Combined movement examination of the human lumbar spine

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This thesis is presented for the degree of
Doctor of Philosophy of
The University of Western Australia

School of Surgery
The University of Western Australia
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Declaration

This is to certify that this thesis does not incorporate, without acknowledgement, any material previously submitted for a degree or diploma from any university and that, to the best of my knowledge and belief, does not contain any material previously published or written by another person except where due reference is made in the text.

Signed

Name: Aubrey Paul Monie

Date: 11.11.16
Abstract

Background

The combined movement examination (CME) of the lumbar spine is an inexpensive, non-invasive clinical test for patients presenting with low back pain (LBP). First described by Brian Edwards nearly 40 years ago Edwards (1979), CME has since been recommended for use by clinicians to assess lumbar spine movement dysfunction because it has the added benefit of assessing the low back function in single, cardinal planes as well as combined planes of movement. With CME, the aim is to better understand how a patient’s LBP responds to mechanical stress and to use this information, along with other components of clinical assessment, to aid clinical reasoning. Although recommended for use in clinical practice, there were no reports of lumbar spine CME reliability nor normal reference range (NRR) values prior to these studies, nor were there reports of abnormal CME patterns suggestive of pathoanatomical diagnosis, nor evidence to suggest that CME facilitates clinical reasoning.

Purpose

The primary aim of this thesis was to determine if using 3-D computer-aided lumbar CME assists in identifying unique, atypical, patterns of movement, and predicting the likely structure(s) causing lumbar spine dysfunction in cases of mechanical LBP.

It was hypothesised that computer-aided lumbar CME is a valid and reliable method of measurement, that a NRR will show decreased range of motion (ROM) with age (and that females have a greater ROM than males in each age group), that lumbar CME data will approximate the NRR following specialist intervention, and that specific CME patterns will exist for the two most common pathoanatomical structures – the facet and intervertebral disc (IVD) and nerve root compression.
Methods

Four primary hypotheses were tested to answer fundamental concepts of lumbar CME including: validity and reliability, changes in age and gender, normal and abnormal movement patterns, and change to movement patterns post-intervention. Further, five secondary hypotheses were tested using pooled outcome data for the purpose of informing future research directions. These related to intervention, changes to low back movement, self-report outcomes, pathology and age group.

Lumbar CME was assessed with a non-invasive 3-D motion tracking system MotionStar™ [Ascension Technology, VT, USA] and dedicated software LabVIEW [National Instruments, TX, USA] to test validity and intra- and inter-session reliability, respectively.

In order to identify atypical CME movement patterns suggestive of pathoanatomical structure, it was necessary to identify a NRR for CME. A convenience sample from the general population – with an average of 19 asymptomatic subjects, for each gender and from each decade of life between 20-70 years of age – was assessed on one occasion to establish a NRR.

Symptomatic cases of mechanical LBP were recruited in collaboration with pain specialists and neurosurgical specialists from a tertiary teaching hospital. Quantitative and qualitative methods were used to report lumbar spine function. Quantitative data was derived from computer-aided lumbar CME. Quantitative and qualitative data was collected using three short questionnaires – the visual analogue scale for pain and stiffness (VASp and VASs), the short form health survey (SF-12) for assessing physical and mental health, and the Roland Morris low back pain and disability questionnaire (RMDQ) for assessing pain and function. These questionnaires form the core set of instruments used in much spine research and informed the investigators of the subject’s
LBP, stiffness, health, function and disability. A minimally clinically important difference (MCID) of 30% was adopted for all outcome measures to report a clinically significant change.

The results provided preliminary evidence to propose specific lumbar CME movement patterns suggestive of pathoanatomical diagnosis – facet, IVD and nerve root compression. Fifteen of the 35 cases were used to inform specific CME patterns for facet, IVD or nerve root compression due to their isolated single structure pathology at a single vertebral segment. Cases with multi-level and/or multi-structure lumbar pathologies were not used when considering specific CME patterns for single pathologies. This was done to control variables by minimising atypical movement due to other structures.

The ability for 13 post-graduate physiotherapy students to discern a clinical diagnosis and putative source of movement asymmetry (pathoanatomy) was examined over three time intervals with CME montages of three different, specific lumbar CME patterns. Cluster analysis was performed on pooled outcome data to test the five secondary hypotheses.

**Results**

Computer-aided CME had acceptable reliability when recording lumbar CME in normal subjects. Asymptomatic lumbar CME movement patterns were essentially symmetrical, had a flexion ROM at least 2.5 times greater than extension, and combined movement directions of approximately 80% that of the sagittal direction. The use of lumbar CME assisted in identifying atypical lumbar movement relative to an age and gender NRR in the majority of clinical cases. Preliminary evidence was identified for CME movement patterns in cases with pathoanatomical diagnoses – facet, IVD and nerve root compression.
Clinical reasoning was facilitated following an education-based program on discrete CME patterns and their putative pathoanatomical basis. Interquartile calculations using pooled outcome data showed that, on average, at 14 weeks post-intervention, CME ROM approached the NRR. Cluster analysis of pooled outcome data, displayed using hierarchical cluster dendrograms, indicated that flexion z-scores could separate cases with LBP due to disc pathology from cases with radiculopathy or facet pathology. Cluster analysis showed that all cases were similar in the relative change in ROM (%) post-intervention when compared by intervention. A sub-group of cases receiving neurosurgery clustered separately to pain management and other cases receiving neurosurgery when compared with their self-report outcomes. Analysis of all cases, by SF-12 mental component summary (MCS) scores, showed a clear separation which confirmed that there is a psychological influence to the way a patient perceives LBP. Age group clusters identified – 28-45 years (n=11), 48-58 years (n=11) and 61-70 years (n=13) – were likely to be due to biological age changes. While no dominant pathology was shown in the middle age group, the youngest contained predominantly radiculopathies, and the oldest included the majority of the bilateral facet joint cases. The results from the cluster analysis in this thesis were specific to the cohort of 35 cases with mechanical LBP and serve to direct future research. However, larger cohort numbers are required to further test these hypotheses.

**Conclusion**

The use of lumbar CME assisted in identifying atypical lumbar movement relative to an age and gender NRR. Preliminary evidence supports specific CME movement patterns in cases of mechanical LBP suggestive of pathoanatomical diagnosis, specifically facet joints, IVD and nerve root compression. Cluster analysis of outcome data suggests that future research should consider factors such as pathology, intervention outcomes,
mental health and age, and sub-groups within these, when investigating mechanical LBP.
Acknowledgements

Many people and organisations have contributed to the successful completion of this PhD thesis. Many thanks are extended to all the volunteers who participated in various studies comprising this project. Without the participation of the volunteers, this project would not have been possible.

My supervisors have supported me throughout the project. Each of the supervisors provided valuable input from their specific area of expertise. Their high standards and expectations provided me with the drive to be productive, maintain momentum and produce work to the best of my ability.

Thank you to my co-ordinating supervisor, Winthrop Professor Kevin Singer, who knew all of the research milestones, hazards and solutions well before they presented. His frequent questioning (often rhetorical) encouraged me to think outside the box, problem solve and develop solutions for the many administrative and scientific challenges presented throughout the candidature. His years of experience in supervising PhD students was reassuring.

Thank you to Dr Roger Price who was a true asset in the planning of the project, data interpretation, recruitment logistics and proof reading of manuscripts. His attention to detail and need to think-out a problem from multiple perspectives has taught me lessons for life.

Thank you to Professor Chris Lind who frequently took the time out of his busy schedule to consider the project’s requirements and answer my many questions. Professor Lind guided me through multiple avenues in a large tertiary hospital to improve participant invitation.
Thank you to the staff in the department of pain management and outpatient ward at Sir Charles Gairdner Hospital (SCGH). Their hospitality, assistance and readiness to volunteer as asymptomatic participants was much appreciated.

Thank you to the Human Research Ethics Committee (HREC) at The University of Western Australia and SCGH, respectively, for assessing and approving my research applications, and to the Graduate Research School staff for their support and guidance.

Thank you to Ray Smith (Scientific Programmer, School of Surgery, The University of Western Australia) for his time and effort to teach me how to use the laboratory equipment and how to process data.

