the moderate to severe DDD levels as categorized by Mimura et al (1994) and Wilke et al (2006). The previously reported changes in load distribution and in the hydrostatic pressure of discs with decreased disc height imply that attempts to improve this post-herniated porcine disc model are warranted. The (ICC) found in this study are considered high and closely match that of Wilke et al (2001) (KAPPA 0.90) and Frobin et al (2001) (KAPPA 0.82). Similar to the results of Tibrewal and Pearcy (1985), the intra-observer error showed a mean maximum difference of 0.49mm from the mean of two readings of 20 measurements and an inter-observer error mean maximum difference of 0.7mm between the two observers.

The immediate future work that arose from this paper was a study aimed at reducing the disc height loss that occurred concurrently with this method of creating herniation. Modifying the loading pattern that has previously been used to cause herniation, artificially hydrating the discs and/or using a different model are potential solutions to the disc height loss problem and will be discussed in chapter 3. In addition, this study prompts numerous avenues of research aimed at bridging the gap between the research and clinical perspectives of low back pain including the links between clinical

patterns and the morphological, neurophysiological and chemical parameters utilized in in-vitro research.

CONCLUSION

The methodology of repeatedly flexing while axially compressing porcine cervical specimens previously used to create herniation is associated with simultaneous moderate disc height loss of the specimen. Similar levels of disc height loss occurred whether the specimens herniated or not. This level of disc height loss has been associated with moderate degenerative changes, less movement-related changes in (IDP) (Adams et al 1994, Adams et al 2000, Rohlmann et al 2005) and an altered load distribution across the spinal segment (Dunlop et al 1984, Adams et al 1988, Adams and Hutton 1980, Adams et al 1994, Adams et al 2000, Rohlmann et al 2005). Efforts to modify the methodology and reduce the disc height loss are warranted when the mechanical changes associated with such disc height loss could potentially impact the results of specific research.

CHAPTER 3

ATTEMPTS TO CREATE DISC HERNIATIONS; SUCCESSES AND FAILURES

ABSTRACT

Background; Disc herniations when created in an in vitro porcine model show a loss of the disc height of the specimens. Such disc height loss is of concern to researchers using the post-herniated porcine specimens as disc height loss has been shown by others to change the load distribution and hydrostatic pressure of the disc. The purpose of this study was to improve the post-herniated porcine model such that the disc height loss that occurred simultaneously with the herniation was reduced.

Methodology; The attempts made to achieve this purpose were broadly categorized as (1) modifying the loading pattern that had previously been used to cause herniation, (2) re-hydrating the discs after a period of testing and (3) using a different model. Dissected from porcine cervical spines, the C3/4 osteoligamentus specimens were potted in ultra high-density polyethylene cups and tested in a servohydraulic jig. In order to track the position of the nucleus radiologically a mixture of barium sulphate, Blue dye, distilled water and the C2/3 nucleus was injected into the C3/4 nucleus. Anterior and lateral x-ray images taken before, during and after testing.

Modifying the loading pattern: Six specimens were repeatedly flexed in a 10^0 range under 1kN of axial compression at a rate of 45^0/s and a frequency of 1Hz. Four other specimens were prepared for higher axial compression testing. The intent was to repeatedly flex these specimens under 2 and 2.596kN of axial compression at a rate of 45^0/s and a frequency of 1Hz for 90 minutes. Three more specimens were tested under torque rather than position control. All three were loaded with 1.472kN of axial compression. The torque levels chosen were based on the torque-angular deformation curve of each specimen. The specimens were repeatedly flexed in a 10^0 range at a frequency of 1Hz.

Re-hydrating the discs: Four porcine C3/4 specimens underwent 90 minutes of repeated flexion testing in a 10^0 range under 1.472 kN of axial compression at a rate of 45^0/s and a frequency of 1Hz. After post-test x-ray images the specimens were placed unloaded in a submersion saline (0.9% NaCl) bath for approximately 12 hours, after which the specimens were re-x-rayed. Three other specimens were injected with 0.1ml of distilled water through a 25 gauge needle after every 30 minutes of repeated flexion of the specimens when under 1 kN of axial compression at a rate of 45^0/s and a frequency of 1Hz. Eight more specimens (6 C3/4 and 2 T7/8) were repeatedly flexed in a 10^0 range under 1.472kN of axial compression at a rate of 45^0/s and a frequency of 1Hz. Three specimens were re-injected with barium sulphate-nucleus mix after 20 minutes of dynamic testing. Five other

specimens were injected with the barium sulphate mix after undergoing sufficient dynamic testing to cause loss of approximately 50% of their pre-test disc height.

Using a different model: Prior to potting the right postero-lateral annulus of five porcine C3/4 specimens was ruptured using a 2 mm blade that was inserted through the left antero-lateral annulus. The specimen then underwent 90 minutes of repeated combined flexion and left side flexion testing in a 10^0 range under 1 kN of axial compression at a rate of 45^0/s and a frequency of 1Hz. In a separate test axial compression of 0.3kN, 1kN and 1.472kN was applied to 3, 4 and 2 T7/8 porcine specimens respectively and the specimens were repeatedly flexed in a 10^0 range at a rate of 45^0/s and a frequency of 1Hz for 90 minutes.

Results; Repeated flexion of porcine cervical specimens (n = 6) under a lower compression level (1kN) resulted in disc herniation but with loss of 50% of the pre-test disc height ($p < 0.001$). Re-hydrating specimens by injecting the disc with a barium sulphate nucleus mix (n = 5) or by placing the specimen unloaded in a submersion saline bath for an extended period of time (n = 4) resulted in a significant increase of the disc height of the specimens. Further flexion testing of the specimens significantly reduced the disc height again. Intermittent saline injection of specimens (n = 3) during the failure procedure did not prevent or reduce the disc height loss that occurred in the absence of saline injections. Using higher compression levels (2 and 2.5kN, n = 4), failure testing under torque control (n = 3), non-physiologically starting the annular rupture (n = 5) and using hypolordotic thoracic porcine spines (n = 12) instead of porcine cervical spines were unsuccessful attempts at creating herniations.

Conclusion; The disc height loss that coexists with the porcine cervical disc herniations that were created with repeated flexion under axial compression is clinically significant. This model appears to be a valuable model for investigation of herniated discs that have substantial disc height loss and the related mechanics (loss of hydraulic behavior, motion, foramen space and changes in facet loading). Consideration of the limitations posed by the concurrent disc height loss prior to future use of this model and methodology are imperative.

INTRODUCTION

Disc herniations have been created in an in vitro porcine model to the detriment of disc height of the specimens. Such disc height loss is of concern to researchers using the post-herniated porcine specimens as disc height loss has previously been shown to change the load distribution and hydrostatic pressure of the disc (Rohlmann et al 2005, Dunlop et al 1984, Adams et al 1988, Adams and Hutton 1980, Adams et al 2000, Adams et al 1994). An example of such research is investigation of the proposed mechanical theory underlying the clinical success of the McKenzie treatment of disc herniation (Derangement syndrome). McKenzie theorized that in a disc with a competent annulus and a hydrostatic nucleus particular repeated movement or sustained positioning will positively change the location of the displaced portion of the nucleus. This theory involves fluid flow, hydrostatic pressure and stress gradients across the disc. These factors will be changed by alterations in disc height and hydration level of the disc. This, coupled with indications that excessive disc height loss is not characteristic of young in vivo herniations (Schiebler et al 1991, Frobin et al 2001, Holodny et al 2000, Taylor and Twomey 1994 and 1985, Adams and Hutton 1985) necessitates attempts to eliminate or reduce the disc height loss that occurs simultaneously with in vitro herniation. An in vitro model that more closely matched the young herniated discs most successfully treated with McKenzie's derangement would facilitate a greater understanding of the proposed mechanical theory underlying the McKenzie approach to disc herniation. The attempts made to achieve this purpose were broadly categorized as (1) modifying the loading pattern that had previously been used to cause herniation, (2) re-hydrating the discs during testing and (3) using a different animal model. Callaghan and McGill (2001) successfully herniated porcine cervical spines by applying low-moderate levels of compression to the specimen while cycling the specimen repeatedly between predetermined flexion and extension angles. Compression levels approximately equivalent to that of standing (867N) and light lifting (1472N) were used, the higher the compression level the lower the number of repetitions required to herniate the disc and the higher the incidence of more severe disc herniation (grade 4 versus grade 3). As shown 1472N of axial compression resulted in moderate levels of disc height loss investigation of the disc height of the post-herniated porcine cervical specimen created with lower axial compression is warranted. Interestingly also warranted is investigation of the impact higher levels of compression would have on disc height. Adams et al (1985) created herniations using higher compression levels and as the number of repetitions required to create herniation is a function of the compression level, higher compression levels could ultimately preserve disc height. A review of Callaghan and McGill (2001) also suggests that torque control of

the test movements is less damaging to the discs, creating grade 3 rather than grade 4 herniations, a finding that led to another possible means of preserving disc height while creating herniations.

Alternative means of re-hydrating the discs was also considered. The dynamic balance between the compression forces on the disc and the disc's osmotic pressure determines the hydration level of the disc in vivo. Hydration of the discs in vivo is primarily achieved by diffusion through the endplates (Adams and Roughley 2006). Traditionally in vitro specimens are wrapped in saline-soaked gauze to minimize water loss during testing. This method of maintaining hydration of the specimens seems inadequate as the dynamic balance between compression and osmotic pressure tips in favor of compression, which results in disc height loss. The additional artificial hydration methods attempted in this study included frequent saline injection during testing, injection of the radio-opaque mix during testing (Adams & Hutton 1985), placing the specimen in a saline submersion bath for a prolonged period of time (Gunning et al 2001) and administering saline to the specimen via an in situ IV line. Three key alternative methods of creating herniation were also worthy of consideration. Brinckmann (1986), Keller et al (1990) and Simunic et al (2001) ruptured the annulus using a scalpel prior to loading the specimens. This start on the herniation process was considered a reasonable method of accelerating the damage and of possibly reducing the disc height loss inherent to the methodology. Following the methodology of Oliphant et al (2006), who created visible disc disruption by injecting cadaveric porcine lumbar discs with nitrogen gas at pressures of 69 kPa, 172 kPa, 345 kPa and 690 kPa, injecting pressured air into the disc was considered a possible means of preserving disc height while accelerating the herniation process. Alternatively porcine thoracic, rather than cervical segments were subjected to the herniation method with the expectation that these spines would herniate sooner than the cervical spines. Clinically, the hypolordotic spine is thought to be predisposed to low back pain (LBP) as it lacks the inbuilt protection of lordosis (Farcy et al 1997, Keegan 1953, LaGrone 1988). The lower thoracic segments of the pig are hypolordotic relative to the cervical spines of the same species.

The purpose of this study was to create herniations in vitro without causing more than 33% of disc height loss. Disc height loss of less than 33% is associated with mild disc degeneration (Wilke et al 2006). The attempts made to achieve this purpose were broadly categorized as (1) modifying the loading pattern that had previously been used to cause herniation, (2) rehydrating the discs during testing and/or (3) using a different model. The hypotheses of this study were that these new methods would create disc herniation prior to causing more than 33% (Wilke et al 2006) of disc height loss.

METHODOLOGY

The preparation of the specimens that is identical across the tests, regardless of the specific differences in subsequent testing, will be described and then followed by the details of the various subsequent tests. The porcine cervical spines had been bagged and frozen immediately post-mortem and thawed at room temperature for 12-15 hours prior to dissection. The C3/4 osteoligamentus specimens were dissected. The adjacent discs were examined for degeneration and all specimens were classified as Grade 1 on Galante's scale of disc degeneration. For the purposes of testing the specimens were wired (18 gauge steel wires) and cemented (non-exothermic dental stone (Denstone ®, Miles Inc., South Bend, IN, USA)) in ultra high-density polyethylene cups. Wood screws were inserted, to a maximum depth of 1 cm, into both the top cup and center of the C3 vertebral body and the bottom cup and center of the C4 vertebral body to ensure bonding. In order to track the position of the nucleus radiologically 0.55ml of a radio-opaque mixture (Barium sulphate-nucleus mixture) was injected into the C3/4 intervertebral disc (IVD) through the anterior annulus using a 20 gauge needle. The mixture included a solution of barium sulphate, Blue dye (250mg Coomassie brilliant blue, 97.25 ml of distilled H_20 and 2.5 ml of methanol) and distilled water, in a 2:1:2 ratio, with which the C2/3 nucleus was mixed. The C2/3 nucleus was the harvested nucleus of the adjacent segment which was mixed with the radio-opaque solution to eliminate the possibility that the less viscous $BaSO_4$ mixture could herniate through annular clefts/ ruptures that the more viscous nucleus would not. The needle aperture was sealed with superglue after the injection. A layer of saline (0.9% NaCl) soaked plastic-backed material and a layer of polythene film were wrapped around the specimens to rehydrate them. A lateral x-ray image (Mercury Modular X-ray, 007 mas, 100ma, 54 kvp) of the specimen was taken in a standard frontal and transverse plane position before it was placed in a servohydraulic dynamic testing jig (model 8511, Instron Canada, Burlington, Ont., Canada), which was modified to apply both axial compression and single plane pure moments simultaneously (Callaghan and McGill 2001). An unconstrained testing apparatus was utilized so the cup containing the C3 segment was fixed in position, the center of the testing plate in line with the geometric center of the C2/3 disc, while the lower cup, containing the C4 segment was free to translate and rotate on a low friction surface. Each specimen was preloaded (260N for 879s) to reverse the effects of freezing and to determine a position of zero torque of the specimen, following which the potted specimens were removed from the jig and anterior and lateral view X-ray images were taken prior to further testing (the pre-test x-ray images). In order to identify the maximum and minimum angles to be used for dynamic failure testing the torque-angular deformation relationship of each specimen was recorded. To do this each specimen was axially compressed to the level that

would be used for the dynamic failure test (see below) and rotated 5 times, in pure flexion at a rate of 0.5^0/s during which the torque-angular deformation curve was plotted. The angle at which the stiffness of the torque – angular deformation curve of the specimen increased was the maximum angle used in the dynamic testing and an angle 10^0 less than the maximum was the minimum angle used. The specimen was removed from the jig and anterior and lateral view X-ray images were taken prior to further testing.

1. Modifying the load (1, 2, 2.596 kN) and control (torque control) used to create herniation

a. Lower Axial Compression

The pre-test storage, preparation, potting and preliminary testing of these specimens was as described above. Six specimens were repeatedly flexed in the 10^0 range (described above) under 1kN of axial compression at a rate of 45^0/s and a frequency of 1Hz. The testing was discontinued when the specimens herniated or lost more than 50% of their pre-test disc height. The x-ray procedure was repeated every 30 minutes during and at the end (post-test x-ray images) of the failure test.

b. Higher Axial Compression

The pre-test storage, preparation, potting and preliminary testing of these specimens was as described above. Four specimens were prepared for this testing. The intent was to repeatedly flex these specimens under 2 and 2.596kN of axial compression at a rate of 45^0/s and a frequency of 1Hz for 90 minutes. All four specimens failed in the preparatory stages of the test.

c. Torque Control

The pre-test storage, preparation, potting and preliminary testing of these specimens was as described above. Three specimens were tested under torque rather than position control. All three were loaded with 1.472kN of axial compression. The torque levels chosen were based on the torque-angular deformation curve of each specimen. The torque level at which the stiffness of the torque – angular deformation curve of the specimen increased was the maximum torque used in the dynamic testing. Minimum torque was identified as the torque that coincided with an angle 10^0 less than that of the angle at maximum torque. The specimens were repeatedly flexed at a frequency of 1Hz. These tests were to run until herniation occurred or 50% of the pre-test disc height was lost.

2. Changing the hydration of the specimen

a. Saline bath

The pre-test storage, preparation, potting and preliminary testing of these specimens was as described above. Four porcine C3/4 specimens underwent 90 minutes of repeated flexion testing in a 10^0 range (as described above) under 1.472 kN of axial compression at a rate of 45^0/s and a frequency of 1Hz. The x-ray procedure was repeated every 30 minutes during the failure test and again after the 90 minutes of testing (post-test x-ray images). The specimens were then placed unloaded in a submersion saline (0.9% NaCl) bath for approximately 12 hours, after which the specimens were re-x-rayed (post-rehydration x-ray images). Specimens that had not herniated during the initial 90 minutes of testing were placed in the testing machine for another 60 minutes flexion testing. The x-ray procedure was repeated every 30 minutes during the continued failure test and again after the 90 minutes of testing (post-test (2) x-ray images).

b. Saline re-injection

The pre-test storage, preparation, potting and preliminary testing of these specimens was as described above. Three specimens were injected with 0.1ml of distilled water through a 25 gauge needle after every 30 minutes of dynamic testing. The specimens were unloaded at the time of injection. The dynamic testing consisted of repeated flexion of the specimens when under 1 kN of axial compression at a rate of 45^0/s and a frequency of 1Hz. The x-ray procedure was repeated every 30 minutes during the failure test and again after the 90 minutes of testing (post-test x-ray images). The testing was stopped when either herniation occurred or injecting the spine did not compensate for the disc height loss.

c. Re-injection with the barium sulphate-nucleus mix

The pre-test storage, preparation, potting and preliminary testing of these specimens was as described above. Eight specimens (6 C3/4 and 2 T7/8) were repeatedly flexed in a 10^0 range under 1.472kN of axial compression at a rate of 45^0/s and a frequency of 1Hz for 90 minutes. Three specimens were re-injected with barium sulphate-nucleus mix after 20 minutes of dynamic testing. Five other specimens were injected with the barium sulphate mix after undergoing sufficient dynamic testing to cause loss of approximately 50% of their pre-test disc height. The specimens were unloaded at the time of injection and the needle aperture was sealed with superglue after injection. The x-ray procedure was repeated every 30 minutes during the failure test and again after the 90 minutes of testing (post-test x-ray images). The

testing was stopped when either herniation occurred or more than 50% of the pre-test disc height was lost again.

d. IV line in situ

An IV line was inserted through a 22 needle into the nucleus of 4 specimens that had been prepared, potted and pre-loaded as described above. The IV line was connected to an infusion pump which administered saline to the disc. Axial compression of 1kN was applied to the specimens and the specimens were repeatedly flexed in a 10^0 range at a rate of 45^0/s and a frequency of 1Hz. The tests were terminated as the IV line did not remain in situ.

3. Changing the model

a. Rupturing the annulus prior to testing

The pre-test storage and dissection of these specimens was as described above. Prior to potting the specimens the right postero-lateral annulus was ruptured using a 2 mm blade that was inserted through the left antero-lateral annulus (Figure 3.1). The depth the blade was inserted was measured on the adjacent exposed disc such that the inner 2/3 of the annulus was ruptured. The position of the blade in the specimen was checked by a cranio-caudal x-ray. Subsequent potting and preliminary testing of the specimens were as described above. Five porcine C3/4 specimens underwent 90 minutes of repeated combined flexion and left side flexion testing in a 10^0 range (as described above) under 1 kN of axial compression at a rate of 45^0/s and a frequency of 1Hz.

b. Delaminate the annulus using pressurized air

Air was injected through a 21-gauge needle into the annulus of 4 C3/4 specimens at a pressure of 60 psi (413.79 kPa) for 2 minutes. These specimens were dissected so that the extent of the damage could be assessed prior to dynamically testing other specimens prepared in this way. Other than the damage created by the needle disc disruption was not observed in these specimens.

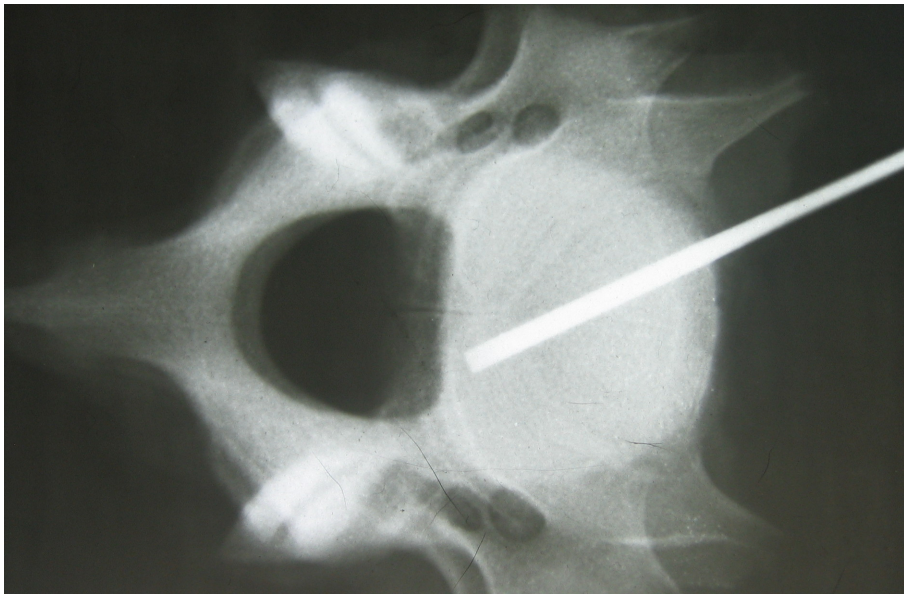

Figure 3.1: A cranial view of a porcine cervical disc being artificially ruptured on its posterolateral aspect.

c. Removal of the nucleus

As re-hydrating the disc with either saline or barium-sulphate mix often resulted in spontaneous herniation, which was likely due to excessive fluid within the annulus, attempts were made to remove the true nucleus of the disc with the intent of replacing it with the barium-sulphate and C2/3 mix. The pre-test storage and preparation of two specimens was as described above. Prior to potting the specimens a 16-gauge needle was inserted into the anterior annulus and nucleus. A suction pump was attached to the needle and up to 30 inHg (101.59 kPa) of suction applied to remove the nucleus of the disc. These specimens were dissected so that the extent of the removal of the nucleus and any evidence of annular damage could be assessed prior to dynamically testing other specimens prepared in this way.

d. Porcine thoracic spines

The hypolordotic T7-8 segments (Figure 3.2) of porcine thoracic spines were dissected from porcine spines that had been bagged and frozen immediately post-mortem and thawed at room temperature for 12-15 hours prior to dissection. The pre-test storage, preparation, potting and preliminary testing of these specimens was as described above for the cervical specimens. Axial compression of 0.3kN, 1kN and 1.472kN was applied to 3, 4 and 2 specimens respectively and the specimens were repeatedly flexed in a 10^0 range (as described above) at a rate of 45^0/s and a frequency of 1Hz for 90 minutes. The x-ray procedure was repeated every 30 minutes during the failure test and again after the 90 minutes of testing (post-test x-ray images).

DATA ANALYSIS

The change in the position of the nucleus was grossly classified according to the discographic scale of Adams et al (1986). Disc height was measured as per Wilke et al (2006) and reported as a percentage of the pre-test disc height (post-preload height). Paired t-tests were used to test if significant changes in disc height and stiffness occurred after the dynamic failure testing of the lower compression group (1kN) and the thoracic spine group relative to the pre-test levels. Repeated measures ANOVAs were used to test if significant disc height and stiffness changes occurred in the saline bath and barium sulphate-nucleus re-injection groups after the initial failure testing, after re-hydration and after continued failure testing.

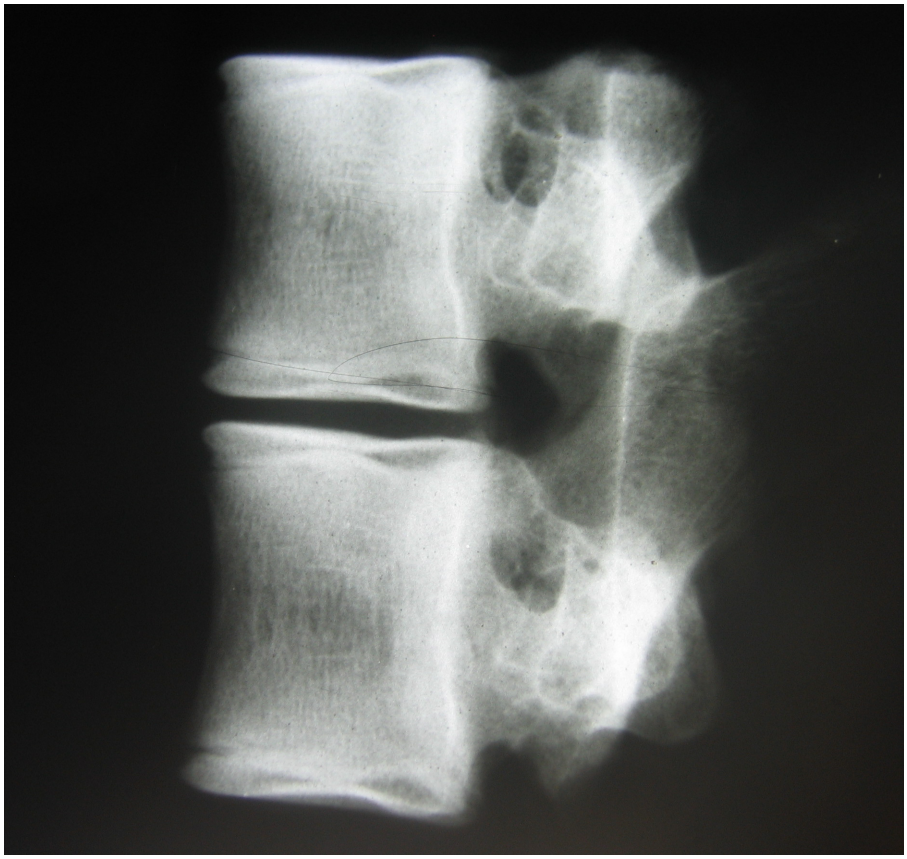

Figure 3.2: A lateral view of a porcine thoracic spine. Note the parallel endplates and hypo-lordotic posture of this specimen.

Model		N	Compression kN	Mvt.
Lower compression		6	1	Flexion
Higher compression		2	2	Flexion
		2	2.596	Flexion
Torque Control		3	1.472	Flexion
Re-hydration	**Saline bath**	4	1.472	Flexion
	Re-inject. with $BaSO_4$	8	1.472	Flexion
	Re-inject. with saline	3	1	Flexion
	In situ IV line	4	1	Flexion
Total change in model	**Thoracic**	3	0.3	Flexion
		4	1	Flexion
		2	1.472	Flexion
	Cut annulus	5	1	Flexion + Side Flexion
	Use air pressure to rupture annulus	4	-	-
	Remove Nucleus	2	-	-

Table 3.1: The methods used to create herniation and the testing protocols for the specimens.

RESULTS

1. Modifying the load (1, 2, 2.5 kN) and control used to create herniation

a. Lower compression

Three of the 6 C3/4 porcine cervical segments tested with repeated flexion under 1kN of axial compression herniated. On average the herniated specimens lost 51.5% (SD 3.24%) of their pre-failure disc height while those that did not herniate lost 55.7% (SD 12.05%) of the pre-test disc height. This disc height loss occurred after an average of 5600 repetitions (SD = 5100 repetitions) of flexion. The stiffness of these specimens increased significantly ($p < 0.01$) over the course of the failure procedure (Table 3.2) from 1.37 Nm/deg. (SD 0.68) to 1.99 Nm/deg (SD 0.67).

b. Higher compression

All 4 of the C3/4 specimens tested under higher axial compression failed due to anterior herniations through the sealed aperture created when injecting the radio-opaque barium-sulphate-nucleus mix into the disc. Two of the specimens were loaded under 2596N while two others failed under 2000N of axial compression. All four failed during the preliminary steps of the tests, prior to repeated testing.

c. Torque control

The creep and increased stiffness of the specimens that occurred during the testing reduced the range of flexion produced by the set torque value such that the test became static flexion. Herniation did not occur and the tests were discontinued after 2 hours of testing.

2. Re-hydrating the specimens

a. Saline baths

On average these 4 specimens lost 57.92% (SD = 12.57%) of their pre-test disc height after 90 minutes of flexion under 1472N of axial compression. After an average of 12 hours (SD 1.63 hour) in a saline bath the loss of disc height was reduced to 11.88% (SD 14.88%) of the pre-test disc height. One of the 4 specimens had herniated after the initial 90 minutes of testing. Three of the 4 specimens underwent an additional 60 minutes of flexion after re-hydration in the saline bath and again lost a similar amount of disc height

(58.33% of their pre-test disc height). At this time two of these three specimens had endplate fractures while the third had herniated. The stiffness of the specimens after the failure procedure (2.40 Nm/Deg SD 0.29) was reduced after 12 hours in the saline bath (1.72 Nm/Deg SD 0.42).

b. Saline injections

Despite the total addition of 0.3ml of saline to each of three C3/4 specimens the disc height loss after 90 minutes of testing in these 3 specimens was almost equivalent (52.64% of pre-test height, SD = 5.21%) to that when the specimens were not injected with saline injection (57.92%). A herniation did not occur in these specimens. An additional 0.1ml of saline did not compensate for the disc height loss that had occurred thus far in the testing.

c. Injection with the barium sulphate-nucleus mix

After 20 minutes of dynamic testing, three C3/4 porcine specimens were injected when unloaded with barium sulphate-nucleus mix. However, all three specimens herniated anteriorly through the sealed injection aperture once the axial compression of 1.472kN was re-applied for further testing. The 5 specimens (3 C3/4 and 2 T7/8) that were subjected to the repeated testing until approximately a 50% reduction of their pre-test disc height occurred regained an average of 30.62% (SD = 3.38%) of the lost disc height after re-injection of the barium sulphate-nucleus mix (x = 0.32ml, SD = 0.15ml). The average post-injection disc height was 81.57% (SD = 11.93%) of the pre-test disc height. No significant differences in the disc height changes between cervical and thoracic segments occurred. All five specimens then underwent further repeated testing. One of the five specimens herniated. While the remaining 4 specimens lost 63.53% (SD = 6.82%) of pre-test disc height after an average of 40 minutes of additional testing without herniating. The tests were stopped because of extreme loss of the posterior disc height (x = 78.13%, SD = 9.24%).

Group		Compression (kN)	Stiffness (Nm/Degree) Mean (SD)							
			Start of failure	End of failure procedure 1 (pre-rehydration)	After re-hydration	End of repeat failure procedure	After rehydration	End of repeat failure procedure	After rehydration	End of repeat failure procedure
Lower compression		1	1.37 (0.68)	1.99 (0.67)	-	-	-	-		-
Re-hydration	**Saline bath**	1.472	1.08 (0.08)	2.40 (0.29)	1.72 (0.42)	3.73 (1.09)	-	-		-
	Re-inject with BaSu	1.472	2.05(0.49)	1.87 (0.45)	1.69 (0.57)	1.62 (0.62)	-	-		-
	Re-inject with saline	1	1.94 (0.44)	1.81 (0.45)	1.82 (0.51)	2.01 (0.68)	1.67 (0.46)	1.77 (0.54)	1.73 (0.39)	1.85 (0.56)
Total change in model	**T/sp**	0.3	1.23 (0.12)	1.57 (0.13)	-	-	-	-		-
		1	1.29 (0.42)	1.95 (0.59)	-	-	-	-		-
		1.472	1.93 (0.22)	2.30 (0.09)	-	-	-	-		-

Table 3.2: The stiffness (Mean, SD) of the specimens at various stages of testing. Failure testing increased the stiffness of the specimens while re-hydration of the specimens decreased the stiffness.

3. Changing the model

a. Cutting the annulus

Three of the 5 porcine specimens (C3/4), in which the posterior annulus had been ruptured, herniated through the anterior sealed aperture of the scalpel used to rupture the posterior annulus. The other two specimens had early nuclear extrusions with total loss of the nucleus into the spinal canal.

b. Delamination of the annulus by pressurized air

This methodology did not result in annuls damage that could have accelerated herniation of the disc. Delamination of the annulus was not observed in any of the four specimens.

c. Removing the nucleus

The nucleus of 4 specimens could be suctioned through a 16- gauge needle with 20 inHg (67.73kPa) of vacuum. However, the aperture, even when sealed, of a 16-gauge needle weakened the annular wall and resulted in spontaneous anterior herniation when the specimen is loaded under 1kN. A vacuum of 30 inHg (101.59 kPa) through smaller needles resulted in damage diffusely along the inner annulus. This methodology could not be used to reduce the increased IDP due to the addition of the barium sulphate – nucleus mix to the nucleus of the disc.

d. Porcine thoracic spines

The ratio of the disc height to the vertebral body width (Frobin et al 1997) of the 12 porcine thoracic specimens (T7/8) tested was not significantly different from that of the porcine cervical spines, even though both measurements were considerably less in the thoracic spines compared to porcine cervical spines. No posterior herniations occurred in the specimens tested. Three of these tests were stopped due to visible instability of the specimens in the cups while nine of these tests were stopped due to the excessive disc height loss visible on examination of the intermittent x-rays taken during the tests. The disc height loss of the nine specimens averaged 58.25% (SD = 11.14%) relative to that prior to failure testing.

The changes in disc height as a result of the various testing protocols are summarized and put in context in figure 3.3.

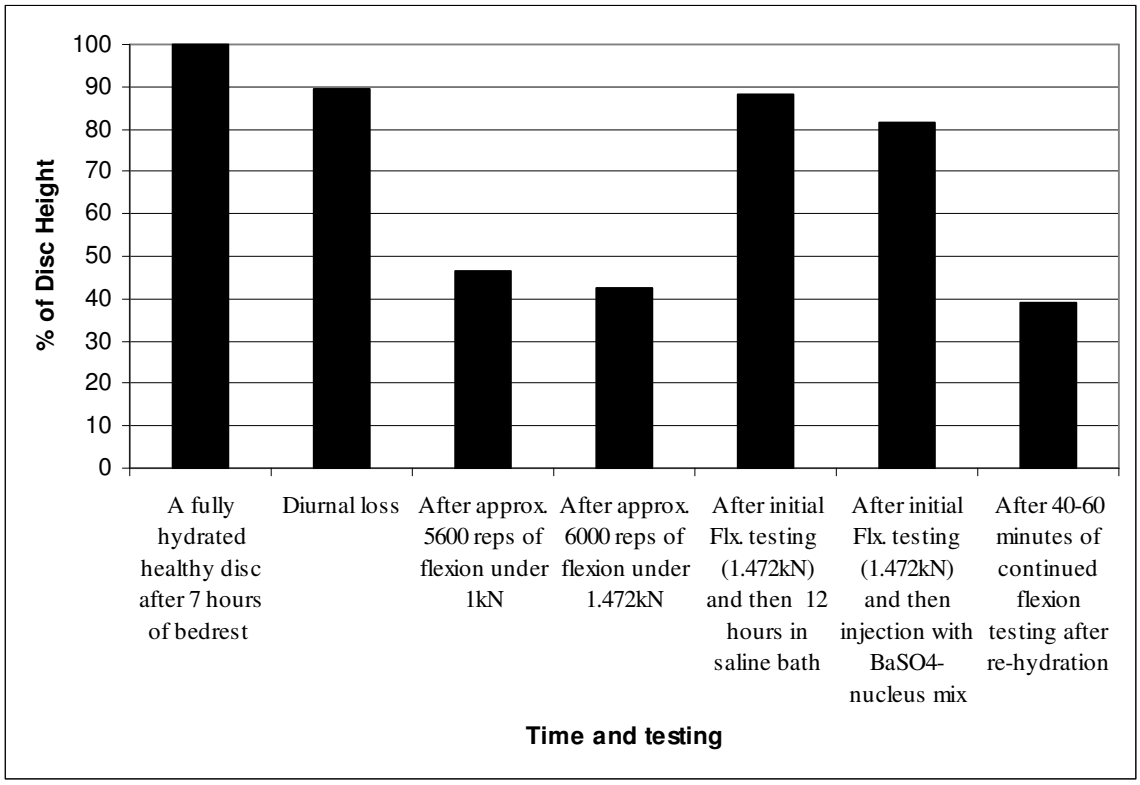

Figure 3.3: A healthy disc is fully hydrated after 7 hours of sleep (Reilly et al 1994) and is shown here as 100% of disc height. Normal diurnal disc height loss is approximately 10%. Specimens tested under either 1 or 1.472kN of axial compression and repeatedly flexed lost more than 50% of the pre-test disc height. Re-hydration of the specimens either by placing the specimen unloaded in a submersion saline bath for approximately 12 hours or by injecting the specimen with $BaSO_4$ –nucleus mix increased the disc height to within 20% of the pre-test height. An additional 60 minutes of flexion testing again reduced the disc height to less than 50% of the pre-test level.

DISCUSSION

This study was designed to investigate possible methods of creating disc herniation without causing concurrent moderate-severe (>33%, Wilke et al 2006) disc height loss. Posterior herniations were created by combining lower axial compression with higher repetitions of flexion, however, the concurrent disc height loss was more than 50% of the pre-test disc height. While the disc height loss that occurred with dynamic testing could be reversed by placing the specimen in a saline submersion bath or re-injecting the specimen with barium sulphate-nucleus mix the disc height could not be maintained within the in vivo diurnal range under continued loading. Furthermore, in a number of specimens that were re-injected with barium sulphate mix premature anterior herniation occurred. Attempts to reduce the intradiscal pressure (IDP) by removing the nucleus from the specimen prior to injecting the specimen with a barium-sulphate stained nucleus failed due to a conflict between aperture size, level of suction and damaging the annulus. Intermittent saline injection during testing did not change the disc height loss that occurred with dynamic testing. Anterior or posterior nuclear extrusion with total loss of the nucleus to outside the annulus occurred after the annulus was artificially ruptured prior to testing and also when higher compression levels were applied during the dynamic testing. The thoracic spines tested did not herniate but lost more than 50% of their pre-test disc height during the dynamic testing. In conclusion, in vitro disc herniations were created but concurrent disc height loss of more than 33% of pre-test disc height occurred. The implications that such disc height loss may have on the results of investigations using this model need to be considered on a study-by-study basis.

The limitations of this study are as previously described. Animal models are a necessary part of the quest for a greater understanding of in vivo biomechanics. These models are controllable, reproducible and cost-effective, unlike specimens from human cadavers where age, exercise level, diet, environment, personal factors and genes add variability to a sample. More specifically, the porcine cervical spine model has been shown to be anatomically, geometrically and functionally similar to human lumbar spines (Yingling et al 1999, Oxland et al 1991) and was not only a reasonable surrogate for human spines but also provided a more reproducible model. Dhillon et al (2001) and (Callaghan and McGill 1995) have shown that freezing does not change the time-dependant behavior nor elastic properties of the frozen specimens. The number of repetitions in this study would precede collagen repair in vivo (Adams and Hutton 1982). The specimen pool was justified by the fact that using an animal model from a population that was homogenous in age, exercise, diet, and breed reduced individual variability of the specimens. Furthermore, the behavior of the specimen in the sample was similar, reducing the need to increase the sample size.

The disc height loss associated with the successful herniation of specimens repeatedly flexed under axial compression levels of 1 and 1.472 kN in this study was more than 50% of the pre-test disc height. As the disc height loss of specimens that did not herniate was very similar to those that did, the herniation cannot account for the loss of disc height. Similarly, Holodyn et al (2000) reported that the displaced portion of the nucleus in herniated discs did not significantly alter disc height. The volume of herniated nucleus was reported to be 1-2% of total disc volume by Holodyn et al (2000) (Table 3.3). Disc height loss has previously been attributed to fluid exchange and creep deformation of the annulus (Koeller et al 1984). As hydration level and disc height loss changes the mechanical behavior of the disc and spinal segment (Panagiotacopulos et al 1987, Andersson et al 1979, Kraemer et al 1985, Gunning et al 2001, Race et al 2000, Simunic et al 2001, Costi et al 2002, Koeller et al 1984, Adams and Roughley 2006, Jensen et al 1994, Adams et al 1994, Adams et al 2000, Rohlmann et al 2005, Dunlop et al 1984, Adams et al 1988, Adams and Hutton 1980, Adams et al 1994, Adams et al 2000, Rohlmann et al 2005) this level of disc height loss is of concern to future use of this post-herniated porcine in vitro model in research to which fluid flow, hydrostatic pressure and stress gradients across the disc are pertinent. The hydration level of the disc decreases as the osmotic pressure of the disc gradually succumbs to sustained axial compression. This loss of fluid from the disc makes it more like an elastic solid than a viscous fluid (Adams et al 1990). Specimens that have been creep loaded seem to have a reduced susceptibility to herniation (Simunic et al 2001, Adams and Hutton 1982, Adams et al 1987). Greater disc height loss is associated with a more fibrosed nucleus (Adams and Roughley 2006, Jensen et al 1994), less movement-related changes in intradiscal pressure (Adams et al 1994, Adams et al 2000, Rohlmann et al 2005) and an altered load distribution across the spinal segment (Dunlop et al 1984, Adams et al 1988, Adams and Hutton 1980, Adams et al 1994, Adams et al 2000, Rohlmann et al 2005). Given the disc height loss associated with this testing this post-herniated in vitro porcine model appears to be representative of a herniated disc that also has the disc height characteristics of a moderately degenerate disc (Wilke et al 2006).