Thanks to Chris Barrett, who was an understudy to Brian Edwards, the father of lumbar CME and a co-author on a publication, who took the time to introduce me to key concepts of 3-D motion tracking for CME of the lumbar spine.

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Thank you to my wife Kalindi for her support through the lengthy candidature and tolerance of my spikes in stress levels and frustration. Without your nod of approval and continual support, this thesis examination would not have been possible.

Last, but not least, thank you to my (now) nine year old daughter Sahlia and seven year old son Tyson. I am truly sorry for all of those times I turned down your requests to play. My grumpy reply while I sat at my desk was a reflection of my frustration. I hope that my passion and dedication to previous studies, this thesis project, and my work as a physiotherapist, has a positive influence on your education and work ethic.
Statement of originality

This thesis is presented for the degree of Doctor of Philosophy (PhD) at The University of Western Australia, through the School of Surgery, Centre for Musculoskeletal Studies.

The research project was developed by the author, in conjunction with supervisors – Winthrop Professor Kevin Singer, Director of the Centre for Musculoskeletal Studies, School of Surgery, The University of Western Australia; Dr Roger Price, Head of Department, Medical Technology and Physics, SCGH, and adjunct associate Professor, School of Physics, The University of Western Australia; and Professor Chris Lind, Consultant Neurosurgeon, SCGH, and Professor, School of Surgery, The University of Western Australia, who have also been involved in editing this thesis.

The recruitment of volunteers and management of this project were the sole responsibility of the author. Volunteers were examined at either The University of Western Australia or SCGH, according to the respective ethical standards and informed consent. The author conducted all aspects of recruitment and examination required for this research project. The author independently analysed the data in consultation with his supervisors and the occasional consultation with Mrs Laura Firth or Mr Marty Firth, statistical consultants for the Centre for Applied Statistics, The University of Western Australia.

The material compromising this thesis is the original work of the author towards the PhD degree, unless otherwise stated. This thesis has not been submitted, either in part or whole, for the award of any other degree at this or any other university.
Ethics

Approval to conduct the studies outlined in this thesis was granted by the HREC of The University of Western Australia. Approval was also granted by the HREC and Research Governance at SCGH, Perth Western Australia. All approved documentation is provided in Appendices I.1-I.15. All participants were provided with a plain language Patient Information Form (PIF, Appendix I.1-2, I.5-6) and a short demonstration of the low back movements required before providing written, informed consent on a Participant Consent Sheet (Appendix I.3-4, 7). Consent from the convenience sample of volunteers was given face-to-face, during personal communication with the author, before examination.

Funding

Funding for incidental expenses related to the studies in this thesis was provided by the Centre for Musculoskeletal Studies, School of Surgery, The University of Western Australia. In addition, equipment was provided by the Centre for Musculoskeletal Studies, and The Department of Medical Technology and Physics, at SCGH. Equipment alterations were made at no cost by the School of Electrical Engineering, The University of Western Australia, and the Department of Medical Technology and Physics, SCGH. The candidate was granted a post-graduate student scholarship by the Graduate Research and Scholarship office at The University of Western Australia (Appendix XV).

Mr Aubrey Paul Monie
November, 2016
Thesis publications

This thesis includes six peer-review journal publications, one text publication and one poster presentation at the Spine Society of Australia National Conference in 2013.

Journal publications


Professional issue


Text book chapter


Poster presentation

Monie A.P., Price RI and Singer KP. Non-invasive lumbar spine movement: Validation of the MotionStar 3-D electromagnetic tracking system and preliminary evidence. Spine Society of Australia, 24\textsuperscript{th} Annual Scientific Meeting. p. 81.
Declaration for thesis containing published work

The published co-authored work in Chapters 4 and 5, the submitted manuscript, Chapter 6 and Appendices VIII, IX, XI and XIII were completed during the course of the thesis investigation. Supervisors provided advice and suggestions for the manuscript prior to submission to journals. The work contained in these chapters was predominantly the author’s own at submission for publication (>75%) with any suggested edits to each paper being made by the author.

Aubrey Paul Monie
PhD Candidate

Winthrop Professor Kevin P. Singer
Coordinating Supervisor
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## Abbreviations and glossary

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<tr>
<td>AP</td>
<td>Anterior-posterior</td>
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<tr>
<td>BMI</td>
<td>Body mass index</td>
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<tr>
<td>CI</td>
<td>Confidence interval (95% CI)</td>
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<tr>
<td>CLBP</td>
<td>Chronic low back pain</td>
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<tr>
<td>CME</td>
<td>Combined movement examination</td>
</tr>
<tr>
<td>CV</td>
<td>Coefficient of variation (%CV)</td>
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<tr>
<td>CT</td>
<td>Computerised tomography</td>
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<tr>
<td>DDD</td>
<td>Degenerative disc disease</td>
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<tr>
<td>EwLSF</td>
<td>Extension combined with left side-flexion</td>
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<tr>
<td>EwRSF</td>
<td>Extension combined with right side-flexion</td>
</tr>
<tr>
<td>FwLSF</td>
<td>Flexion combined with left side-flexion</td>
</tr>
<tr>
<td>FwRSF</td>
<td>Flexion combined with right side-flexion</td>
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<tr>
<td>HREC</td>
<td>Human Research Ethics Committee</td>
</tr>
<tr>
<td>ICC</td>
<td>Intraclass correlation coefficient</td>
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<tr>
<td>IVD</td>
<td>Intervertebral disc</td>
</tr>
<tr>
<td>IVF</td>
<td>Intervertebral foramen</td>
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<tr>
<td>L4-5</td>
<td>Lumbar segment comprising of the 4(^{th}) and 5(^{th}) levels</td>
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<tr>
<td>LBP</td>
<td>Low back pain</td>
</tr>
<tr>
<td>L1</td>
<td>First lumbar vertebra</td>
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<tr>
<td>LSC</td>
<td>Least significant change</td>
</tr>
<tr>
<td>LSF</td>
<td>Left side-flexion</td>
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<tr>
<td>MRI</td>
<td>Magnetic resonance imaging</td>
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<tr>
<td>MCID</td>
<td>Minimal clinically importance difference</td>
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<tr>
<td>MCS</td>
<td>Mental component summary</td>
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<tr>
<td>MT</td>
<td>Manual therapy</td>
</tr>
<tr>
<td>NRR</td>
<td>Normal reference range</td>
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<tr>
<td>NSCLBP</td>
<td>Non-specific chronic low back pain</td>
</tr>
<tr>
<td>NSLBP</td>
<td>Non-specific low back pain</td>
</tr>
<tr>
<td>P1</td>
<td>Movement to the point that pain is initially felt</td>
</tr>
<tr>
<td>PA</td>
<td>Posterior-anterior</td>
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<tr>
<td>PCS</td>
<td>Physical component summary</td>
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<td>Posterior superior iliac spine</td>
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<tr>
<td>RMDQ</td>
<td>Roland Morris low back pain and disability questionnaire</td>
</tr>
<tr>
<td>R2</td>
<td>Movement is restricted/stopped due to resistance (not pain)</td>
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RSF  Right side-flexion
SCGH  Sir Charles Gairdner Hospital
SD  Standard deviation from the mean
SF  Side-flexion
SF-12  Short form health survey with 12 questions
VASp  Visual analogue scale for pain
VASs  Visual analogue scale for stiffness
z-score  Standard score for normally distributed data

**Glossary of definitions**

**Box plots:** Box plots are used in this thesis to summarise outcome data. The standard format uses horizontal lines which from the top represent the maximum, 25\textsuperscript{th} percentile, median, 75\textsuperscript{th} percentile and minimum values.

**Cardinal planes:** There are three cardinal planes of the human body. The sagittal plane divides the body into left and right – spine movement in the sagittal plane from a neutral position can be flexion (forward) or extension (backward). The coronal plane divides the body into anterior (dorsal) and posterior (ventral) – spinal movement in this plane is side-flexion (SF) to the left (LSF) or right (RSF). The transverse plane divides the body into superior and inferior – movement in this plane is rotation to the left or right.

**Combined movement:** Combined movement of a vertebral segment refers to movement in more than one cardinal plane, simultaneously. A spinal segment (or region) can be flexed or extended, and side-flexed to the left or right, simultaneously.