Unlike in the current study, compression levels of greater than 2000N, in combination with flexion of the specimen, have previously created herniations (Adams and Hutton 1985). In the current study premature spontaneous herniations occurred under axial compression of 2000N or 2596N, prior to being repeatedly flexed. Adams and Hutton (1985) injected the specimens with radio-opaque fluid but did not report the quantity nor the size of the needle used to inject the fluid. The increase in IDP that resulted from the addition of the barium sulphate-nucleus mix was not quantified in the current study but this increase in IDP coupled with the weakened annulus because of the needle aperture may account for the discrepancy between the results of these studies. Callaghan and McGill (2001) compared the repetitions required and the damage created in porcine cervical

spines when using position control versus torque control. Position control produced more consistent but severe levels of damage, which was what prompted the use of torque control in this study. Due to the increased stiffness of the specimens over the course of the testing the flexion angles that the predetermined torque levels rotated the specimen between were reduced, resulting in a static flexion test which did not create herniations. A prolonged lack of consensus on the issue of whether positional or torque control is more representative of in vivo movement exists in the spine literature, which in itself is indicative of a lack of convincing evidence towards one control system or the other. Reviewing the neurophysiology literature emphasizes the lack of agreement on a particular control system. Regulation of motor control has been linked to internal models in the central nervous system (Gribble and Scott 2002, Li et al 2001). These internal models, which are thought to be parameter specific (velocity, direction), interact to produce a movement. Arm movement has been extensively studied from a movement control perspective. Almieda et al (1995) report similar EMG patterns despite different kinematic patterns of the shoulder and elbow joints and suggest that movements are controlled by muscle activation patterns that are planned for the expected torque requirements of the task. Similarly, Gottlieb et al (1997) hypothesize that voluntary rapid arm reaching movements use feed-forward control of dynamic joint torque. However, are the changes seen in the torque the result of a change of the motor control pattern driven by a change in joint position? Scott et al (1997) has shown a change in the central nervous system neural activity when hand orientation changed in a given task. The sensitivity of the internal model to position has also been reported by Tong et al (2002) and Hwang et al (2003). Hwang et al (2003) suggested that the internal model, sensitive to both position and velocity, compute the torque required to produce the movement. Others promote an equilibrium control strategy (Massion et al 1992, Gharfouri and Feldman 2001, St-Onge et al 1997) whereby torques are determined by that required to achieve equilibrium of a limb or trunk. Based on the current literature it appears that this issue cannot be categorically decided upon as the literature appears contradictory, indicative of the complexity of the control issue. Searching for one control system is likely oversimplifying a multi-factorial, hierarchical and potentially variable issue.

A number of the attempts that were made in this study to re-hydrate the specimen to counter the disc height loss associated with this testing methodology were, from one perspective, successful. Gunning et al (2001) super-hydrated porcine cervical spines by immersing the specimens unloaded in a saline bath for 6 hours while Simunic et al (2001) fully hydrated discs, increasing disc height by 2-2.5mm, by immersing them unloaded for at least 20 hours in saline solution. Both Gunning et al (2001) and Simunic et al (2001) were increasing the hydration level of healthy specimens. In the current study, 12 hours in a saline bath increased the mean disc height from 57.92% to 88.12% (2-3mm) of the pre-test disc height in specimens that had already lost disc height due to dynamic

Level	Volume (mm^3) Mean ± SD	
	Herniated Material	Normal Disc
L1-2	-	13663 ± 2553
L2-3	278 ± 71	15669 ± 2584
L3-4	593 ± 292	19375 ± 3502
L4-5	549 ± 329	19580 ± 2622
L5-S1	366 ± 212	19067 ± 3166

Table 3.3: The volume of the herniated material and of the discs in 44 patients was determined from MRI and reported by Holodyn et al (2000). The volume of the porcine cervical disc is considerably smaller (endplate area = 500mm^2 (Yingling et al 1999) x disc height = 5.10-6.10mm (Tampier et al (in press) = 2550-3050mm^3) than that of the human lumbar disc.

testing. While the disc height after the saline submersion bath was at a physiological level, the disc height loss after further testing was more rapid than on first testing. These results are in keeping with those of van der Veen et al (2005) who reported an increase in disc height on unloading specimens while in a saline bath but not a corresponding increase in nuclear pressure, indicating incomplete recovery of the nucleus. Decay of the specimens would have to be considered if 12 hours in a saline bath was required to reverse the disc height loss that occurred with every 40-60 minutes of dynamic testing. Re-injecting the specimens with up to 0.55ml of the barium sulpahte-nucleus mix returned disc height to within the diurnal levels of disc height variation. The volume added to the porcine disc is approximately 20% (Endplate area = 500mm^2 (Yingling et al 1999) x disc height = 5.10-6.10mm (Tampier et al (in press) = 2550-3050mm^3) of the total volume of the disc. Andersson and Schultz (1979) considered the mechanical properties of cadaver lumbar spine motion segments before and after saline injection (volume not reported). The twelve out of 16 specimens that accepted and retained the saline had increased stiffness and 37-83% higher IDP on re-loading. In the current study the stiffness of the segments was decreased by re-hydrating the specimens but increased again in many specimens after continued failure testing.

Brinckmann (1986) divided the posterior annulus of cadaver lumbar specimens (minus 1mm), before loading the specimens with 1-2kN and measuring the disc contours. A number of the specimens were injected with 1.5-2ml of saline, reloaded and re-measured. Brinckmann (1986) reported that leakage into the radial fissure in the annulus did not occur in any of the 10 specimens. Unlike Brinckmann (1986), who loaded the specimens under axial compression in neutral posture, the specimens in this study were loaded under axial compression in flexed postures. The nucleus of 2 of the specimens extruded through the posterior radial fissure, rupturing the outer 1mm of the annulus. Similar results were reported by Simunic et al (2001) who fully divided the annular wall and loaded the specimens under axial compression and full flexion. In the current study the nuclear extrusion exceeded the intended extent of damage and the ability to produce herniated discs without compromising disc height was still at large. The curvature of the lumbar spine varies greatly within the population. Many clinicians believe that discs of hypolordotic spines are predisposed to disc herniation. The porcine thoracic spine is hypo-lordotic and was readily available for testing; however, these specimens did not herniate more readily than the porcine cervical spines. In fact, a herniation did not occur in the thoracic specimen group. The posterior annulus of the porcine thoracic specimens is thicker, the specimens were stiffer and the thoracic disc height was less than of the porcine cervical discs. It is currently unknown whether the same is true of human discs of hypo-lordotic versus normal or hyper-lordotic spines. This finding warrants further investigation.

The continued pursuit of successful methods of creating herniation in the absence of disc height loss is warranted and potentially vital to a more thorough understanding of in vivo disc failure and the mechanism of treatment responses. As there is ever increasing pressure on clinicians to adhere to evidence-based practice the importance of practice-based evidence is paramount. The challenge to bridge the gap between the expertise and understanding offered by various disciplines is posed to all and if accepted will ultimately accelerate the prevention and treatment of LBP. A more thorough understanding of the post-herniated porcine cervical model has been attained and a framework for interpretation of the results of future research using this model has been provided. This model is a model for herniated discs that have moderate disc height loss. Knowing this and the implications associated with disc height loss, investigation of the mechanical theory (movement or positioning can change the location of the displaced portion of nucleus in a herniated disc) credited with the clinical success of the McKenzie approach is acceptable as the limitations of the model are understood.

CONCLUSION

Alternative methods of creating disc herniation in porcine specimens were attempted. The disc height loss that coexists with the porcine cervical disc herniations created with repeated flexion under axial compression remained clinically significant. This model appears to be a valuable model for investigation of herniated discs that have substantial disc height loss and the related mechanics (loss of hydraulic behavior, motion, foramen space and changes in facet loading). Consideration of the limitations posed by the concurrent disc height loss prior to future use of this model and methodology are imperative.

CHAPTER 4

THE MCKENZIE APPROACH TO DISC PROLAPSE – EVIDENCE OF A MECHANICAL FOUNDATION?

ABSTRACT

Background; The McKenzie approach supposes that the location of the displaced portion of the nucleus in a prolapsed disc can be changed, moving it away from pain sensitive structures, by repeated movement or sustained positioning. This theory, which is fundamental to the McKenzie approach, has not been scientifically tested. Previous research has established that repeated flexion or combined flexion and side flexion under low-moderate levels of axial compression can create disc prolapse, causing a portion of the nucleus to displace posteriorly. In essence, this study investigates whether extension or combined extension and side flexion can move the displaced portion of nucleus towards the centre of the disc.

Methodology; The C3/4 segments of 18 porcine cervical spines were dissected and potted in ultra high-density polyethylene cups for testing. In order to track the position of the nucleus radiologically 0.55ml of a radio-opaque mixture was injected into the C3/4 intervertebral disc (IVD) through the anterior annulus using a 20 gauge needle. Anterior and lateral view X-ray images were taken prior to testing to establish that the disc was healthy and that the nucleus was contained within the annulus. The specimens were placed in a servo hydraulic dynamic testing jig. Each specimen was preloaded (260N for 879s) to reverse the effects of freezing, following which the potted specimens were removed from the testing machine and anterior and lateral view X-ray images were taken in a standard frontal and transverse plane position prior to further testing (the pre-test x-ray images). The maximum and minimum angles to be used for dynamic failure testing were identified from the angle of increased stiffness on the torque-angular deformation curve of each specimen which was recorded while the specimen was axially compressed (1472N) and rotated in the plane of the test motion (either pure flexion or combined flexion and side flexion) at a rate of 0.5^0/s. The failure test involved repeatedly flexing or flexing and side flexing the specimens under 1.472kN of axial compression at a rate of 45^0/s and a frequency of 1Hz until prolapse or severe structural damage (severe disc height loss, retrolisthesis) of the specimen occurred. The x-ray process was repeated at 10 minute intervals for the first 30 minutes of testing and subsequently at 30 minute intervals. Post-failure x-ray images were taken after completion of the failure test (post-failure images). Specimens that had prolapsed were immediately put through a reversal test, which consisted of repeated extension or combined extension and side flexion of the specimen at a rate of 45^0/s and a frequency of

1Hz. The reversal testing was discontinued when the displaced portion of the nucleus appeared to move more anteriorly towards the centre of the disc or when the condition (disc height loss/ retrolisthesis) of the specimen hindered interpretation of the results. Post-reversal x-ray images were taken after completion of the reversal test (post-reversal images). An independent radiologist reviewed the pre-test x-ray images to assess whether the specimens contained healthy discs prior to testing and then compared the pre-test and post-failure x-ray images to determine if a portion of the nucleus had been displaced posteriorly ± laterally during the failure test. In specimens that were considered prolapsed the radiologist reviewed the post-reversal x-ray images to provide an opinion on whether the displaced portion of the nucleus had moved more anteriorly, towards the centre of the disc. Disc height of the specimens was measured by the investigator according to Wilke et al (2006).

Results; High correlation between the results of the radiographer's review of the x-ray images on two occasions was found. Based on a radiologist's review of the x-ray images, all eighteen specimens contained healthy discs prior to testing and two of the eighteen specimens had endplate fractures while eleven of the eighteen specimens had prolapsed after the failure testing. The disc height loss measured by the investigator of the prolapsed group of eleven after the failure test was 53.03% (SD 18.00%). According to the radiologist there was a positive clinical change in the displaced portion of the nucleus in five of the eleven prolapsed specimens after the reversal testing while in the remaining six the position of the displaced portion of the nucleus did not change. The results of the reversal testing provided a sub-classification of the group of eleven such that the prolapsed discs that centralized with reversal testing had significantly less disc height loss ($p < 0.01$) after the failure procedure than those that did not centralize. Neither the classification of the herniation (circumferential or radial) nor the angle of lordosis of the specimens were linked to the behavior of the specimens.

Conclusion; This study provides a proof of the principle on which the McKenzie disc approach is based. The principle being that a displaced portion of nucleus can be directed back towards the center of the disc in response to particular active and passive movements/ positions. Successful reversal of all disc prolapses was not found. Future studies investigating the stresses driving the reversal will enhance our understanding of the mechanics of healthy and pathological discs while studies investigating optimal doses and techniques of the McKenzie protocol will improve the selectivity and efficacy of treatment.

INTRODUCTION

Little consideration has been given scientifically to the reversal of the failure process of the lumbar discs even though numerous studies have investigated the loading mechanisms necessary to cause disc failure (Callaghan and McGill 2001, Aultman et al 2005, Andersson 1981, Brinkmann 1986, Kelsey et al 1984, Gordon et al 1991, Lu et al 1996). This being despite the fact that the reduction of symptoms and signs related to such disc damage is a key objective of the daily work of many physiotherapists. It has been proposed that the success of the McKenzie approach, considered one of the most widely accepted physiotherapy approaches in diagnosis and management of low back pain (LBP) (Varamini and Jam 2005), has to do with movement/position-related reversal of the disc damage. Specifically, it is thought that the portion of the nucleus that has left the centre of the disc and resides in the annulus is directed back towards the center in response to particular active and passive movements/ positions. To date no one is able to state categorically whether the conceptual model for the derangement syndrome is correct. Lack of quantification of the clinical theory of treating disc damage limits the understanding and selectivity of such practices. Furthermore, in the absence of a thorough understanding of the mode of affect (mechanical, sensory, psychological) of this movement based approach the treatment may be sub-optimal.

The McKenzie approach is an established international clinical assessment and treatment technique. One aim of this approach is to identify and reduce prolapsed/herniated discs. Generally, the foundation of this technique is clinical experience coupled with a theorized explanation. The founder of this movement-based technique suggests that the identification and treatment of disc prolapse is based on the mechanical response of the nucleus pulposus of the IVD to repeated movement and sustained positioning. Centralization, a term coined by McKenzie, is the reduction of the most distal symptom with repetition of a particular movement or by sustaining a particular position, the movement and position being particular to the location of the displaced portion of the nucleus. The centralizing direction of movement most commonly involves extension movements (Donelson et al 1991). McKenzie proposed that the direction of movement that centralizes the symptoms precisely corresponds with the direction in which a portion of the nucleus has abnormally migrated and that successful centralization is dependant on a hydrostatically intact nucleus that is contained within the outer annulus. The clinical and prognostic value of this technique has been the topic of some investigation. Adams et al (1996) reported patients had decreased pain scale readings, increased lumbar range of motion, reduced trunk muscle activity and elevated levels of substance P following a 6-week treatment program of the McKenzie extension procedures. Long et al (2004) reported that over 90% of those assigned to a McKenzie exercise regime reported their symptoms

were better or resolved after 2-weeks of the program. Alexander et al (1998) were able to predict in 91% of cases using this repeated movement assessment technique those that would respond to non-surgical treatment of disc prolapse. Similarly, Long (1995) found that centralizers reported significant deceases in their maximum pain ratings and had a higher return-to-work rate than non-centralizers. Donelson et al (1990) reported that clients, who could not achieve centralization of symptoms as a result of repeated movements, did not respond well to conservative therapy and generally had a poor treatment outcome. Donelson et al (1997) investigated the theory that centralization is dependent on a competent annulus (the outer border not breached) in a study that investigated the correlation of the McKenzie classification of the symptom response to movement to whether or not the annulus was competent, as determined by discogram. Ninety-one percent of those that centralized had an intact annulus suggesting possible grounds for this component of the McKenzie theory. The issue of a competent versus a breached annulus raises the terminology issue. In clinical circles, posterior displacement of a portion of the nucleus that does not breach the outer annulus is referred to as a 'prolapsed' disc while posterior displacement of a portion of a nucleus through a breached outer border of the annulus is referred to as a 'herniated' disc. However, in research there appears to be an absence of this distinction and the term 'herniation' tends to be used to cover this spectrum of disc damage. As it has been shown that centralization is dependent on a competent annulus (Donelson et al 1997) the term disc 'prolapse' will be used to signify this distinction.

From a biomechanical perspective, the McKenzie explanation appears possible given the design of the disc, the movement-related changes in the internal pressure of the disc and the movement-related potential for annular damage. In its simplest form, the disc can be considered a pressure vessel whereby the nucleus is the pressurized fluid and the annulus and vertebral endplates make up the walls of the vessel (Brinkmann and Grootenburg 1991). As a viscous fluid, the nucleus does not possess a shape of its own but rather takes on the shape of the vessel within which it is contained. The change in shape and position of the nucleus associated with movement depends on a change in shape created by the annulus and/or the endplates. But the mechanics are not straightforward. Flexion causes lowering of the anterior aspect of the vertebral body and rising of the posterior aspect, which results in increased compression of the anterior annulus along with increased tension of the posterior annular fibers. Adams and Hutton (1982) reported that in full flexion a 50% increase in the posterior annular height and a 30% decrease in the height of the anterior annulus occur relative to their respective heights in erect standing. They also reported that the posterior annulus thins to 67% of its thickness in lordotic posture while the anterior annulus buckles inward from the additional compression during flexion. This would depend greatly on the amount of viscous "healthy" nucleus

and state of the disc. Brault et al (1997), Schnebel et al (1988) and Fennel (1996) have all reported a posterior shift, of less than 1.0mm, of the nucleus on flexion in healthy discs. More recently Lyndsay et al (2007) reported posterior migration of the peak pixel intensity of the nucleus on MRI in sitting in lumbar flexion relative to that when sitting in extended posture, standing and in prone extension. This change in position of the nucleus has been classed by others as a change in shape of the nucleus in full flexion in a healthy IVD (Hickey and Hukins1980b). Flexion postures also cause an increase in the hydraulic stress (flow-related) on the posterior annulus and a large increase in the in vivo nuclear pressure (static), the latter is largely due to the increased compression caused by the increased ligament tension and contraction of the back muscles. Thus many mechanical changes occur with flexion: the posterior fibers of the annulus have to resist increased internal pressure; increased hydraulic pressure of the nucleus; and increased tension of the annular fibers. Supporting the argument that a portion of nucleus will displace into the area of the annulus that is under maximum combined stress are the findings of Aultman et al (2005) (Appendix A), a study in which specimens that were repeatedly flexed where the flexion axis was moved 30^0 to the left of the sagittal plane developed right postero-lateral disc herniations, predictable in 15 out of 16 specimens. Specifically, the site of the nucleus breach of the inner annulus was determined by the bending axis, a finding also reported by Tsantrizos et al (2007). Looking more closely at the specific cause of annular damage McNally et al (1993) identified stress concentrations in the area of eventual herniation in their study that looked at the failure of motion segments. They suggested stress concentrations; defined as a greater maximum annular vertical stress than the mean stress in the nucleus, result in the outer lamellae bulging outward while the inner lamellae are collapse inward. The stress concentrations were identified at the anatomic boundary between the nucleus and inner annulus, a location that the authors suggested could initiate sequential failure. Recent research by Tampier et al (in press) suggests that the tension and eventual rupture of the annulus may not be not be an accurate component of the mechanism of herniation. The formation of clefts between the annular layers was identified on repeated flexion of porcine cervical specimens under axial compression of 1472N and suggests that the hydraulic stress plays a large role in the displacement of a portion of the nucleus. Future research addressing the interplay of the mechanical stresses on the annulus is required before the mechanism of herniation can be stated definitely.

As suggested by the work of Brinckmann (1986), flexion is a necessary component of posterior disc herniation creation. Axial compression of human motion segment specimens in which the posterolateral aspect of the annulus had been damaged by cutting the annulus, except for the outer 1mm, did not result in an extrusion of disc material at the site of annular damage. Brinckmann (1986) concluded that a radial division of the annulus under compression in a neutral spine position is

not sufficient to produce a clinically relevant disc herniation. A number of mechanical concepts can explain why flexion is a necessary component of posterior disc failure. Considering the disc to be a pressure vessel, with solid non-permeable walls, camouflages the complexity of the interaction between the fluid and solid components of the disc. More accurately, the disc should be considered as a porous elastic solid containing a viscous incompressible fluid. The theory of the deformation of a porous elastic solid containing a viscous incompressible fluid developed by Biot (1941, 1956, 1957) has been applied to the IVD given its biphasic behavior (fluid (nucleus) and solid (collagen layer of the annulus) phases) (Simon et al 1985). A component of that theory and of particular interest to this discussion is the permeability of the annulus and consideration of how the annular permeability changes with position of the spine and with the damage previously described. The permeability (k_a) of the annulus is related to the effective radius, a, of the pores by;

$$k_a = (a^2.\ \psi^f\ ) / (8.\delta^2.\mu_f) \qquad \text{Eqn. 4.1}$$

whereby ψ^f is the porosity of the annulus, μ_f is the viscosity of the fluid and the δ is the tortuosity factor, which is the distance actually traversed by a fluid particle relative to the thickness of the tissue (Holmes and Mow, 1990). The porosity of the annulus may differ in neutral and flexed positions of the disc as voids between the annular fibers are exposed when the posterior annulus is tensioned in flexion and the increased porosity may enable displacement of a portion of the nucleus.

Darcy's law offers yet another mechanical explanation for the necessity of flexion to the development of disc herniation. The rate at which fluid can flow into or out of the annulus under a pressure driving force depends on the hydraulic permeability (k_a) and the hydrostatic pressure difference P as follows:

$$J = (k_a/h)*(P) \qquad \text{Eqn. 4.2}$$

where 'h' is the total thickness of the perfused tissue. The hydrostatic pressure within the nucleus increases drastically in a flexed position of the in vivo spine and could help fluid flow into the annular layers.

Central to the McKenzie theory of assessment and rehabilitation of disc prolapse is a clinician's apparent ability to influence the location of a displaced portion of nucleus in the annulus. If the combination of increased compression of the anterior aspect of the disc and increased tension of the posterior annulus results in a stress gradient causing the flow of the nucleus in flexion - is there a corresponding stress gradient that could reverse the nuclear infiltration of the annulus? Adams et al

(2000) investigated the effects of backward bending on healthy lumbar IVD and found that 2^0 of extension increased the maximum compressive stress within the posterior annulus by an average of 16%, compared with the neutral posture, in degenerated and healthy IVD. Backward bending tended to reduce the compression in the anterior annulus and nucleus (to 0.5 MPa) and increase it (to 2.75 MPa) in the posterior annulus, creating a greater stress gradient across the disc than in a neutral position. However, in degenerate spines the results were more variable. In 7 of 19 degenerated specimens extension caused a reduction, of up to 40%, in the maximum compression in the posterior annulus while in the other degenerate discs the compression was increased by 43% relative to the neutral posture. Adams et al (2000) suggest that the variability in the stress gradient across the disc created by extension may be a reasonable explanation for the variance in the success and failure of the McKenzie approach in reducing disc prolapse. Figure 4.1 shows how this mechanical theory can impact the results of McKenzie approach. The stress gradient in an extended posture of a healthy spinal segment and a segment that has lost disc height are shown. The loss of disc height associated with degeneration can change the contact points of the spinal segments which would move the centre of rotation more posteriorly. This would result in increased compressive loading of the neural arch and increased tension of the anterior and posterior annulus. According to this theory the decompression of the posterior annulus may allow the displaced portion of the nucleus to track more anteriorly towards the centre of the disc. In summary, the clinically proposed mechanism of the success of the McKenzie approach to disc derangement has been considered from a mechanical point of view and such consideration has shown that the proposed mechanisms have merit. Quantitative investigation of the mechanical ability of repeated movement/sustained positioning to influence the location of the displaced portion of the nucleus was central to this study.

A number of different disc lesions have been described. The formation of distinct fissures in the annulus, radial and circumferential, has been previously reported (Adams and Hutton 1985, Thompson et al 1990, Brinckmann and Horst 1985, Brinckmann and Grootenboer 1991, Haefeli et al 2006) and defined as fissures that are perpendicular to the endplates and those that run along the circumference of the disc respectively. Also previously reported are rim lesions (Osti 1990) which are radial tears at the periphery of the annulus adjacent to the endplates. More recently Tampier et al (in press) identified the formation of small clefts in between the layers of the annulus through which the nucleus pulposus was 'pumped' in porcine cervical spines tested under axial compression and repeated flexion (Callaghan and McGill 2001). Once a pocket of nucleus within a cleft acquired enough pressure a new cleft was formed in the weakest part of the layer and the cycle repeated. A portion of the nucleus was eventually displaced to the outer annulus as propagation of the cleft

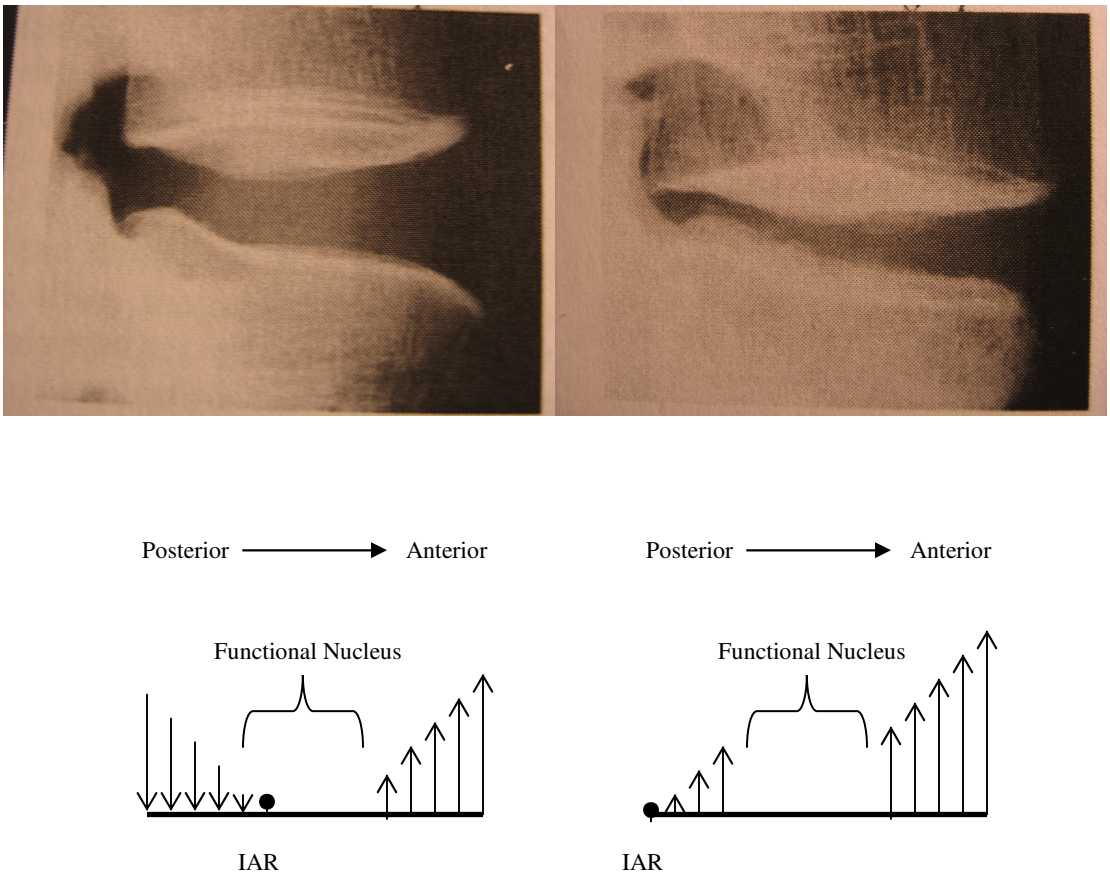

Figure 4.1: A schematic of the stress gradient across the disc during extension of (a) a hydrated healthy disc and (b) a dehydrated, degenerated disc. In a healthy hydrated disc the instantaneous axis of rotation (IAR) of extension lies in the posterior aspect of the nucleus. As disc height decreases the contact of the articular surfaces of the facet joint increases moving the IAR more posteriorly. The anterior annulus and posterior annulus are both under tension as the neural arch shields the posterior annulus from compression. Adams et al (2000) suggests that this may account for the success of the McKenzie extension approach.

formation continued peripherally. Whether cleft formation is the only pathway through which a portion of the nucleus becomes displaced, a precursor to rupture or a totally separate process, rupture and cleft formation possibly occurring under different conditions will be the topic of future research. The circumferential or radial displacement of the nucleus was considered in this study in reference to the changes caused by the extension treatment of the specimens.

The curvature of the lumbar spine varies greatly within the population. Hypolordotic posture reflects a flexion pattern of stress on lumbar tissues in ADL (Scannell and McGill 2003). Many clinicians believe this predisposes discs of hypolordotic spines to disc herniation. Furthermore, the hypolordotic spine is thought to be predisposed to mechanical low back pain (MLBP) as it lacks the inbuilt protection of lordosis (Farcy et al 1997, Keegan 1953, LaGrone 1988). As identification of individual characteristics that predispose to particular forms of MLBP would be a key step in the prevention of MLBP the link between the lordotic angle of the porcine spines and the prevalence to prolapse was considered.

The objective of this study was to create in vitro disc prolapse in porcine cervical specimens, to investigate the effects of extension and/or side bend movements on the position of the displaced portion of the nucleus in the annular layers. The hypotheses of this study were:

H: Repeated motion, opposite to the motion that caused the disc to prolapse, would reverse the position of the displaced portion of nucleus.

H: Discs that would not respond to reversal testing would have a portion of the nucleus displaced circumferentially in the annulus or have a full herniation, with the displaced portion of the nucleus breached through the outer annulus

H: The specimens that prolapse will have been dissected from hypolordotic spines.

METHODOLOGY

Creating disc prolapse (Failure procedure)

The C3-6 segments of 18 porcine cervical spines were dissected from porcine spines that had been bagged and frozen immediately post-mortem and thawed at room temperature for 12-15 hours prior to dissection. A lateral x-ray image (Mercury Modular X-ray, 007 mas, 100ma, 54 kvp) of the C3-6 intact segments was taken before the C3/4 osteoligamentus specimens (intact IVD, facets and intervertebral ligaments) were dissected. The method used to create disc prolapse is based on that of Callaghan and McGill (2001). The C2/3 and C4/5 IVDs were examined for degeneration and all specimens were classified as Grade 1 on Galante's scale of disc degeneration. For the purposes of testing, the specimens were wired with 18 gauge steel wires and cemented with non-exothermic

dental stone (Denstone ®, Miles Inc., South Bend, IN, USA) into ultra high-density polyethylene cups. The anterior wiring was changed to improve the visibility of the nucleus in the lateral x-ray images such that a wire ran through the top of the C3 and bottom of the C4 vertebral bodies. Wood screws were inserted, to a maximum depth of 1 cm, into both the top cup and center of the C3 vertebral body and the bottom cup and center of the C4 vertebral body to ensure bonding. In order to track the position of the nucleus radiologically 0.55ml of a radio-opaque mixture ($BaSO_4$ mixture) was injected into the C3/4 IVD through the anterior annulus using a 21 gauge needle. Two of the specimens were injected with a solution of barium sulphate, Blue dye (250mg Coomassie brilliant blue, 97.25 ml of distilled H_20 and 2.5 ml of methanol) and distilled water, in a 2:1:2 ratio, while the remaining 16 were injected with the same solution with which the C2/3 nucleus was mixed. The C2/3 nucleus was the harvested nucleus of the adjacent segment which was mixed with the radio-opaque solution to eliminate the possibility that the less viscous $BaSO_4$ mixture could herniate through annular clefts/ ruptures that the more viscous nucleus would not. The needle aperture was sealed with superglue after the injection. A layer of saline (0.9% NaCl) soaked plastic-backed material and a layer of polythene film were wrapped around the specimens to rehydrate them. The specimen was placed in a servo hydraulic dynamic testing machine (model 8511, Instron Canada, Burlington, Ont., Canada), which had been modified to apply both axial compression and single plane pure moments simultaneously (Callaghan and McGill 2001). An unconstrained testing apparatus was utilized so the cup containing the C3 segment was fixed in position, the center of the testing plate in line with the geometric center of the C2/3 disc, while the lower cup, containing the C4 segment was free to translate and rotate on a low friction surface. Each specimen was preloaded (260N for 879s) to reverse the effects of freezing during which time the Instrom found a position of zero torque for the specimen. Following preload the potted specimens were removed from the jig and anterior and lateral view X-ray images were taken prior to further testing (the pre-test x-ray images). Moulds supporting the upper and lower cups were made to maintain a standard frontal and transverse plane position of the specimen during the x-ray process. As earlier pilot work had shown that the position of the nucleus was not altered whether the specimen was x-rayed in a pre-determined sagittal plane position or as determined by the stiffness of the specimen after testing it was decided not to fix the sagittal plane position of the specimens for x-ray. The argument for this being that clamping the specimens into a pre-determined position would have undesirably varied the compression of the specimens at the time of x-ray and additionally made it more difficult to maintain the standard frontal plane position of the specimens during the x-ray process. Disc height measurements would not be altered by a change in the sagittal plane angle during x-ray as it is averaged across the disc. In order to identify the maximum and minimum angles to be used for dynamic failure testing the torque-

angular deformation relationship of each specimen was recorded. To do this each specimen was axially compressed (1472N) and rotated 5 times, in either pure flexion or combined flexion and side flexion (depending on the direction of the test motion), at a rate of 0.5^0/s during which the torque-angular deformation curve was plotted. The angle at which the stiffness of the torque – angular deformation curve of the specimen increased was the maximum angle used in the dynamic testing and an angle 10^0 less than the maximum was the minimum angle used. The first 4 specimens were repeatedly flexed under axial compression of 1472N at a rate of 45^0/s and a frequency of 1Hz and the repeated test motion of the remaining 14 was combined flexion and side flexion also under 1.472kN of axial compression at a rate of 45^0/s and a frequency of 1Hz. The x-ray process was repeated at 10 minute intervals for the first 30 minutes of testing and subsequently at 30 minute intervals. If early tracking of a portion of the nucleus was identified in a specimen more frequent x-rays were taken to prevent full herniation, rather than prolapse, of the specimen (Table 4.1). Post-failure x-ray images were taken after completion of the failure test (post-failure images).

Reversal Testing

Prolapse was defined as a posterior/lateral shift of the nucleus of at least 50% (2-3mm) of the pre-test width of the annulus. Specimens that had prolapsed were immediately put through a reversal test which consisted of 10^0 (from the position of zero torque determined during preload) of repeated extension or combined extension and side flexion of the specimen at a rate of 45^0/s and a frequency of 1Hz. In accordance with the McKenzie approach, the direction of motion that changes the location of the displaced portion of the nucleus precisely corresponds with the direction in which a portion of the nucleus has abnormally tracked. Combined extension and side flexion was the movement used for the reversal test if the student investigator determined from the post-failure anterior x-ray image that a portion of the nucleus was displaced laterally as well as posteriorly, as seen on the lateral x-ray image. Axial compression of 260N was used for the reversal test after two tests at higher compression levels (867 and 1472N) raised concerns about disc height loss. The reversal testing was discontinued when the displaced portion of the nucleus appeared to move more anteriorly towards the centre of the disc or when the condition (disc height loss/ retrolisthesis) of the specimen hindered interpretation of the results. Post-reversal x-ray images were taken after completion of the reversal test (post-reversal images).

DATA ANALYSIS

Given the importance of the consecutive x-ray images for each specimen in this study steps were taken to avoid errors and confirm that erroneous positioning of the specimens for x-ray would not hinder the interpretation of results. The x-ray images were taken when the specimens were held in supporting moulds in standard frontal and transverse positions. Furthermore a series of x-ray images were taken of one specimen in a number of rotated positions (Figure 4.2). The changes in the position and dimension of the nucleus and wires were considered. The position of the nucleus could be manipulated by consecutive 5^0 rotations of the specimen in the supporting moulds. However, notable change in the position of the wiring is visible with this degree of rotation, which provided the control marker needed to ensure such rotational changes in the position of the specimen on x-ray would not be mis- interpreted as 'real' change. The change in the wiring position in the pre-test, post-failure and post-reversal x-ray images for each specimen was minimal.

Creating disc prolapse (Failure procedure)

An independent radiologist reviewed the x-ray images on two occasions, 3 months apart, to

(1) Determine that the pre-test discs were healthy: based on the disc height of each specimen, the nucleus of each specimen being contained within the annulus and the relative position of the vertebrae (no spondylo- or retro-listhesis; Spondylolisthesis being the anterior displacement of t),

(2) Classify the type of failure that occurred with failure testing (no failure, endplate fracture, prolapse, herniation, retrolisthesis) and to categorize the disc height loss of the specimens.

Reversal Testing

The independent radiologist reviewed the post 'reversal' testing x-ray images to

(3) Categorize the loss in the disc height of each specimen as mild, moderate or severe

(4) Determine whether the location of the displaced portion of the nucleus in the posterior annulus of each specimen had changed and if so in what direction

(5) Determine the change in vertebral body alignment relative to that of the post-failure testing alignment (spondylo- or retro-listhesis)

Specimen	Reps. of Test Motion		Reversal Test Compression (N) [N/T = not tested]	Reps. of 'Treatment' Motion		Sustained Extension (Mins)
	Flexion	Flexion/ Side Flexion		Extension Reps.	Extension/ Side flexion	
K	5400		N/T	-		
L	5400		867	1200		
N	5400		1472	1200		
M18	10800		N/T	-		
O11		9000	260	900		
O13		3600	260	1800		
O132		5400	260		1800	
O16		5400	N/T	-		
O162		1800	260		1800	
O173		14400	N/T			
O18		1800	260		2700	
O23		2400	260	900		
			606	2700		
O232		4500	N/T	-		
O233		4500	N/T	-		
O24		900	260	-	-	15
			260		900	
O242		1800	260		5100	
O25		3300	N/T	-		
N32		2700	260		900	

Table 4.1: While under 1472N of axial compression the repetitions and direction of motion used to create disc prolapse are shown. Subsequent reversal test specifications are shown on the right-hand side of the table. Testing of specimens that had not failed but that had severe disc height loss was discontinued.