**Change scores:** Change scores for low back ROM, patient-reported LBP, low back stiffness, physical and mental health, and disability, compared to pre-intervention baseline scores, were employed. For lumbar ROM change scores, standard deviations (SD) were used by reporting the magnitude and direction of change in post-\textit{z}-score
values. For self-report questionnaires, change scores were reported using percentage (%).

**MCID:** This difference for all self-report outcome measures was set at \(\geq 30\%\) (Ostelo et al., 2008).

**Source structure:** The word source or source structure is used to describe the anatomical structure responsible for eliciting the sign (restricted CME movement) or reported symptom. For example, in the case of a painful left lumbar spine during lumbar extension, the left facet joint may be the source of the pain.

**Statistical significance:** Statistical significance was defined throughout this thesis with \(p<0.05\) representative of a meaningful difference.

Note that the terms computer-aided combined movement examination (CME) and CME are used interchangeably in this thesis. For clarification, the results and conclusions in Studies 1-3 are reported using data obtained by a computer-aided 3-D motion tracking system, accurate to 0.6° in the x, y and z axes. This was required to improve the objectivity of the CME studies. However, clinical CME is used without motion tracking apparatus (Maitland, 1997).
1. Introduction

1.1. The low back pain problem
Low back pain is a major public health system problem. It is one of the five most common reasons for physician consultation, with a lifetime prevalence as high as 85% (Joud et al., 2012). More than 90% of all episodes of back pain are probably attributable to mechanical causes, but the precise pathoanatomic lesion is rarely identifiable (White and Gordon, 1982). Establishing the source of pain is important if specific interventions for LBP are to be effective (Cornwall et al., 2006).

Chronic LBP (CLBP) is usually defined as back pain which persists beyond 3 months (Last and Hulbert, 2009, Chou, 2010). However, a distinction based solely on LBP duration may not be sufficient, as several factors including the overall effect of LBP, site, severity and disability could also be considered (Balagué et al., 2011). CLBP often includes psychological factors such as higher than usual levels of stress, depression and/or anxiety, with the potential for fear-avoidance and catastrophising behaviours (Besen et al., 2015, Deyo, 2015). Furthermore, social factors involving relationships, family, work and navigating the medico-legal system may amplify or prolong pain (Deyo, 2015).

1.2. Recommendations for the management of LBP
Authoritative groups such as the United Kingdom’s NICE agency (NICE, 2009), the American College of Physicians or American Pain Society (Chou et al., 2007), The Australian National Health and Medical Research Council (NHMRC, 2004b), as well as systematic reviews, for example Dagenais et al. (2010), provide best practice guidelines to assist in managing patients with LBP.
Minimum clinical assessment of LBP includes obtaining a medical history and a physical examination (NHMRC, 2004b, SAH, 2011). Physical examination incorporates a movement assessment which can include a multi-planar CME (Edwards, 1979, Barrett et al., 1999), soft tissue palpation, passive movement examination and neurological screening if implicated. Together, the history and physical examination should result in a provisional diagnosis (Maitland, 1997, Sahrmann and Van Dillen, 2015).

CLBP is increasingly recognised as being more than simply anatomical (Deyo, 2015). A biopsychosocial model with a multidisciplinary approach is therefore widely accepted and recommended for use (Waddell, 1992, Deyo, 2015). However, it would be a mistake to assume that psychosocial factors such as depression and work dissatisfaction are important contributors to LBP. Psychosocial factors influence people’s response to pain, rather than being a cause of pain (Adams et al., 2002, p.1). This thesis investigation focusses on lumbar pathoanatomical factors contributing to LBP and how these affect CME.

Assessing lumbar spine movement in the clinical setting to investigate dysfunction and to monitor changes in spinal movement characteristics of individuals over time is routine clinical practice (Maitland, 1997, Lyle et al., 2005, Ha et al., 2013, Laird et al., 2014). These movements are often in the cardinal planes – sagittal, coronal and transverse. Single plane movements are often unrepresentative of the actual movements of the lumbar spine so, while still important, have limited value in assessing lumbar function (Pearcy and Hindle, 1989). Every day, asymptomatic lumbar spine movements are fluid, often using multiple planes of movement simultaneously (Figure 1.1). The reasons for this are both functional and anatomical (Little et al., 2007). One examination method, originally described by Edwards (1979), which assesses both planar and combined plane (physiological) positions is the CME. The CME sequentially examines
the patient’s ability to actively side-flex the lumbar spine while in flexed, neutral and extended positions.

Figure 1.1. Examples of activities in everyday life.
The functional movements illustrated use movements in multiple planes, requiring coupled movement of the lumbar spine, with combinations of flexion or extension with side-flexion and rotation.

Recommendations for assessment and treatment of LBP have not changed noticeably in the last 2 decades. An assessment tool which informs clinical reasoning and directs the clinician towards an evidence-based provisional diagnosis would putatively result in fewer patients in the non-specific CLBP sub-group (NSCLBP) improve treatment approaches, speed recovery and ultimately save time and resources for both the patient and the health system.
1.3. This thesis investigation

The genesis of this thesis investigation is the pioneering work of Perth physiotherapist, Brian C. Edwards (1942-2003) who first described and recommended the use of a CME for the lumbar spine (Edwards, 1979). The studies comprising this thesis will test if the CME is a useful clinical tool. If the examination proves to be reliable, it could be supported for greater use in the clinical setting as an inexpensive, non-invasive method for assessing, informing a provisional diagnosis, and reassessing mechanical low back conditions.

The following primary hypotheses were made:

1. Computer-aided CME is a valid and reliable test for measuring lumbar ROM in single and combined planes of movement.

2. Normal CME will reduce in ROM with age, and females will have slightly more ROM than males in each decade of life from 20-69 years.

3. CME changes after specialist intervention approaching the NRR.

4. Preliminary CME patterns are will be identified which are specific to facet pathology, IVD pathology and nerve root compression.

The results from these studies may be used to formulate additional hypotheses to further the understanding of CME and its use in clinical assessment of mechanical LBP. An additional five secondary hypotheses were made to test with outcome data and cluster analysis. These were:

1. Patients presenting with low back dysfunction from different pathoanatomical structures will result in clusters by pathoanatomy when analysing data for
flexion depending on the location of the lumbar pathology relative to the sagittal axis of rotation (Appendix VI, Figure 5).

2. Surgery cases will result in greater improvements in self-report outcome measures compared with cases receiving pain management intervention.

3. Surgery cases will achieve greater improvements in ROM compared to pain management intervention, primarily because the restricting anatomical structure is removed.

4. A review of the literature led to the hypothesis that the psychological state of patients will have an effect on self-report outcome data. Those with low SF-12 MCS will score lower than those with average SF-12 MCS scores on the self-report outcome measures.

5. Older subjects, >50 years of age, will make up the majority of the radiculopathies due to the increased likelihood of age-related degenerative disc disease (DDD) and facet degeneration leading to degenerative canal or intervertebral foramina (IVF) stenosis.

Chapter 2 reviews the relevant literature by describing the LBP problem and its prevalence in the community, reporting the key structures implicated in mechanical LBP, the recommended guidelines for clinical assessment, and finally the literature reporting CME of the lumbar spine.

Two pilot studies were completed to validate a tri-planar goniometer, which in turn was used to validate the MotionStar 3-D tracking system (Appendix II) and to test the reliability of 3-D motion tracking of lumbar CME (Appendix III).
Chapter 3 of this thesis describes the methods employed in each study, followed by scientific investigations in Chapters 4-8.

Chapter 4 reports the reliability of computer-aided 3-D motion tracking of lumbar CME, a normal reference range and two case studies to illustrate the clinical utility of CME. Chapter 5 and 6 report preliminary evidence for specific CME movement patterns in cases receiving pain management and neurosurgical intervention, respectively. In each study, three cases with different pathoanatomical presentations are used to illustrate CME patterns. Chapter 7 reports the clinical use of lumbar CME without 3-D tracking data, and how clinical reasoning may be facilitated using the proposed CME movement patterns suggestive of pathoanatomy. Chapter 8 uses pooled data from chapters 5 and 6 to test the five secondary hypotheses.

Chapter 9 discusses the results of the studies in the light of the hypotheses, addresses the major themes of the thesis, acknowledges the limitations in both methods and results, and suggests the clinical implications of the results. A final chapter reports the conclusions which can be drawn from this thesis and recommends avenues for future research to continue the investigation of CME as a non-invasive, inexpensive, clinical assessment for mechanical LBP.

1.4. Summary
This thesis-based evaluation will systematically test the utility of the CME as a diagnostic tool for specific lumbar spine pathology following a staged series of preliminary investigations. Characteristic CME movement patterns will be examined for facet joint, IVD and nerve root pathologies respectively.
2. Review of the literature

2.1. Overview
The literature review in this chapter is tailored to relate specifically to mechanical LBP and/or low back hypomobility due to lumbar pathology, clinical assessment of the symptomatic low back, including CME, and speculating the likely pathoanatomical diagnosis of mechanical LBP. Mechanical LBP includes lumbar strain, symptomatic IVD, with or without radiculopathy, facet joint osteoarthritis and spinal stenosis, which collectively account for ≥95% of cases of LBP (Borenstein, 2013). It is not the intention to describe the details of lumbar spine neuroanatomy LBP from non-mechanical factors such as psychosocial factors, interventions or outcomes.

2.2. The prevalence and incidence of LBP
Musculoskeletal conditions rank in the top five of Australia’s total burden of disease groups (AIHW, 2016a) (Figure 2.1). Of the total Australian population in 2011-12, 13.6%, three million people, reported having low back problems which accounted for a quarter of all long-term conditions due to injury (ABS, 2012).

LBP is one of the most costly musculoskeletal pain syndromes of modern society and is the most common cause of absence from work, with up to 84% of people having LBP at some time in their life (Freburger et al., 2009, Balagué et al., 2011) and with the highest prevalence amongst women and those aged 40-80 years (Manchikanti et al., 2014). In Australia, national health survey statistics for 2011-12 show that there are almost equal amounts of people working with and without back problems. The prevalence of benign LBP appears to decrease with age after a peak in the sixth decade; however, the incidence of severe LBP continues to increase with age (Dionne et al., 2006) (Figure
Recent research shows that LBP causes more years lived with disability than any other health condition (Kamper et al., 2015).

Figure 2.1. Top five total burden of disease in Australia, 2011.
A total of 12% of the Australian population reported back problems according to 2011-12 self-report estimates, ranking it amongst the top five total burden of disease (AIHW, 2016b).

Figure 2.2. Prevalence of all back problems in Australia, by age.

Despite these statistics, there has been little progress over the past 15-30 years in making an accurate diagnosis and providing improved treatment strategies for LBP.
patients (Buchbinder et al., 2010, Witenko et al., 2014). Indeed, Lee and Vasudevan (2012) report that the prevalence of LBP has been increasing over the last decade despite increasing costs associated with LBP patients, low back surgery and the pursuit of improved LBP management.

Some 40-70% of those experiencing LBP seek health care (Joud et al., 2012), with the majority being labelled non-specific LBP (NSLBP). NSLBP is described as LBP without an identifiable pathoanatomical source, and has a lifetime prevalence of 80% (Smith et al., 2014). Approximately 90% of acute episodes of NSLBP recover in 6 weeks; however, the majority of people who experience their first episode of NSLBP will develop LBP lasting longer than 1 year (Hestbaek et al., 2003).

An estimated 23% of acute LBP sufferers develop CLBP, with 11-12% of the population being disabled by it (Balagué et al., 2011). CLBP is defined as back pain which has lasted greater than 12 weeks (Chou, 2010). The CLBP sub-group of patients utilise the majority of resources, and current recommendations are for a multimodal, multidisciplinary, biopsychosocial approach (O'Connor et al., 2015). Germon and Hobart (2015) argue that management of LBP has lost sight of the basic clinical tenet of making a diagnosis, perhaps because it is very difficult. They state that in the United Kingdom, and many other countries, some guidelines recommend, and even discourage, attempts to make a diagnosis. Unfortunately, the consequence of this illogical approach is that many people receive non-specific advice, and non-specific treatment (Germon and Hobart, 2015).

Evidence-based approaches for the assessment and management of CLBP in primary care settings have been investigated and are recommended (Chou et al., 2007, NICE, 2009, SAH, 2011). The quest for the ingredients for economically viable and effective multidisciplinary management of CLBP continues (Clark et al., 2016). A reliable
A diagnostic tool for assessing low back problems would speed clinical decision making, leave fewer patients in the NSCLBP sub-group, improve treatment approaches, speed recovery, and ultimately save time and resources for both the patient and the health system.

2.3. Pathoanatomical structures contributing to mechanical LBP
Establishing the pathoanatomical diagnosis of mechanical LBP is important if specific interventions are to be developed (Cornwall et al., 2006). However, the literature indicates that history and clinical examination are of dubious value in the determination of a pathoanatomical diagnosis (Hutson and Ward, 2016). Unfortunately, of those patients who present to the health practitioner with LBP, approximately 80-95% are grouped into a NSCLBP sub-group. NSCLBP means that the source structure producing the pain has not been identified, and their LBP has persisted beyond 12 weeks, respectively (Ehrlich, 2003, O'Sullivan, 2005, ABS, 2013). Without an identifiable source structure, treatment directed specifically to the source of the pathology is not possible, often resulting in pragmatic management (Ehrlich, 2003).

A study by Kent and Keating (2004) reported that of 651 Australian primary care clinicians, 93% treat their NSCLBP patients differently, depending on the individual patient’s signs and symptoms. This is despite the lack of evidence for different patient sub-group characteristics or management. Often surgery for CLBP is offered as a last measure, yet this is usually costly and often fails to provide permanent relief (Ehrlich, 2003, Atkinson and Zacest, 2016). For example, results from trials comparing intensive rehabilitation and spinal fusion surgery have shown similar clinical improvement in short-term and long-term follow-up (Kent and Keating, 2004).
Virtually every structure in the lumbar spine has been implicated as a potential source of LBP, including the thoracolumbar fascia, ligaments, muscles (Cornwall et al., 2006) and kissing spinous processes also known as Baastrup’s disease (Hutson and Ward, 2016). Numerous anatomical innervation studies, provocative diagnostic stimulation and blocks have been used over the decades to report pain and common referral patterns from various lumbar structures (Bogduk, 1985, Groen et al., 1987, Schwarzer et al., 1994, Fukui et al., 1997, O’Neill et al., 2002, Edgar, 2007, Laplante et al., 2012, Bogduk et al., 2013). Studies report that in the primary care setting the specific disease or spinal abnormality cannot be reliably identified and attempts to identify the pathoanatomical structure causing LBP have not been validated (Koes et al., 2006, Chou et al., 2007).

Other studies show that specialist diagnostic procedures, such as discography, facet blocks and intra-articular injections, can reveal the source of LBP in 90% of patients (DePalma et al., 2011, Laplante et al., 2012). The precise location of a patient’s LBP can predict its source (Laplante et al., 2012). Recognising pain referral patterns characteristic for IVD and facet joints may sharpen the evaluating clinician’s diagnostic skills (Laplante et al., 2012). DePalma et al. (2011) state that the belief that 90% of LBP cannot be diagnosed is dated and the converse is in fact true.

Sinister conditions may also masquerade as LBP. Side-effects of medication, vascular pathology or neoplasms may mimic musculoskeletal conditions (Greenhalgh and Selfe, 2015). Tumours and infections can affect any of the structures in the low back. However, these are uncommon as a cause of LBP in primary care – the prevalence of tumours in LBP is 0.7% and that of infection 0.01% – and most patients have some other disorder that is the basis of their LBP (Adams et al., 2002).
More than 90% of all episodes of LBP are probably attributable to mechanical causes; however, as mentioned, the precise pathoanatomic lesion is rarely identifiable (White and Gordon, 1982). The two structures in the lumbar spine, identified as the source of the majority of LBP, are the facet joints and IVD (DePalma et al., 2011, Laplante et al., 2012) (Figure 2.3). Osseo-ligamentous tissues and the disc anulus are the primary contributors to spinal stiffness (Cunningham et al., 2007, Little et al., 2007). After considering the prevalence of anatomical structures implicated in CLBP, Hutson and Ward (2016) state that, for every 10 patients seen clinically with CLBP, on average four will have discogenic pain, another four will have facet pain, and the remaining two cases will most likely be sacroiliac-related.
In the older aged population, point prevalence estimates suggest that up to 20% of individuals are affected by degenerative lumbar conditions. Spinal stenosis in this population has become the leading cause of spinal surgery (Mannion et al., 2010). With lumbar segment degeneration and spinal canal or IVF narrowing, the exiting nerve root is susceptible to compression (Steurer et al., 2010, van der Windt et al., 2010) (Figure 2.3. Axial cryosection through L3-4 depicting the IVD, paired zygapophysial (facet) joints, and pre-vertebral and post-vertebral musculature. [D=IVD; Z=zygapophyseal joints; P=psoas muscles; M=multifidus muscles]. (Images used with permission from: Dr Groen. University of Utrecht. The Netherlands).
This nerve root compression, causing radiculopathy, is prevalent in 3-5% of the population (Tarulli and Raynor, 2007).