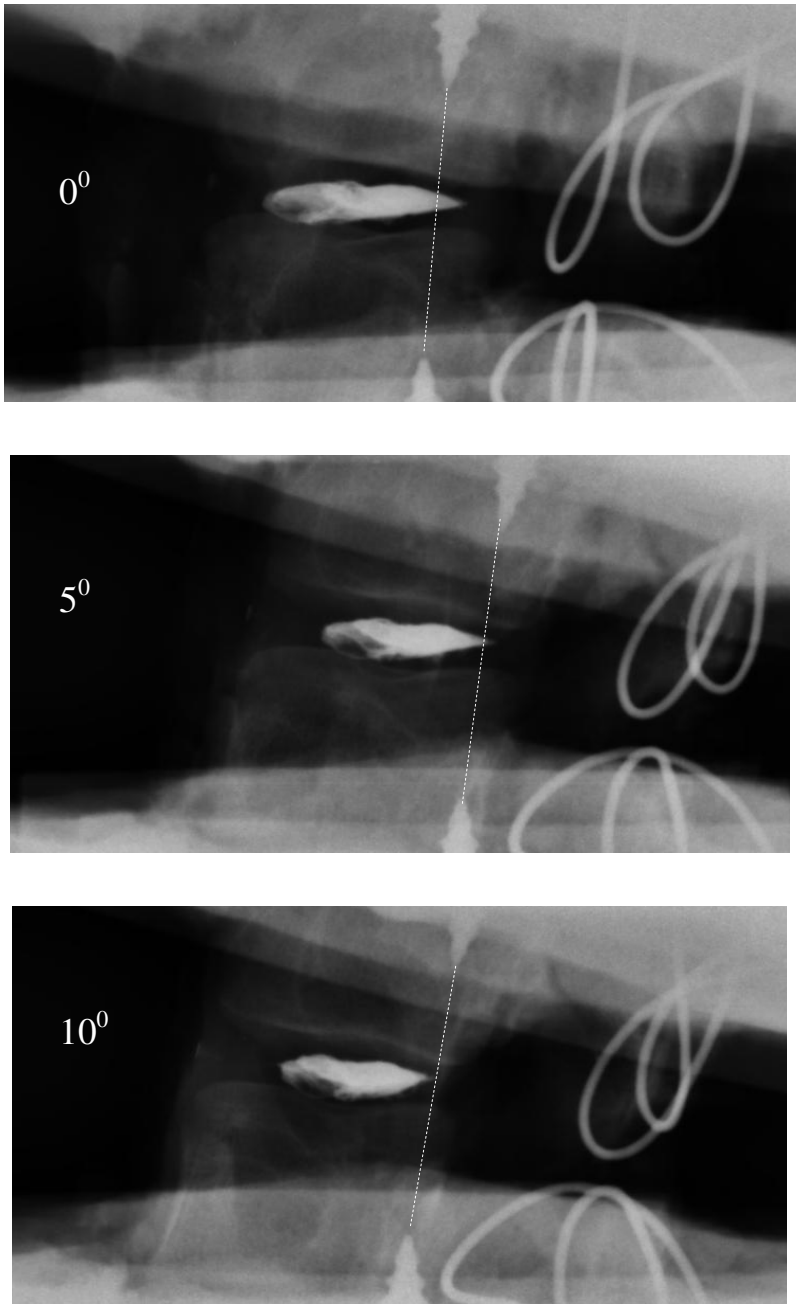

Figure 4.2; A series of lateral x-ray images were taken of one specimen in varying rotational positions. A line drawn between the tips of the screws provided a line of reference. The screws are positioned in the center of the upper and lower cup and therefore equidistant from the x-ray plate in all images. The position of the nucleus in the lateral x-ray image could be manipulated by a 5^0 rotation of the specimen in the supporting moulds. However, notable change in the position of the wiring is visible with this degree of rotation, which provided the control marker needed to ensure such rotational changes in the position of the specimen on x-ray would not be missed and interpreted as 'real' change.

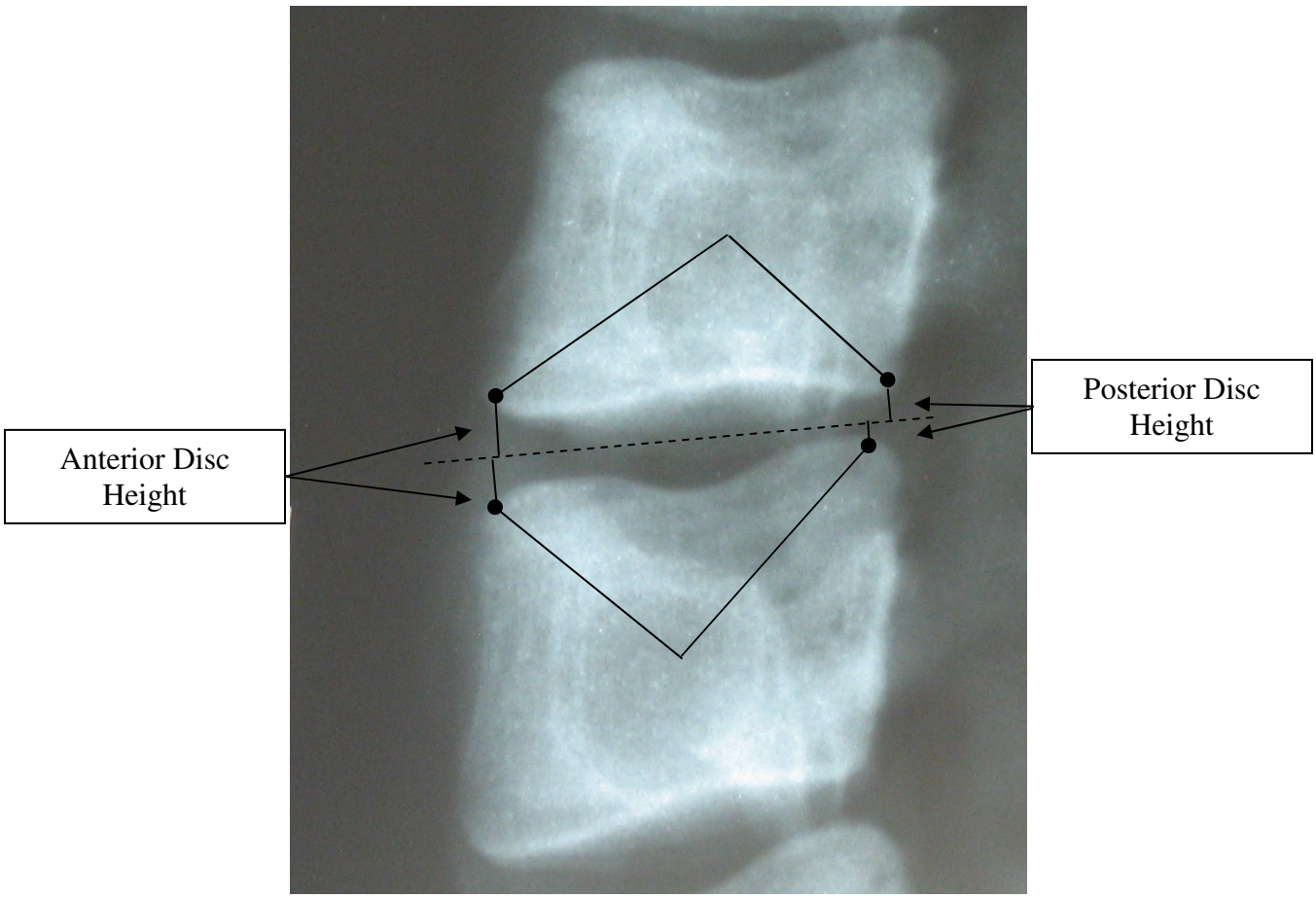

Figure 4.3: As per Wilke et al (2006) anterior disc height is calculated as the sum of the perpendicular distances from the anterior edges of the superior and inferior vertebral bodies (anterior black dots) to the midplane of the disc (dashed line). The edges of the vertebral bodies are identified as the points that are furthest from the center of the vertebral body. Posterior disc height is the sum of the distances from the posterior edges of the superior and inferior vertebral bodies (posterior black dots) to the midplane of the disc. Both in Wilke et al (2006) and in the current study the disc height was measured directly from the lateral x-ray images by each observer. Wilke et al (2006) expressed disc height as a percentage of the normal values reported by Frobin et al (1997). In the current study disc height loss was expressed as a percentage of the pre-test disc height (post-preload) and the anterior and posterior disc heights were averaged to give one figure for disc height loss.

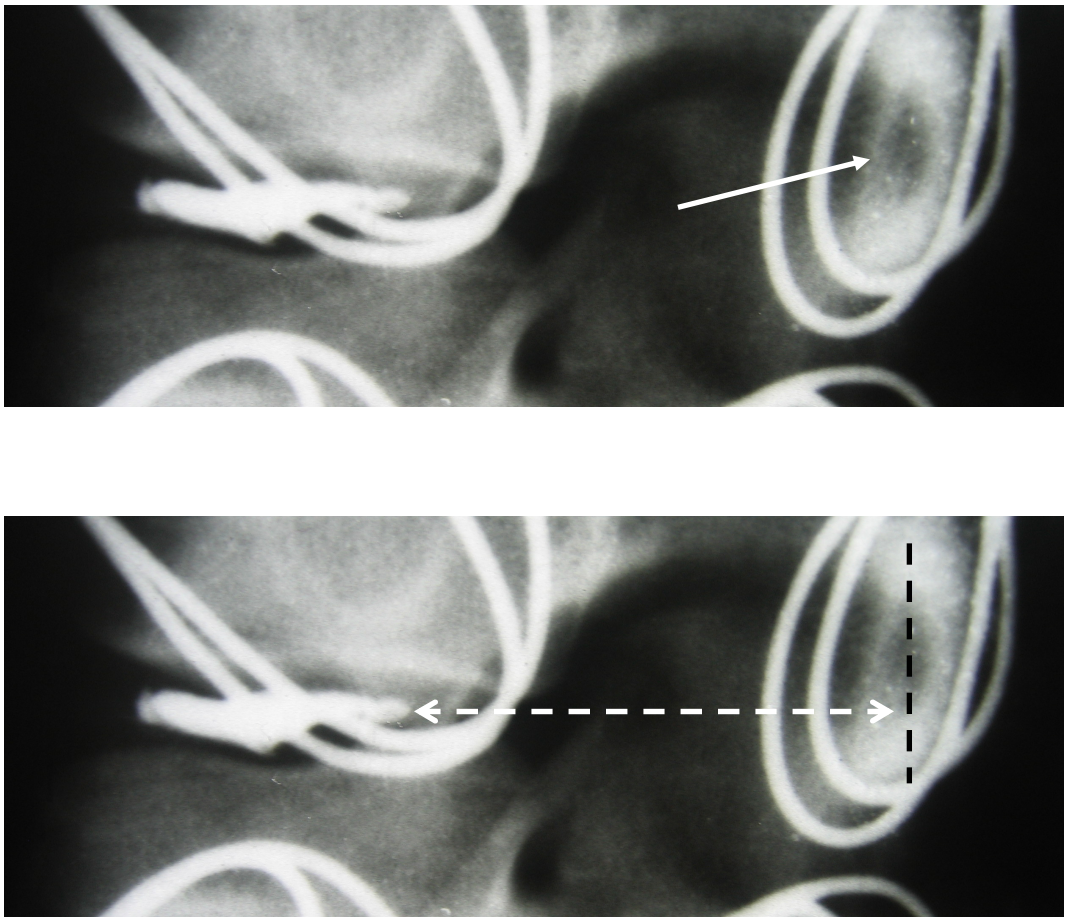

Figure 4.4: The position of the nucleus was measured relative to the inferior articular process of the C3 segment (black dashed line). The distance from the anterior and posterior borders (while dashed line) of the nucleus relative to this line were compared to the equivalent distance on subsequent x-ray images.

The pre-test (post-preload), post-failure and post-reversal disc height of the specimens was measured by the investigator on the lateral x-ray images according to Wilke et al (2006) (Figure 4.3). The post-failure and post-reversal disc height was normalized to the disc height of the pre-test (post-preload) disc height measurements. The position of the posterior margin of the nucleus was measured relative to the inferior articular process of the C3 segment by the student investigator (Figure 4.4). The stiffness of the specimens was recorded as the slope of the line joining the minimum to maximum angles of the repeated motion on the torque-deformation curve. The mean stiffness over the first 10 cycles of the dynamic test was considered the pre-test stiffness and was compared to that of the last ten cycles, the final stiffness. The lordotic angles of the C3-C6 spine and of the C3/4 specimen were measured. The lordotic angle of the spine was defined as the angle between the superior endplate of the C3 vertrbral body and the inferior endplate of the C6 vertebra (Figure 4.5) while the lordotic angle of the specimen was defined as the angle between the superior endplates of the C3 and C4 vertebral bodies (Figure 4.6).

A two-way Pearson's correlation was used to compare the radiologists' review of the x-ray images on two occasions. Differences in disc height and stiffness of specimens after the failure testing were considered using a paired t-test. Repeated measures ANOVA was used to test for significant group differences (reversed or not) in disc height and stiffness of prolapsed specimens after failure and reversal testing. Analysis of the ability of disc height post failure testing to predict the specimen response to reversal testing was performed using discriminate testing. A one-way ANOVA was used to test for group differences (prolapsed or not) in the lordosis angle of the specimens.

RESULTS

The correlation of the radiologist review of the x-ray images on two occasions was found to be high. Perfect correlation scores on the health of the specimens prior to testing and on the number of endplate fractures after failure testing was found. Significant correlation of whether the specimens prolapsed or not was (p, 0.01, r = 0.9), one specimen that was considered not prolapsed on the first review was considered prolapsed on the second. The investigator had deemed it not prolapsed at the time of testing and had not put it through the reversal test. The correlation of whether the posterior margin and the posterior volume of the displaced portion of nucleus changed after the post reversal testing was r = 0.83 and r = 1 respectively. On the second review the radiologist deemed that the posterior border of two specimens did not change after the reversal testing. He reported a clinically significant change in the posterior volume of the nucleus in both of these specimens on both reviews.

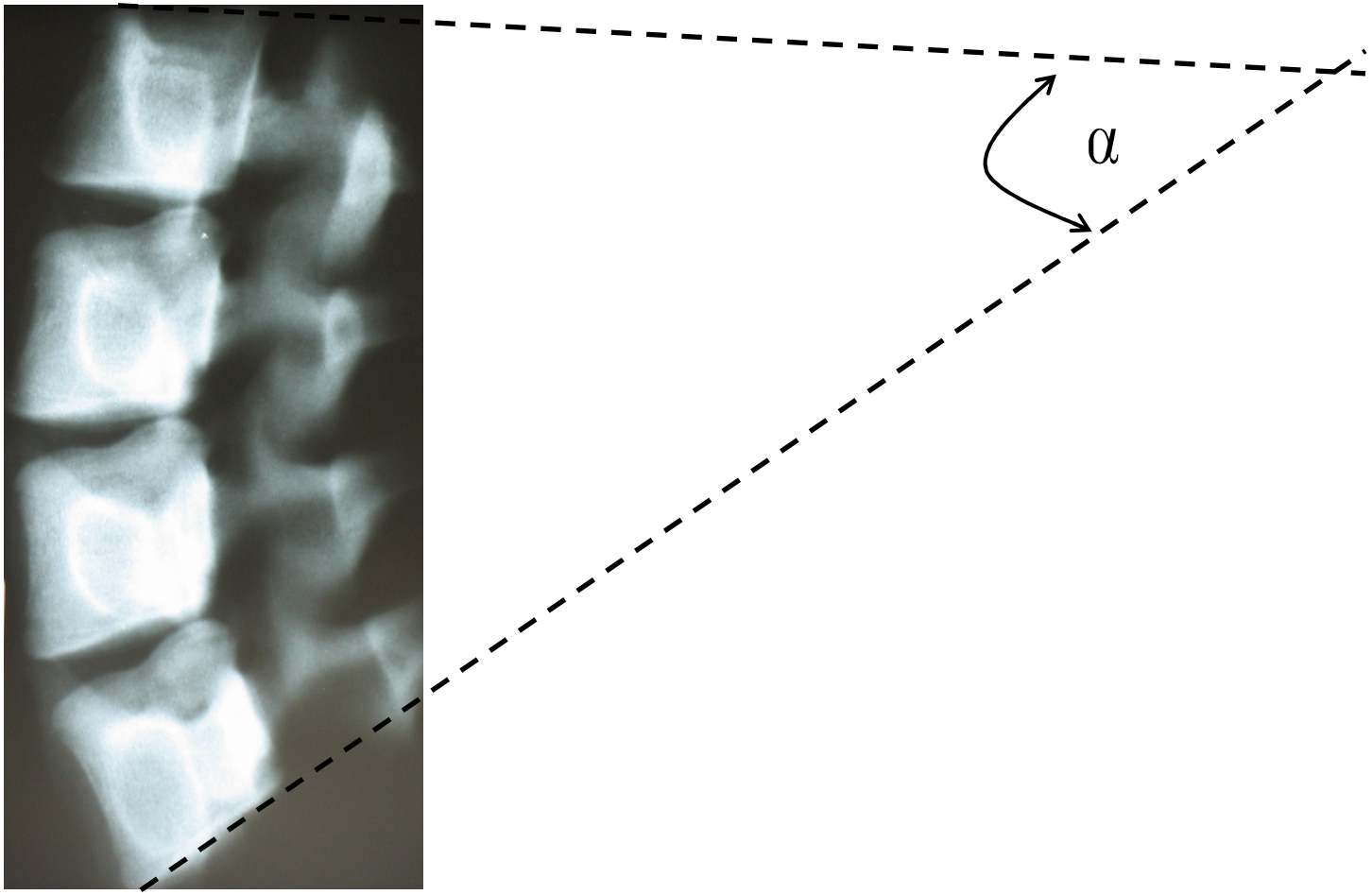

Figure 4.5: The lordotic angle of the porcine spines was measured as the angle (α) between the between the superior endplate of the C3 vertrbral body and the inferior endplate of the C6 vertebra. In this example the lordotic angle is 38^0.

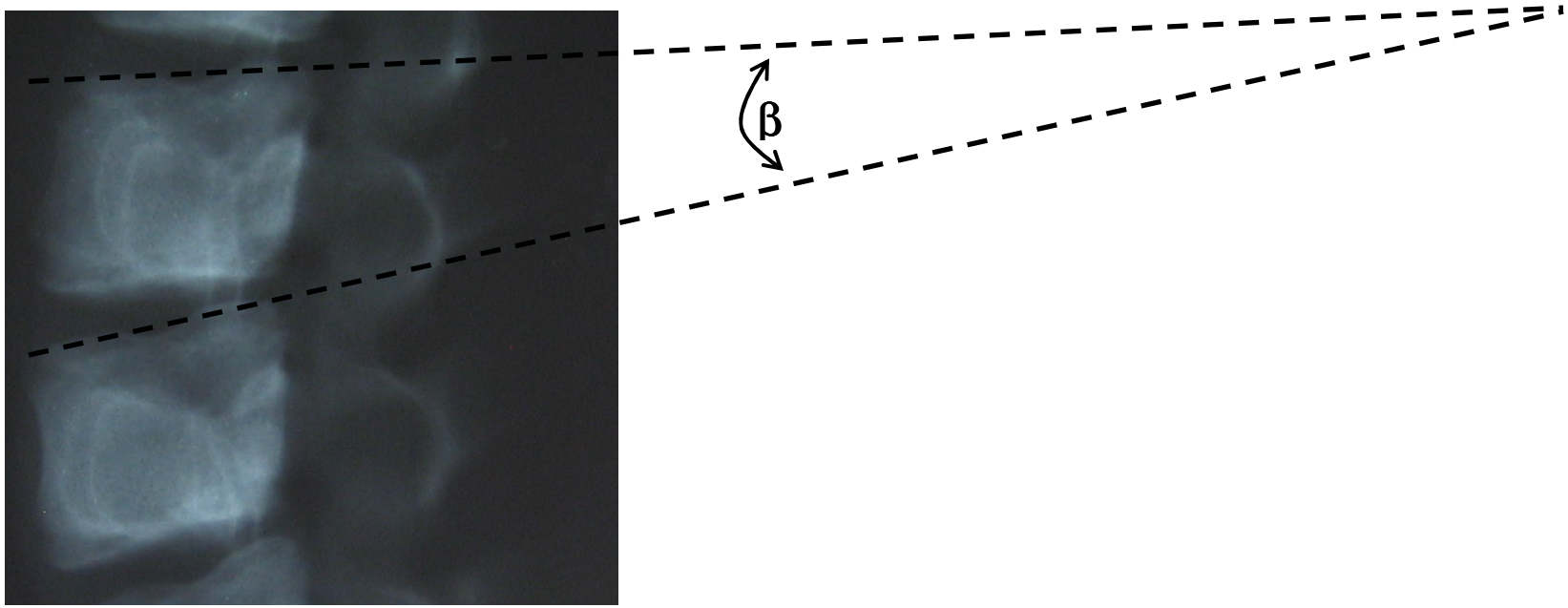

Figure 4.6: The lordotic angle of the C3/4 porcine specimens was measured as the angle (β) between the superior endplates of the C3 and C4 vertebra. In this example the lordotic angle is 11^0.

The radiologist's first review of the x-ray images are shown on table 4.2. After the failure testing, two specimens had endplate fractures while eleven of the eighteen specimens had prolapsed. The mean disc height loss of all specimens, measured according to Wilke et al (2006)by the investigator, was 55.89% (SD 15.59%) and that of the specimens that prolapsed was 53.03% (SD 18.00%). Significant increases ($p < 0.001$) from 1.31Nm/degree (SD 0.42) to 2.44Nm/degree (SD 0.633) in the stiffness of the 18 specimens after the failure procedure were found. Seven of the 18 specimens that underwent failure testing did not go on to have 'reversal' testing as 2 of the 7 had endplate fractures with severe loss of disc height after the failure procedure while the 5 others not did not prolapse.

According to the radiologist there was a positive clinical change in the displaced portion of the nucleus in five of the eleven prolapsed specimens after the reversal testing (Figures 4.8- 4.12; note some variation in the photographic magnification of the images exists and thus measurements should not be taken directly from these photographs) while in the remaining six the position of the displaced portion of the nucleus did not change (Figure 4.13). The disc height loss of the 11 specimens after reversal testing was 46.49% (SD 27.46%) with no significant differences found between the specimens that reversed that those that did not. Interestingly, the post-reversal results provided a sub-classification of the 11 post-failure prolapsed specimens. A significant difference ($p < 0.01$) in the post-failure disc height was found between the prolapsed discs that reversed and those that did not (Figure 4.14). The change in position of the anterior and posterior margins of the nucleus was of mean magnitude of 2mm (SD 2.2mm) and 2.6 mm (SD 1.3 mm) respectively (Note the width of the posterior annulus in these specimens was in the range of 4-6mm). No change was identified in the posterior margin of the nucleus in two of the five prolapsed specimens after the reversal testing. The review by the radiologist was not only based on the posterior margin but also on posterior volume and clinically important changes. The specimens that did not respond to reversal testing had prolapsed to a greater extent (more volume posteriorly and to apparent outer border of annulus) then those that did respond. The stiffness of the specimens increased over the course of the reversal test from 1.27 to 1.88 Nm/degree when loaded under 260N of axial compression. When considered separately the number of repetitions of motion (failure procedure or reversal testing), the direction of motion (flexion versus combined flexion and side flexion), or the maximum range of the repeated motion did not have a significant effect on the changes in disc height (post-failure procedure or post-reversal testing) or distinguish between those that did not prolapse, those that prolapsed and reversed and those that prolapsed but did not reverse. Three of the six discs that did not respond to 2 of those that did respond reversal testing had circumferential prolapses but no consistent herniation pattern was found to distinguish between the discs that responded and those that did not respond to reversal testing. The lordosis angles of the spines and segments ranged from $28\text{-}42^0$ and $7\text{-}22^0$ respectively

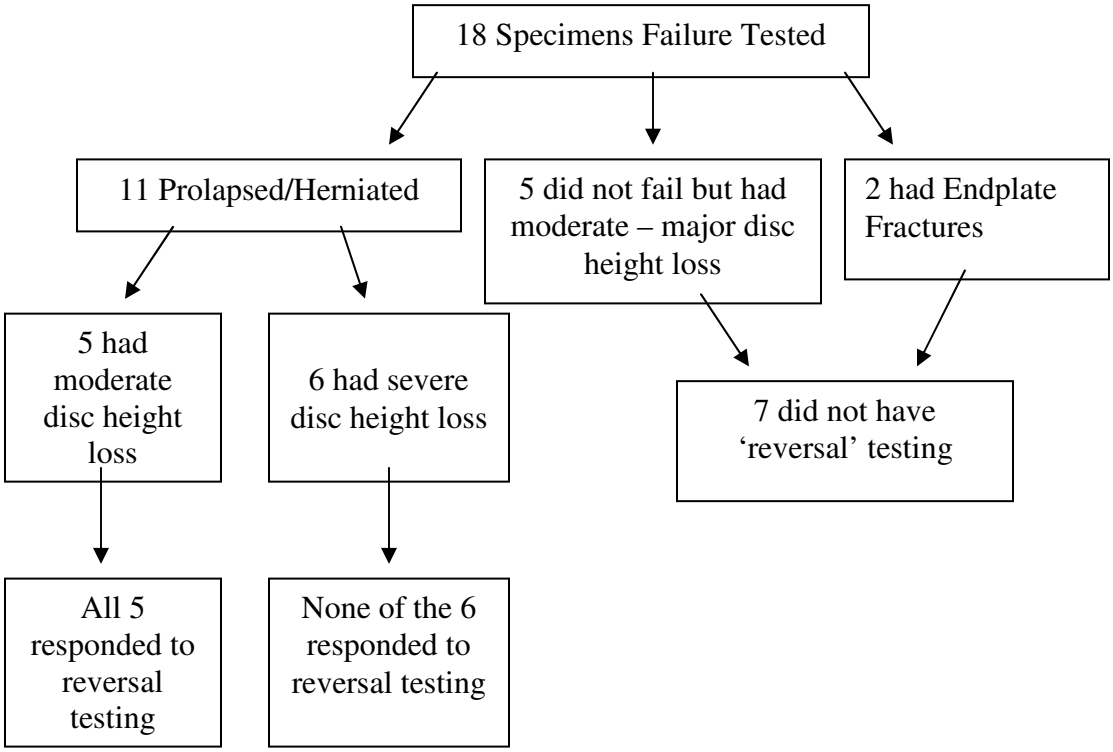

Figure 4.7: An overview of the results of the failure procedure and of the reversal testing is shown. Of note are the 5 prolapsed discs that had moderate posterior disc height loss and responded positively to reversal testing versus the 6 prolapsed discs that had severe posterior disc height loss and did not respond to reversal testing.

Specimen	Post Failure test					Post 'Reversal' test				
	Endplate Fracture Y/N	Posterior migration of nucleus Y/N	Lateral migration of nucleus Y/N	Retro-listhesis	Disc height loss	Reversal of posteriorly migrated nucleus	Reduced posterior volume of migrated nucleus	Reversal of l;aterallymigrated nucleus	Increased retro-listhesis	Disc height change
K	Y	N	N	Y	Severe	N/T				
L*	N	Y	N	N	Mod.	Y	Y	Y	N	N
N*	N	Y	N	N	Mod.	Y	Y	N	N	N
M18	N	N	N	N	Mild	N/T				
O11**	N	Y	N	N	Severe	N	N	N	N	↑
O13**	N	Y	Y	Y	Severe	N	N	N	Y	↑
O132**	N	Y	N	Y	Severe	N	N	N	N	N
O16	Y	Y	N	Y	Severe	N/T				
O162*	N	Y	N	N	Mod.	Y	Y	Y	N	↑
O173	N	N	Y	Y	Mod.	N/T				
O18*	N	Y	Y	N	Mod.	Y	Y	N	Y	↑
O23*	N	Y	N	N	Mod.	Y	Y	N	N	↓
O232	N	N	N	N	Mild	N/T				
O233	N	N	Y	Y	Mild	N/T				
O24**	N	Y	Y	N	Severe	N	N	N	N	↑
O242**	N	N	Y	N	Severe	N	N	N	Minor	↑ ant.
O25	N	N	N	N	Mild	N/T				
N32**	N	Y	N	Y	Severe	N	N	N	N	N

Table 4.2: An independent radiologist's opinion of the changes in the condition of the disc and the change in the position of the "nucleus" with each stage of testing as seen in the x-ray images are outlined. * Five of the 11 prolapsed discs responded to reversal testing. ** The 6 prolapsed discs that did not respond to reversal testing had all lost more disc height after the failure procedure than those that reversed

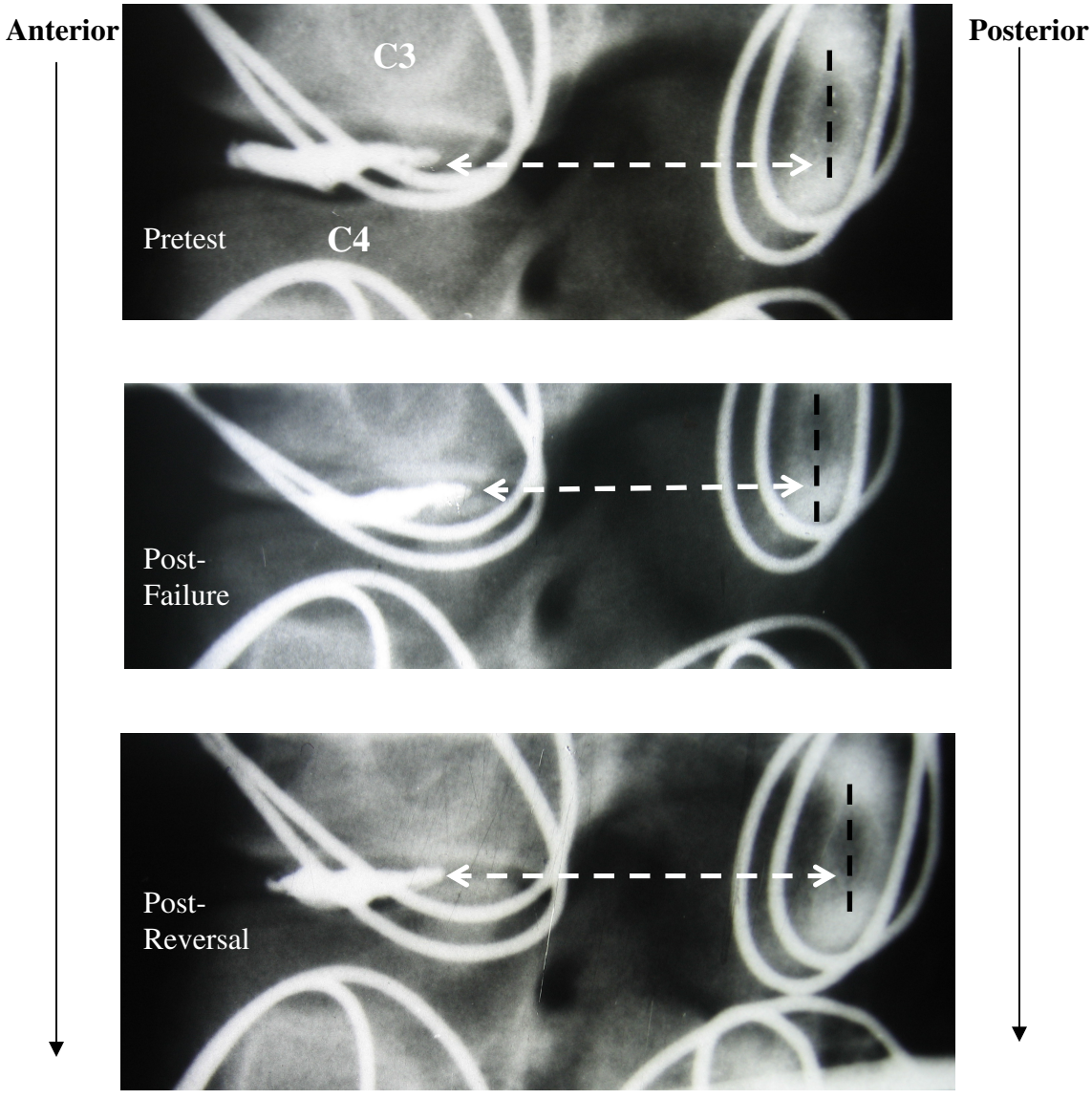

Figure 4.8; The pretest, post-failure and post-reversal lateral images of specimen L. This specimen was one of the five that responded to the reversal testing according to the radiologist. The distance from the posterior margin of the nucleus to the inferior articular process of C3 (black vertical line) is indicated by the white dashed line.

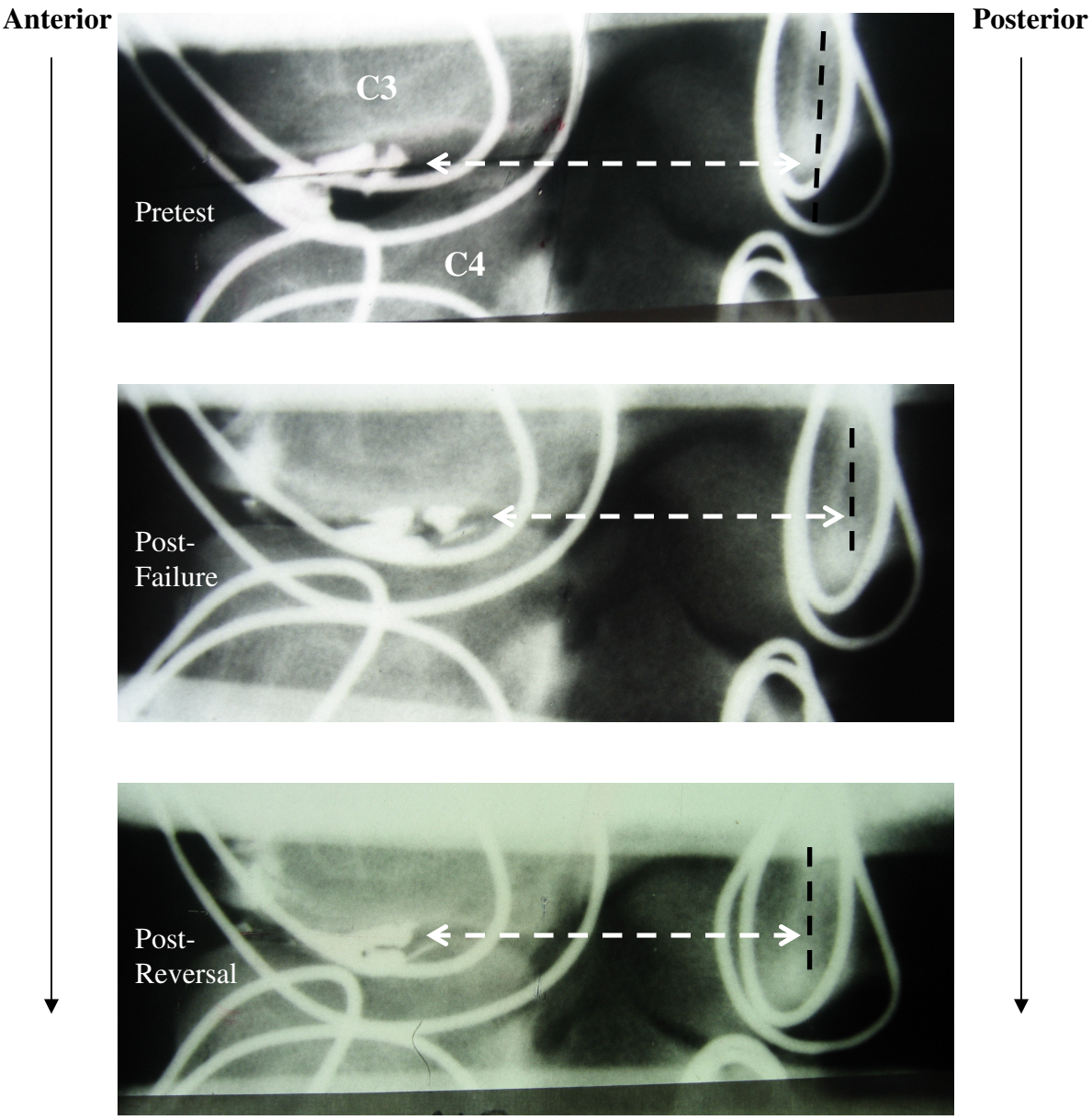

Figure 4.9 The pretest, post-failure and post-reversal lateral images of specimen N. This specimen was one of the five that responded to the reversal testing according to the radiologist. The distance from the posterior margin of the nucleus to the inferior articular process of C3 (black vertical line) is indicated by the white dashed line.

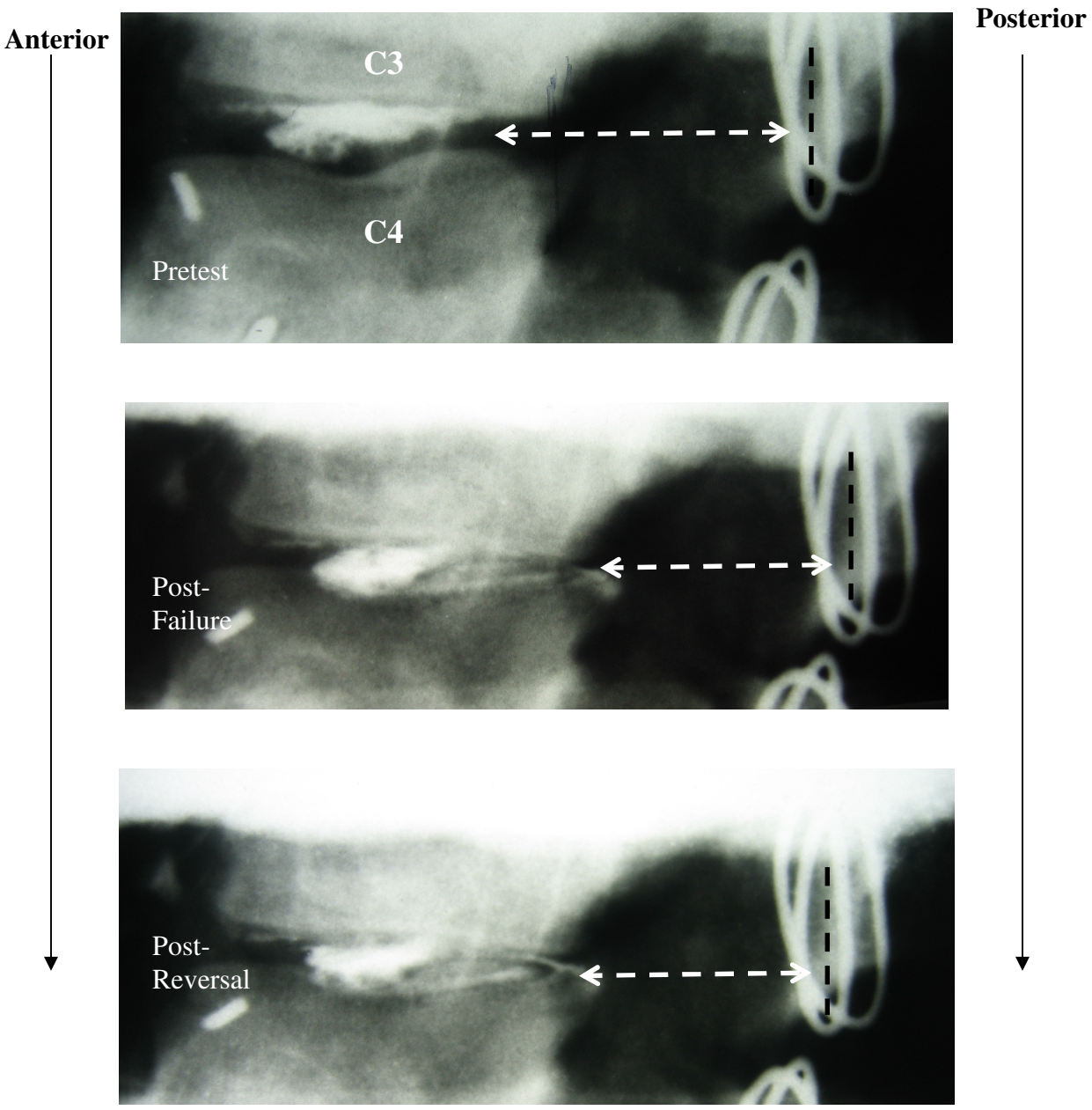

Figure 4.10 The pretest, post-failure and post-reversal lateral images of specimen O162. This specimen was one of the five that responded to the reversal testing according to the radiologist. The distance from the posterior margin of the nucleus to the inferior articular process of C3 (black vertical line) is indicated by the white dashed line.

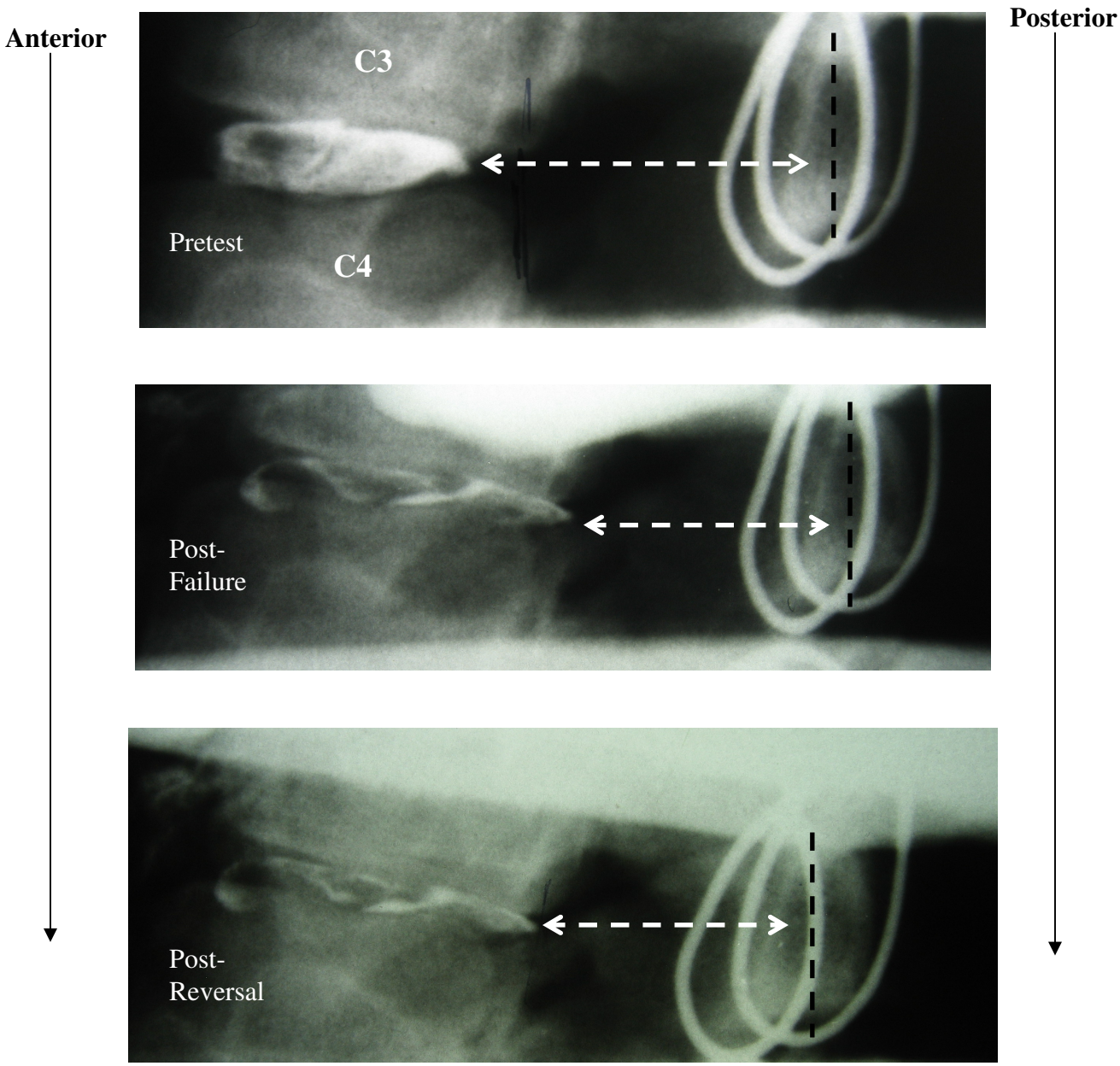

Figure 4.11 The pretest, post-failure and post-reversal lateral images of specimen O18. This specimen was one of the five that responded to the reversal testing according to the radiologist. The distance from the posterior margin of the nucleus to the inferior articular process of C3 (black vertical line) is indicated by the white dashed line.

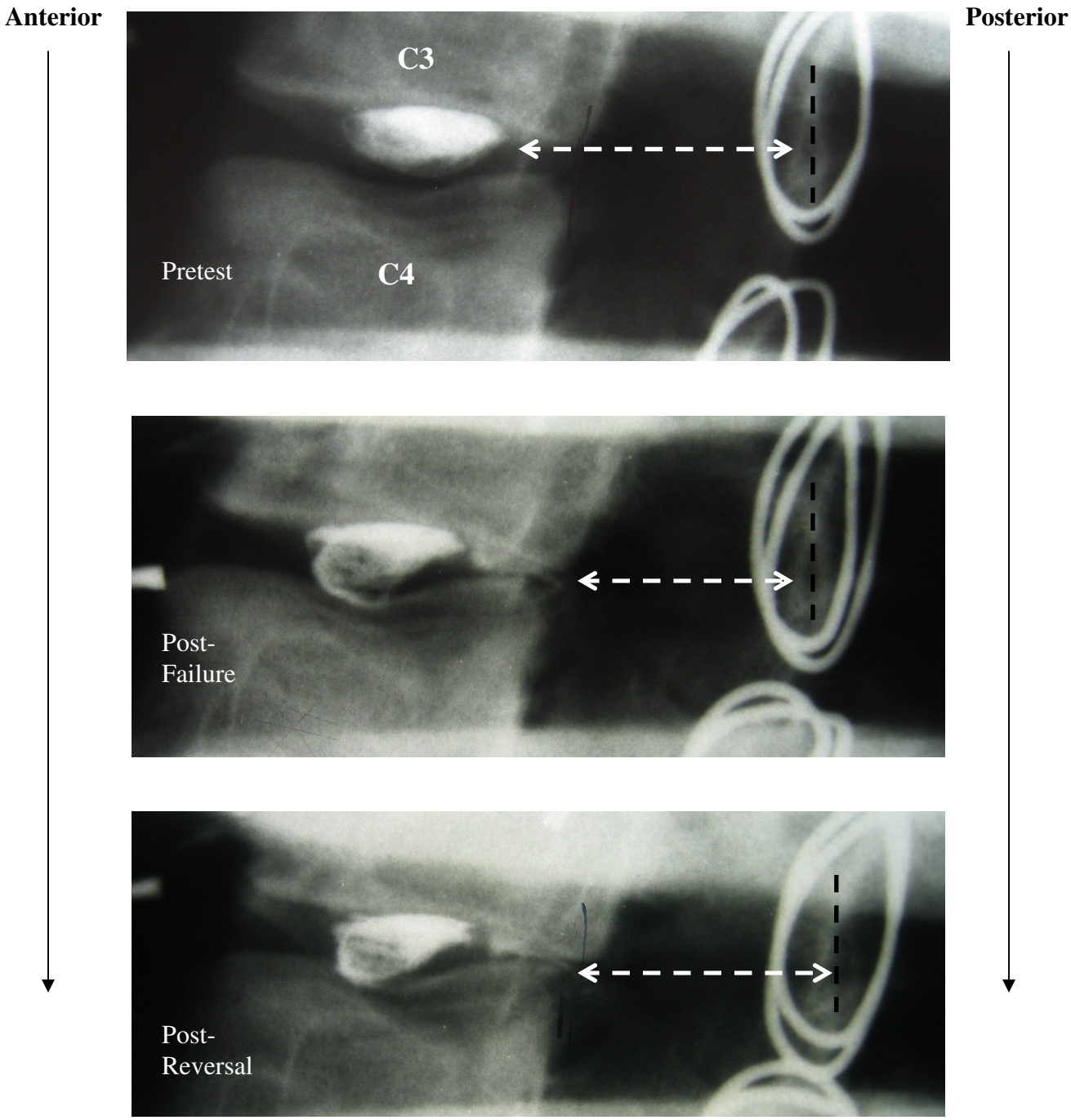

Figure 4.12 The pretest, post-failure and post-reversal lateral images of specimen O23. This specimen was one of the five that responded to the reversal testing according to the radiologist. The distance from the posterior margin of the nucleus to the inferior articular process of C3 (black vertical line) is indicated by the white dashed line.

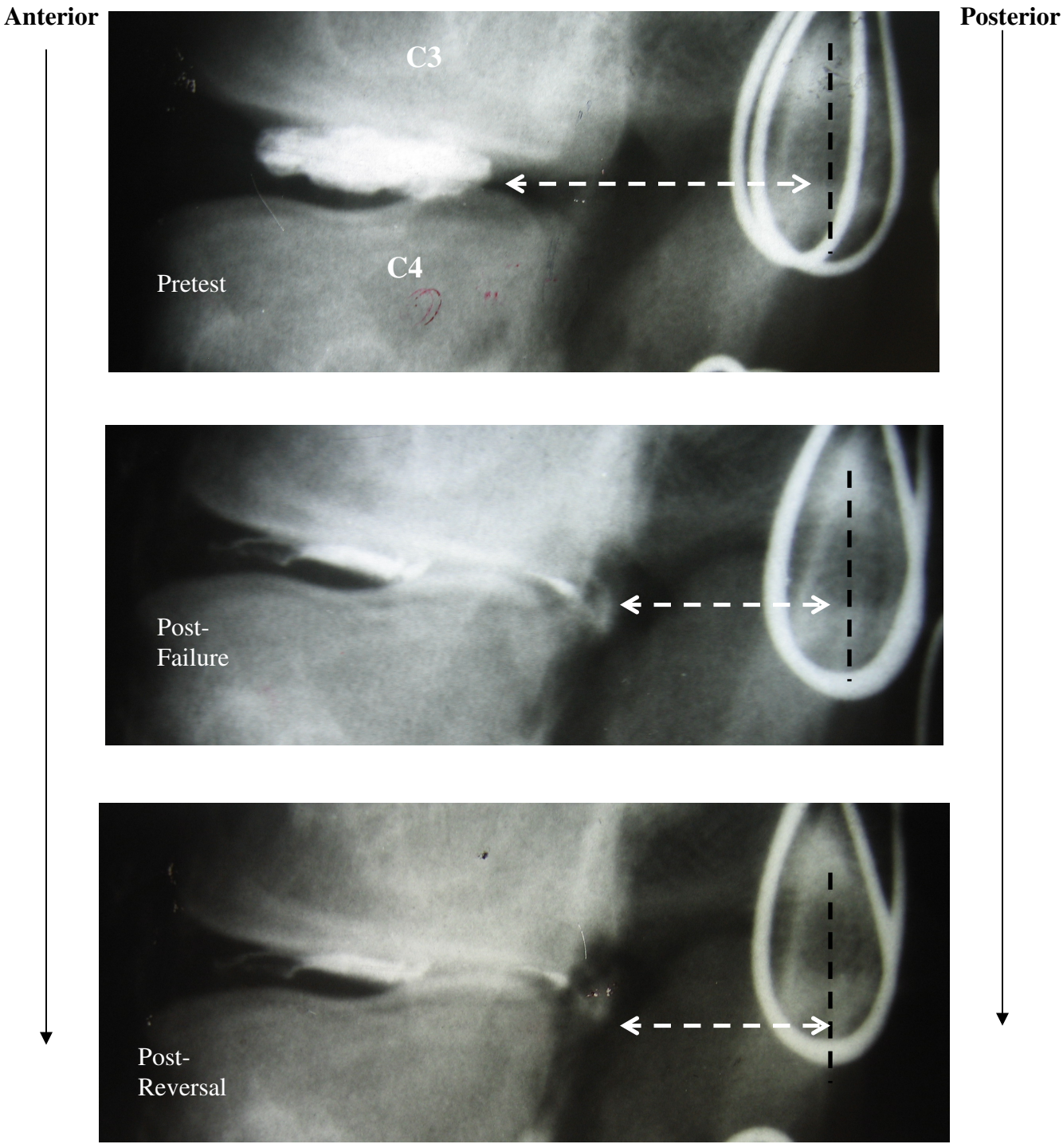

Figure 4.13; The pretest, post-failure and post-reversal lateral images of specimen O132. This specimen is one of the six prolapsed discs that the radiologist's regarded as not changed after the reversal test. The distance from the posterior margin of the nucleus to the inferior articular process of C3 (black vertical line) is indicated by the white dashed line.

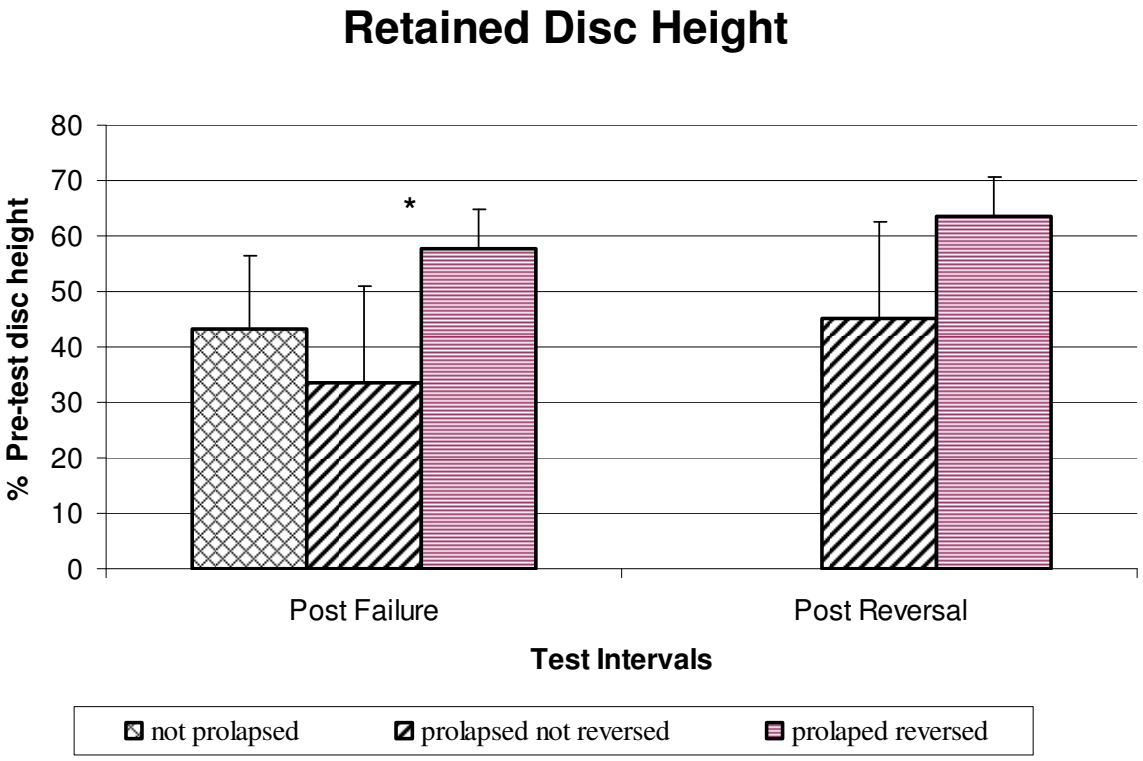

Figure 4.14: The disc height, measured according to Wilke et al (2006), after the failure procedure and after the reversal testing of the three categories of specimens is shown. The specimens that prolapsed and responded to reversal testing had significantly more disc height after the failure procedure then those that did not respond to reversal testing.

and did not distinguish between the groups. As seen in table 4.1 the reversal testing that centralized the disc prolapses did so with movements in the same plane but opposite direction to the movement that caused the prolapse, in all but one case. In the case of O23, a specimen that failed with combined flexion and lateral flexion, was 'reversal' tested with pure sagittal extension as the prolapse appeared in the sagittal plane only.

DISCUSSION

Repeated extension (or combined extension and side bend) following posterior (or posterolateral) disc prolapse was found to make a positive clinical change in the displaced portion of the nucleus in a number of discs. In so doing this study is a proof of the principle on which this aspect of the McKenzie approach is based. To the author's knowledge this is the first scientific evidence supporting the theory that repeating movements opposite to those that caused posterior migration of the nucleus can alter the prolapsed material. The results indicate that the McKenzie approach works on some prolapsed discs and not on others. Consideration of the changes in disc height of the specimens during the testing procedures offers some understanding of the varied success of this approach. The disc height loss after the failure procedure distinguished between specimens that responded to reversal testing and those that did not respond. Classifying the type of prolapse as circumferential or radial did not throw consistent light on the varied success nor did consideration of the lordosis angle of the specimens. These findings provide support for the postulated mechanism of the success of the McKenzie approach. The success of this approach is likely modulated by a number of factors, disc height loss being one such factor.

The limitations of this study include the use of an animal model. Animal models are a necessary part of the quest for a greater understanding of in vivo biomechanics and are helpful in understanding clinical observations. These models are controllable, reproducible and cost-effective, unlike specimens from human cadavers where age, exercise level, diet, environment, personal factors and genes add variability to a sample. More specifically, the porcine cervical spine model has been shown to be anatomically, geometrically and functionally similar to human lumbar spines (Yingling et al 1999, Oxland et al 1991) and was not only a reasonable surrogate for human spines but also provided a reproducible model. The use of an in vitro model to replicate an in vivo phenomenon hinders the interpretation of such work in an in vivo context. Dhillon et al (2001) and Callaghan and McGill (1995) have shown that freezing does not change the time-dependant behavior nor elastic properties of the frozen specimens and thus freezing is not considered to significantly alter the results obtained from this in vitro model. The number of repetitions of motion applied to the specimens in

this study would occur in less than 1 week when flexion/extension cycles occur at a rate of 6 motions per minute (Brinckmann et al 1988). This timeframe would precede the minimum 500 days required for collagen repair and proteoglycan turnover in vivo (Adams and Hutton 1982, Urban et al 2004). The disc height loss associated with this testing methodology could be considered excessive relative to young in vivo non-degenerate prolapsed discs. Previous failed/ partially successful attempts to improve this included intermittently unloading the specimen in a saline submersion bath, injecting the disc during the test and leaving an IV line in situ hooked up to an infusion pump during the test. While re-hydrating the disc either in a saline submersion bath or by injecting the disc increased the disc height, a short period of further testing resulted in an almost immediate loss of disc height. Knowing the disc height loss issues associated with this methodology provided a framework for interpretation of the results. To facilitate x-ray tracking of the nucleus it was necessary to add 0.55ml of a radio-opaque mixture to the nucleus of the disc. This changes the intradiscal pressure (IDP) and the stiffness of the segment. Decreasing the volume of the radio-opaque mixture was not an option as it reduced the resolution of the x-ray images. There was concern that the increase in IDP would result in increased frequency of endplate fracture but this was not found. The link between diagnostic imaging findings and the various clinical presentations of clients with a disc prolapse is still lacking. Given the difficulty of interpreting quantified changes in the position of the nucleus qualitative analysis was the only option available. The independent radiologist's analysis of the x-ray images was from a clinical perspective, that being changes that could have clinical significance.

Previous studies have investigated components of the mechanical theory to which the success of the McKenzie approach is attributed. Donelson et al (1997) investigated and supported the theory that discs that respond to McKenzie have a competent annulus. The basis for the study was McKenzie's speculation that the discs that respond to the repeated movement approach have a hydrostatic nucleus and an intact outer border of the annulus, collectively described as the dynamic internal disc model. Donelson et al (1997) in a prospective study evaluated the existence of a relation between patterns of pain response during a clinical mechanical assessment (McKenzie approach) and annular competence findings of discography. The symptoms of 50% of the subjects centralized during the McKenzie assessment. Seventy four percent of these subjects had positive discograms (reproduced pain pattern) and 91% of the 74% had a competent annulus. The strong predictive value of centralization has been shown previously (Long 1995, Donelson et al 1990) whereby centralizers, especially those with only 1-4 weeks of symptoms, have faster recoveries than non-centralizers. While these studies support the clinical utility of the McKenzie approach they do not provide insight into the mode of effect of this approach. The current study shows a reduction of the prolapsed nucleus in a number of specimens, which could account for the reduction in symptoms caused by

nociceptor stimulation in the posterior annulus, posterior longtitudinal ligament or nerve root that is seen clinically. Mechanically Adams et al (2000) suggested that stress shielding by the neural arch could account for the success of extension exercises in vivo as loss of disc height decreased the more commonly increased posterior annular stress associated with extension. The disc height of the prolapsed specimens increased marginally after the reversal testing which supports the argument that stress shielding may be part of the mechanisms underlying the success of the McKenzie approach. However, in the current study the specimens that responded to the reversal testing were not the specimens that had lost most disc height, which prompts further consideration of the possible mechanism. Flexion increases compression of the anterior annulus while reducing compression and causing tension of the posterior annulus (Shah 1980, Adams and Hutton 1982). There is increased hydraulic stress on the posterior annulus as fluid moves away from the high anterior compression. Brault et al (1997) and others (Schnebel et al 1988, Fennel et al 1996) have shown, using magnetic resonance imaging (MRI) pixel intensity changes, that the nucleus of the lumbar discs moves posteriorly by approximately 1mm on flexion of the spine segments. The reverse is seen on extension of healthy spine segments. Extension involves a posterior sagittal rotation and a small posterior translation. It is associated with increased compressive stress within the posterior annulus and a reduction in the compressive stress in the anterior annulus and nucleus (Adams et al 2000). In such a case it is mechanically understandable that in a healthy disc the posterior aspect of the nucleus could move more anteriorly with extension. The difficulty arises when trying to explain how a displaced portion of the nucleus that lies within the posterior annulus would move more anteriorly. Figure 4.15 shows an example of the order of selection of the McKenzie procedures associated with varying levels of severity of disc prolapse. The order suggests that the effectiveness of treatment is based on achieving a greater range of extension, even if under a lower level of axial compression. As shown in Figure 4.16 a very extended disc would have a greater stress gradient within the posterior annulus than a disc that is under a higher level of compression but is less extended. The fluid and displaced portion of the hydrostatic nucleus within the posterior annulus will move from the higher level of compression to a lower level of compression within the posterior annulus. The hydraulic pressure would be in an anterior direction. Based on this argument the discs that have greater disc height are more likely to reverse as greater extension of the segments can occur before the facet joints bring the range to a halt and also the stress in the posterior annulus is compressive rather than tensile (figure 4.1). Such a mechanism would also explain why in cases with milder disc prolapse extending in standing rather than lying appears more effective. The overall compression of the posterior annulus may need to be increased as the stress gradient in lying is only sufficient to move the displaced nucleus partially back to the center of the disc. Two factors, possibly combined to some degree, may

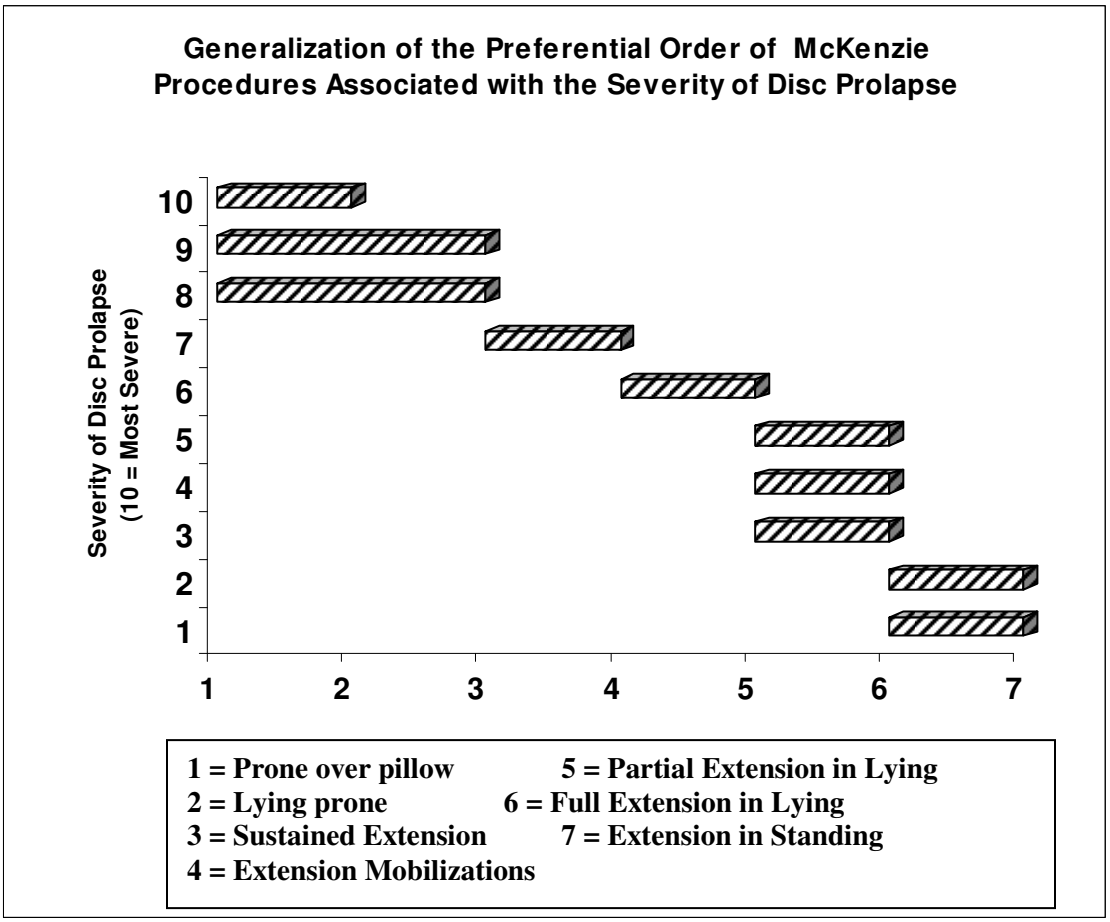

Figure 4.15: shows an example of the McKenzie procedures selected according to the severity of the disc prolapse. The list is not intended to be all inclusive nor is the order set in stone. The order suggests that the effectiveness of treatment is based on achieving a greater range of extension, even if under a lower level of axial compression.

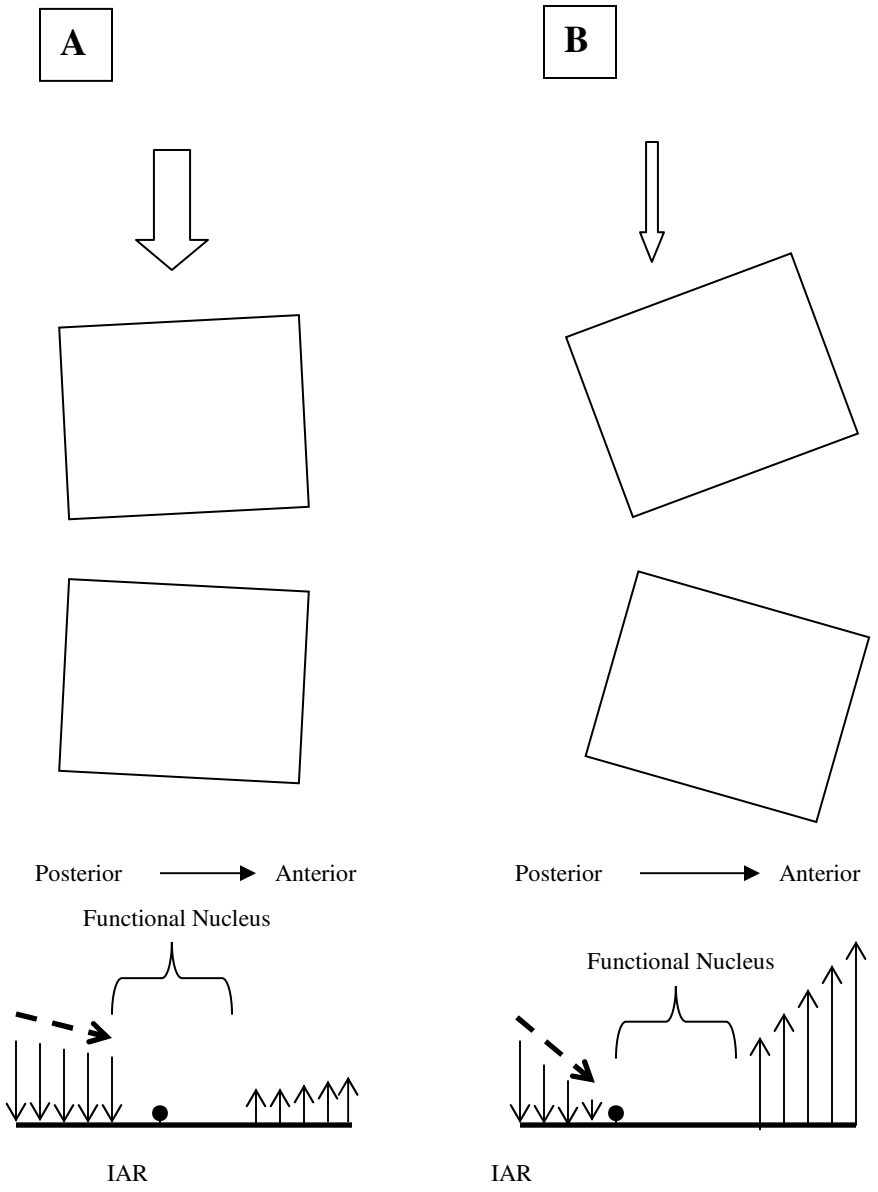

Figure 4.16 A schematic of the stresses in the annulus of the disc under different loading patterns is shown. The rectangular boxes represent the vertebral bodies. In case A, intended to represent an attempt at extension in standing in an acute disc prolapse, the disc is loaded under axial compression in a slightly extended position. In this case there is a relatively high compressive force but not a large stress gradient within the posterior annulus. In case B, representing the greater extension that can often be achieved in acute disc prolapse with prone lying, extension mobilizations and extension in lying relative to that in a standing position, the stress gradient (Black dashed arrow) across the posterior annulus is higher. The displaced portion of nucleus will move away from the higher stress in the outer posterior annulus. The hydraulic pressure is in an anterior direction.

account for the change in position of the anterior margin of the nucleus. Firstly, it has been shown that the nucleus of the lumbar discs moves posteriorly on flexion of the spine segments (Brault et al 1997, Schnebel et al 1988, Fennel et al 1996, Lyndsay et al 2007). Given the high number of repetitions of flexion used in this study there may be more than normal buckling of the inner anterior annulus causing the nucleus to move a little more posteriorly than previously reported. The increase in stiffness of the specimens, a measure of the damage of the IVD (Thompson et al 1990), did not correlate with the disc height loss nor did it distinguish between the categories of specimen behavior. The change in stiffness levels was similar to that reported by Callaghan et al (2001) and Drake et al (2005), both of which used comparable models and testing parameters. The increase in stiffness of in vitro specimens is usually explained to result from a change in the contact points across the segment associated with a decrease in disc height. However, the displaced portion of the nucleus within the posterior annulus may alter the torque required to rotate the specimen. It is interesting to link this to clinical practice as it has been suggested that the prolapsed portion of nucleus mechanically blocks the posterior sagittal rotation of the specimen. Due to the variability in the degree to which these specimens are prolapsing it is not possible to make a categorical link between the size of the displaced portion and the increase in the stiffness of the specimens.

Worthy of consideration is the cost of extension which may be beneficial for a prolapsed disc but it imposes increased stress on other spinal tissues. Extension of the spine involves posterior sagittal rotation and posterior translation, the limit of which is marked by an inelastic, non-recoverable deformation of the motion segment at some angle between 3^0 and 8^0 (Adams et al 1988). The spinous process, the interspinous ligaments and the facet joints may be damaged in extension depending on the spacing between the spinous processes. Hedman et al (1997) in their cadaveric study of male human spines found that sitting in extended postures increased the facet forces in the L4-5 IV joints. The articular surfaces of the facet joints did not show signs of fracture after extension testing in this study. Future studies will address the balance between movements that help the disc but hinder the health of other tissues.

Identifying biomechanical factors that can be used to predict ones risk of developing LBP is central to successful prevention of LBP. Based on a clinical observation spine posture seems to be one such biomechanical factor, for example individuals with hypolordotic spines seem to have a predisposition to disc prolapse. Sahrmann (personal communication with McGill) has also observed that individuals with hypolordotic spines tend to be the ones that have had a laminectomy to remove extruded nuclear material. Scannell and McGill (2003) investigated and supported the hypothesis that individuals with hypolordotic spines have increased flexion stresses in sitting, standing and walking relative to hyperlordotic spines. Individuals with hypolordotic lumbar spines sat in more lumbar

flexion than non-hypolordotic spines, increasing their risk of flexion related tissue damage, for example disc prolapse. The lordosis angles were measured in this study in an attempt to gain further insight into posture related risk. In the current study the lordotic angle of the spine or the segment did not distinguish between the outcome groups nor was there a link between either angle and the number of repetitions required to cause the disc to prolapse. The in vivo range of lordosis of the human lumbar spines has previously been reported to vary between 16 and 70^0 (Hansson et al 1985, Murrie et al 2002), which is much greater than the variability seen here in the porcine cervical osteoligamentus spines ($28 – 42^0$). At this point the link between the amount of lordosis and the potential for prolapse remains with the stresses imposed on the spine (Scannell and McGill 2003).

We confirm that the McKenzie approach to the treatment of disc prolapse works in some discs. The displaced portion of nucleus in the annulus was at least partially returned to the normal central position of the disc. The factor that distinguished between those that responded and those that did not respond to reversal testing were the post failure disc height. Loss of disc height has previously been shown to alter a number of mechanical characteristics of the segments including: the viscoelastic properties of the nucleus such that the nucleus behaves more as a solid then a fluid; the intradiscal pressure; the extension axis of rotation of the specimen. This study sheds light on a theory that has been unsubstantiated in physiotherapy for over 20 years. It is a starting point from which numerous other studies will develop. Two logical lines of research arise from this study – investigation of (1) the stresses driving the return of the displaced portion of nucleus towards the center of the disc and (2) the most optimal 'dose' and technique of extension and combined extension and side bend to prevent and rehabilitate disc prolapse.

CONCLUSION

This study has provided evidence of a mechanical foundation for the success of the McKenzie derangement approach in specific individuals, which is aimed at resolving symptoms and signs related to disc prolapse. The displaced portion of nucleus was returned towards a more normal central position in a number of prolapsed discs. A number of other discs, that tended to loose more disc height, did not respond to the reversal testing, a finding that will spark further investigation of the selection of such practice and the stress gradient driving the movement of the nucleus. Also outstanding are studies investigating the recommended McKenzie 'doses' in acute, subacute and chronic phases of rehabilitation (sustained versus repeated extension), the impact such 'doses' may have on the health of other spinal tissues and the role of repeated extension in the prevention of recurrence of aberrant displacement of the nucleus.

CHAPTER 5

MCKENZIE EXTENSION EXERCISES – QUANTIFIED AND CONSIDERED

ABSTRACT

Background; The McKenzie approach to the treatment and prevention of mechanical low back pain is a two-tiered approach that involves identifying the appropriate exercises to be performed by the client as well as the specific motions, postures and loads to be avoided in order to accelerate recovery and reduce the risk of recurrence of the condition. Extension and extension with side bend motion are movements recommended in disc prolapse rehabilitation and prevention by the McKenzie approach. The kinematics of these exercises has not been quantified nor evidence supporting clinical selection of the exercises considered, both of which could improve the effectiveness and safety of the practice. The spinal range in a number of activities of daily living was also measured.

Methodology; Twenty asymptomatic subjects volunteered to participate in this study and performed frequently prescribed McKenzie exercises and a selection of activities of daily living during which a 3-SPACE Isotrak system measured their three dimensional lumbar kinemetics. One subject underwent a series of McKenzie exercises while electromyography and three-dimensional lumbar motion were measured.

Results; Mean peak extension of extension in standing and extension in lying exercises were within 3% (SD 22.33%) of each other. An additional 6.75% (SD 11.18%) of extension occurred when extension in lying was combined with a Grade 3 Maitland extension mobilization to L3. The peak extension during the extension in lying exercise was increased after the mobilization relative to the pre-mobilization range. The mean peak right side bend in the right side glide exercise, normalized to the full right side bend range, was 61% (SD 17.4%). The mean peak left side bend position at peak extension in extension left side glide was only 21% of the full left side bend range. The L4-5 forces at the position of peak extension in extension in lying and extension in standing were 828.97N and 1368.86N respectively. The shear forces in these exercises are less than 100N, considered low shear levels relative to the previously reported spine shear tolerance. The peak flexion ranges of the activities of daily living investigated match those previously used to create disc prolapse when applied under moderate load and high repetitions.

Conclusions; For the first time the lumbar spine ranges achieved in commonly prescribed McKenzie exercises have been quantified. The results of this study enhance clinical practice by

providing quantitative evidence of the relative peak motion of the McKenzie exercises. The compressive and shear forces identified in the case study increase our understanding of the potential kinematic and kinetic differences in these exercises. The peak flexion ranges of the ADL investigated match those previously used to create disc prolapse, although the prolapses were created under extremely high repetitions of these ranges. This would imply that educating clinicians and individuals regarding the need to eliminate/modify these causative/aggravating factors is important, until further research provides a greater understanding of the repetitions of these motions required to cause or exacerbate disc prolapse is completed.

INTRODUCTION

The McKenzie approach to the treatment and prevention of mechanical low back pain (MLBP) is a two-tiered approach that involves identifying the appropriate exercises to be performed by the client as well as the specific motions, postures and loads to be avoided in order to accelerate recovery and reduce the risk of recurrence of the condition. The McKenzie approach supposes that the displaced portion of nucleus in a prolapsed disc can be moved back towards the center of the disc using movements and positions specific to the direction of disc prolapse. In general, for a posterior or posterolateral disc prolapse the recommended exercises involve extension movements while flexion movements are those to be avoided. Extensive research on the efficacy of McKenzie exercises has been documented (Faas 1996, Philadelphia Panel 2001, van Tulder et al 2000) but the kinematics and kinetics of these exercises have not been quantified.

The importance of quantifying these frequently prescribed exercises is highlighted by research that has identified risks and benefits of extended postures. Generally, posture affects the hydraulics of the disc and the disc-facet joint load sharing. Adams and Hutton (1985) found lumbar lordosis to be inversely proportional to intradiscal pressure (IDP), the more extended the sitting or standing lumbar posture the lower the IDP relative to sitting or standing in flexed postures. Adams et al (1999) reported that reduced lumbar lordosis was predictive of serious MLBP (defined as LBP requiring medical attention or time off work) in a group of 403 healthy volunteers recruited for a prospective 3-year study. They suggest that the curvature of the lumbar spine in lordosis allows it to bend and absorb more strain energy than if it were simply compressed thus acting as a shock absorber. The disc shear and ligament forces were higher in the flexed posture than in the extended posture. These results coincide with those of Burton et al (1994) who reported that compressive forces within the disc are lessened in extended postures. Larsen et al (2002) reported significantly fewer persons reported MBLP in a group that performed 15 repetitions of extension in lying in the morning and evening in comparison to a group that did not perform these exercises.

While reducing IDP, disc shear and ligament forces could be considered assets of extended postures extension has also been associated with a number of biomechanical liabilities. It has been identified as being injurious to the facet joints (Shirazi-Adl 1991, 1994, Dunlop et al 1984). Under a 10Nm extensor moment the L4-5 facet articular processes carried a contact force of approximately 90N in comparison to zero contact force at the L4-5 level under a flexor moment of 10Nm (Shirazi-Adl 1994). Addition of compression increased these contact forces in extension while it had no effect on them in small degrees of flexion. In full extension the inferior articular processes abut on the lamina below bringing extension to a halt and sclerotic changes can be seen in this area. The extension forces are transmitted through both the articular surfaces of the facet joints and the capsular

ligaments that enclose the joints. These facet joint capsules are strained at end of range movements and are pain sensitive. Signs of excessive facet joint loading are splitting of the articular cartilage, especially in the superior articular processes, and thickening of the subchrondral bone. In extension the restraining forces from the superior articular processes of the facet are transmitted through the pars interarticularis and may result in spondylolysis (fracture of the pars interarticularis), and eventually lead to spondylolisthesis (slippage of one vertebrae of the one below). Furthermore, extension can also crush the interspinous ligament complex at the level of the "kissing" spinous processes. In summary, extension of the spine can have a positive effect on the disc but these benefits must be weighed against the possible liabilities of extension. An understanding of the biomechanics of the McKenzie exercises will assist in selecting the appropriate exercises, which could improve the effectiveness and safety of clinical practice.