Prevalence figures for the structure responsible for producing pain, the source structure, are therefore important in assessing the utility of a clinical test in the diagnosis of CLBP, and can be used to guide clinical decision making (Hutson and Ward, 2016).

2.4. Clinical assessment of LBP

Recommended clinical assessment of LBP includes obtaining a medical history and a physical examination (NHMRC, 2004b, SAH, 2011), which includes active movements of the low back, soft tissue palpation, passive movement examination and neurological screening if implicated. Bauer et al. (2016) report that on average ROM tests are more reliable than tests which consider how the patient moves and controls their low back movement or proprioceptive tests. This study’s statistical analysis was questioned by Sabour (2016), stating that descriptive statistics cannot substitute clinical judgement,
and an individualised approach should be used to assess reproducibility. However, clinicians use physical examination procedures routinely when making clinical decisions, yet limited data, such as age- and gender-matched reference ranges for ROM and speed for common assessment tasks such as planar or combined lumbar movement, sit-to-stand, climbing stairs, walking and running, are available to guide the decision making process (Lyle et al., 2005). Together, the history and physical assessment should result in a provisional diagnosis (Chou et al., 2007). Progressing to treatment without a thorough assessment or without correlating symptoms with examination findings increases the prospect of misdiagnosis and mismanagement.

Useful clinical clues may be provided by various signs and symptoms. For example, for patients >65 years, pain not increased by straining and not worsened with lumbar flexion may suggest facet joint pain (Hutson and Ward, 2016). Discogenic LBP is often described as a band-like distribution, worse in the morning, worse with strain, and aggravated by lumbar flexion (Hutson and Ward, 2016). For disc herniation, the highest incidence is between the ages 30-40 years, and clinical clues include LBP, leg pain, deep tendon reflex loss and muscle weakness (Deyo et al., 1990). Where indicated, focussed spine imaging assists with diagnosis and staging interventions (Deyo et al., 2014), with the accepted caveat that pain and symptoms do not correlate well with imaging (Maus, 2010).

On average, people with LBP have reduced lumbar ROM (Laird et al., 2014). McGregor et al. (1997) compared the movement of 138 subjects with previously published normal data, and reported that patients with lumbar stenosis, disc prolapse and DDD are often hypomobile. However, they did not report individual differences between pathologies, stating that motion characteristics may not be very sensitive in categorising individual patients, limiting clinical usefulness for diagnostic purposes.
Despite efforts to assess and diagnose accurately in the clinical setting, three systematic reviews report there is no physical examination technique, laboratory test nor imaging modality that can precisely identify the pathoanatomical reason for LBP, distinguish the culprit from other potential structures, or predict response to therapeutic intervention (Hancock et al., 2007, Sehgal et al., 2007, Falco et al., 2012). More recently, Dewitte et al. (2015) stated that there is no persuasive scientific evidence currently available to underline the discriminative value of clinical tests for LBP. Therefore where the condition is complex, not responding to treatment, or where symptoms masquerade as more sinister pathology, referral to appropriate health professionals is encouraged (Greenhalgh and Selfe, 2015).

2.4.1. Biomechanics of a lumbar movement examination
During sagittal plane movement the axis of rotation is not fixed. As a superior vertebra flexes forward on an inferior vertebra, its axis of rotation moves slightly anterior in the nucleus pulposis. During lumbar extension, the axis of rotation moves slightly posteriorly, relative to neutral standing (Adams et al., 2002). The normal spine has a lordotic curve in the lumbar region. There are variations on the degree of the normal curve which distributes forces across the spinal column. It is disruption of this equilibrium by pathological processes or, in most cases aging, that results in deformity (Roussouly and Nnadi, 2010).

2.4.2. Lumbar extension
The neural arch resists approximately 60-70% of the applied bending moment when extended to its elastic limit, resulting in facet joint loading. Beyond this, damage can be detected after an average of 5° (range 3-8°) into the elastic limit (Adams et al., 2002). The rest of the resistance to lumbar extension comes from the IVD and the anterior longitudinal ligament (Adams et al., 2002). Concentrations of compressive stress appear
in the posterior anulus after just 2° of lumbar extension (Adams et al., 1994). Functional movements which are likely to increase loads towards the end of available lumbar extension include standing, walking, running, ceiling painting, hanging clothes on a line, shaving, washing hair, high jumping, fast bowling at cricket and playing overhead sports such as tennis and volleyball (Bible et al., 2010, Merlino and Perisa, 2012).

2.4.3. Lumbar flexion
In a normal lumbar segment, resistance to early flexion is provided by the IVD and ligamentum flavum. Between half flexion and full flexion, the IVD resists more than the posterior ligaments due to anterior IVD compression and posterior IVD tension. Ligament tension increases rapidly in the last few degrees of flexion, so that in full flexion the facet joint capsules, posterior IVD and posterior ligaments share the resistance in decreasing degrees (Adams et al., 2002). The similarity in lumbar flexion between cadaveric and living joints suggests that the ROM is influenced strongly by mechanical properties of the segment rather than muscle activity and length (Adams et al., 2002). Functional tasks which approximate end-of-range flexion include lifting from a squat, putting shoes on, gardening, sitting-to-stand and sports such as gymnastics, lawn bowls, cycling and rowing (Bible et al., 2010).

2.4.4. Coupled movements of the lumbar spine segments
Coupled motion in the lumbar spine is the consistent association of one motion (SF or rotation) about the other motion. Side-flexion of the lumbar spine has not been studied to the same depth as sagittal plane movement. Symmetry suggests that most of the spine’s resistance to SF comes from compression of the ipsilateral facet and IVD and stretching of the contralateral facet and IVD (Adams et al., 2002). In an original contribution to the study of mechanics of the spine in the context of scoliosis, Lovett (1903) states that SF and rotation occur together. With every rotational movement, there
is also SF, vice versa. Lovett concludes that there are only three fundamental types of movement – flexion, extension and SF with rotation (Figure 2.5).

Coupled movement of a lumbar segment

Figure 2.5. Illustration of a lumbar segment in LSF combined with left rotation.

Legaspi and Edmond (2007), in a systematic review of 24 articles on lumbar coupled motion, concluded that there was little agreement across the articles as to the specific characteristics of coupled motion. Further, they caution the physical therapist to this inconsistency when applying coupled motion concepts during clinical assessment and treatment of LBP patients. The manual therapist, using techniques which require patient positioning in lumbar SF or rotation, cannot assume that one rule fits all, but must consider each patient as an individual case. The risk of assuming a coupled motion, or normal facet morphology, is increased when attempting high velocity, low amplitude manipulative techniques towards end-of-range lumbar positions.

2.5. Active movement examination of the lumbar spine

Physical examination incorporates an active movement assessment which can be planar (Pearcy and Hindle, 1989, Madson et al., 1999, Troke et al., 2005), multi-planar (Edwards, 1979, Brown, 1988, Barrett, 1995) or functionally specific such as rolling in bed or sitting-to-stand (Bible et al., 2010, Pfingsten et al., 2014). A planar movement
examination of the lumbar spine involves asking the patient to move their low back in the three cardinal planes – sagittal, coronal and transverse (Figure 2.6A). A multi-planar test combines two or three planes of movement. For example, during a CME the position of extension and SF is used (Figure 2.6B). A quadrant test (also known as Kemp’s test) attempts to achieve movement in all three planes, sequentially and cumulatively, such as flexion, before adding SF, followed by rotation. Functional tests are specific to the patient’s lifestyle and may include everyday tasks such as sitting-to-stand, rolling in bed or putting on lace-up shoes, as well as patient-specific tasks such as kicking a ball, diving or hitting a golf ball (Figure 2.6C).