McKenzie promotes teaching clients to avoid particular movements or positions that will exacerbate their condition. Flexion motion, or flexion with side bend and rotation, when combined with modest compression loading have been identified as causative factors in the production of disc prolapse (Callaghan and McGill 2001, Gordon et al 1991, Aultman et al 2005). Compression of a neutral spine, however, rather than a flexed spine, was not sufficient to produce a disc prolapse in a study by Brinkmann (1986). Combined these studies suggest that while higher compression levels accelerate the production of disc prolapse (Callaghan and McGill 2001) particular kinematics are vital to the loading mechanism that will result in disc prolapse. Eliminating damaging motion is considered fundamental to prevention and rehabilitation of the symptoms and signs related to disc prolapse (McKenzie 2002). Previous studies have reported on the spine kinematics of workplace tasks (Marras et al 1995, Granata et al 1999, Punnett et al 1991) but little research describing the kinematics of everyday activities exists. Dolan et al (1988) investigated the lumbar curvature during commonly adopted sitting and standing postures and reported that many of the postures investigated resulted in reduced lumbar lordosis in comparison to erect sitting and standing. There was a consistent preference for flexed postures; sitting on the floor with the knees drawn in produced the most lumbar flexion (34^0), findings that suggest that the kinematics of activities in daily living (ADL) may be problematic. However, Godin et al (2003) used a video based 3D postural sampling approach to evaluate spine loads during home-based activities (cooking, cleaning, laundry, vacuuming, gardening, cutting the grass and tending to an outdoor swimming pool) and reported that despite high compression loading during these ADL the lumbar spine was in a neutral position (for 85.3% of the testing time) which suggests that the kinematics may not be problematic. Generally, clinicians rely on experience to identify specific functional activities that may exacerbate discogenic pain. Documentation of spine motion during ADL would be helpful. Many clients report "doing nothing"

prior to an episode of discogenic pain; yet unbeknownst to them they may have replicated the injury mechanism through dynamic motion or static postures. The second question addressed in this study was related to the spine kinematics of (ADL), specifically - do seemingly benign activities of daily living (ADL) involve sufficient levels of flexion, side bend and rotation to cause/exacerbate disc prolapse?

The objectives of this study were to quantify the kinematics of commonly prescribed McKenzie exercises and techniques as well as the kinematics of particular ADL. The McKenzie exercises considered were as follows; extension in standing (EIS); extension in lying (EIL); extension mobilization in EIL; EIL after mobilization; right side glide in standing (RSG); EIL in left side glide (ELSG). The ADL considered were Toilet Flush From Standing; Toilet Flush From Sitting; Reaching to a coffee table with the left hand; Reaching to a coffee table with the right hand; Reaching to answer phone when sitting at a desk; Reaching to answer phone when sitting at a desk and then leaning onto right armrest; Reaching to answer phone when sitting at a desk then leaning onto right armrest and cross left leg over right.

Specifically the hypotheses were;

H: The extension of EIL would be greater than that of EIS

H: The range of extension would be increased during and after extension mobilization

H: The peak flexion, ± side bend and rotation, ranges of the ADL investigated will match those previously used to create disc prolapse.

The information gained from this research will increase clinicians' understanding in advance of making decisions about prescription of McKenzie exercises and will put clinicians in a better position to weigh the potential benefits and risks of the exercise approach. An increased understanding of the spinal motion that occurs during these particular ADL will either prompt clinicians to include advice regarding such activities or render such advice as unnecessary.

METHODOLOGY

Subjects

Twenty subjects, asymptomatic at the time of testing, volunteered to participate in this study. Asymptomatic subjects were chosen as the severe intensity of the pain during an acute disc prolapse combined with the risk of exacerbating the condition deemed such a symptomatic subject pool as inappropriate for this exploratory study. All 20 subjects were clients of a physiotherapy clinic, 6 of the 20 had previously participated in a MLBP treatment program at the clinic, 4 of the 20 had a

history of MLBP but were not treated at the clinic for MLBP, while the remaining 10 did not have a history of MLBP and were treated at the clinic for non-LBP related issues. (5 males and 5 females each with a positive history of MLBP, mean age 42.8 and 47.8 years respectively: 5 males and 5 females with a negative history of MLBP, mean age 43.6 and 42 respectively), This study was approved by the University Office of Research Ethics.

Procedures

The kinematics of the lumbar spine were measured with a 3-SPACE Isotrak system (Polhemus Navigation Systems, Colchester, VT) where a source producing a high-frequency magnetic field was secured to the pelvis of subjects with straps around the back and between the legs. A sensor module was placed over the T12 spinous process. In this way, only rotational displacement of the ribcage (sensor module) relative to the pelvis (source) was detected. This system measured the three directional cosines about the orthogonal axes of flexion-extension, lateral bending, and axial rotation. The zero position of the 3-space system was a template position (source and sensor aligned horizontally) used for all subjects. This device has its own A-D converter, sampling at 60 hertz, while storing the three angular measurements on a computer.

Three repetitions of each of the following motions were performed;

Establish full active range of motion (ROM trials)

Subjects performed full active sagittal plane movements (flexion, extension) followed by full active frontal plane movement (right and left side bend) starting in a standing position. They were instructed to keep their knees straight during the movements and to determine their own speed of movement but were advised that they would be prompted if they were going too slowly. Three trials of both sagittal and frontal plane movement were collected.

Extension in standing (EIS)

Subjects were instructed to bend backwards, while keeping their knees straight, supporting their trunk by keeping their hands on their hips and remain in the extended position for the count of 3 (Figure 5.1).

Overhead reaching

This is not a McKenzie exercise but was included as it was of interest to the researchers. From a natural standing position the subject reached both hands towards the ceiling while continuing to look

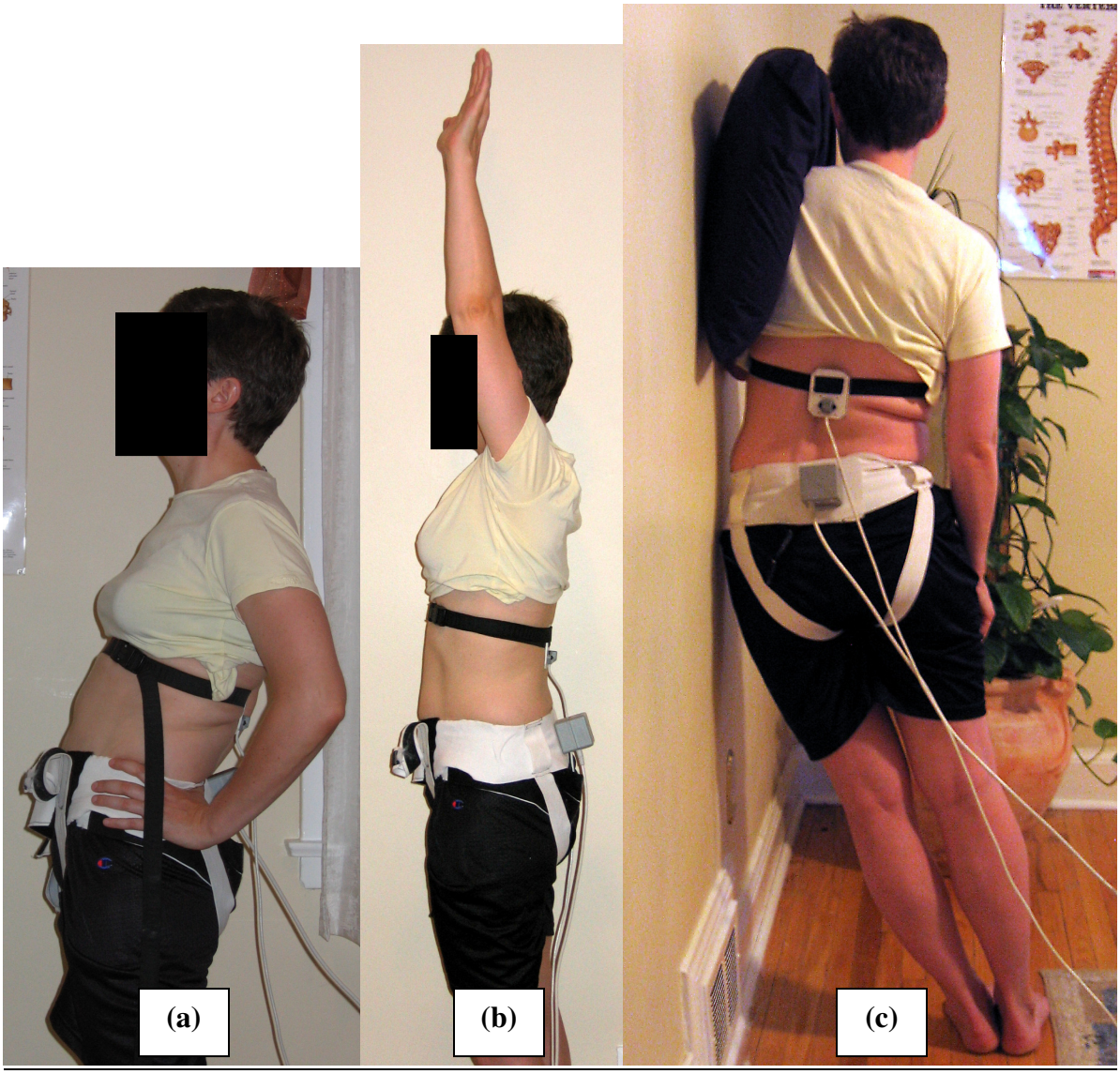

Figure 5.1: The exercises performed from an upright position are shown. (a) Extension in standing (EIS): The subject supported her trunk by keeping her hands on her hips while bending backwards (b) Overhead Reach (OHR): From a natural standing position the subject reached both hands towards the ceiling while continuing to look straight ahead (c) Right side glides in standing (RSG): The subject stood with her feet 30cm from a wall, rested her left shoulder against the wall and moved her hips to the wall.

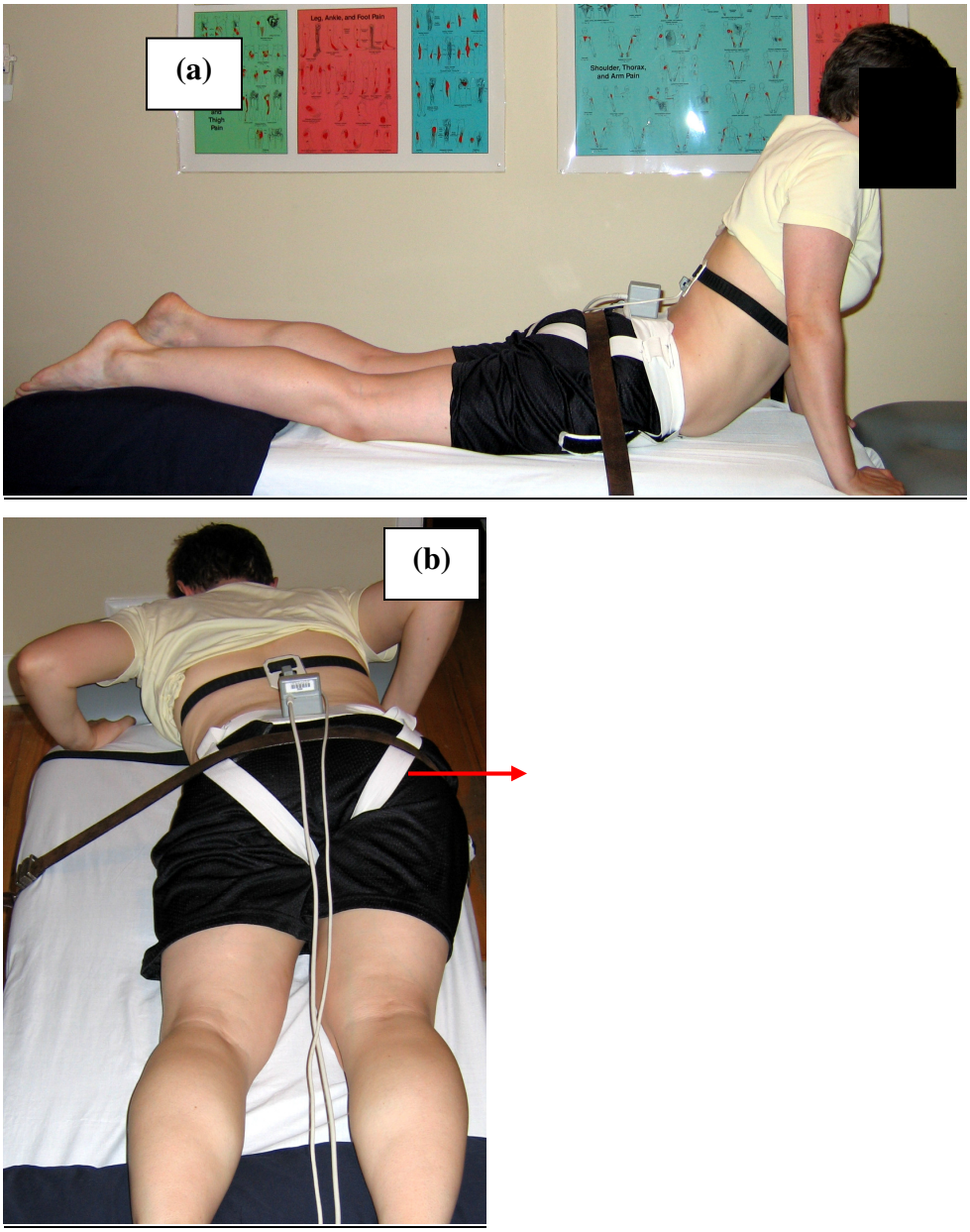

Figure 5.2: Two of the four exercises performed from a prone lying position are shown. (a) Extension in Lying (EIL): The subject used her arms to raise her trunk off the bed (b) EIL in left side glide (ELSG): The starting position of the exercise is shown. While in a prone position the subject's hips were translated maximally to the right by the student investigator (left side glide in McKenzie nomenclature) putting the subject's lumbar spine into a left side bend position. From this starting position the subject repeated an EIL.

straight ahead. Once in this position the subject held the position for the remainder of the 6 second trial (Figure 5.1).

Right side glides in standing (RSG)

The subject stood with their feet one foot from a wall, with their left side closest to the wall. Keeping their feet in that position he/she leaned towards the wall, resting his/her shoulder against the wall. Starting in this position the subject then moved his/her hips to the wall (Right slide glide in McKenzie nomenclature), keeping his/her feet and shoulder in the starting position, held this position for the count of 3 and then returned to the starting position (Figure 5.1).

Extension in lying (EIL)

The subject started in prone lying (face down), with his/her hands under the shoulders, the pelvis strapped to the bed. Using his/her arms the subject raised the trunk off the bed as far as possible and held that position for the count of 3 (Figure 5.2).

Extension mobilization in extension

While the subject was in the extended position of the EIL exercise the student investigator performed Grade 3 Maitland extension mobilizations on L3 (L3 was the most accessible spinous process in this position with the 3-Space equipment in place). Maitland extension mobilization is a physiotherapy technique that involves the therapist applying intermittent low amplitude passive oscillations to, in this case, the posterior aspect of the spinous process with the goal of subsequently increasing the range of active motion in the direction of the mobilization. The purpose of the technique is to passively move the vertebra in the desired direction, in this case extension.

EIL after mobilization

Three EIL trials were repeated after the mobilization trials.

EIL in left side glide (ELSG)

While in a prone position the subject's hips were translated maximally to the right (left side glide in McKenzie nomenclature) by the student investigator and in a translated position the subject performed an EIL again (Figure 5.2).

CASE STUDY

In order to understand these exercises further one subject underwent the series of exercises while electromyography and three-dimensional lumbar motion were measured. The purpose of this case study was to gain a preliminary understanding of the magnitudes of the differences in the L4/5 forces during these exercises. Future research with various subject pools (LBP, classified LBP, hypolordotic versus hyper-lordotic) will expand the information gained here.

L4-5 JOINT FORCES

The subject, a 35 year-old female (height of 172 cm and weight of 68.18kg) had no history of low back pain. The exercises performed were EIS, a Modified RSG, EIL without reinforcement (Pelvis not strapped), EIL and ELSG. EIS, EIL and ELSG were performed as previously described. The modified RSG exercise was performed in standing as a translation, as a posed to side bending while leaning against the wall as in figure 5.1(c). The subject translated her pelvis to the left. The EIL without reinforcement exercise is similar to the EIL exercise but without the addition of a restraining strap fixed around the subject's pelvis. The kinematics of the lumbar spine were measured with a 3-SPACE Isotrak system as described above. Seven channels of EMG were collected bilaterally by applying disposible Medi-Trace surface electromyogram (EMG) electrodes (Ag-AgCl) to the skin over the following muscles using Ag-AgCl electrodes from the following muscles; Rectus abdominis (right-RRA, left-LRA) placed 3 cm lateral to umbilicus, aligned straight up and down; External oblique (right-REO, left-LEO) placed 15cm lateral to umbilicus, orientated diagonally down and in (lateral electrode superior); Internal oblique (right-RIO, left-LIO) placed 1 cm inferior and medial to the ASIS orientated horizontally (or with the medial electrode slightly superior); Latissimus dorsi (right LD, left LD) placed lateral to T9 over the muscle belly; Thoracic extensors (erector spinae) - (right-RTE, left-LTE) -5cm lateral to T9, follow muscle bulk up and out; and Lumbar extensors (erector spinae) at L3 and L5 - (right-RL3E, left-LL3E, right-RL5E, left-LL5E)-3cm lateral to L3 and L5 respectively, follow muscle bulk up and out. The subject performed a number of maximum contraction tasks (MVCs) to allow normalization for each channel. Two abdominal MVC procedures were performed. The subject maintained a bent-knee sit-up posture while making maximal trunk flexion, right side flexion, left side flexion, right trunk rotation and left trunk rotation exertions against the resistance offered by a research assistant. The other abdominal exertion, also isometric, consisted of the research assistant resisting the subject's trunk flexion and rotation from below up while the subject was supported on a plinth in a supine position with her hips flexed to 90^0. Extensor muscle MVCs were collected with the subject's trunk cantilevered over the edge of a bench and a

research assistant resisting the subject's extensor effort. The MVC for latissimus dorsi was collected while the subject's arms were in 90^0 of abduction and external rotation and the research assistant resisted shoulder adduction. The EMG signals were amplified to produce approximately ±2.5V and then A/D converted with a 12 bit, 16 channel A/D convertor at 2048 Hz. The EMG signal was full-wave rectified, low-pass filtered using a second-order Butterworth filter (cut-off frequency of 2.5Hz) and then normalized to the maximum amplitudes during the MVC trials.

The reaction forces and moments acting at the L4-5 intervertebral joint (IVJ) were calculated through a linked segment model that uses external force measures, subject kinematics and anthropometrics (McGill and Norman 1985, McGill 1992 and Cholewicki and McGill 1996). External forces were not measured directly using a force plate in this study but estimated and considered in all trials. The proportion of the head, arms and trunk (HAT) weight transmitted through the hands in the EIL exercise was measured by placing the hands on a weighing scale while performing the EIL exercise. Based on this measure 20% of HAT weight was considered an external force to the L4/5 for the exercises performed in lying, the remaining 80% supported through the hands. Segment kinematics coordinates were calculated using a computer software program from side-view photographs, taken at start and finish positions of each exercise. The anthropometric data used in the model is that of the 50^{th} percentile male and was not changed for this subject. These reaction forces and moments are inputted into an anatomically detailed lumbar spine model that uses the 3-SPACE lumbar posture and position data to identify the segmental position of each lumbar vertebra and calculates the initial compression and shear forces at the L4/5 IVJ. The orientation of the vertebrae allows calculation of muscle and ligament lengths and their velocity of length change. The restorative moment created by the passive tissues is calculated knowing the orientation of the vertebrae and the stress-strain relationships of the passive tissues. The moment allocated to the muscle is determined and inputted into a 'distribution-moment model' which calculates muscle force and stiffness profiles for each muscle. The moments were balanced in the EIS trials and the average gain applied to balance these moments was then applied to all other trials. The normalized EMG signals as well as the calculated muscle length and velocity of contraction are used to calculate the active muscle force. The L4-5 forces equal the sum of the forces from the linked-segment model and the muscle and passive tissue forces.

Activities of Daily Living (ADL)

All subjects performed the following tests and repeated each test three times.

Toilet Flush From Standing

Subjects stood 30cm from a bench that was 65cm high (standard height of toilet handle). Subjects were instructed to press lightly with their right hand on a small object placed on the left hand side of the bench as though flushing a toilet and then to return to the upright position. Movement of their feet was not allowed.

Stand Up, Flush Toilet And Wash Hands Starting In A Sitting Position

Subjects sat with their back to the bench described above and were instructed to stand up, to turn to the right, press lightly with their right hand on a small object placed on the left hand side of the bench and then step forward and mimic washing their hands in a sink as though in a washroom.

Coffee Table Reach Left

Subjects stood 60cm from a bench that was 50cm high and were instructed to pick up a small object, which was on the left hand side of the bench, with their left hand. The subjects were allowed to move their feet if they wanted but did not have to do so. Once they had picked up the object they were to stand upright until the end of the trial.

Coffee Table Reach Right

Again subjects stood 60cm from a bench that was 50cm high and were instructed to pick up a small object, which was on the left hand side of the bench, using their right hand. The subjects were allowed to move their feet if they wanted but did not have to do so. Once they had picked up the object they returned to the starting position until the end of the trial.

Answer Phone At Desk

Subjects sat at a desk (70cm high) with their feet on the floor and arms resting on the desk. They were instructed to pick up a phone handset placed 24cm from the edge of the desk with their left hand and to talk on the phone for the remaining duration of the test.

Answer Phone At Desk And Lean Onto Right Armrest

Subjects were instructed to answer the phone as described in the previous task and then to lean onto the right armrest while talking on the phone.

Answer Phone At Desk, Lean Onto Right Armrest And Cross Left Leg Over Right

Subjects were instructed to answer the phone and lean onto the right armrest while talking on the phone as described in the previous task and to then cross their left leg over their right leg.

DATA ANALYSIS

Group effects (gender and LBP history) were investigated using a two-way analysis of variance, ($P< 0.05$). A repeated measures ANOVA was used to analyze the means of peak extension during the exercises and peak flexion during the ADL. For each of the McKenzie exercises subjects' peak extension, averaged across the three trials, was normalized to their maximum extension angle in the ROM trials. Total lumbar spine extension range was converted to segmental extension based on the in vivo results of Pearcy et al (1984) and Pearcy and Tibrewal (1984) (Table 5.1). The equivalent in vitro ranges are calculated from the in vivo ranges. As in vitro ranges are reported relative to elastic equilibrium, a 14.1^0 adjustment to the in vivo range was made (Andersson et al (1979) and in vitro segmental distributions calculated (Adams 1994). Similarly for each of the ADL, subjects' peak flexion, averaged across the three trials, was normalized to their maximum flexion angle in the ROM trials. Much of the research to date on the creation of disc prolapse has involved in vitro segmental testing, rather than whole spine in vivo testing. This in vivo data was converted to the equivalent lumbar spine in vitro ranges (Adams and Dolan 1991) in order to facilitate interpretation. The need for a conversion formula arises because the scale of flexion for a cadaveric specimen is different from the scale for a living person. 0% flexion is the elastic equilibrium position in the case of a cadaveric specimen while it is the standing position in the case of a living person. Likewise 100% flexion differs between cadaveric and living specimens, in the former case it is the elastic limit of the specimen while it is the extreme toe-touching position in the case of the latter.

Development of the conversion formula (Figure 5.3): Assuming that the in vivo and in vitro ranges are linearly related:

$$T = k_1 + k_2 \times V \qquad \text{(Eqn. 5.1)}$$

where T is the in vitro percent flexion, V is the in vivo percent flexion and k_1 and k_2 are constants.

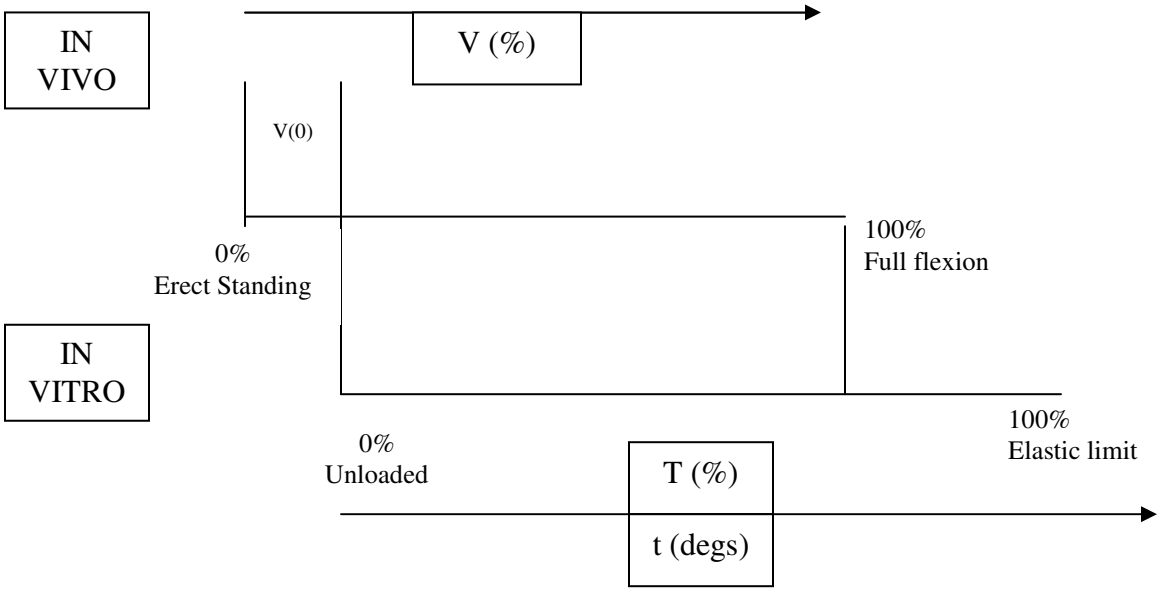

Figure 5.3: Schematic of the differences in the in vivo versus in vitro lumbar spine range of flexion in vivo (From Adams and Dolan 1991).

Let v equal the in vivo range of flexion in degrees, t equal the in vitro range of flexion in degrees and $v_{(0)}$ equal the in vivo flexion angle in degrees that corresponds to the unloaded spine in vitro. With reference to the figure above:

$$\text{when } T = 0,\ V = 100 \times v_{(0)} / v$$
$$\text{when } V = 100,\ T = 100 \times [v - v_{(0)}] / t$$

Substituting into equation (1) gives

$$T = -100 \times v_{(0)} / t + (v/t) \times V \qquad \text{(Eqn. 5.2)}$$

Based on the results of this study the mean in vivo range of flexion (v) equals 51.5^0 (mean age of participants = 44yrs): while suitable values of $v_{(0)}$ and t, taken from the literature, are 14.1^0 and 47.3^0 respectively (Adams 1986). The equation becomes

$$T = -29.8 + 1.09 \times V. \quad \text{(Eqn. 5.3)}$$

This conversion formula gives the in vitro equivalent of the in vivo spinal motion. The segmental motion of the L4/5 segment, the most frequent lumbar disc to prolapse, was calculated from the distribution of total in vitro range of spine motion according to Adams and Hutton (1986).

RESULTS

McKenzie Exercises

Group differences (gender/ low back pain history) in spinal motion were not found. The mean peak extension, normalized to full range of extension in standing, achieved in each McKenzie exercise is shown in figure 5.4 and the equivalent segmental in vitro ranges shown in table 5.1. Mean lumbar extension while resting in prone lying (54.36% SD 15.07%) and in relaxed standing posture (55.32%, SD 10.36%) were almost equivalent while the mean peak extension during the EIL exercise was only 3% (SD 22.33%) more than that of EIS. An extension mobilization in an EIL position increased lumbar extension by a further 6.75% (SD 11.18%). This 6.75% was equivalent to an increase of just under 5^0 of extension. Post mobilization the mean peak extension of EIL had increased by 3.71% (SD 9.93%) relative to that prior to the mobilization. The mean peak extension

of overhead reaching (72.10% SD 17.76%) was greater than that of relaxed standing but less than the mean peak extension of EIS. A large variation in the range of motion achieved by individual subjects exists. Significant correlations between the peak extension in a number of the positions and exercises were found (Table 5.2). However, subjects who had the greatest extension range in standing did not consistently achieve the greatest peak extension in each exercise. Variability was also found in the side bend ranges achieved. The mean peak right side bend in the RSG exercise, normalized to the full right side bend range, was 61% (SD 17.4%), while the mean peak side bend at peak extension in ELSG was a right side bend of 8.70% (SD 23.58%) of maximum right side bend (Figure 5.5). 70% of subjects were in a right side bend position (16.73% of full range, SD 21.43%) at peak extension of ELSG. The full range of right side bend correlated ($r = 0.635$) significantly ($p< 0.01$) with the right side bend range achieved in the RSG exercise.

Case Study Results

The sagittal plane kinematics and normalized EMG data during the EIS, EIL without reinforcement, EIL and ELSG exercises are shown in figures 5.6-5.9 along with the peak muscle compressive force and the combined axial compression at peak extension of the exercise. The L4/5 compression at the peak of the extension motion was less than the peak compression due to the muscle component during the exercises performed from a prone lying position. The frontal and sagittal plane kinematics along with the EMG and kinetic data in the modified RSG exercise are shown in figure 5.10. The normalized (to MVC trials) maximum level of muscle activation (%) during the trial and the muscle activation (%) at the peak range of motion during each of the McKenzie exercises is shown in table 5.3. Abdominal muscle activation dominated the EIS exercise while extensor muscle activation was more dominant than abdominal muscle activation during the EIL without reinforcement, EIL and ELSG exercises. The L4/5 axial compression and shear forces are shown in table 5.4. The compressive force at the peak extension range of EIL was just under 60% of that at the peak extension of EIS. The peak extension range of the EIL and EIS exercises was equivalent.

ADL Results

Individual variations in standing position and in the range of flexion from standing (full flexion) occurred, but significant group (gender/ low back pain history) differences in these variables were not found. The mean peak flexion angle achieved during each ADL, normalized to each subject's full flexion, ranged from 56.4-78.6% (Figure 5.11). Much of the research to date on the creation of disc prolapse has involved in vitro segmental testing, rather than whole spine in vivo testing. Converting

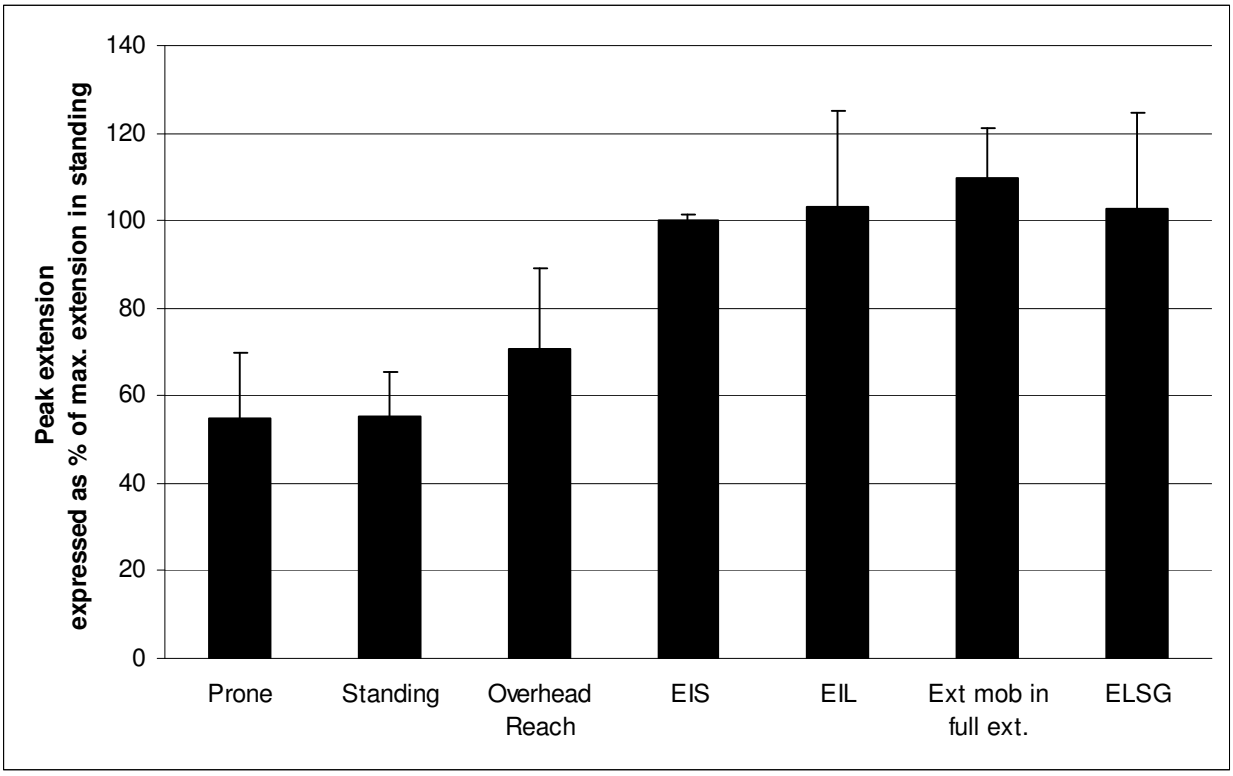

Figure 5.4: The mean peak extension (one standard deviation bar) achieved in each exercise is shown. Each subject's peak extension was normalized to their maximum extension in standing. Lumbar extension in prone lying and in standing was similar. Peak extension during the EIL exercise produced 3% more extension than that of EIS while an extension mobilization in an EIL position increased lumbar extension by a further 6.75%.

	Extension in standing	Overhead reach	Extension in prone lying	Extension in prone with extension mobilization	Extension in prone after extension mobilization	Extension in prone in a left side glide
In Vivo Extension ROM (degs.) Relative to Standing						
Degrees of Extension of Lumbar Spine	20.89	11.22	22.43	27.72	25.42	23.87
L4/5	2.61	1.40	2.80	3.47	3.18	2.98
L5/S1	6.53	3.51	7.01	8.66	7.94	7.46
In Vitro Extension ROM (degs.) relative to Elastic Equilibrium						
Degrees of Extension of Lumbar Spine (In vitro)	34.99	25.32	36.53	41.82	39.52	37.97
L4/5	5.25	3.80	5.48	6.27	5.93	5.69
L5/S1	7.58	5.49	7.91	9.06	8.56	8.23

Table 5.1: The lumbar spine extension motion, relative to the relaxed standing posture, and the segmental distribution at L4/5 and L5/S1 of peak extension during each exercise are shown. Total lumbar spine extension range was converted to segmental extension based on the in vivo results of Pearcy et al (1984) and Pearcy and Tibrewal (1984). The equivalent in vitro ranges are calculated from the in vivo ranges. As in vitro ranges are reported relative to elastic equilibrium, a 14.1^0 adjustment to the in vivo range was made (Andersson et al (1979) and in vitro segmental distributions calculated (Adams 1994).

		Prone	Standing	Overhead Reach	EIS	EIL	ELSG	EIL with mob.
Prone	Pearson's Correlation	1	.651**	.520*	.469*	.594**	.462*	.397
	Sig. (2-tailed)	-	.001	.018	0.16	.005	.035	.075
Standing	Pearson Correlation	.651**	1	.794**	.596**	.580**	.496*	.472*
	Sig. (2-tailed)	.001	-	.000	.004	.006	.022	.031
Overhead Reach	Pearson Correlation	.520*	.794**	1	.356	.448*	.381	.321
	Sig. (2-tailed)	.016	.000	-	.113	.041	.089	.156
EIS	Pearson Correlation	.469*	.596**	.356	1	.695**	.621**	.643**
	Sig. (2-tailed)	.032	.004	.113	-	.000	.003	.002
EIL	Pearson Correlation	.594**	.580**	.448	.695**	1	.893**	.902**
	Sig. (2-tailed)	.005	.006	.041	.000	-	.000	.000
ELSG	Pearson Correlation	.462*	.496*	.381	.621**	.893**	1	.895**
	Sig. (2-tailed)	.035	.022	.089	.003	.000	-	.000
EIL with mob.	Pearson Correlation	.397	.472*	.321	.643**	.902**	.895**	1
	Sig. (2-tailed)	.075	.031	.156	.002	.000	.000	-

**Correlation is significant at the 0.05 level (2-tailed)

* Correlation is significant at the 0.01 level (2-tailed)

Table 5.2: The correlation levels of subjects peak extension in each exercise are shown.

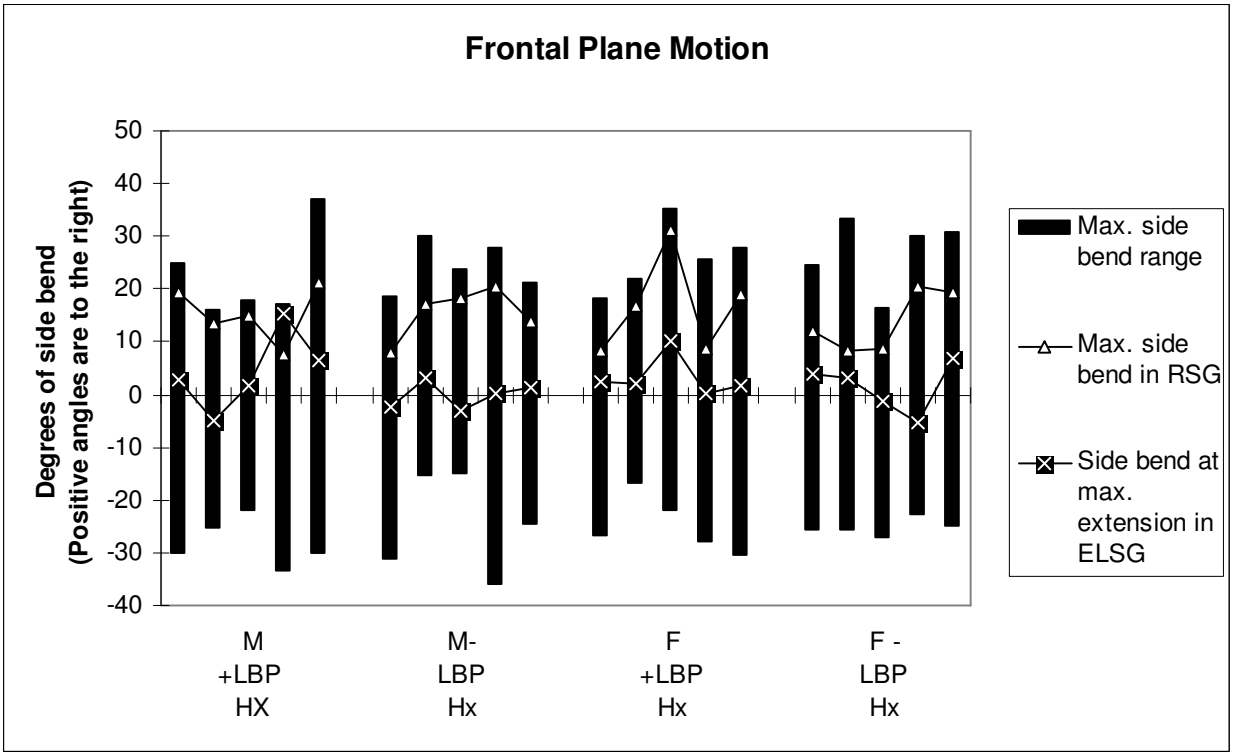

Figure 5.5: The limits of the black bars show each subject's full range of right side flexion (positive angle) and full range of left side flexion (negative angle) ranges achieved in standing. Superimposed are the peak ranges of side flexion achieved during the right side glide (RSG) exercise and at peak extension in the extension left side glide (ELSG) exercise. A mean of 61% (SD 17.4%) of the full right side flexion range was achieved in RSG. Seventy percent of the subjects were in right side bend (16.73% of Max. range) at the peak of extension in the ELSG exercise.

the in vivo spinal ranges to the equivalent in vitro segmental ranges shows the mean peak L4/5 flexion ranged from 3.95^0 – 6.71^0 in these ADL. In addition to pure flexion, flexion combined with side bend and/or rotation motion is another known mechanism to create disc prolapse. Table 5.5 shows the L4/5 side bend and rotation at peak flexion in each ADL. The kinematics of flexion, side-bend and rotation angles are also similar to those previously used to create disc herniation when applied with high repetitions and moderate compression.

DISCUSSION

To the authors knowledge this is the first time the lumbar spine motion during a number of commonly prescribed McKenzie exercises has been quantified. Peak extension in EIL was greater than that of EIS, as hypothesized, but only by a mean of 3% (SD 22.33%) of the maximum extension range. Significant statistical differences in the peak motion during these exercises were not found except between the peak extension in EIL and EIL with mobilization of L3 (6.75% difference (SD 11.18%)). As expected extension mobilizations in EIL resulted in greater peak extension than that of EIL alone and subsequently

EIL had increased peak extension. Over 50% of the full right side bend range was achieved in the RSG exercise. The mean peak side bend at peak extension in ELSG was a right side bend of 8.70% (SD 23.58%) of maximum right side bend. 70% of subjects were in a right side bend position (16.73% of full range, SD 21.43%) at peak extension of ELSG. EIL from a position of left side flexion (ELSG) did not result in less peak extension than that of EIL. The large variability in the peak ranges achieved during these exercises could not be accounted for by gender or low back pain grouping. The L4-5 compressive forces at the peak extension of EIL were less than 60% of that in EIS. It appears that substantial lumbar flexion occurs during what many would consider to be benign ADL. Given the magnitudes of motion documented in this work potentially injurious kinematics were identified in these simple ADL. This would imply that educating clinicians and individuals regarding the need to eliminate/modify these causative/aggravating factors is important, until further research provides a greater understanding of the repetitions of these motions required to cause or exacerbate disc prolapse is completed.

A limitation of this study is that the subjects did not necessarily have a history of disc prolapse and that the subjects were not in the acute phase of disc prolapse. Clinically, there is no doubt that individuals with an acute posterior disc prolapse cannot achieve these ranges of extension. The purpose of this study was to gain insight into the relative ranges of extension during these exercises when subjects were in a pain-free state. The impact that a painful condition, especially disc prolapse,

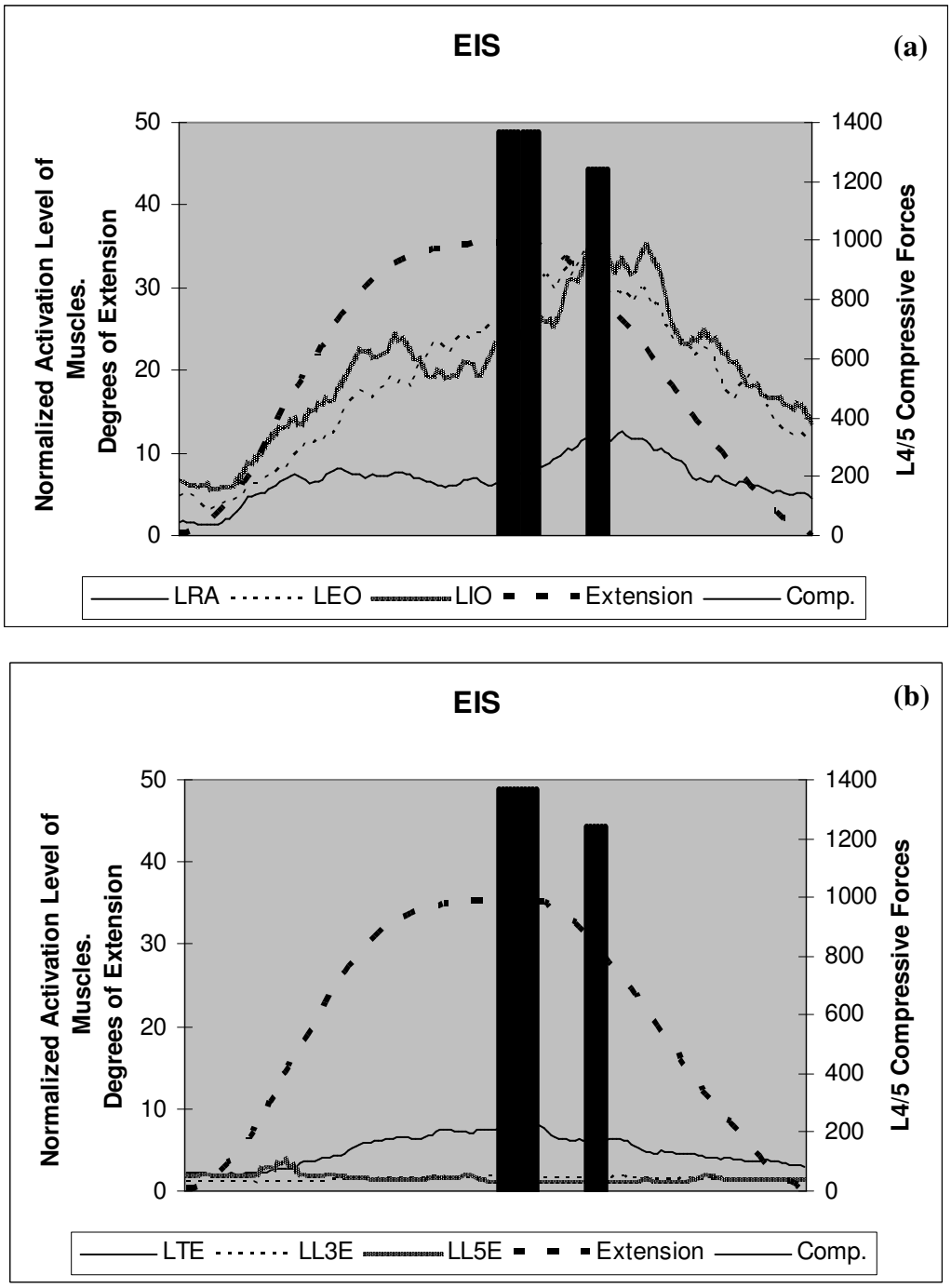

Figure 5.6: showing muscle activation of the left (a) abdominal and (b) extensor muscles during the EIS exercise. Muscle activation, expressed as a percentage of the activation level during MVC trials, and sagittal plane displacement (dashed black line), expressed in degrees relative to the standing position, are both shown on the primary x-axis. The peak axial compression (N) (column) is shown on the secondary x-axis. The wide column represents the sum of the compression due to the muscle activation and the reaction forces at peak extension. Peak muscle compression is shown as the narrow column.

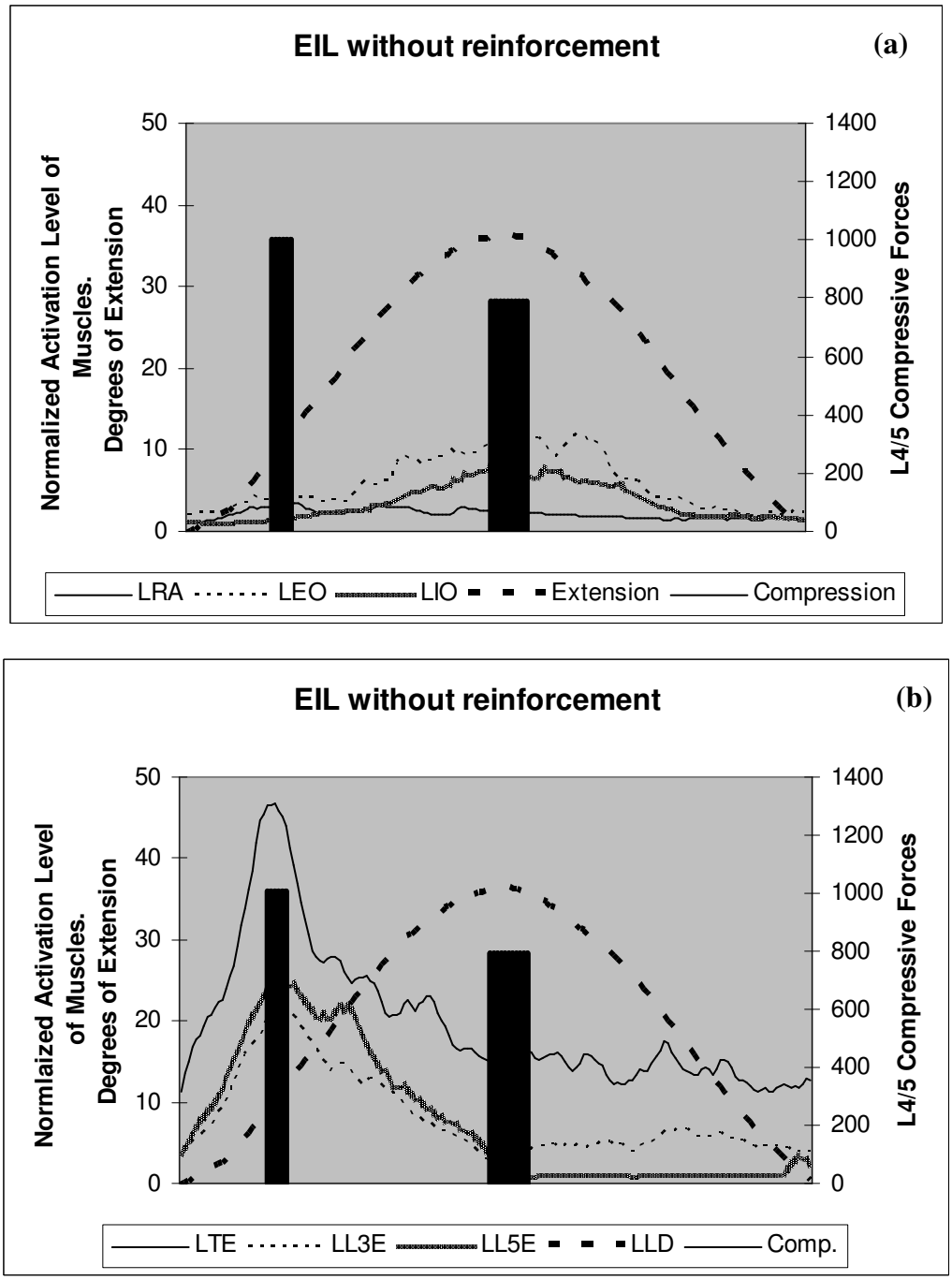

Figure 5.7: showing muscle activation of the left (a) abdominal and (b) extensor muscles during the EIL without reinforcement exercise. Muscle activation, expressed as a percentage of the activation level during MVC trials, and sagittal plane displacement (dashed black line), expressed in degrees relative to the standing position, are both shown on the primary x-axis. The peak axial compression (N) (column) is shown on the secondary x-axis. The wide column represents the sum of the compression due to the muscle activation and the reaction forces at peak extension. Peak muscle compression is shown as the narrow column.

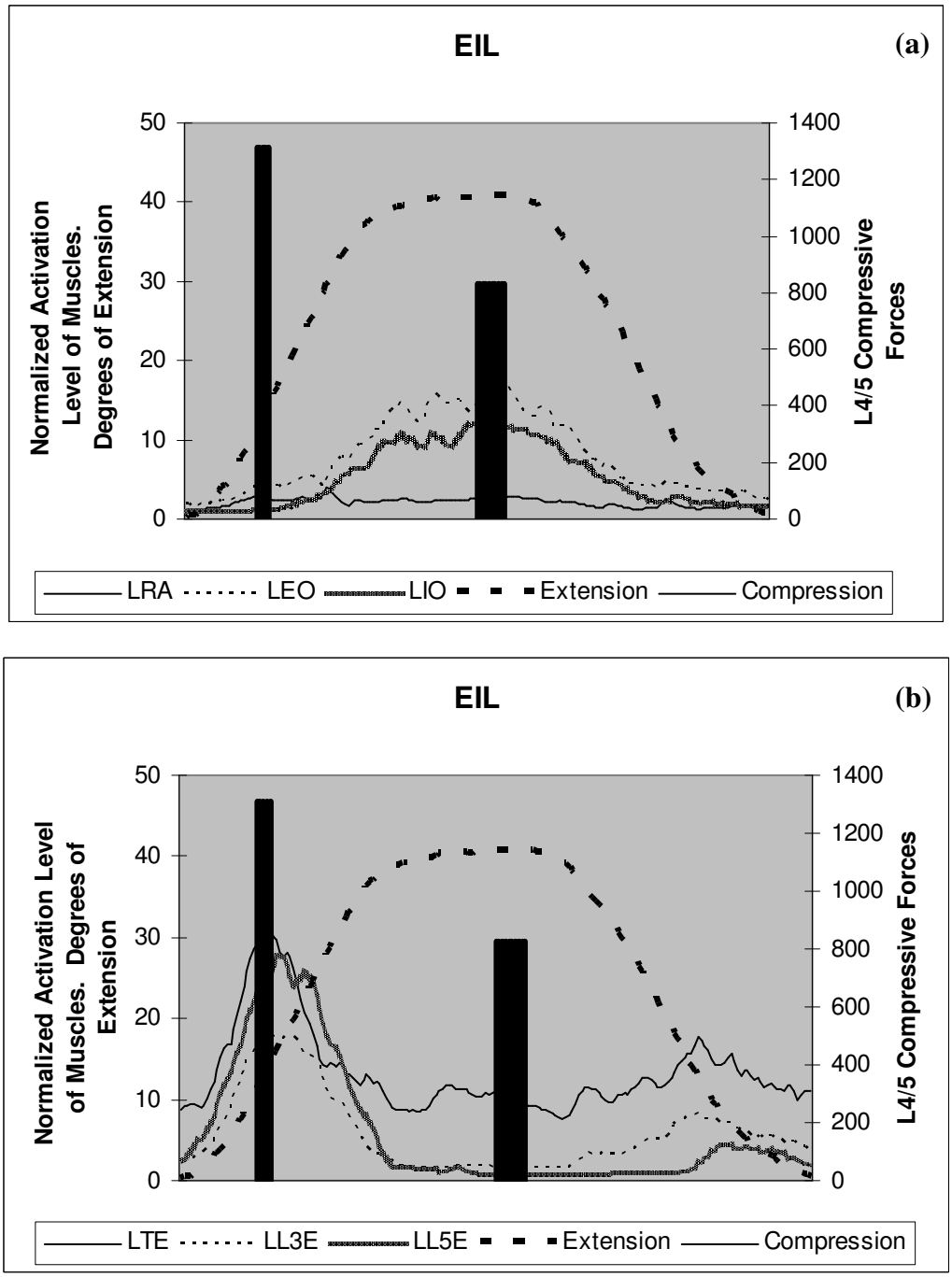

Figure 5.8: showing muscle activation of the left (a) abdominal and (b) extensor muscles during the EIL exercise. Muscle activation, expressed as a percentage of the activation level during MVC trials, and sagittal plane displacement (dashed black line), expressed in degrees relative to the standing position, are both shown on the primary x-axis. The peak axial compression (N) (column) is shown on the secondary x-axis. The wide column represents the sum of the compression due to the muscle activation and the reaction forces at peak extension. Peak muscle compression is shown as the narrow column.

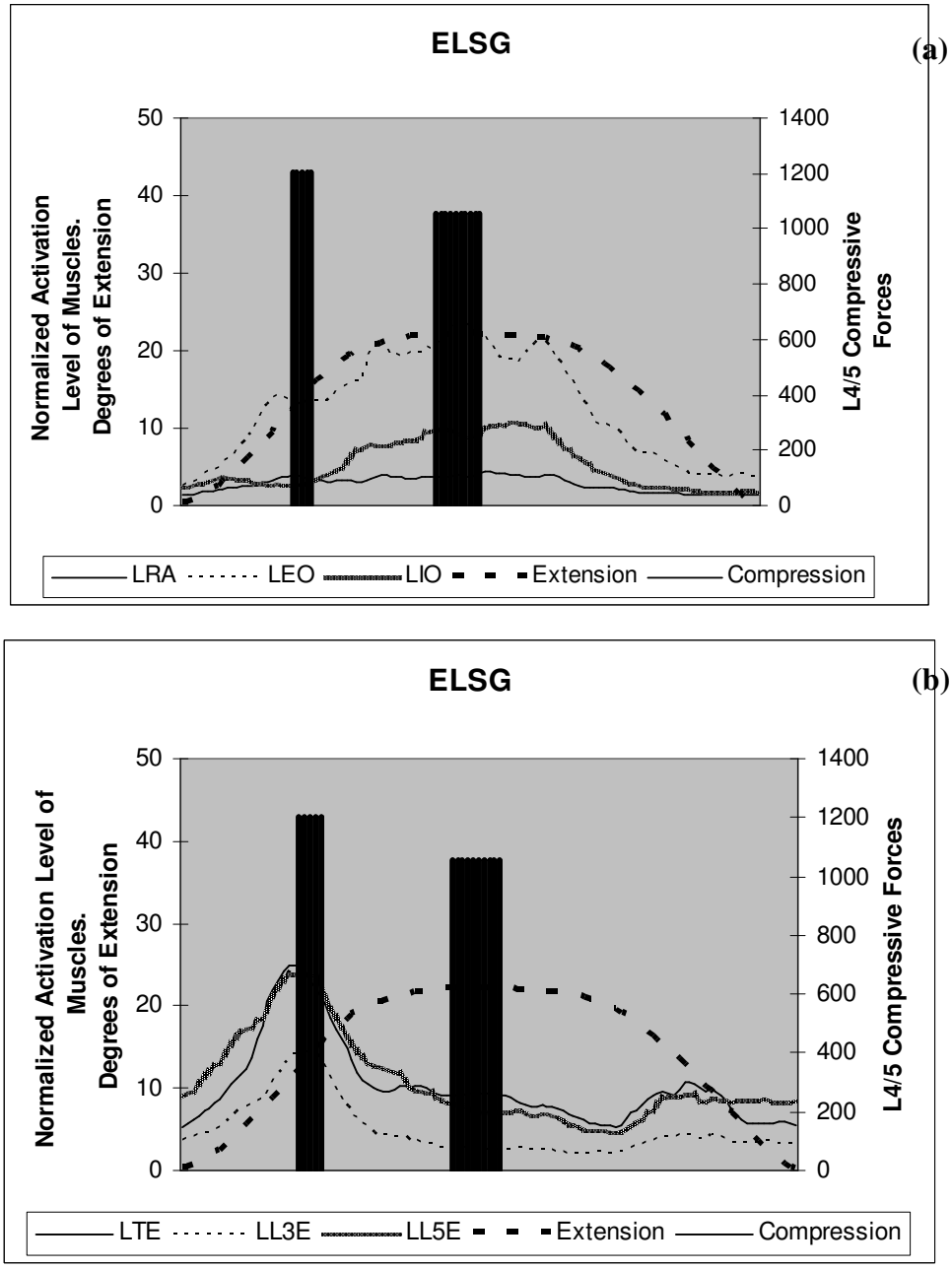

Figure 5.9: showing muscle activation of the left (a) abdominal and (b) extensor muscles during the ELSG exercise. Muscle activation, expressed as a percentage of the activation level during MVC trials, and sagittal plane displacement (dashed black line), expressed in degrees relative to the standing position, are both shown on the primary x-axis. The peak axial compression (N) (column) is shown on the secondary x-axis. The wide column represents the sum of the compression due to the muscle activation and the reaction forces at peak extension. Peak muscle compression is shown as the narrow column.

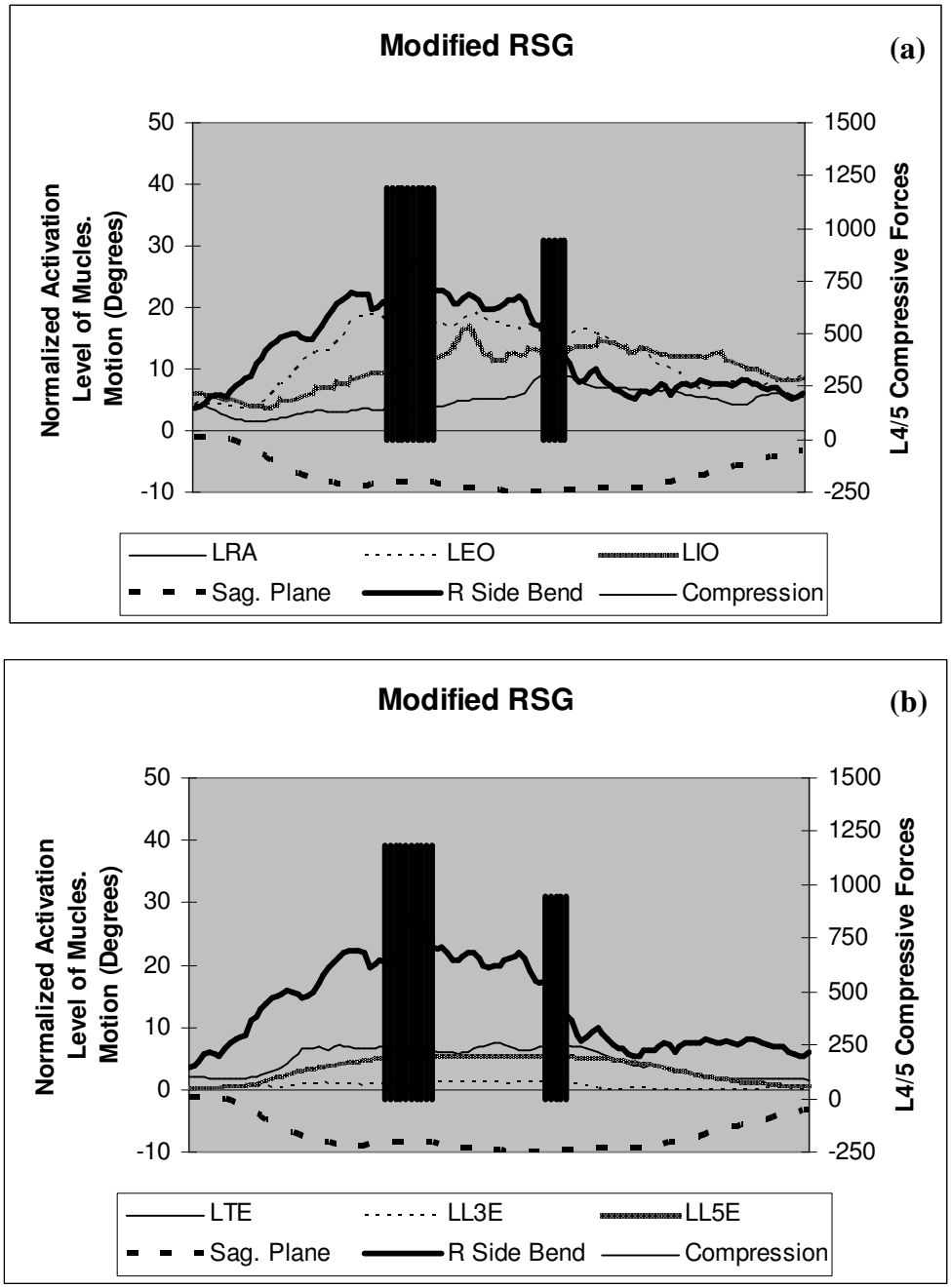

Figure 5.10: showing muscle activation of the left (a) abdominal and (b) extensor muscles during the modified RSG exercise. Muscle activation, expressed as a percentage of the activation level during MVC trials, flexion (dashed black line) and right side bend displacement (solid bold black line) expressed in degrees relative to the standing position, are both shown on the primary x-axis. The peak axial compression (N) (column) is shown on the secondary x-axis. The wide column represents the sum of the compression due to the muscle activation and the reaction forces at peak right side bend. Peak muscle compression is shown as the narrow column.

Muscles	EIS		EIL Without Reinforcement		EIL		ELSG		Modified RSG	
	Max.	At Peak Extension	Max.	At Peak Extension	Max.	At Peak Extension	Max.	At Peak Extension	Max.	At Peak RSB
	% Of Maximum Activation In MVC Trials									
RRA	27.59	20.94	4.77	2.63	5.28	5.14	5.66	4.24	6.84	5.39
LRA	12.58	7.38	3.53	2.35	3.94	2.58	4.45	3.87	9.03	3.92
REO	20.62	17.83	7.84	6.59	12.91	11.59	12.49	10.76	6.07	5.60
LEO	34.60	25.57	12.20	11.45	17.77	16.63	23.65	22.43	19.32	16.90
RIO	15.90	12.15	6.74	6.12	8.84	8.32	15.41	11.43	9.79	9.29
LIO	39.00	23.83	8.15	7.43	12.37	11.56	10.66	9.29	16.79	11.12
RLD	4.99	4.26	9.30	7.81	6.91	4.99	22.94	19.71	2.56	2.22
LLD	9.85	8.08	16.01	12.47	11.90	11.30	13.46	9.65	15.93	14.08
RTE	10.97	10.38	30.99	16.53	23.76	10.68	37.68	24.04	5.42	4.93
LTE	8.17	7.95	46.71	14.85	30.57	10.11	24.94	9.21	7.597	6.48
RL3E	9.94	2.05	23.49	3.06	18.58	1.89	16.86	3.54	3.26	2.98
LL3E	1.79	1.73	22.14	3.18	18.05	1.67	14.43	2.65	1.25	1.05
RL5E	7.45	2.87	28.97	6.97	29.86	2.65	21.40	4.81	12.60	11.55
LL5E	3.54	1.15	25.23	0.88	27.85	0.68	23.92	7.38	5.48	5.34

RRA - Right Rectus Abdominis, LRA - Left Rectus Abdominis, REO - Right External Oblique, LEO - Left External Oblique, RIO - Right Internal Oblique, LIO - Left Internal Oblique, RLD - Right Latissimus Dorsi, LLD – Left Latissimus Dorsi, RTE – Right Thoracic Extensors, LTE – Left Thoracic Extensors, RL3E – Right Lumbar Extensor at L3, LL3E – Left Lumbar Extensor at L3, RL5E – Right Lumbar Extensor at L5, LL5E – Left Lumbar Extensor at L5

Table 5.3 : The normalized (to MVC trials) maximum level of muscle activation (%) during the trial and the muscle activation (%) at the peak range of motion during each of the McKenzie exercises is shown. Abdominal muscle activation dominated the EIS exercise while extensor muscle activation was more dominant than abdominal muscle activation during the EIL without reinforcement, EIL and ELSG exercises.

may have on these ranges will be the topic of future work. Furthermore the L4-5 shear and compression forces were calculated on only one subject. This serves the purpose of taking a first look at the relative L4-5 forces during these exercises and provides an increased understanding of the mechanics underlying the preferential order of McKenzie treatment of derangement. A more thorough understanding will be gained by future investigation of these forces in subjects with classified derangements and LBP as well as at various stages of rehabilitation.

This study has provided clinicians with a greater understanding of the relationship between the kinematics and kinetics of these exercises. The results have shown that the mean peak extension in EIL is marginally greater than EIS, although considerable variability exists. However, the L4-5 compression at the peak EIL position was less than 60% of that at the peak EIS position. Lower compression in EIL may explain the preference for prescribing this McKenzie exercise during the acute phase of disc prolapse. EIL was not performed as a passive exercise by this subject, which is in keeping with the results of Fiebert & Keller (1994). Fiebert & Keller (1994) reported that the erector spinae muscles are even more active in EIL than during standing, EIS or prone lying suggesting that EIL is not a passive exercise. These findings are echoed in the current study. The compression loading of the spine during EIL may not be as low as assumed clinically, although still lower than that of EIS in this subject and much lower than that of numerous other commonly prescribed exercises (Kavcic et al 2004, Callaghan et al 1998) and much lower than the NIOSH recommended limit of 3400N (NIOSH 1981). The peak compression levels in standing of this subject are lower than those previously reported to occur by Callaghan and McGill (1999), understandable given the smaller female subject in this study relative to the larger male subjects in Callaghan and McGill (1999). The effect of the McKenzie approach, not just EIL, on pain ratings, return to work and as a predictor of outcome (Brotz et al 2003, Donelson et al 1990, Long 1995, Clare 2004, Aina et al 2004) has been extensively documented in the literature. The literature also contains reports of the benefits of extension relative to flexed postures: lower intradiscal pressure, lower disc shear and ligament forces than in flexed postures (Adams and Hutton 1985, Adams et al 1999, Burton et al 1994, Hedman et al 1997, Nachemson 1960, Williams et al 1991) and increased disc height (Magnusson et al 1996). However, extension comes with a price and has been identified as being injurious to the facet joints (Shirazi-Adl 1991, 1994, Dunlop et al 1984). It is important to note that the L4-5 compression and shear forces were calculated at the peak extension positions of these exercises. Only the compressive and shear forces due to the muscle contraction are known in the range between the start and peak extension position.

Sitting, leaning onto an armrest while crossing one leg over the other resulted in the most lumbar flexion in comparison to the other ADL investigated. In comparison to previous in vitro research the

segmental flexion angles, but not the number of repetitions, of these ADL are similar to those used to create a prolapse. In the study of Callaghan and McGill (2001) the flexion angles tested were approximately 50% of the elastic limit of a specimen. Flexion angles of 31.69-55.88% of the elastic limit were reached in the ADL investigated in this study. An even more potent in vitro mechanism to create disc prolapse is flexion combined with side bend and/or rotation, motion that occurred in all of these ADL (Aultman et al 2005, Andersson 1981, Kelsey et al 1984, Shirazi-Adl 1989). Gordon et al (1991) produced disc ruptures by combining rotation ($1\text{-}3^0$), flexion (7^0) and compression (1334 N) within physiological ranges. Likewise Lu et al in their 1996 finite element model study found flexion (7^0), rotation (2^0) and compression of a saturated disc to result in initiation and propagation of an annular fissure. The results of these studies suggest that individuals are more at risk of disc herniations when their total spine or segmental flexion is combined with compression and rotation. Likewise, Shirazi-Adl (1989) found increased strain in annular fibers when both rotation and side bend were combined with a given level of flexion and compression. Using a nonlinear finite element model, tensile strains of about ten percent in the disc fibers were predicted under maximum loads simulating symmetrical lifting. These strains were increased to twenty percent when the lift became asymmetrical. Equivalent levels of annular strain were predicted by different combinations of side bend and rotation. Shirazi-Adl (1989) found identical maximum fiber strain by reducing flexion, compression and shear loading while increasing side bend and rotation angles. For example, the predicted annular strain under pure sagittal flexion of 12^0 under 4000N compression resulted in approximately 10% annular strain, the equivalent strain was also predicted by combining only 7.5^0 of flexion with 1.2^0 rotation and 3.7^0 lateralbend under 2000N compression. Similar combinations of side bend and rotation occurred in this study. Modifying ADL to eliminate flexion and flexion combined with side bend and rotation will reduce the risk of developing disc prolapse. Changing lumbar posture from flexion to extension will also change the coupled motion of the specimens eliminating problematic kinematics (Cholewicki et al 1996) The implication of the results of this study is not that every individual should attempt, or needs to, totally avoid flexion or combined flexion and side-flexion rather, the results apply to individuals who have a history of disc prolapse. This advice will extend to those who have a predisposition to the development of disc prolapse, as determined by future research.

The lumbar spine ranges achieved in commonly prescribed McKenzie rehabilitative and preventative exercises have been quantified. The results of this study enhance clinical practice by providing quantitative evidence of the order of peak extension of the McKenzie exercises. The clinical reasoning practice model encourages clinicians to make direct links between the specific needs of each client and the specific treatment chosen for that client. To date, the selection of these

	A/P Shear (N)	Compression (N)	M/L Shear (N)
Standing (start of EIS)	7.56	831.52	-3.15
At Peak extension in EIS	-86.69	1368.86	-19.39
Start of EIL without reinforcement trial	-2.06	425.90	-4.59
Peak EIL without reinforcement	75.35	792.22	3.51
Start of EIL trial	20.88	322.55	10.40
Peak EIL belt	88.29	828.97	15.83
Start ELSG trial	20.22	402.25	10.71
Peak ELSG	88.26	1052.03	16.18
Start of Modified RSG trial	10.04	845.61	-2.79
At Peak side bend in Modified RSG	36.29	1187.06	26.88

Table 5.4: The L4-5 shear and compression forces at the start and peak motion of each exercise. Negative A/P shear is a net posterior shear of the trunk with respect to the pelvis. Negative M/L shear is a shear of the trunk to the left relative to the pelvis.

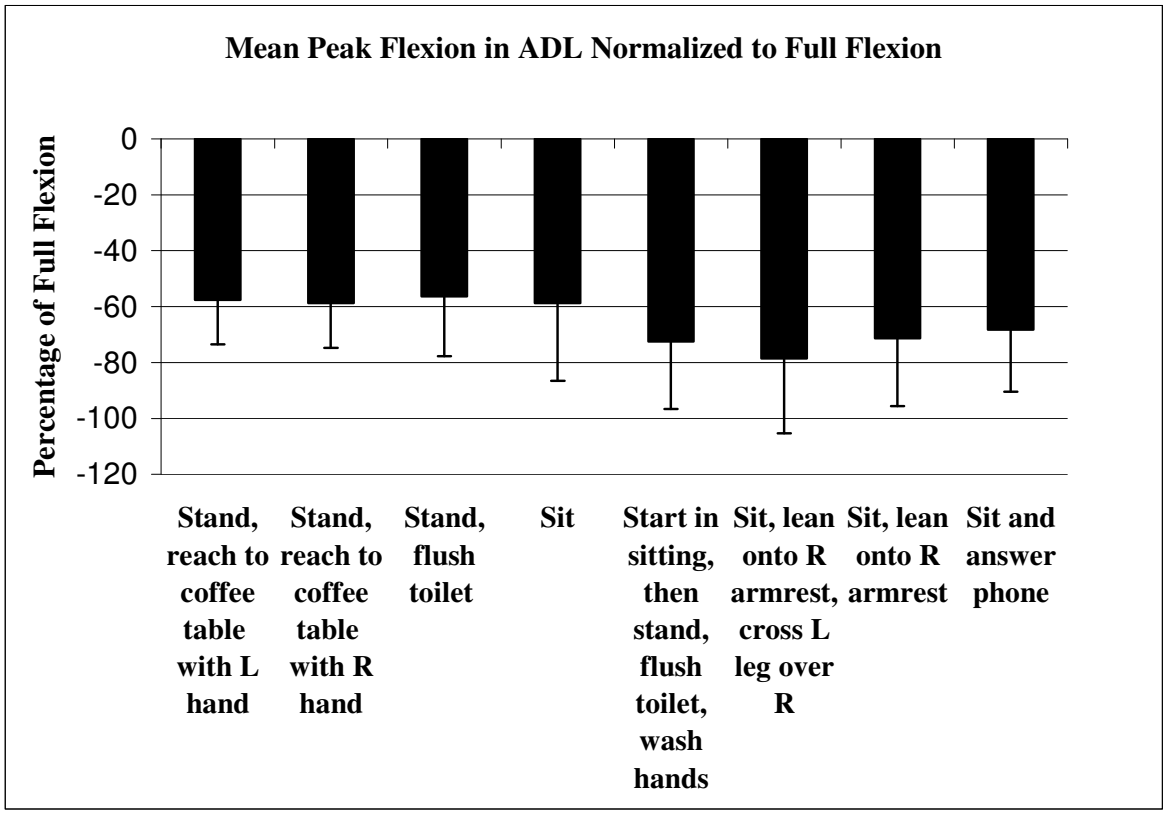

Figure 5.11: The mean (+ one SD bar) of the normalized peak flexion in each ADL is shown. Subjects' peak flexion angles were normalized to their full flexion range in standing. As flexion angles are negative throughout this paper the percentages are also shown to be negative.

	Stand, reach to coffee table with L hand	Stand, reach to coffee table with R hand	Stand, flush toilet	Sit, flush toilet, wash hands	Sit, lean onto R armrest, cross L leg over R	Sit, lean onto R armrest	Sit and answer phone
Peak flexion (relative to standing (deg)							
Mean at L4/5	7.26	7.34	7.06	9.16	9.82	9.00	8.62
SD	1.82	2.19	2.21	3.38	3.23	3.27	2.98
Side bend at peak flexion (deg)							
Mean at L4/5	0.98	0.84	0.79	0.68	1.15	1.03	0.69
SD	0.86	0.54	0.63	0.80	0.81	0.75	0.53
Rotation at peak flexion (deg)							
Mean at L4/5	0.61	0.68	0.62	0.52	1.03	0.93	0.72
SD	0.47	0.62	0.56	0.51	0.73	0.68	0.57

Table 5.5: The L4/5 distribution (White and Panjabi (1978) of the mean side bend and rotation that occurred at peak flexion for each ADL is shown. Gordon et al (1991) produced disc herniation by combining 1-3^0 of rotation, 7^0 of flexion and 1334 N of compression at high repetitions.

exercises was totally based on clinical experience. Also of value to clinical practice is the finding that left side flexion was not consistently maintained throughout the ELSG movement. ELSG, a treatment technique that combines left side flexion and active extension, is usually used to reverse a left posterolateral disc prolapse. Seventy percent of subjects were in a right side bend at peak extension highlighting the need to monitor clients closely when prescribing exercises. Acute observation requires the development of clinical skill and experience, which remain an important component of research-enhanced practice. Extension mobilizations in a position of EIL increased the peak extension while the spine was being mobilized. The peak range of extension in EIL was marginally greater after the mobilizations indicating a carryover effect. The long-term effect of mobilizations on the peak range of extension in EIL was not investigated.

CONCLUSION

For the first time the lumbar spine ranges achieved in commonly prescribed McKenzie exercises have been quantified. The results of this study enhance clinical practice by providing quantitative evidence of the relative peak motion of the McKenzie exercises. The compressive and shear forces identified in the case study increase our understanding of the potential kinematic and kinetic differences in these exercises. The peak flexion ranges of the ADL investigated match those previously used to create disc prolapse, although the prolapses were created under extremely high repetitions of these ranges. This would imply that educating clinicians and individuals regarding the need to eliminate/modify these causative/aggravating factors is important, until further research provides a greater understanding of the repetitions of these motions required to cause or exacerbate disc prolapse is completed.

CHAPTER 6

THESIS DISCUSSION AND CONCLUSION

The macroscopic goals of this thesis were to attain a greater understanding of disc failure and the mechanics of its clinical treatment responses. The purpose of these goals was to provide a foundation for evidence-based practice, a goal that was successfully achieved. This thesis ultimately challenged and increased our understanding of pathological discs while simultaneously adding information to assist clinical decision making. Several new contributions to the existing knowledge of lumbar spine biomechanics and clinical concepts of treating disc prolapse have been made and will be discussed on a study-by-study basis followed by discussion on a topic-by-topic basis.

Creating Disc 'Herniation' In Vitro: Is There In Vivo - In Vitro Disc Height Equivalency?

This in vitro study challenges the camouflage offered by the multidisciplinary use of similar terminology. Appling Wilke et al's (2006) classification of disc height loss to in vitro herniated discs links the damage created in vitro to that existing in vivo. The classification system of Wilke et al (2006) has been shown to be in good agreement with the macroscopic degree of in vivo degeneration as determined from MRI. The first study of this series broadens our understanding of the disc herniations created in vitro and enhances our knowledge of the mechanics of post-herniated in vitro porcine discs while moving closer to unification of research and clinical perspectives of 'herniated' discs. The methodology of repeatedly flexing while axially compressing porcine cervical specimens previously used to create herniation was associated with simultaneous moderate disc height loss of the specimens. This level of disc height loss has previously been shown to be associated with less movement-related changes in intra-discal pressure and an altered load distribution across the spinal segment. The study provides a multidisciplinary perspective on the limitations of the model and promoted efforts to modify the methodology and reduce the disc height loss when the mechanical changes associated with such disc height loss could potentially impact the results of specific research.

H: A modified version of the in vitro methodology of Callaghan and McGill (2001) would produce disc herniations in porcine cervical spines without concurrently causing moderate to severe disc height loss (>33% of pre-test disc height).

Result: The methodology successfully herniated discs, however, almost 60% of disc height loss occurred simultaneously.

Attempts To Create Disc Herniations; Successes And Failures

Continuing from the previous study which used disc height loss to sub-classify post-herniated in vitro discs, the second in vitro study in this thesis pursues alternate methods of creating herniation with the goal of creating herniation prior to causing more than thirty three percent of pre-test disc height loss of the specimens. The successes and failures of this study provide insight into the mechanics associated with various in vitro methodologies and the mechanical implications of specimen and methodological modification. Posterior herniations were created by combining lower axial compression with higher repetitions of flexion, however, the concurrent disc height loss was more than 50% of the pre-test disc height. While the disc height loss that occurred with dynamic testing could be reversed by placing the specimen in a saline submersion bath or re-injecting the specimen with barium sulphate-nucleus mix the disc height could not be maintained within the in vivo diurnal range under continued loading. The relatively hypolordotic thoracic spines tested did not herniate but lost more than 50% of their pre-test disc height during the dynamic testing. In conclusion, in vitro disc herniations were created but concurrent disc height loss of more than 33% of pre-test disc height occurred. This study indicated that the in vitro model used in the first in vitro study was the best-available. Knowing the disc height loss associated with this methodology provided a framework for interpretation of the results of the subsequent and third in vitro study in this thesis.