CME is considered more reflective of functional movements of the lumbar spine than planar movements (Edwards, 1979). Maitland (1997) suggested that clinically useful information is provided by assessing the quality, range and pain response to combinations of flexion and SF/rotation in the low lumbar spine. However, examination of the patient by individually stressing the spine in all three planes of movement simultaneously, such as the Quadrant test, is likely to be difficult for even the asymptomatic spine, and makes interpretation of the pathoanatomy for symptomatic

Figure 2.6. Planar movement of the lumbar spine.
(A) The three cardinal planes used for a planar movement examination. (B) A CME using two planes – extension plus LSF. (C) An example of a patient-specific functional movement examination, hitting a golf ball.
patients increasingly difficult. Combining rotation as a third plane of movement is more difficult to perform for a patient, difficult to control as an examiner, and incorporates a more passive component to the active test (Maitland, 1997). Interestingly, a recent systematic review used five studies to conclude that the Quadrant test (combining extension, SF and rotation) for the lumbar spine has limited evidence to support its use and has poor diagnostic value (Stuber et al., 2014). This is partly due to the lack of good quality studies meeting inclusion criteria, varied examination methods and generally low reported specificity. Specific functional tests, such as activities of daily living and work-related tasks, are useful and recommended as part of a structured assessment; however, they have an inherent problem to differentiate patients with LBP from normal, and problems with inter-rater reliability (Pfingsten et al., 2014). In addition, patient-specific function will vary between genders, age groups and lifestyles.

2.5.1. Variation in lumbar movement with age and gender
In a study of 104 asymptomatic volunteers, lumbar spine ROM decreased with age and no significant differences were reported in relation to gender (Dvořák et al., 1995). In a study by McGill et al. (1999), 12 healthy elderly volunteers (mean age 69 years) showed decreased sagittal and coronal plane movement with age but not axial rotation. In a more recent study of 323 asymptomatic volunteers aged 25-75 years, Dreischarf et al. (2014) report a 20% reduction of lordosis, 12% loss of flexion, and 31% decrease in extension ROM between the age groups 20-29 years and those older than 50 years, respectively.

2.5.2. CME of the lumbar spine
There is limited literature available on CME of the lumbar spine or the use of the MotionStar tracking device for lumbar spine examination. A review of the literature describing 3-D motion tracking and CME was performed using internet-based search
tools (Ovid Medline, Web of Knowledge and CINAHL) which identified approximately 60 articles reporting 3-D spinal tracking systems, lumbar spine movements, combined movements, lumbar diagnostics, lumbar assessment, lumbar mechanics, spinal coupled movements, or more specifically CME.

A hand search of key papers and texts by leading authorities was also conducted. Studies from the late 1990s, and later, were reviewed. Many 3-D lumbar spine tracking studies were published in the late 1990s when accurate and reliable motion tracking technology became available. Of these, very few investigations have specifically used 3-D motion tracking systems to record lumbar spine movements during CME. Eight key original articles were used in this review of lumbar CME.

The CME examines patients by asking them to move from a comfortable standing position to flexion and extension. At each of the three positions (neutral, flexion and extension) the patient is guided by cues into LSF and RSF (Figure 2.7). Edwards (1979), Maitland (1997), (Brown, 1988), (Barrett, 1995) and McCarthy (2010) accurately describe the CME procedure and discuss its clinical significance in terms of reduced ROM and treatment choices; however, the reliability and diagnostic value of the examination is not reported. Barrett et al. (1999) reported on lumbar CME using the 3-D Fastrak™ [Polhemus, Vermont, USA] motion tracking system. Their study assessed the intra-examiner reliability of the CME in asymptomatic and LBP subjects. They showed acceptable intra-examiner reliability in detecting reduced spinal movement in LBP subjects and recognised that symptomatic spines have reduced spinal movement compared to asymptomatic spines. There was no follow-up study demonstrating exactly what these differences are, or why, and whether or not invasive pain intervention or neurosurgery normalises the lumbar spine movement patterns.
Several studies report evidence to show that patients with LBP show decreased lumbar CME (Barrett, 1995, Hidalgo et al., 2014a, Hidalgo et al., 2014b, Hidalgo, 2015). Brown (1988) concluded that changes occur to normal patterns of CME when pathology is present, typified by signs and symptoms. Brown also suggests that the likely causative structure at fault may be speculated on from its anatomical location in the motion segment, and that every condition will have a different pattern in variance to that found in the norm. However, to designate abnormal lumbar ROM presupposes knowledge of normal ROM (McGregor et al., 1997). A review of the literature suggests that normal lumbar CME is not defined for different age groups and gender, and causative structures and their resulting abnormal CME patterns are not described.
Brown also states that the amount of change to CME movement patterns from the normal appear to correspond with the severity, site and nature of LBP, speculating that CME allows greater insight into the ways of reasoning and forming a clinical diagnosis. Additionally, Brown states that the risk of making a wrong treatment choice is greatly reduced by analysing a patient’s CME pattern. Lumbar CME and passive pain provocative tests were also used by Hidalgo et al. (2014a) to report acceptable inter-examiner reliability and good validity in identifying the main provocative movement pattern and lumbar segmental level, and also showed this combined assessment helped to distinguish between participants with and without LBP. However, there was no speculation as to the pathoanatomical diagnosis responsible for LBP. Hidalgo et al. (2014b) report that ROM and speed data provide useful information to discriminate between asymptomatic cases and cases with LBP. They conclude that targeting specific interventions, such as manual therapy (MT), might be better suited to patients with specific movement characteristics. However, in all of these studies, quantitative data on how lumbar CME is affected, the effects of intervention on lumbar CME, and proposed pathoanatomical structures have not been reported.

From this review it is clear that apart from the thesis investigation by Barrett 1997, there has been no comprehensive study describing a NRR for lumbar spine CME movement patterns, nor any study that predicts the diagnosis for patients with specific types of LBP presentations using CME, for example facet joint pathology, IVD pathology and nerve root compression.

2.5.3. Measuring ROM of the lumbar spine
Invasive techniques have been used in an attempt to accurately measure lumbar spine movements (Gregersen and Lucas, 1967, Hanley et al., 1976). With the advance of technology, non-invasive methods of kinematic measurement of the human lumbar
spine using 3-D tracking systems with skin mounted sensors have been shown to be valid methods of measuring lumbar kinematics (Barrett, 1995, Mieritz et al., 2012, Ha et al., 2013, Hidalgo et al., 2014b). However, measurement of spine movement using skin-mounted sensors in rotation includes large movements of the skin and thorax, and overestimate movements measured by radiographs (Adams et al., 2002).

As a clinical outcome measure, ROM has the advantage of being more objective and quantifiable than measures of symptoms such as pain scores (McGregor et al., 1997). Lumbar speed measures are a complex neuromuscular synergistic co-ordination, requiring motivation, skill, strength and flexibility, and metabolic support. Speed is sensitive to any of these factors, and not specific to the cause of impairment (McGregor et al., 1997). A systematic review of 3-D motion tracking of the lumbar spine by Mieritz et al. (2012) concludes that most 3-D tracking systems used for lumbar motion measures may be considered reliable enough to be used for research purposes on the group level, but it is uncertain if they can be used for assessing the individual patient.

2.5.4. Statistics relevant for movement analysis

Descriptive statistics are used in studies reporting 3-D motion tracking of the lumbar spine for validation purposes (Mannion and Troke, 1999, Sutherland et al., 2008, Ha et al., 2013), reliability studies (Barrett et al., 1999, Larivièrè et al., 2014), quantifying movement (Cohn et al., 1989, Gatton and Pearcy, 1999, Van Herp et al., 2000) and cohort comparisons (Barrett et al., 1999, Vogt et al., 2001, Hidalgo et al., 2014b).