H: The hypotheses of this study were that (1) modifying the loading pattern that had previously been used to cause herniation, (2) rehydrating the discs during testing and/or (3) using a different model, would create disc herniation prior to causing more than 33% of disc height loss.

Result: These attempts either failed to produce herniations or did so with more than 33% of disc height loss.

McKenzie Treatment Of Disc Prolapse –Evidence Of A Mechanical Foundation?

The focus of the third study was the mechanical investigation of the McKenzie clinical theory of the treatment response seen in vivo in prolapsed discs. This study is a proof of the principle on which this aspect of the McKenzie approach is based and provides, to the author's knowledge, the first scientific evidence supporting the theory that repeating movements opposite to those that caused posterior displacement of a portion of the nucleus can positively change the position of the displaced portion of nucleus. The results indicate that the McKenzie approach works on some prolapsed discs and not on others. Consideration of the changes in disc height of the specimens during the testing procedures (Table 6.1) offers some understanding of the varied success of this approach and exposes

a vast area of future research that will refine the clinical approach and mechanical understanding of this specific disc pathology.

H: Repeated motion, opposite to the motion that caused the disc prolapse, will reverse the disc prolapse.

Result: Based on a radiologists review reversal testing involving repeated movements opposite to those that created the disc prolapse reversed the displacement to a clinically important level in 5 of the 11 specimens but did not have an effect on the other 6 prolapsed discs.

H: Discs that do not respond to reversal testing will have extensive circumferential annular tears or will have a herniation with extrusion of nuclear material.

Result: A number of the discs that did not respond to reversal testing had clear circumferential prolapses but the pattern was not consistently a distinguishing factor between the discs that responded and those that did not respond to reversal testing.

H: The specimens that prolapse will have been dissected from hypolordotic spines.

Result: The Lordosis angles of the specimens did not distinguish between the groups.

McKenzie Extension Exercises – Quantified And Considered

The study described in chapter 5 provides a first look at the current in vivo application of this approach. The lumbar spine ranges achieved in commonly prescribed McKenzie rehabilitative and preventative exercises and those that occur in seemingly benign ADL were quantified. This study has provided clinicians with a greater understanding of the relationship between the kinematics and kinetics of these exercises. The results of this study enhance clinical practice by providing quantitative evidence of the order of peak motion of the McKenzie exercises as well as highlighting ADL that involve levels of flexion, side bend and rotation sufficient to cause disc damage and even prolapse.

H: The extension of EIL would be greater than that of EIS

Result: Mean peak extension of extension in lying (EIL) exercises was on average 2^0 greater than that of EIL.

H: The range of extension would be increased during and after extension mobilization

Result: An additional 5^0 of extension occurred when EIL was combined with a Grade 3 Maitland extension mobilization to L3. The EIL peak extension was increased after the mobilization relative to the pre-mobilization range.

H: The peak flexion, ± side bend and rotation, ranges of the ADL investigated match those previously used to create disc prolapse.

Result: This hypothesis was accepted. However it is recognized that previously these kinematics have produced herniation when applied repeatedly under moderate levels of axial compression.

Disc Height Loss

Disc height loss was an issue that was relevant to all three in vitro studies in this thesis. Based on the existing literature disc height was identified as a factor that could categorize the coexisting disc damage created by in vitro testing in a manner that was clinically and mechanically relevant. The disc height loss in all three studies is compared in table 6.1 and is similar across the three studies. The standard deviation of the specimens in study three was larger than that in studies 1 and 2. However, the failure testing in study 1 and for most of the study 2 tests was for a fixed time while the number of repetitions of failure testing in study three was more variable as the test was stopped when prolapse was identified. In order to attain a greater understanding of the disc height loss associated with this methodology one specimen was axially compressed for 120 minutes under 1472N. Pre- and post- compression lateral x-ray images were taken and the axial creep of the specimen was recorded every 30 minutes. Figure 6.1 shows the progressive disc height loss of the specimen under an axial compression load. The specimen was preloaded with 260N for 15 minutes prior to being loaded with 1472N of compression. The interval of in which the greatest amount of axial creep occurred was the first 30 minutes of loading the specimen with 1472N of axial compression. However, the specimen lost almost equivalent amounts of disc height under lower axial compression (260N) for a shorter period of time (Pre-load). The rate of creep decreased over time, which echoes the findings of Reilly et al (1994). It is also in keeping with Darcy's Law which states that as the hydrostatic pressure decreases the rate of fluid flow will decrease. A total of just less than 5mm of axial creep occurred in this disc over 90 minutes of testing, which is greater than the 1.5mm of diurnal disc height loss reported by Adams et al (1987). Adding repeated flexion to the loading pattern, as was the case in these studies, further reduced the disc height of the specimens as the increased hydrostatic pressure would promote further fluid flow out of the disc.

The results of the reversal testing (reversed or not) provided a sub-classification of the specimens. The post-failure disc height of the two reversal groups was significantly different. The group of specimens that responded to the extension loading was the one that has retained most disc height. This is in keeping the mechanical theory underlying the reversal of the displaced portion of nucleus that has been proposed in this thesis. The theory attributes the change in position of the displaced

	STUDY 1	STUDY 2	STUDY 3
All specimens	57.08% (11.19%)	54.53% (8.19%)	55.89% (15.59%)
All prolapsed specimens	60.14% (7.50%)	53.33% (10.90%)	53.03% (18.00%)
All non-prolapsed specimens	55.53% (12.11%)	55.13% (7.30%)	56.67 (13.23%)
All prolapsed and reversed specimens *	-	-	42.17% (6.91%)
All prolapsed and not reversed specimens	-	-	66.43% (17.43%)

Table 6.1: The mean (SD) disc height loss, measured according to Wilke et al (2006), of all specimens in studies 1 and 3 and a number of specimens in study 2 are compared. As study 2 included a number of different testing protocols only those specimens that underwent similar testing to the specimens of study 1 and 3 are included in the results of study 2. The difference in disc height loss between these groups is not statistically significant (one-way ANOVA, $p < 0.05$). The response of the study 3 specimens to the reversal testing provided a sub-classification of the total study 3 specimens, namely those that did not prolapse, those that prolapsed and reversed and those that prolapsed and did not reverse. *Significant disc height differences between the sub-classified groups were found.

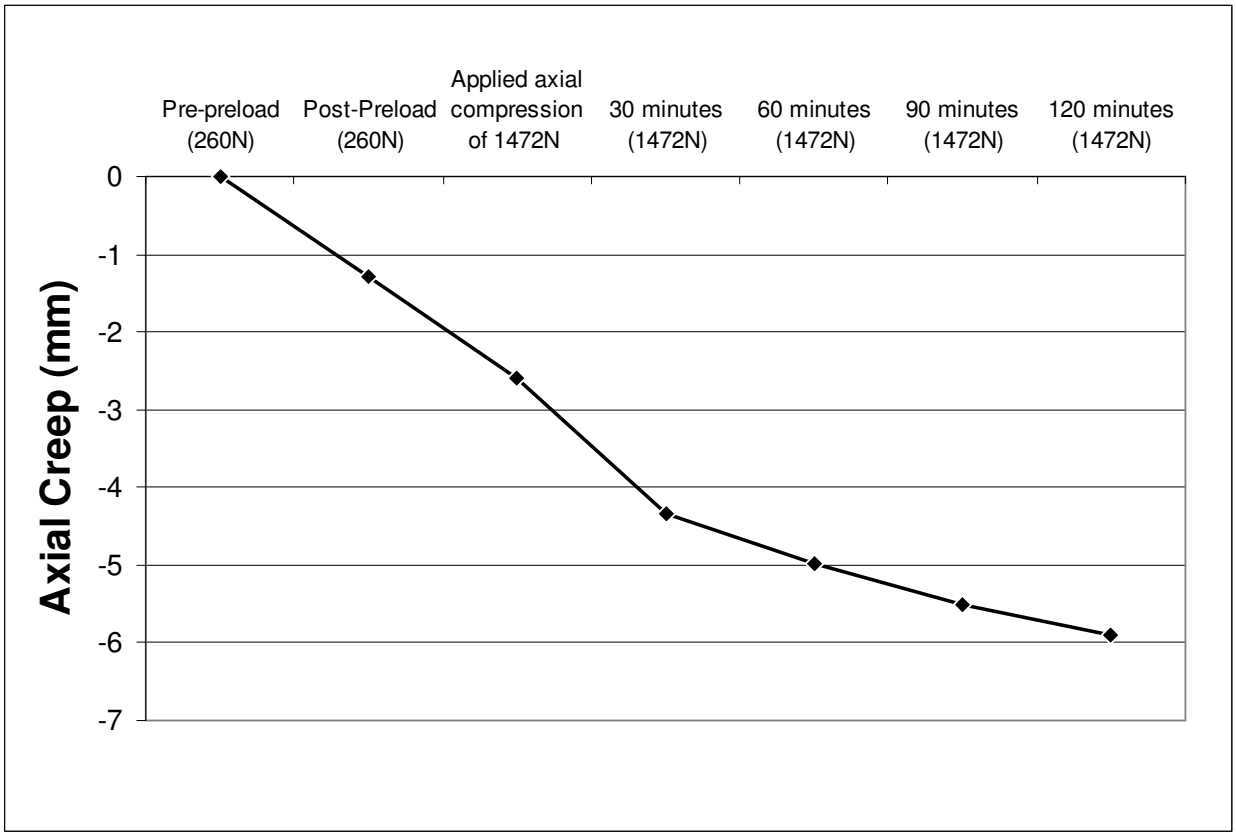

Figure 6.1 shows the progressive axial creep of one specimen when loaded in a position of minimal torque under axial compression. The specimen was preloaded with 260N for 15 minutes prior to being loaded with 1472N of compression. The greatest axial creep occurred in the first 30 minutes of loading, echoing the findings of Reilly et al (1994). Relative to the post-preload disc height, which was considered the pre-test disc height in the in vitro studies in this thesis, a total of just less than 5mm of axial creep occurred in this disc over 90 minutes of testing, which is greater than the 1.5mm of diurnal disc height loss reported by Adams et al (1987). After 120 minutes of axial compression the creep of 6mm, as recorded by the Instron, is equivalent to 67% of disc height loss when measured according to Wilke et al (2006) and reported as a percentage loss of the post-preload disc height.

portion of nucleus to a compressive stress gradient in the posterior annulus. The fluid and displaced portion of the hydrostatic nucleus within the posterior annulus will move from the higher level of compression to a lower level of compression within the posterior annulus. The hydraulic pressure would be in an anterior direction. Based on this argument the discs that have greater disc height are more likely to reverse as greater extension of the segments can occur before the facet joints bring the range to a halt and also the stress in the posterior annulus is compressive rather than tensile.

These studies have increased our understanding of this in vitro model, an understanding that facilitates more accurate interpretation of the results of research using the model. Consideration of the limitations posed by the concurrent disc height loss and disc prolapse is imperative on a study-by study basis.

The Angle of Lumbar Lordosis

The angle of lumbar lordosis was an issue that was addressed in three of the studies as identifying biomechanical factors that can be used to predict ones risk of developing LBP is central to successful prevention of LBP. Prompted by the clinical thinking that not all spines are equal and that hypolordotic spines are more predisposed to disc prolapse than spines with greater lordotic angles, attempts were made to create herniations in the thoracic rather than cervical porcine specimens as these specimens are more hypolordotic. These attempts failed. While the hypolordotic spines may not be more predisposed to disc prolapse the result should be considered knowing that the posterior annulus of the porcine thoracic specimens is thicker, the specimens were stiffer and the thoracic disc height was less than of the porcine cervical discs. Given this further investigation of the clinical theory was warranted. The lordotic angles of the spines and specimens in the third study were measured but again failed to distinguish between the different specimen responses to failure or reversal testing. Once again it may be that hypolordotic spines may not be more predisposed to disc prolapse but the variation in the in vivo range of lordosis of human lumbar spines (16 and 70^0 , Hansson et al 1985, Murrie et al 2002) is much greater than the variability seen here in the porcine cervical osteoligamentus spines ($28 – 42^0$). It may be that the range of lordosis within the porcine specimens is not large enough to address this question. Finally, in keeping with the same clinical perspective it was considered in the in vivo study whether individuals with hypolordotic spines had less extension in their lumbar spines relative to those with greater lumbar lordosis in standing. The lumbar posture in standing of the subjects in study 4 correlated significantly with the peak extension in the McKenzie exercises was not consistently indicative of the range of extension the lumbar spine could achieve.

Extension

Central to this thesis was the role of extension in the reversal of disc prolapse. The McKenzie approach supposes that in a posterior or posterolateral disc prolapse the displaced portion of nucleus in a prolapsed disc can be moved back towards the center of the disc using extension movements and positions. Ten degrees of extension, relative to a position of zero torque, was used to in the in vitro study to change the position of the displaced portion of the nucleus in the posterior annulus. In comparison, the in vitro equivalent of the peak in vivo extension ranges achieved in the McKenzie exercises in study 4 are repeated in Table 6.2. The extension range used in the in vitro study was marginally greater than that identified at the L5/S1 segment when the spine is mobilized in an extension in lying (EIL) position. Facet joint damage was not identified after extension in the in vitro study suggesting that the range used in the in vitro study under the low compressive loads was not excessive. Based on clinical experience this degree of extension could not be achieved in the early stages of disc prolapse. Future research will need to decipher the range that is most appropriate to the investigation of the treatment of disc prolapse created in vitro and also that used in vivo.

This dual in vitro-in vivo approach bodes well towards achieving the ultimate goal of evidence-based practice but there is no doubt that investigating clinical concepts through in vivo and in vitro studies has limitations. In vivo research is limited by the pragmatic boundaries of subject numbers and ethical subject inclusion-exclusion criteria. The clinical concepts investigated in this study are based on the recognition of patterns in clients with acute disc prolapse - a most unsuitable subject pool. One may consider the use of an in vitro animal model to investigate concepts that apply to the treatment of humans a limitation. Further to that already stated, it should be highlighted that investigating the mechanics of the IVD was what was fundamental to enhancing our understanding of the clinical concepts in question. Rather than considering the use of an in vitro animal model a limitation of this study, considering it vital to the feasibility and quality of this research may be more appropriate.

This research has provided a foundation for a large body of future studies. Numerous avenues of research aimed at bridging the gap between the research and clinical perspectives of low back pain including the links between clinical patterns and the mechanical, morphological, neurophysiological and chemical parameters utilized in research have been highlighted. Understanding the mechanics of herniated discs with varying degrees of disc height loss, including loss of hydraulic behavior, motion, foramen space and changes in facet loading will be valuable to clinical practice. Investigation of the selection of the practice of the McKenzie approach and the stress gradient driving the movement of the nucleus is of utmost importance. Also outstanding are studies investigating the recommended

Study 3; In Vitro Extension ROM (degs.)						
Porcine C3/4	10					
Study 4: In Vitro Equivalent of the In Vivo Extension ROM (degs.)						
	Extension in standing	**Overhead reach**	**Extension in prone lying**	**Extension in prone with extension mobilization**	**Extension in prone after extension mobilization**	**Extension in prone in a left side glide**
Degrees of Extension of Lumbar Spine (In vitro)	34.99	25.32	36.53	41.82	39.52	37.97
L4/5	5.25	3.80	5.48	6.27	5.93	5.69
L5/S1	7.58	5.49	7.91	9.06	8.56	8.23

Table 6.2: The in vitro range of extension used in study 3 is compared to the in vivo segmental ranges achieved in the McKenzie exercises in study 4. Total lumbar spine in vivo extension ranges were converted to segmental extension based on the in vivo results of Pearcy et al (1984) and Pearcy and Tibrewal (1984). The equivalent in vitro ranges are calculated from the in vivo ranges. As in vitro ranges are reported relative to elastic equilibrium, a 14.1^0 adjustment to the in vivo range was made (Andersson et al (1979) and in vitro segmental distributions calculated (Adams 1994).

McKenzie ‘doses’ in acute, subacute and chronic phases of rehabilitation (sustained versus repeated extension), the impact such ‘doses’ may have on the health of other spinal tissues and the role of repeated extension in the prevention of recurrence of displacement of a portion of the nucleus.

APPENDIX A

The direction of progressive herniation in porcine spine motion segments is influenced by the orientation of the bending axis.

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Clin Biomech (Bristol, Avon). 2005 Feb;20(2):126-9.

Abstract

Background. It has been shown that disc herniations are a cumulative injury created by repetitive flexion motion while under modest compressive loads. There is a lack of data linking the direction of nucleus tracking to the orientation of the bending motion axis. Our purpose was to determine if the direction that the nucleus tracks through the annulus during progressive herniation is predictable from the direction of bending motion (i.e. a specific side with posterio-lateral herniation).

Methods. Matched cohorts (n = 16) of porcine cervical spine (C3/4 and C5/6) motion segments were potted in aluminum cups and bent at an angle of 30° to the sagittal plane flexion axis while under a sustained compressive load of 1472 N.

Findings. The direction of bending motion affected the tracking pattern of the nucleus through the annular fibres in a predictable pattern. Specifically, bending the motion segments at an angle of 30° to the left of the sagittal plane flexion axis biased the movement of the nucleus toward the posterior right side of the disc in 15 of the 16 specimens.

Interpretation. Based on this animal model, shown to have similar biomechanical behaviour to humans, the direction that the nucleus tracks through the annular fibres appears to be dependent upon the direction of bending motion. This may have implications on both herniation prevention and rehabilitation of posterio-lateral bulges and herniations.

Introduction

Clinically, disc herniation is most commonly observed posteriorly or posterio-laterally (Cruess and Rennie, 1984; White and Panjabi, 1978). Although a consistent method for generating disc herniations has been established (Gordon et al., 1991; Callaghan and McGill, 2001), there is a lack of research documenting whether the type of motion can influence the direction that the nucleus tracks (i.e. posterio-lateral). Using cadaveric lumbar motion segments with intact passive tissues and facets, Adams and Hutton (1985) demonstrated that repetitively loading the spine results in delamination of the annulus. Specifically, when the spine is loaded asymmetrically, the nucleus tracks along a radial fissure formed in the contralateral corner of the disc suggesting that annular delamination is load/direction dependent. This phenomenon was demonstrated while motion segments were cyclically compressed at a pre-determined flexion angle, therefore few specimens failed by disc prolapse. Callaghan and McGill (2001) determined that posterior disc herniations are consistently created with repetitive flexion under modest static compressive forces. Their data suggest that disc herniations are an injury that result from cumulative bending trauma and can initiate after only 5870 cycles of flexion/extension while under a compressive load of only 867 N. However, when the compressive load was increased to 1472 N, the incidence of disc herniations also increased and the extent of tissue damage was more severe. With this background, an asymmetrical loading pattern combined with repetitive flexion is needed to assess whether changing the bending axis influences the direction of progressive tracking. If so, this would provide evidence to justify specific primary and secondary prevention approaches together with specific motion based rehabilitation strategies.

The purpose of this study was to determine if the direction of nucleus tracking during progressive herniation is dependent upon (and therefore predictable from) the plane of bending motion. It was hypothesized that repetitively bending the motion segment oriented at the *x* -axis minus 30° (terminology according to Panjabi et al., 1974) would bias the progressive tracking of the nucleus towards the posterior right side of the disc. In orthopaedic terms, the motion was about the sagittal plane flexion axis that had been turned 30° (Fig. 1). The motion examined in the present study is a common movement in real working life as documented by Fathallah et al. (1998) and shown in Fig. 2.

Methods

Sixteen porcine cervical spines were obtained and frozen immediately post-mortem in doubled polyethylene bags at –20 °C. The spines were matched for age (approximately 6 months old), weight (80 kg), diet, and physical activity. Prior to testing, the specimens thawed for 12–15 h to ensure the disc had reached room temperature. They were then dissected into motion segments of two vertebrae (C3/4 and C5/6). The intervening disc, facet joints, and passive tissues of the two vertebrae remained intact. The data set consisted of 12 C3/4 and 4 C5/6 motion segments. Fixation of the specimens was achieved by four 18-gauge wires which were looped bilaterally around the anterior processes and the lamina and looped through holes in aluminum cups. A "pastry bag" was used to inject non-exothermic dental stone (Denstone®, Miles, South Bend, IN, USA) all around the base of the vertebra and within the cups to further prevent any motion at the cranial and caudal ends of the motion segments. Finally, a screw was also used to fasten the top aluminum cup to the cranial end of the motion segment, protruding a maximum of 1 cm into the centre of the vertebral body. To assist subsequent investigation of the direction of nucleus tracking, a solution of 0.25 mL barium sulphate (radio-opaque), 0.15 mL water, and 0.15 mL blue dye (Coomassie Brilliant Blue G-mix: 0.25% dye, 2.5% MeOH, 97.25% distilled water) was injected into the centre of the discs through the anterior annulus wall. Although radiographs were not obtained in this experiment, barium sulphate was included because this consistency and viscosity of solution was known to track through the annulus only if a fissure was present (Callaghan and McGill, 2001). To maintain hydration, the specimens were wrapped in bench-top paper, wet with a physiologic saline solution (0.9% NaCl), and enclosed in polythene film. The direction of nucleus tracking was documented from visual inspection and digital photographs of the discs after sectioning the motion segments. A servohydraulic dynamic testing machine (model 8511, Instron Canada, Burlington, Ont., Canada) was used to apply a compressive pre-conditioning load of 300 N to all specimens for 15 min prior to testing. The pre-conditioning countered any swelling that had occurred post-mortem and due to freezing. During the preload, a custom jig (Callaghan and McGill, 2001) controlled using a servo-motor, found a position of zero torque, or elastic equilibrium, which was used as the zero position (neutral lordosis) for the passive and dynamic testing protocol. Each specimen underwent a passive range of motion test performed under 1472 N of compression. The passive test brought the specimens through five repeats of their passive range of motion at a rate of 0.5°/s. The axis for the range of motion was oriented

30° from the anatomical sagittal plane flexion/extension axis. Again using the Instron, a dynamic test repetitively bent 16 specimens (about the 30° axis) at a rate of 0.5 Hz for 6000 cycles while under 1472 N of compression. The bending 'elbow' of the angle vs. torque curve, corresponding to the point of increased stiffness in bending, was used as a limit for the range of motion in the dynamic test. The specimens were brought back to neutral (the position of elastic equilibrium) for each cycle.

Results

Bending the motion segments about an axis oriented 30° to the left of the sagittal plane flexion axis resulted in the focused nucleus tracking toward the posterior right side of the disc in 15 of the 16 trials (94%) (Fig. 1). Specifically, while 14 of the 15 were very distinct in the nucleus track, one of the 15 had a bulge as expected on the right posterio-lateral side of the disc, but with more diffuse leakage of the dye around the annulus. The one specimen that did not show a distinct track simply demonstrated general failure with leakage of the dye in all directions.

Discussion

These data suggest that the direction of progressive posterio-lateral herniation, at least in porcine segments, is dependent upon (and therefore predictable from) the direction of bending motion. Repetitively bending the motion segments at an angle of 30° to the left of the sagittal plane flexion axis biased the tracking of the nucleus toward the posterior right side of the disc. Clearly, repetitive flexion under a modest compressive load remains a convincing mechanism for disc prolapse (Callaghan and McGill, 2001). The initiation and progression of a herniation involves the nucleus tracking through a fissure in each concentric layer of annular fibres as suggested by Adams and Hutton (1985) and seen in the present study. Discovering that the side that the nucleus tracks is dependent upon the direction of bending motion is of use in understanding injury mechanics (particularly progressive disc herniation) and therefore in developing methods of injury prevention. It is plausible that an observed posterio-lateral herniation or bulge on one side of the disc would be linked to dominant bending to the other side

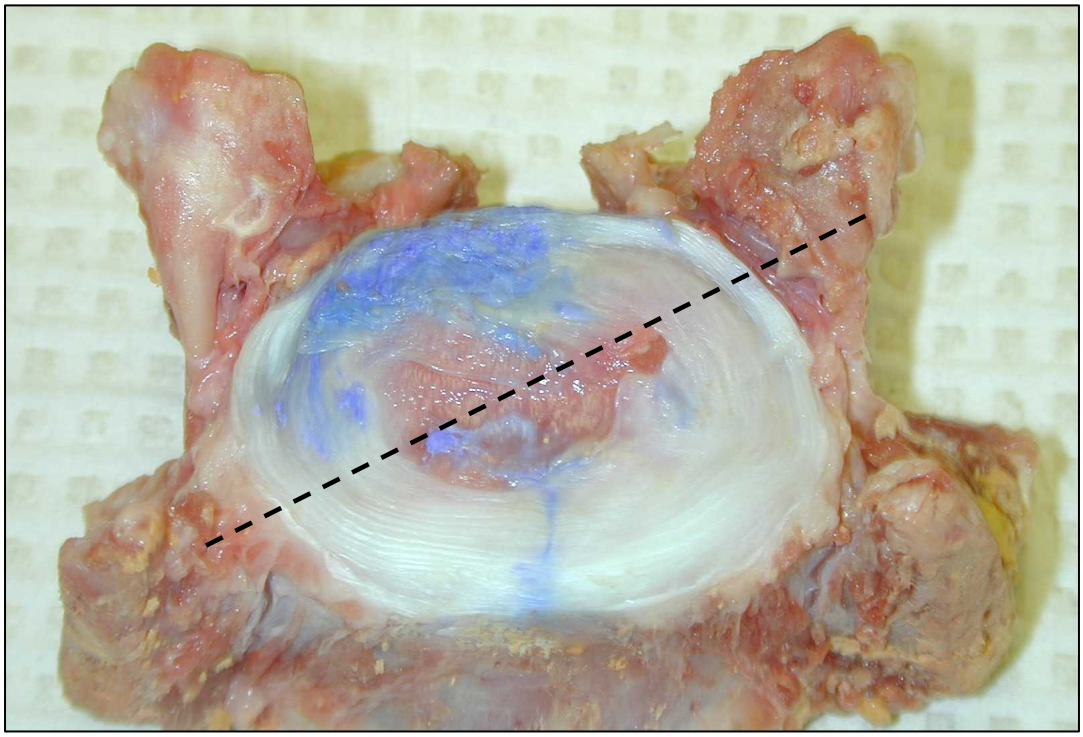

Fig. 1: Photograph of C4 sectioned from a C3/4 motion segment. Bending the motion segments about an axis oriented 30° to the left of the sagittal plane flexion axis resulted in the nucleus tracking toward the posterior right side of the disc.

Fig. 2: An industrial worker performing a repetitive task involving combined flexion with lateral bend is demonstrative of the complex motion pattern examined in the present study.

in real life (Adams and Hutton, 1985). Based on retrospective injury records, Fathallah et al. (1998) reported that industrial workers at high and medium risk of low back disorders performed complex motion patterns involving combined lateral and twisting velocities similar to the motion pattern examined in the present study. The observation of one specimen that exhibited diffuse annular failure could have been due to several variables. It is possible that the quality of the disc was compromised and that undetected damage existed in this specimen prior to the experiment. It is unlikely that the other discs were damaged given their origin and similar behaviour.

The primary limitation of this study was the use of an animal model to portray the response of the human lumbar spine. Young, healthy, human cadavers matched for age, diet, weight, and physical activity are simply not available. However, the porcine spine appears to be a useful surrogate to the human lumbar spine in both anatomical features (Oxland et al., 1991) and biomechanical behaviour (Yingling et al., 1999). Although no study has examined the orientation of the annular fibres in pig discs, our hand-protractor measurements suggest they are similar. The similarities of a pig neck and human lumbar spine should not be surprising since a large extensor moment is required to hold up the cantilevered head of a quadruped which results in huge compressive forces on the cervical vertebrae similar to the compressive forces on the human lumbar spine from supporting the weight of the upper body and head. Pigs also exercise a 'rooting' behaviour hence the robust extensor anatomy and restricted motion similar to a human lumbar spine. In summary, a porcine cervical spine model provides a homogeneous subject sample for a matched group control study such as this and appears to have similar failure mechanics to a young adult human.

In conclusion, repetitive bending (flexion with some lateral bend) produces tracking of the nucleus toward the opposite posterior side of the disc. This information may be useful for both diagnostic and prevention issues and introduces a new question: If the direction that the nucleus tracks is dependent upon the direction of bending motion, can prophylactic motion patterns be defined that would slow the progression of a prolapsed disc to a herniation?

APPENDIX B

Information and Consent FormORE File #: 11198

Study Title: Investigation of the lumbar spine range and combination of motion during activities of daily living and during prescribed exercise.

This study represents the student investigator's (Joan Scannell) PhD research and will be supervised by Professor McGill, Occupational Biomechanics and Safety Laboratory, Department of Kinesiology, University of Waterloo.

Purpose: The purpose of this study is to investigate the ranges and combinations of lumbar movement utilized during activities of daily living and during clinically recommended exercises. This study will use 3-dimensional spine motion monitoring equipment to quantify movement patterns and these movement patterns will be investigated in future research. Simply, an instrument will be strapped to your back which will measure your spine motion during the assessment and treatment procedures. You will receive a trace and Joan Scannell will discuss your results with you. This may lead to better treatment protocols in the future.

All study participants will be assigned a code, which will be attached to the results to assure your anonymity. All data will be kept confidential and stored for an indefinite period in a secure, locked location at the University of Waterloo Spine Biomechanics Laboratory. This data may be used in future studies, but will remain accessible only to the members of the Spine Biomechanics Laboratory, and all participants will remain anonymous in any future analysis of the data.

What we are asking you to do: You will be asked to perform your usual tasks but while wearing the spine motion monitor. This light device consists of a sensor (approximately 2cm x 2cm x 2cm) and a source (approximately 4cm x 4cm x 4cm) both of which will be attached to your back with straps. This test will add about 30 minutes to your usual appointment time.

Specifically the tasks you will be requested to perform are:

1. Stand in your natural standing posture for 10 seconds
2. Bend forwards, backwards and side to side
3. Reach with each hand separately to objects placed in front of you, while in a standing position and then in a sitting position
4. Reach both hands overhead
5. Bend backwards, supporting your trunk with your hands on your hips and keeping your knees straight
6. Leaning your shoulder onto a wall move your hips to the wall
7. Lie face down while the physiotherapist palpates your lumbar spine
8. Perform a sloppy push up by lying face down, place your hands under your shoulders, keep your hips on the bed and push your upper body up with your arms.
9. Perform a sloppy push up while the physiotherapist glides assists the extension of your spine

10. Allow Joan Scannell to reposition your hips after which you will repeat a sloppy push up.

Participants who are not eligible for the study are

1. **individuals that currently have acute low back pain**
2. **individuals whose activities or movements are currently restricted by medical condition.**

Should an emergency situation arise in which emergency medical attention is required the emergency services will be contacted to arrange for emergency transportation via ambulance to hospital.

Every attempt will be made to accommodate the schedule of the study participants. If participants have any questions regarding the study, the contents of this form or the conditions of participation please contact Joan Scannell at 416 207-9395 or Professor Stuart McGill at 888-4567 ext 6761.

Risks with your participation: You are simply requested to wear the measuring device. There are no known risks associates with this. Please ask for answers to any questions that you may have.

Consent Form

I have read the information about the procedures and risks involved in this study, and have received satisfactory answers to my questions related to this study. The specific details of the study have been explained to me both orally and in written form on the previous pages. I understand that my anonymity will be assured unless I give specific written permission for my name to be used and that I may withdraw my participation at any point in the study without penalty by simply asking to do so.

This study has been reviewed by, and received ethics clearance through the Office of Research Ethics at the University of Waterloo. If I have any questions or concerns resulting from my participation in this study I can contact the director at 888-4567 ext. 6005 with the ORE file number at the top of the page. I am aware that I may report any concerns or complaints from any participation in this study to the Office of Research Ethics, University of Waterloo.

Participant Name; _____ Date ;___/ /__

D M Y

Participant 's Signature: _____

Participant Tel:_____ Participant e-mail:_____

Witness Signature:_____ Date ;___/ /__

D M Y

Participant Code: ____ (office use)

References

Adams M.A.(1995) Mechanical testing of the spine: an appraisal of methodology, results and conclusions. *Spine* 20: 2151-2156

Adams, M.A., Bogduk, N., Burton, K., Dolan, P. (2002) The biomechanics of back pain. Churchill Livingstone.

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

Adams M.A.,.Dolan P. (1991) A technique for quantifying the bending moment acting on the lumbar spine in vivo. *J.Biomechanics* 24:117-26.

Adams M.A., Dolan P (1995) Recent advances in lumbar spinal mechanics and their clinical significance. *Clinical Biomechanics* 10: 3-19

Adams M.A., Dolan P. (1996) Time-dependant changes in the lumbar spine's resistance to bending. *Clinical Biomechanics* 11: 194-200

Adams M.A., Dolan P., Hutton W.C., Porter R.W. (1990) Diurnal changes in spinal mechanics and their clinical significance. *The Journal of Bone and Joint Surgery* 72B: 266-270

Adams M.A., Dolan P., Hutton W.C., Porter R.W. (1988) The lumbar spine in backward bending. *Spine* 13: 1019-1026

Adams M.A, Green T.P., Dolan P. (1994) The strength in anterior bending of lumbar intervertebral discs. *Spine* 19: 2197-2203

Adams M.A., Hutton W.C. (1980) The effect of posture on the role of the apophyseal joints in resisting intervertebral compressive force. *Journal of bone and joint surgery* 62B: 358-362

Adams M.A., Hutton W.C. (1981) The relevance of torsion to the mechanical derangement of the lumbar spine. *Spine* 6*:* 241-248

Adams M.A, Hutton W.C. (1982) Prolapsed intervertebral disc: A hyperflexion injury. *Spine 7*: 184-191

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

Adams M.A., Hutton W.C. (1986) Has the lumbar spine a margin of safety in forward bending? *Clinical Biomechanics* 1: 3-6

Adams M.A., Hutton W.C., Scott J.R.R. (1980) The resistance to flexion of the lumbar intervertebral joint. Spine 5: 245-253

Adams M.A., Mannion A.F., Dolan P. (1999) Personal risk factors for first-time low back pain. *Spine* 24: 2497-2505

Adams M.A, May S., Freeman B.J.C., Morrison H.P., Dolan P. (2000) Effects of backward bending on lumbar intervertebral discs. *Spine* 25: 431-437

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

Adams M.A, McNally D.S., Chinn H., Dolan P (1994) Posture and the compressive strength of the lumbar spine. *Clinical Biomechanics* 9: 5-14

Adams M.A., Roughley P (2006) What is intervertebral disc degeneration and what causes it? *Spine* 31: 2151-2161

Adler JH, Schoenbaum M, Silberberg R, (1983) Early onset of disk degeneration and spondylosis in sand rats. Vet Pathol 20: 13-22

Aina A., May S., Clare H. (2004) The centralization phenomenon of spinal symptoms- a systematic review. *Manual Therapy* 9: 134-143

Alexander A.H., Jones A.M., Rosenbaum Jr D.H. (1998) Nonoperative management of herniated nucleus pulposus: patient selection by the extension sign: Long-term follow-up. *Orthopedic Review* 21: 181-188

An H.S., Anderson P.A., Haughton V.M., Iatridis J.C., Kang J.D., Lotz J.C., Nataragjan R.N., Oegema T.R., Roughley P, Setton L.A., Urban J.P., Videman T. Andersson G.B.J., Weinstein HJ.N. (2004) Introduction: Disc degeneration: Summary (focus issue on disk degeneration) *Spine.* 29: 2677-2678

An H.S., Masuda K. (2006) Relevance of in vitro and in vivo models for intervertebral disc degeneration. *The Journal of Bone and Joint Surgery* 88: 88-94

Anderson G.D., Izzo M.W., Hall D.J., Vaccaro A.R., Hilibrand A., Arnold W., Tuan R.S. Albert T.J. (2002) Comparative gene expression profiling of normal and degenerative discs. *Spine* 27: 1291-1296

Andersson G.B., Schultz A.B. (1979) Effects of fluid injection on mechanical properties of intervertebral discs. *Journal of Biomechanics* 12: 453-458

Andersson G.B. (1981) Epidemiological aspects of low-back pain in industry. *Spine* 6: 53-60

Argoubi M., Shirazi-Adl A. (1996) Poroelastic creep response analysis of a lumbar motion segment in compression. *Journal of Biomechanics* 29: 1331-1339

Auerbach J.D., Johannessen W, Borthakur A, Wheaton A.J., Dolinskas C.A., Balderston R.A., Reddy R, Elliott D.M., (2006) In vivo quantification of human lumbar disc degeneration using T_{1p}-weighted magnetic resonance imaging. *Eur Spine* 3: 338-344

Aultman C.D., Scannell J.P., McGill S.M (2005) The direction of progressive herniation in porcine spine motion segments is influenced by the orientation of the bending axis. *Clinical Biomechanics* 20: 1-2

Aoki Y., Takahashi Y., Takahashi K., Chiba T., Kurokawa M., Ozawa T., Moriya H. (2003) Sensory innervation of the lateral portion of the lumbar intervertebral disc in rats. *The Spine Journal* 4: 275-280

Bardet J.P. (1992) A Viscoelastic model for the dynamic behavior of saturated poroelastic soils. *Journal of Applied Mechanics* 59: 128-135

Bass E.C., Duncan N.A., Hariharan J.S., Dusick J, Bueff H.Ulrich, Lotz J.C.: (1997) Frozen storage affects the compressive creep behavior of the porcine intervertebral disc: *Spine* 22*:* 2867-2876

Battie M., Videman T., Parent E. (2004) Lumbar disc degeneration: Epidemiology and genetic influences. *Spine* 29: 2679-2690

Battie M.C., Videman T., Gibbons L.E. (1995) Determinants of lumbar disc degeneration. A study relating lifetime exposures and magnetic resonance imaging findings in identical twins. *Spine* 20: 2601-2612

Belytschko T., Kulak R.F., Schultz A.B. (1974) Finite element stress analysis of an intervertebral disc. *Journal of Biomechanics* 7: 227-285

Benneker L, Heini P, Anderson S, Alini M, Ito K (2005) Correlation of radiographic and MRI parameters to morphological and biochemical assessment of intervertebral disc degeneration. *Eur Spine* 14: 27-35

Berlemann U, Gries N, Moore R (1998) The relationship between height, shape and histological changes in early degeneration of the lower lumbar discs. *Eur Spine* 7: 212-217

Berry R. (1961) Genetically controlled degeneration of the nucleus pulposus in the mouse. *Journal of BoneJoint Surgery* 43B: 387-393

Bibby SR, Jones DA, Ripley RM, Urban JPG. (2005) Metabolism of the intervertebral disc: Effects of low levels of oxygen, glucose, and pH on rates of energy metabolism of bovine nucleus pulposus cells. *Spine* 30: 487-496

Bibby S.R., Urban J.P.G. (2004) Effects of nutrient deprivation on the viability of intervertebral disc cells. *Eur Spine Journal* 13: 695-701

Binkley J., Finch E., Hall J., Black T., Gowland C. (1993) Diagnostic classification of patients with low back pain: Report on a survey of Physical Therapy Experts. *Physical Therapy*. 73: 138-155

Birney T.J., White J.J., Berens D, Kuhn G. (1992) Comparison of MRI and discography in the diagnosis of lumbar degenerative disc disease: *Journal of Spinal Disorders*: 5: 417-423

Biot M.A. (1956) General solutions of the equations of elasticity and consolidation for a porous material. *Journal of Applied Mechanics* 78: 91-96

Biot M.A. (1957) The elastic coefficients of the theory of consolidation. *Journal of Applied Mechanics* 57: 594-601

Boden S.D., Davis D.O., Dina T.S., Patronas N.J., Wiesel S.W. (1990) Abnormal magnetic resonance scans of the lumbar spine in asymptomatic subjects. *Journal of Bone and Joint Surgery* 72: 403-408

Boden S.D. (1996) Current concepts review – The use of radiographic imaging studies in the evaluation of patients who have degenerative disorders of the lumbar spine: *Jornal of Bone and Joint Surgery* 78: 114-124

Bogduk N, Twomey L. (1991) Clinical anatomy of the lumbar spine, 2nd Edn, *Churchill Livingstone:*

Bogduk N. (1980) Lumbar dorsal ramus syndrome. *Med J Aust* 2*: 537-541*

Bogduk N, Wilson A.S., Tynan W (1982) The human lumbar dorsal rami. *J Anat* 134: 383-*397*

Bogduk N. (1983) The innervation of the lumbar spine. *Spine* 8: 286-293

Bogduk N. (1990) Pathology of lumbar disc pain*. J Manual Medicine* 5: 72-79

Bogduk N. (1997) Clinical anatomy of the lumbar spine and sacrum. 2nd Edn, *Churchill Livingstone*

Bogduk N., Amevo B., Pearcy M. (1995) A biological basis for instantaneous centres of rotation of the vertebral column. *Proc Instn Mech Engrs*. 209: 177-183

Boos N., Rieder R., Schade V., Spratt K.F., Semmer N., Aebi M. (1995) The diagnostic accuracy of magnetic resonance imaging, work perception, and psychosocial factors in identifying symptomatic disc herniations. *Spine* 20: 2613-2625

Boos N., Weissbach S, Rohrbach H, Weier C, Spratt K, Nerlich A (2002) Classification of age-related changes in lumbar intervertebral discs. *Spine* 27: 2631-2644

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

Brault J.S., Driscoll D.M., Laakso L.L., Kappler R.E., Allin E.F., Glonek T. (1997) Quantification of lumbar intradiscal deformation during flexion and extension, by mathematical analysis if magnetic resonance imaging pixel intensity profiles. *Spine* 22: 2066-2072

Brennan G.P., Fritz J.M., Hunter S.J, Thackeray A, Delitto A, Erhard R.E.(2006) identifying subgroups of patients with acute/subacute "nonspecific" low back pain. *Spine* 31: 623-631

Brinckmann P, Biggemann M, Hilweg D. (1988) Fatigue fracture of human lumbar vertebrae. *Clinical Biomechanics* 21: 809-812

Brinckmann P., Horst M. (1985) The influence of vertebral body fracture, intradiscal injection, and partial discectomy on the radial bulge and height of human lumbar discs. *Spine* 10: 138-145

Brinckmann P. (1986) Injury of the annulus fibrosis and disc protrusions. An investigation on human lumbar discs. *Spine* 11: 149-153

Brinckmann P., Grootenboer H. (1991) Change of disc height, radial disc bulge and intradiscal pressure from discectomy. *Spine* 16: 641-646

Brotz D., Kuker W., Maschke E., Wick W., Dichgams J., Weller M. (2003) A prospective trial of mechanical physiotherapy for lumbar disk prolapse. *Journal of Neurology* 250: 746-749

Brown M.D. (1971) The pathophysiology of disc disease. Symposium on disease of the intervertebral disc. *Orthopedic Clinics of North America* 2: 359-370

Brown T., Hansen R.J., Yorra A.J. (1957) Some mechanical tests on the lumbosacral spine with particular reference to the intervertebral discs. *Journal of bone and joint surgery* 39A: 1135-1164

Buckwalter J.A. (1995) Spine update: Aging and degeneration of the human intervertebral disc. *Spine* 20: 1307-1314

Burton A.K., Tillotson K.M., Boocock M.G. (1994) Estimation of spinal loads in overhead work. *Ergonomics* 37: 1311-1321

Bushell G.R., Ghosh P, Taylor T.K.F., Akeson W.H. (1977) Proteoglycan chemistry of the intervertebral disks. *Clin Orthop 129*: 115-123

Butler D.S., Trafimow J., Andersson G., McNeill T, Huckman M (1990) Disc degenerate before facets. *Spine* 15:111-113

Callaghan J.P., McGill S.M. (1995) Muscle activity and low back loads under external shear and compressive loading. *Spine* 20 (9): 992-998

Callaghan J.P., Patla A.E., McGill S.M. (1999) Low back three-dimensional joint forces, kinematics, and kinetics during walking. *Clinical Biomechanics* 14: 203-216

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

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

Callaghan J.P., Gunning, J.L., McGill S.M. (1998) The relationship between the lumbar spine load and muscular activity during extensor exercise *Physical Therapy* 78: 8-18

Cassidy, J.J., Hiltener, A. Baer, E. (1989) Hierarchical structure of the intervertebral disc. *Connective Tissue Research* 23: 75-88

Chan D., Song Y., Sham P. Cheung K.M.C. (2006) Genetics of disc degeneration. *European Spine Journal* 3: 317-325

Cholewicki J., Crisco J.J. Oxland T.R., Yamamoto I., Panjabi M.M. (1996) Effects of posture and structure on three-dimensional coupled rotations in the lumbar spine. *Spine* 21: 2421-2428

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

Cihangiroglu M., Yildirim H., Bozgeyik Z., Senol U., Ozdemir H., Topsakal C., Yilmaz S. (2004) Observer variability based on the strength of MR scanners in the assessment of lumbar degenerative disc disease. *European Journal of Radiology* 51: 202-208

Cinotti G., Della Rocca C., Romeo S., Vittur F., Trasimeni G. (2005) Degenerative changes of porcine intervertebral disc induced by vertebral endplate injuries. *Spine* 30: 174-180

Clare H., Adams R., Maher C.G. (2004) A systematic review of efficacy of McKenzie therapy for spinal pain. *Australian Journal of Physiotherapy* 50: 209-216

Clare H.A., Adams R., Maher C.G. (2004) Reliability of the McKenzie spinal pain classification using patient assessment forms. *Physiotherapy* 90: 114-119

Cole T.C, Ghosh P, Taylor T.K. (1986) Variations of the proteoglycans of the canine intervertebral disc with ageing. Biochim Bio phys Acta 880: 209-219

Costi J, Hearn T, Fazzalari N (2002) The effect of hydration on the stiffness of intervertebral discs in an ovine model. *Clinical Biomechanics* 17: 446-455

Crock A.O. (1986) Internal disc disruption. A challenge to disc prolapse fifty years on. *Spine* 1: 650-653

Cruess R.L., Rennie W.R.J., Adult Orthopaedics, (1984), Churchill Livingstone Inc., New York.

DeLeo J.A.(2006) Basic science of pain. *The Journal of Bone and Joint Surgery* 88: 58-62

Delitto A, Erhard R.E, Bowling R.W. (1995) A treatment-based classification approach to low back syndrome: Identifying and staging patients for conservation treatment. *Phys. Ther.* 75: 470-485

Deyo R.A. (1986) Early diagnosis evaluation of low back pain. *J Gen Intern Med.* 1: 328-338

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

Dolan P., Adams M.A., Hutton W.C. (1988) Commonly adopted postures and their effect on the lumbar spine. *Spine* 13(2): 197-201

Donelson R, Silva G, Murphy, K (1990) Centralization phenomenon. Its usefulness in evaluating and treating referred pain. *Spine* 15: 211-213

Donelson R., Aprill C., Medcalf R., Grant W. (1997) A prospective study of centralization of lumbar and referred pain. A predictor of symptomatic discs and annular competence. *Spine* 22: 1115-1122

Donelson R., Grant W., Kamps C. (1991) Pain response to sagittal end-range spinal motion. A prospective, randomized, multicentered trail. *Spine* 16: S206-S212

Downing A.M., Hunter D.G. (2003) Validating clinical reasoning: a question of perspective, but whose perspective? *Manual Therapy* 8: 117-119

Drake J, Aultman C.D., McGill S, Callaghan J.P. (2005) The influence of static axial torque in combined loading on intervertebral joint failure mechanics using a procine model. *Clinical Biomechanics* 20:1038-1045

Dunlop R, Adams M, Hutton W (1984) Disc space narrowing and the lumbar facet joints. *The Journal of Bone & Joint Surgery* 66: 706-710

Edgar M.A., Nundy S., (1964) Innervation of the spinal dura mater. *J Neurol Neurosurg Psychiat* 29: 530-534

Faas A. (1996) Exercises: Which ones are worth trying, for Which Patients, and When? *Spine* 21: 2874-2879

Fairbank J.C.T., Park W.M., McCall I.W., O'Brien J.P. (1981) Apophyseal injections of local anesthetic as a diagnostic aid in primary low-back pain syndromes. *Spine 6*: 598-605

Farcy J.P., Schwab F. (1997) Management of flatback and related kyphotic decompensation syndromes. *Spine* 22: 2452-2457

Farfan H.F., Osteris V., Lamy C. (1976) The mechanical etiology of spondylolysis and spondylolisthesis. *Clinical Orthopedics and Related Research* Jun: 40-55

Fathallah F.A., Marras W.S., Parnianpour M. (1998) The role of complex, simultaneous trunk motions in the risk of occupation-related low back disorders, *Spine* 23: 1035-1042

Fazey P.J., Song S., Monsas A., Johansson L., Haukalid T., Price R.I., Singer K.P. (2006) An MRI investigation of intervertebral disc deformation in response to torsion. Clinical *Biomechanics* 21: 538-542

Fennel A.J., Jones A.P., Hukins D.W.L. (1996) Migration of the nucleus pulposus within the intervertebral disc during flexion and extension of the spine. *Spine* 21: 2753-2757

Fiebert I., Keller C.D. (1994) Are "passive" extension exercises really passive? *J Orthop Sports Phys Ther*. 19: 111-116

Freeman B.J.C. (2003) Does intradiscal electrothermal therapy denervate and repair experimentally induced posterolateral annular tears in an animal model? *Spine* 28: 2602-2608

Fritz J.M., Delitto A, Erhard R.E. (2003) Comparison of classification-based physical therapy with therapy based on clinical practice guidelines for patients with acute low back pain: a randomized clinical trial. *Spine* 28: 1363-71

Frobin W, Brinckmann P, Biggemann M, Tillotson M, Burton K (1997) Percison measurement of disc height, vertebral height and sagittal plane displacement from lateral radiographic views of the lumbar spine. *Clinical Biomechanics* 12: S1-S63

Frobin W, Brinckmann P, Kramer M, Hartwig E (2001) Height of lumbar discs measured from radiographs compared with degeneration and height classified from MR images. *Eur Spine* 11: 263-269

Giles L.G.F., Kaveri M.J.P. (1991) Lumbosacral intervertebral disc degeneration revisited: A radiological and histological correlation. *Journal of Manual Medicine* 6: 62-66

Ghosh P, Taylor T.K., Braund K.G., Larsen L.H. (1976) The collagenous and non-collagenous protein of the canine intervertebral disc and their variation with age, spinal level and breed. *Gerontology* 22: 124-134

Ghosh P. (1990) Basic biochemistry of the intervertebral disc and its variation with ageing and degeneration. *Journal of Manual Medicine* 5: 48-51

Godin C, Andrews D.M., Callaghan J.P., (2003) Cumulative low back loads of non-occupational tasks using a 3-dimensional video-based posture sampling approach. *Proceedings of XXIV Association of Canadian Ergonomists (ACE) Conference.* London Ontario, Canada 687-696

Galante J.O.(1967) Tensile properties of the human lumbar annulus fibrosis. *Acta Orthop Scand* Suppl. 100: 1-91

Goggin J.E., Li A.S., Franti C.E., (1970) Canine intervertebral disk disease: characterization by age, sex, breed, and anatomic site of involvement. *Am J Vet Res* 31: 1687-1692

Gordon S.J., Yang K.H., Mayer P.J., Mace A.H., Kish V.L., Radin E.L (1991) Mechanism of disc rupture - A preliminary report. *Spine* 16: 450-455

Gower W.E., Pedrini V, (1969) Age related variation in protein polysaccharides from human nucleus pulposus, annulus fibrosis and costal cartilage. *J Bone Joint Surg* 51A: 1154-1162

Granata K.P., Marras W.S., Davis K.G. (1999) Variation in spinal load and trunk dynamics during repeated lifting exertions. *Clin Biomech (Bristol, Avon).* 14: 367-375

Groen G, Baljet B, Drukker J (1990) The nerves and nerve plexuses of the human vertebral column. *Am J Anat* 188: 282-296

Grunhagen T. Wilde G., Mokhbi D., Shirazi-Adl S. Urban J.P. (2006) Nutrient supply and intervertebral disc metabolism. *The Journal of Bone and Joint Surgery* 88: 30-35

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

Gunzburg R, Parkinson R, Moore R et al (1992) A cadaveric study comparing discography magnetic resonance imaging, histology, and mechanical behavior of the human lumbar disc. *Spine* 17: 417-426

Haefeli M., Kalberer F., Saegesser D., Nerlich A., Boos N., Paesold G. (2006) The course of macroscopic degeneration in the human lumbar intervertebral disc. *Spine* 31: 1522-1531

Haher T.R., Felmy W., Baruch H., Devlin V., Welin D., O'Brien M., Ahmad J., Valenza J., Parish S (1989) The contribution of the three columns of the spine to rotational stability - A biomechanical model. *Spine* 14: 663-670

Hansson T., Bigos S., Beecher P., Wortley M. (1985) The lumbar lordosis in acute and chronic low-back pain. *Spine* 10: 154-155

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

Hendry N. (1958) The hydration of the nucleus pulposus and its relation to intervertebral disc derangement. *J Bone Joint Surg Br.* 40B: 132-144

Hickey D.S., Hukins D.W (1980a) X-ray diffraction studies of the arrangement of collagenous fibres in human fetal intervertebral disc. *Journal of Anatomy* 131: 81-90

Hickey D.S., Hukins D.W.L. (1980b) Relation between the structure of the annulus fibrosus and the function and failure of the intervertebral disc. *Spine* 5: 100-116

Hirsh C, Ingelmark B.E., Miller M., (1963) The anatomical basis for low back pain. *Acta Orthop Scandinav* 33: 1-17

Hirsch C., Schajowicz F. (1952) Studies on structural changes in the lumbar annulus fibrosus. *Acta Orthop Scandinav* 22: 184-189

Hirsh C, Schajowicz F. (1952) Studies on structural changes in the lumbar annulus fibrosus. *Acta Orthop Scandinav*. 22: 184-189

Hockaday J.M., Whitty C.W.M., (1967) Patterns of referred pain in the normal subject. *Brain* 90: 481-496

Holm S., Holm A.K., Ekstrom L. (2004) Experimental disc degeneration due to endplate injury. *J Spinal Disord Tech*. 17: 64-71

Holmes M.H., Mow V.C. (1990) The Nonlinear Characteristics of Soft Gels and Hydrated Connective Tissues in Ultra filtration. *Journal of Biomechanics* 23: 1145-1156

Holodny A., Kisza P., Contractor S., Liu W. (2000) Does a herniated nucleus pulposus contribute significantly to a decrease in height of the intervertebral disc? *Diagnostic Neuroradiology* 42: 451-454

Iatridis J.C., Setton L.A., Weidenbaum M., Mow V.C. (1997) The viscoelastic behavior of the non-degenerate human lumbar nucleus pulposus in shear. *Journal of Biomechanics* 30: 1005-1013

Iatridis J.C., Gwynn I (2004) Mechanisms for mechanical damage in the intervertebral disc annulus fibrosis. *Journal of Biomechanics* 37: 1165-1175

Jackson H.C., Winkelmann R.K., Bickel W.H. (1966) Nerve endings in the human lumbar spinal column and related structures. *J Bone Joint Surg*. 48A: 1272-1281

Jensen M.C., Brant-Zawadzki M.N., Obuchowski N., Moodic M.T., Malkasian D., Ross J.S. (1994) Magnetic resonance imaging of the lumbar spine in people without low back pain. *New England Journal of Medicine* 331: 69-73

Jette A.M, Smith K, Haley SM, Davis KD. (1994) Physical therapy episodes of care for patients with low back pain. *Physical Therapy.* 74: 101-110

Jette AM. (1989) Diagnosis and classification by physical therapists: a special communication. *Physical Therapy.* 69: 967-969

Kaapa E, Holm S, Inkinen R, Lammi M.J, Tammi M, Vanharanta H (1994) Proteoglycan chemistry in experimentally injured porcine intervertebral disk. *Spinal Disorder* 7: 296-306

Kavcic N, Grenier S, McGill S (2004) Quantifying tissue loads and spine stability while performing commonly prescribed low back stabilization exercises. *Spine* 29: 2319-2329

Keegan J.J. (1953) Alterations of the lumbar curve related to posture and seating. *Journal of bone and joint surgery* 35A: 589

Keller T.S., Hansson T.H., Holm S.H., Pope M.M., Spengler D.M. (1989) In vivo creep behavior of the normal and degenerated porcine intervertebral disk: A preliminary report. *Journal of Spinal Disorders* 1: 267-278

Keller T.S., Holm S.H., Hansson T.H., Spengler D.M. (1990) The Dependence of Intervertebral Disc Mechanical Properties on Physiologic Conditions. *Spine* 15: 751-761

Kellgren J, Lawrence J (1952) Rheumatism in miners. *X-ray study* 9:197-207

Kellgren J, Jeffery M, Ball J (1963) The epidemiology of chronic rheumatism. *Atlas of standard radiographs of arthritis* 14-19

Kelsey J.L., Gittens P.B., White III A.A., et al (1984) An epidemiologic study of lifting and twisting on the job and risk for acute prolapsed intervertebral disc. *Journal of Orthopedic Research* 2: 61-66

Kent P. Keating J. (2004) Do primary-care clinicians think that nonspecific low back pain is one condition? *Spine* 29: 1022-1031

Kettler A., Wilke HJ. (2006) Review of existing grading systems for cervical or lumbar disc and facet joint degeneration. *Eur Spine* 15: 705-718

Kilby J., Stignant M., Roberts, A (1990) The reliability of back pain assessment by physiotherapist using a "McKenzie Algorithm" *Physiotherapy* 76: 579-583

Koeller W, Funke F, Hartmann F. (1984) Biomechanical behaviour of human intervertebral discs subjected to long lasting axial loading. *Biorheology 21: 675-686*

Konno S, Kikuchi S, Nagaosa Y, (1994) The relationship between intramuscular pressure of the paraspinal muscles and low back pain. *Spine 19*: 2186-2189

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

Kroeber M.W., Unglaub F., Wang H. (2002) New in vivo animal model to create intervertebral disc degeneration and to investigate the effects of therapeutic strategies to stimulate disc regeneration. *Spine* 27: 2684-2690

Kulak R.F., Belytschko T.B., Schultz A.B., Galante J.O. (1976) Non-linear behavior of the human intervertebral disc under axial load. *Journal of Biomechanics* 9: 377-386

Lagrone M.O, Bradford D.S., Moe J.H, Lonstein J.E, Winter R.B, Ogilvie J.W (1988) Treatment of symptomatic flatback after spinal fusion. *J Bone Joint Surgery* 70: 569-580

Landis J, Koch G (1977) The measurements of observer agreements for categorical data. *Biometrics* 33: 159-174

Lane N, Nevitt M, Genant H, Hochberg M (1993) Reliability of new indices of radiographic osteoarthritis of the hand and hop and lumbar disc. *J Rheumatol* 20: 1911-1918

Lappalainen A, Kaapa E, Lamminen A, Laitinen O, Gronblad M (2002) The diagnostic value of contrast-enhanced magnetic resonance imagining in the detection of experimentally induced annular tears in sheep. *Spine* 27: 2806-2810

Larsen K., Weidlick F. Leboeuf-Yde C, (2002) Can passive prone extensions of the back prevent back problems? A randomized, controlled intervention trial of 314 military conscripts. *Spine* 27: 2747-2752

Lawrence J.S. (1969) General osteoarthrosis in a population sample. *Ann. Rheum. Dis.* 90: 381-389

Leahy J.C., Hukins D.W.L. (2001) Viscoelastic properties of the nucleus pulposus of the intervertebral disk in compression. *Journal of Materials Science: Materials in Medicine* 12: 689-692

Li C.S.R., Padoa-Schioppa C., Bizzi C. (2001) Neuronal correlates of motor performance and motor learning n the primary motor cortex of monkeys adapting to an external force field. *Neuron* 30: 593-607

Lin H.S., Liu Y.K., Adams K.H. (1978) Mechanical response of the lumbar intervertebral joint under physiological (complex) loading. *Journal of bone and joint surgery* 60A: 41-55

Lipson S.J., Muir H. (1981) Proteoglycans in experimental intervertebral disc degeneration. *Spine* 6: 194-210

Lisi A.J., O'Neill W.O., Lindsey D.P., Cooperstein R., Cooperstein E., Zuckerman J.F. (2006) Measurement o in vivo lumbar intervertebral disc pressure during spinal manipulation: A feasibility study. *Journal of Applied Biomechanics* 22: 234-239

Long A., Donelson R., Fung T. (2004) Does it matter which exercise? A randomized control trial of exercises for low back pain. *Spine* 29: 2593-2602

Long A.L. (1995) The centralization phenomenon. Its usefulness as a predictor of outcome in conservative treatment of chronic low back pain (A pilot study). *Spine* 20: 2513-2521

Long D, BenDebba M, Torgenson W (1996) Persistent back pain and sciatica in the United States: patient characteristics. *Journal of Spinal Disorders* 9: 40:58

Lu M.Y., Hutton W.C., Gharpuray V.M. (1996a) Do bending, twisting and diurnal fluid changes in the disc affect the propensity to prolapse? A viscoelastic finite element model. *Spine* 21: 2570-2579

Lu M., Hutton W., Gharpuray V (1996) Can variations in intervertebral disc height affect the mechanical function of the disc. *Spine* 21: 2208-16

Lyndsay, A., Hancock, E., Agouris, I., Smith, F., MacSween, A. (2007) The response of the nucleus pulposus of the lumbar intervertebral discs to functionally loaded positions. *Spine* 32: 1508-1512

Madan S., Rai A., Harley J (2003) Interobserver error in interpretation of the radiographs for degeneration of the lumbar spine. *Spine* 23: 51-56

Magnusson, M. Aleksiev A.R., Spratt K.F., Lakes R.S., Pope M.H. (1996) Hyperextension and spine height changes. Spine 21: 2670-2675

Markolf K.L., Morris J.M. (1974) The structural components of the intervertebral disc. *J Bone Joint Surgery* 56A: 675-87

Marras W.S. and Granata, K.P. (1997) Changes in trunk dynamics and spine loading during repeated exertions. *Spine* 22: 2564 – 2570

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

McCall I.W., Park W.M., O'Brien J.P. (1979) Induced pain referral from posterior lumbar elements in normal subjects. *Spine:* 4: 441-446

McGill S.M., Norman R.W. (1985) Dynamically and statically determined low back moments during lifting. *Journal of Biomechanics* 18: 877-885

McGill S.M. (1992) A myoelectrically based dynamic three-dimensional model to predict loads on lumbar spine tissues during lateral bending. *Journal of Biomechanics* 25: 395-414

McGill S.M. (2002) Low Back Disorders.: Evidence-based prevention and rehabilitation. Human Kinetics

McKenzie R. (2002) Treat Your Own Back. *Spinal Publications Ltd.*: Waikanae, New Zealand

McKenzie R.A. (1981) The Lumbar Spine - Mechanical Diagnosis and Therapy. Spinal Publications: *Spinal Publications Ltd:* Waikanae, New Zealand:

McKenzie R.Z., May S. ((2003) The lumbar spine: Mechanical diagnosis and therapy. 2nd Edition, Spinal Publications: *Spinal Publications Ltd:* Waikanae, New Zealand:

McKenzie R.Z. (1981) The lumbar spine: Mechanical diagnosis and therapy. *Spinal Publications Ltd:* Waikanae, New Zealand:

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

McNally D.S., Adams A.H., Goodship A.E. (1993) Can intervertebral disc prolapse be predicted by disc mechanics? *Spine* 18: 1525-1530

Melrose J., Ghosh P., Taylor T.K, Hall A., Osti O., Vernon-Roberts B., Fraser R. (1992) A longitudinal study of the matrix changes induced in the intervertebral disc by surgical damage to the annulus fibrosis. *J Orthop Res* 10: 665-76

Milette P., Fontaine S., Lepanto L., Cardinal E., Breton G. (1999) Differentiating lumbar disc protrusions, disc bulges, and discs with normal contour but abnormal signal intensity: magnetic resonance imaging with discographic correlations. *Spine* 24: 44-53

Mimura M., Panjabi M.M., Oxland T., Crisco J., Yamamoto I., Vasavada A. (1994) Disc degeneration affects the multidirectional flexibility of the lumbar spine. *Spine* 19 (12) 1371-1380

Moskowitz R.W., Ziv I., Denko C.W., Boja B., Jones P.K., Adler J.H. (1990) Spondylosis in sand rats: a model of intervertebral disc degeneration and hyperostosis. *J Orthop Res* 8: 401-411

Murrie V.L., Wilson H., Hollingworth W., Antoun N.M., Dixon A.K. (2002) Supportive cushions produce no proactical reduction in lumbar lordosis. *British Journal of Radiolody* 75: 536-538

Nachemson A. (1960) Lumbar intradiscal pressure. Experimental studies on post-mortem material. *Acta Orthop Scand* 43: 1-104

Naylor A. (1976) Intervertebral disc prolapse and degeneration. The biochemical and biophysical approach. *Spine* 1: 108-114

Naylor A., (1971) The biochemical changes in the human intervertebral disc in degeneration and nuclear prolapse. *Orthop Clin North Am.* 2: 343-358

NIOSH, 1981. Work Practices Guide for Manual Lifting, NIOSH Technical Report No. 81-122, US Department of Health and Human Services, National Institute for Occupational Safety and Health, Cincinnati, OH.

Oliphant D., Frayne R., Kawchuk G. (2006) A New Method of Creating Internal Disc Injuries of Various Grades. *Clin. Biomechanics* 21: 21-5

O'Sullivn P.B. (2000) Lumbar segmental "instability" clinical presentation and specific stabilizing exercise management. *Manual Therapy.* 5: 2-12

Osti O., Vernon-Roberts B., Moore R., Fraser R.(1992) Annular tears and Disc Degeneration in the Lumbar Spine. *The Journal of Bone and Joint Surgery* 678-682

Osti O.L., Vernon-Roberts B., Fraser R.D. (1990) Anulus tears and intervertebral disc degeneration: an experimental study using an animal model. *Spine* 15: 762-767

Oxland T., Panjabi M., Southern E., Dananceau J. (1991) An anatomic basis for spinal instability: a porcine trauma model. *J Orthop Res* 9: 452-462

Panagiotacopulos N.D., Pope M., Bloch R., Krag M. (1987) Water content in human intervertebral discs. Part II. Viscoelastic behavior. *Spine* 12: 918-924

Panjabi M.M., White A.A., Brand R.A., A note on defining body parts configurations, *J. Biomechanics* 7: 385--387

Pearch M., Portek I., Shepherd J. (1984) Three-dimensional x-ray analysis of normal movement in the lumbar spine. *Spine* 9: 294-297

Pearcy M.J., Tibrewal S.B. (1984) Axial rotation and lateral bending in the normal lumbar spine measured by three-dimensional radiography. *Spine* 9: 582-587

Pfeiffer M., Griss P., Franke P. (1994) Degeneration model of the porcine lumbar motion segment: effects of various intradiscal procedures. *Eur Spine Journal* 3: 8-16

Pfirrmann C.W.A., Metzdorf A., Zanetti M., Hodler J., Boos N. (2001) Magnetic resonance classification of lumbar intervertebral disc degeneration. *Spine* 26: 1873-1878

Philadelphia Panel (2001) Evidence based clinical practice guidelines on selected rehabilitation interventions for low back pain. *Physical Therapy* 81: 1641-1674

Pollintine, P. Dolan P., Tobias J., Adams M.A. (2004) Intervertebral disc degeneration can lead to "stress-shielding" of the anterior vertebral body: A cause of osteoporotic vertebral fracture? *Spine* 29: 774-782

Pollintine P., Przybyla A.S., Adams M.A. (2003) Neural arch load-bearing in old and degenerated spines. *Journal of Biomechanics* 37: 197-204

Punnett L., Fine L.J., Keyserling W.M., Herrin G.D., Chaffin D.B., (1991) Back disorders and non neutral trunk postures of automobile assembly workers. Scandinavian Journal of Work and *Environmental Health* 17: *337-346*

Race A., Broom N., Robertson P. (2000) Effects of loading rate and hydration on the mechanical properties of the disc. *Spine* 25: 662-669

Reilly T., Tynell A., Troup J.D.G. (1984) Circadian variation in human stature. *Chronobiology International* 1: 121-126

Riddle D.L. (1998) Classification and low back pain; a review of the literature and critical analysis of selected systems. *Physical Therapy.* 78: 708-37

Riches P.E., Dhillon N., Lotz J., Woods A.W., McNally D.S. (2002) The internal mechanics of the intervertebral disc under cyclic loading. *Journal of Biomechanics* 35: 1263-1271

Roberts S., Evans H. Trivedi J. Menage J. (2006) Histology and pathology of the human intervertebral disc. *Journal of Bone Joint and Surgery* 88: 10-14

Robinson M.G. (1994) The McKenzie method of spinal pain management. Grieves Modern *Manual Therapy* Churchill Livingstone. 753-769

Rohlmann A., Zander T., Schmidt H., Wilke H., Bergmann G. (2005) Analysis of the influence of disc degeneration on the mechanical behavior of a lumbar motion segment using the finite element method. *Journal of Biomechanics 39:* 2484-2490

Roughley P. (2004) Biology of Intervertebral disc aging and degeneration: Involvement of the extra cellular matrix. *Spine* 29:1-20

Rousseau M.A., Bradford D.S., Hadi T.M., Pedersen K.L., Lotz J.C. (2006) The instant axis of rotation influences facet forces at L5/S1 during flexion/extension and lateral bending. *Spine* 3: 299-307

Sahrmann S.A. (1988) Diagnosis by the physical therapist – a prerequisite for treatment: a special communication. *Physical Therapy.* 68: 1703-1706

Sahrmann S.A. (2002) Diagnosis and treatment of movement impairment syndromes. St. Louis: Moseby

Sambrook P.M., Macgregor A.J.,Spector T.D. (1999) Genetic influences on cervical and lumbar disc degeneration a magnetic resonance imaging study in twins. *Arthritis Rheum* 42: 366-372

Scannell J.P., McGill S.M. (2003) Lumbar posture - Should it, and can it, be modified? A study of Passive Tissue Stiffness and Lumbar Position During activities of daily living. *Physical Therapy* 83: 907-917

Schiebler M., Grenier N., Fallon M., Camerino V., Zlatkin M., Kressel H. (1991) Normal and degenerated intervertebral disk: In vivo and in vitro MR imaging with histopathologic correlation. *American Journal of Radiology Pictorial Essay* 157: 93-97

Schnebel B.E., Simmons J.W., Chowing J., Davidson R. (1988) A digitizing technique for the study of movement of intradiscal dye in response to flexion and extension of the lumbar spine. *Spine* 13: 309-312

Schneiderman G., Flannigan B., Kingston S., Thomas J., Dillin W., Watkins R. (1987) Magnetic resonance imaging in the diagnosis of disc degeneration: Correlation with discography. *Spine* 12: 276-281

Silberberg R., Aufdermaur M., Adler J.H. (1979) Degeneration of the intervertebral disks and spondylosis in aging sand rats. *Arch Pathol Lab Med* 103: 231-235

Silberberg R., Gerritsen G. (1976) Aging changes in intervertebral discs and spondylosis in Chinese hamsters. *Diabetes* 25: 477-483

Shah J.S. (1980) Structure, morphology and mechanics of the lumbar spine. In Jayson MIV (ed) The lumbar spine and backache, *2nd edn. Pitman, London,* Chapter 13, 359-405

Shah J.S., Hampson W.G., Jayson M.I.V. (1978) The distribution of surface strain in the cadaveric lumbar spine. *J Bone Joint Surg*. 60B: 246-251

Shirazi-Adl A., Shrivastava S.C., Ahmed A. (1984) Stress analysis of the lumbar disc-body unit in compression: A three-dimensional nonlinear finite element study. *Spine* 9: 120-134

Shirazi-Adl A. (1989) Strain in fibers of a lumbar disc. Analysis of the role of lifting in producing disc prolapse. *Spine* 14: 96-103

Shirazi-Adl A. (1991) Finite-element evaluation of contact loads on facet of an L2-L3 lumbar. *Spine* 16: 533-541

Shirazi-Adl A. (1994) Biomechanics of the lumbar spine in sagittal/lateral moments. *Spine* 19: 2407-2414

Simon B.R., Wu J.S.S., Carleton M.W., et al (1985) Poroelastic dynamic structural models of Rhesus motion segments. *Spine* 10: 494-507

Simunic I., Broom N., Robertson P. (2001) Biomechanical factors influencing nuclear disruption of the intervertebral disc. *Spine* 26:1223-1230

Smeathers J.E., Joanes D.N. (1988) Dynamic compressive properties of human lumbar intervertebral joints: A comparison between fresh and thawed specimens. *Journal of Biomechanics* 21: 425-433

Smyth M.J., Wright V. (1959) Sciatica and the intervertebral disc. An experimental study. *J.Bone Joint Surg.* 40A: 1401-1418

Snook S.H., Webster B.S., McGarry R.W., Fogleman M.T., McCann K.B. (1998) The reduction of chronic nonspecific low back pain through the control of early morning lumbar flexion. A randomized controlled trial. *Spine* 23: 2601-2607

Sobajima S., Kompel J.F., Kim J.S., Wallach C.J., Robertson D.D., Vogt M.T., Kang J.D., Gilbertson L.G. (2004) A Slowly Progressive and Reproducible Animal Model of Intervertebral Disc Degeneration Characterized by MRI, X-Ray, and Histology. *Spine* 30: 15-24

Stevens B.J., McKenzie R.A. (1988) Mechanical diagnosis and self treatment of the cervical spine. In: Grant R (ed) Physical therapy of the cervical and thoracic spine. *Churchill Livingstone, New York,* Chapter 14, 271-289

Stillwell D.L. (1957) Regional variations in the innervation of deep fasciae and aponeuroses. *Anat Rec* 127: 635-653

Stokes I., Iatridis J. (2004) Mechanical conditions that accelerate intervertebral disc degeneration: Overload versus immobilization. *Spine* 29: 2724-32. Review

Sullivan J., Farfan H., Kahn D. (1971) Pathologic changes with intervertebral joint rotational instability in the rabbit. *Can J. Surg*. 14: 71-79

Swartz J. (1993) Protrusion, extrusion....confusion! *Seminars in Ultrasound, CT and MRI.* 14: 383-384

Tampier C., Drake, J., Callaghan, J.P. and McGill S.M. (2006) Progressive Disc Herniation: An investigation of the mechanism using histochemical and microscopic techniques. In Press

Taylor J., Twomey L. (1994) The effects of aging on the intervertebral discs. In *Grieve's modern manual therapy*, Churchill Livingstone. 177.

Taylor J.R., Twomey L.T. (1985) Vertebral column development and its relation to adult pathology. *The Australian Journal of Physiotherapy* 31: 83-87

Taylor J.R., Twomey L.T. (1986) Age changes in the lumbar zygopophyseal joints: observations on structure and function. *Spine* 11: 739-745

Taylor J.R. (1990) The development and adult structure of lumbar intervertebral discs J. *Manual Medicine* 5: 43-47

Taylor T.K.F., Little K (1965) Intercellular matrix of the intervertebral disk in ageing and in prolapse. *Nature* 208: 384-386

Tertti M., Paajanen H., Laato M., Aho H., Komu M., Kormano M. (1991) Disc degeneration in magnetic resonance imaging, A comparative biochemical histologic, and radiologic study in cadaver spines. *Spine* 16: 629-634

Thompson J., Pearce R., Schechter M., Adams M., Tsang I., Bishop P. (1990) Preliminary evaluation of a scheme for grading the gross morphology of the human intervertebral disc. *Spine* 15: 411-415

Tibrewal S., Pearcy M. (1984) Lumbar intervertebral disc heights in normal subjects and patients with disc herniation. *Spine* 10: 452-454

Tsantrizos A., Ito K., Aebi M., Steffen T. (2007) Internal strains in healthy and degenerated lumbar intervertebral discs. *Spine* 30: 2129-2137

Twomey L.T., Taylor J.R. (1983) sagittal movements of the human lumbar vertebrae column: a quantitative study of the role of the posterior vertebral elements. *Archives of Physical Medicine and Rehabilitation* 64: 322-325

Urban J., Smith S., Fairbank J. (2004) Nutrition of the Intervertebral Disc. *Spine* 29: 2700-9. Review

Urban J., Maroudas A. (1980) The chemistry of the intervertebral disc in relation to its physiological function. *Clin Rheum Dis 6:* 51-76

Varamini A, Jam B. (2005) A need for a shared paradigm in the physical therapy classification and treatment of mechanical low back pain: *Orthopedic Division Review:* Mar/Apr. 40-43

Van der Veen A.J., Mullender M., Smit T., Kingma I., van Dieen J. (2005) Flow-related mechanics of the intervertebral disc: The validity of an in vitro model *Spine* 30: 1-12

van Tulder M., Malmivaara A., Esmail R., Koes B. (2000) Exercise therapy for low back pain: A systematic review within the framework of the cochrane collaboration back review group. *Spine* 25: 2784-2796

Videman T., Nurminen M. (2004) The occurrence of anular tears and their relation to lifetime back pain history: A cadaveric study using barium sulfate discography. *Spine* 29: 2668-2676

Weishaupt D., Zanetti M., Hodler J., Min K., Fuchs B., Pfirrmann C., Boos N. (2001) Painful lumbar disk derangement: Relevance of endplate abnormalities at MR imaging. *Radiology* 218: 420-427

White A.A., Panjabi M.M., Clinical Biomechanics of the Spine, (1978), J.B. Lippincott Co., Philadelphia.

Wilder D.G., Pope M.H., Frymoyer J.W. (1988) The biomechanics of lumbar disc herniation and the effect of overload and instability. *Journal of Spinal Disorder* 1: 16-32

Wilke H., Rohlmann F., Neidlinger-Wilke C., Werner K., Claes L., Kettler A. (2006) Validity and interobserver agreement of a new radiographic grading system for the intervertebral disc degeneration: Part 1 Lumbar spine. *Eur Spine* 15: 720-730

Wilke H., Neef P., Caimi M., Hoogland T., Claes L. (2006) New in vivo measurements of pressures in the intervertebral disc in daily life. *Spine* 1-22

Williams M.M., Hawley J.A., McKenzie R., van Wijmen P.M. (1991) A comparison of the effects of two sitting postures on back and referred pain. *Spine* 16: 1185-1191

Yahia L H, Rhalmi S, Isler M, (1992) Sensory innervation of human thoracolumbar fascia: an immunohistochemical study. *Acta Orthop Scandinav* 63: 195-197

Yingling V.R., Callaghan J.P., McGill S.M. (1997) Dynamic loading affects the mechanical properties and failure site of porcine spines. *Clinical Biomechanics* 12: 301-305

Yingling V.R., Callaghan J.P., McGill S.M. (1999) The Porcine cervical spine as a model of human lumbar spine: An anatomical, geometric, and functional comparison. *Journal of Spinal Disorders* 12: 415-423

Yoshizawa H., O'Brien J P., Thomas-Smith W., Trumper M. (1980) The neuropathology of intervertebral discs removed for low-back pain. *J Path* 132: 95-104

Yu, S., Haughton V.M., Sether L.A., Ho K.C.,Wagner M. (1989) Criteria for classifying normal and degenerated lumbar intervertebral disks. *Radiology* 170: 523-523

Yussen P.S, Swartz J (1993) The acute lumbar disc herniation: Imaging diagnosis. *Seminars in Ultrasound, CT and MRI* 14: 389-398

Ziv I., Maroudas C., Robin G., Maroudas A. (1993) Human facet cartilage: swelling and some physiochemical characteristics as a function of age: Part 2 age changes in some biophysical parameters of human facet joint cartilage. *Spine* 18: 136-146

Ziv I., Moskowitz R.W., Kraise I., Adler J.H., Maroudas A. (1992) Physiochemical properties of the aging and diabetic sand rat intervertebral disc. *J Orthop Res 10*: 205-210