A systematic review by Mieritz et al. (2012), investigated reliability and error of 3-D lumbar motion tracking. They found no difference in the level of reproducibility between LBP and no LBP groups, and recommend that future reproducibility studies examine kinematic parameters, such as coupled motion or smoothness of movement in addition to ROM, to derive measurement error and to provide estimates of acceptable
intra- and inter-rater reliability. Additionally, this review suggested that Pearson correlation coefficient is not appropriate for reliability studies. An intraclass correlation coefficient (ICC) value above 0.7 is considered to demonstrate acceptable reliability to be used for human research purposes (Mieritz et al., 2012).

In addition, least significant change (LSC) or MCID are descriptive statistics employed to determine successful clinical outcomes or meaningful change (Carragee and Cheng, 2010, Nelson et al., 2010). For example, if lumbar flexion for patient with LBP was measured as 30°, and a LSC for lumbar flexion is calculated as 7°, then a retest ROM of 35° would be considered a clinically insignificant improvement. However, a retest ROM of 48° would be considered a significant improvement, potentially due to an intervention aimed at increasing lumbar flexion ROM. Further, Ostelo et al. (2008) propose a MCID to be 30% or more for several self-report LBP questionnaires. A clinically significant improvement may be higher for certain interventions depending on multiple factors including risk, cost and psychosocial factors (Carragee and Cheng, 2010).

Judging whether a particular value for a variable is typical or atypical compared to other values of a normally distributed population can be facilitated by converting raw scores such as weight, height or ROM into z-scores. By calculating a z-score an individual’s score can be compared to the population using units of SD. One great advantage of z-scores is that they facilitate comparison of scores from populations with different means and SDs (Boslaugh, 2012). An individual’s performance, such as ROM, can be expressed relative to their age- and gender-matched NRR, indicating the magnitude of their movement direction in SD (+ or -) from the NRR mean (Bonnick and Lewis, 2013). For ease of comparison, 68% of the normal distribution lies within plus or minus
one \((\pm 1)\) SD of the mean \((-1 \leq z < +1)\), and 95\% lies within \(\pm 2\) SD of the mean \((-2 < z \leq +2)\). A z-score = \((\text{individual’s score} - \text{population mean})/\text{SD}\).

Cluster analysis can be used with multivariate data to discover natural similarities amongst patient populations. The hierarchical cluster analysis, using Euclidean distances, are sensitive to differences in elevation as well as profile shape (Blashfield, 1980). Using cluster analysis, data related to gender, height and weight have been identified as factors related to spinal posture (Roussouly et al., 2005, Smith et al., 2008). The use of cluster analysis has also shown that patients with spinal pain can be grouped into these three clusters (Fanciullo et al., 2003). Clusters can be used to guide future research and investigate the benefits of specific therapies (Fanciullo et al., 2003).

### 2.5.5. Learning clinical reasoning

In the clinical setting, even with new knowledge, a clinician is encouraged to analyse and consider the patient’s assessment findings. Two common models for clinical reasoning include hypothetico-deductive and pattern recognition. Hypothetico-deductive reasoning starts by hypothesising the cause of the presenting condition, followed by a process of elimination through assessment and reassessment. Pattern recognition requires the clinician to make decisions based on previous, similar presentations.

The best indicator of correct diagnosis and improved patient management is the quality of the differential diagnosis concerning the cause or nature of the patient’s condition (Jones, 1992) and the response to treatment. This involves rapid, non-verbal, intuitive cognition, a process used by the experienced clinician (Elstein, 2009, Langridge et al., 2015). Although several possible reasons could easily be proposed as the source of a patient’s condition, it is reported that between three and five diagnostic hypotheses are
common, and this may be linked to various factors, including short-term memory (Elstein, 2009).

By slowing down and verbalising clinical reasoning, experts can teach learners. When learners do the same, their reasoning can be assessed and improved with feedback (Pinnock and Welch, 2014). Competence in clinical reasoning is acquired by practising under supervision with effective feedback, and trainees can learn clinical reasoning effectively if teachers provide guidance in making diagnostic decisions (Pinnock and Welch, 2014).

2.6. Summary
This chapter has reviewed the literature relevant to LBP prevalence, pathoanatomical diagnosis, structures limiting lumbar spine movement, clinical assessment of LBP, and CME of the lumbar spine. The emphasis was on movements specific to CME and proposed structures which may limit symptomatic and asymptomatic lumbar spine CME.

From the evidence reported in this literature review, it is reasonable to speculate on pathoanatomical structures leading to reduced CME. It is also possible to hypothesise CME movement patterns specific to pathoanatomy such as facet joint pain or hypomobility, IVD pathology and nerve root compression. However, to date, there is no literature reporting a CME NRR for ages 20-69 years and both genders, and no evidence to support CME patterns specific to lumbar pathology and mechanical LBP.
3. Methods

3.1. Overview
This chapter describes the research method used in each of the studies in this thesis. Each section explains why the study was performed and how the study progresses the investigation of CME, the investigation of specific symptomatic CME patterns, and the potential use of CME for the assessment of mechanical LBP, including as a clinical reasoning tool in its examination in a clinical setting.

These studies used quantitative and qualitative methods to report lumbar spine function in asymptomatic and symptomatic populations. Quantitative data was collected using a MotionStar, 3-D motion tracking system and its integrated software LabVIEW V5.0. Quantitative and qualitative data were collected using three short validated questionnaires – VASp, VASs for reporting, SF-12 for assessing physical and mental health and the RMDQ for assessing pain and function (Deyo et al., 1998). These questionnaires form the core set of instruments used in much spine research and were used to inform the investigators of the subject’s pain, stiffness, health (with respect to LBP), function and disability.

3.2. Research design
Two pilot studies used a repeated measures design in validation studies to test the accuracy of a measuring device. These are reported in the appendices.

Five studies formed chapters in this thesis, with three subsidiary published papers presented in related appendices. Study 1 is an observational study on CME NNR using asymptomatic volunteers (reported on in Chapter 4). Studies 2 and 3 are clinical test-retest, observational studies of two cohorts receiving two different interventions – invasive pain management approaches (Chapter 5) or neurosurgical procedures (Chapter
6), respectively. Study 4 is a test-retest, observational study of clinicians’ responses to hypothetical scenarios of mechanical LBP where CME models may be identified in clinical practice (Chapter 7), and Study 5 reports cluster analysis results for outcome data from Studies 2 and 3 (Chapter 8).

3.3. Purpose and research hypotheses

3.3.1. Pilot studies

Pilot study I: Validation of a 3-D motion tracking system (Appendices II and III)

In order to test the reliability and precision of a MotionStar 3-D motion tracking system prior to a series of clinical studies, a tri-axial goniometer with known accuracy and precision was used to establish a baseline reference. This validation investigation hypothesised that the triaxial protractor is a reliable device for use when validating the MotionStar 3-D recording system. It is also hypothesised that the error reported from this investigation of the triaxial protractor is similar to the \( \leq 0.1^\circ \) error reported by Barrett (1995), who adopted a comparable tracking system in a thesis investigation of lumbar CME.

Pilot study II: Reliability of computer-aided CME (Appendix III)

In order to perform a series of clinical studies using the MotionStar 3-D motion tracking system, the device was tested for reliability and precision. This validation investigation hypothesised that the MotionStar 3-D tracking device and its integrated purpose-designed software is a reliable device for measuring angle of rotation in the three cardinal planes – sagittal, coronal and transverse. It is hypothesised that repeated measures will have a coefficient of variation (CV) of \(<5\%\), which for the largest movement of lumbar flexion in normal subjects is approximately \(2.5^\circ\). This was proposed as an acceptable error for the purposes of this study.
3.3.2. Main studies

Study 1: The CME NRR (Chapter 4)

The purpose of this chapter is to report the development of a CME NRR for both genders for each decade of life from 20-69 years. It was hypothesised that normal CME will be symmetrical (left versus right), flexion ROM will be greater than extension ROM, and that global (360°) CME patterns will reduce in size, illustrating a decreased ROM in all directions with increased age, and that females will have greater ROM than males for any given age decade.

Additionally, this chapter reports preliminary proof of concept in a case study of two patients with LBP, and the use of CME as a tool to identify atypical or symptomatic movement patterns, and monitors changes post-intervention relative to their respective age- and gender-matched NRR.

Study 2: Pain management cohort (Chapter 5)

The purpose of this study was to assess the use of computer-aided CME to measure change in low back movement following invasive pain management intervention in cases of lumbar spondylosis. Additionally to use the CME NRR (Chapter 4) to compare and contrast movement patterns identified from three specific pathoanatomical structures – IVD, facet joint, and nerve root compression. It was hypothesised that for patients with mechanical LBP arising from disc, facet or nerve root compression, a unique CME movement signature will be identified. It was also hypothesised that self-report questionnaire scores will improve after intervention, and CME ROM will change in the direction of the pathoanatomical structure after intervention by normalising towards each patient’s matched NRR.
**Study 3: Neurosurgery cohort (Chapter 6)**

The purpose of this study was to report the use of computer-aided CME to measure change in low back ROM in cases of lumbar spondylosis, before and after neurosurgical intervention. The CME NRR (Chapter 4) was used to compare and contrast movement patterns identified from specific cases whose primary pathology involved either IVD or nerve root. It was hypothesised that for patients with mechanical LBP arising from disc or nerve root compression, a CME movement signature will be identified similar to that presenting in the pain medicine cohort (Chapter 5) for disc and nerve root pathology. It was also hypothesised that self-report questionnaire scores will improve after intervention, and CME will change after intervention, by normalising towards each patient’s matched NRR.

**Study 4: Clinical reasoning for CME of mechanical LBP (Chapter 7)**

The purpose of this study was first to determine if clinicians could hypothesise pathoanatomical structure(s) contributing to mechanical LBP by viewing a montage of a model patient performing three abnormal CME movement patterns. Secondly, after obtaining baseline assessments from the clinicians, to measure if their clinical reasoning changed after teaching general principles of CME and following a discussion about the putative specific CME movement patterns suggestive of pathoanatomical diagnosis, identified in Chapters 5 and 6. It was hypothesised that after being exposed to specific CME movement patterns, the clinician will be able to propose facet joint or IVD pathology or nerve root compression for CME patterns consistent with these structures.

**Study 5: Cluster analysis of pooled outcome data from Studies 2 and 3 (Chapter 8)**

The purpose of this study was to use pooled outcome data from Studies 2 and 3 to answer the third primary hypothesis, that lumbar CME changes after specialist
intervention, approaching the NRR. Additionally, to answer five secondary hypotheses related to CME, pathology, intervention and self-report outcome scores.

3.4. **Subject recruitment**  
**Study 1: The CME NRR**  
A convenience sample of 192 healthy subjects participated in the NRR study. Subjects were recruited from the community, staff and students from The University of Western Australia, and staff from SCGH, and examined at either location. All participants received an information sheet (Appendix I.1, I.5) and provided written consent (Appendix I.3, I.7) for the respective testing location. Approval for this study was obtained from the university’s HREC [RA/4/1/6020] (Appendix I.8) and SCGH HREC [2014-009] (Appendix I.9).

Participant inclusion criteria were:

- Age within the CME NRR of 20-70 years.
- No significant episode of LBP requiring treatment in the previous 6 months.
- A body mass index (BMI) ≤ 30. Obesity is reported to decrease accuracy of sensor placement (Snider et al., 2011) and may restrict true limits of spinal movement.

Exclusion criteria were:

- Current or previous back pain requiring treatment in the preceding 6 months.
- Previous injection or surgical treatment for the low back, sacroiliac joints or hips.
Study 2: Pain management cohort

Cases for the pain management study (n=17) were selected from a private physiotherapy practice and SCGH pain management department. All participants received an information sheet (Appendix I.2, I.6) and were provided written consent (Appendix I.4, I.7) at the respective testing locations. Approval was obtained from the university’s HREC [RA/4/1/6020] (Appendix I.8) and SCGH HREC [2014-009] (Appendix I.9).

Participant inclusion criteria were:

- Age within the CME NRR of 20-70 years.
- Patient had been diagnosed by a pain specialist as having pain originating from the low back.
- Patient had radiological imaging of the low back for retrospective evaluation.
- Patient was scheduled for pain management, injection procedure for lumbar structure/s, with the use of radiographic imaging for facet joint, IVD or nerve root structures (Fig 3.1).

Exclusion criteria were:

- Previous lumbar spine, pelvis or hip surgery.
- Unable to stand independently, unaided, for 2 minutes continuously.
- Nurse on duty or examiner considered the patient unable to attend examination due to the complexity of the patient’s condition and/or comorbidities.
Figure 3.1. Patient set-up for CME in the pain management department, SCGH.

**Study 3: Neurosurgery cohort**

Cases for the neurosurgery study (n=18) were selected from a private physiotherapy practice and SCGH outpatient neurosurgery department. All participants received an information sheet (Appendix I.2, I.6) and provided written consent (Appendix I.4, I.7) at the respective testing locations. Approval was obtained from the university’s HREC [RA/4/1/6020] (Appendix I.8) and SCGH HREC [2014-009] (Appendix I.9).

Inclusion criteria were:

- Aged within the CME NRR, 20-69 years.
- Patient had been diagnosed by a neurosurgeon as having pain originating from the low back.
- Patient had radiological imaging of the low back for retrospective evaluation.
- Patient was scheduled for neurosurgery, addressing facet joint, IVD or nerve root structures.

Exclusion criteria were:

- Previous lumbar spine, pelvis, or hip surgery.
- Unable to stand independently, unaided, for 2 minutes continuously.
- Neurosurgeon or examiner considered the patient unable to attend examination due to the complexity of the patient’s condition and/or comorbidities.

### 3.5. Computer-aided CME instruction

The method employed for the placement and fixation of the MotionStar tracking system sensors, at first lumbar (L1) and S1 spinal levels, is described in detail in Appendix IV. The examiner demonstrated all eight movements of the CME, explained where the examiner will be standing and how they will guide the patient through the eight movements.

Before beginning any of the CME movements, the patient was asked to stand in a relaxed standing position looking straight forward while the tracking system collected data for five seconds at a rate of 50Hz. This was done to establish a zero reference point [0,0] and to allow calculation of the patient’s lumbar lordosis. The patient was then asked to register the most painful and the stiffest CME movement direction, and to report this to the examiner at the end of the CME. Less severe symptoms, in other CME movement directions were not recorded. The precise location – laterality, region of lumbar spine, gluteal, hamstring, or leg – was not recorded.
3.5.1. Instruction for flexion, and flexion with SF
The examiner stood on the patient’s left side and provided the following instructions – “place your arms across your chest, soften your knees by bending them a little, lean down as though you are reaching for the floor, allow your low back to bend, stop when you reach your comfortable limit”. The examiner counted out 3 seconds when the patient reached their comfortable limit, which facilitated post-hoc data reduction. The examiner then used their right hip against the patient’s left hip, and the examiner’s right hand on the patient’s right hip to stabilise the pelvis, before asking the patient to “side-bend to the left within your comfortable limits”, the examiner’s left hand was simultaneously placed on the lateral aspect of the patient’s right shoulder to guide the patient into flexion with added LSF (FwLSF). The position was held for 3 seconds before the patient was allowed to return to upright standing. The patient was allowed a minimum of 3, and up to 10 seconds to recover if they expressed or looked as if they had experienced discomfort. This break also allowed data to return to baseline [0,0]. During this time, the examiner walked around and stood on the right side of the patient. The procedure and instructions were repeated for the movements of flexion, and then flexion with added RSF (FwRSF), with a mirror image of the examiner’s manual guidance. The movement into FwLSF and FwRSF was active, guided by the examiner without a passive over-pressure component.

3.5.2. SF
With the examiner standing behind and slightly to the left of the patient, the examiner placed their left hand on the patient’s left hip, and their right hand on the patient’s right shoulder with the following instruction – “without leaning forwards or backwards, lean to your left by running your left hand down the outside of your leg. Reach as far as possible, within your comfortable limits”. After a count of 3 seconds in LSF the patient
was instructed to return to upright standing for a minimum of 3 seconds. The procedure and instructions were repeated for RSF.

3.5.3. Extension and extension with SF
With the examiner standing behind the patient, the following instruction was given – “lean backwards as far as you can, allow your low back to bend within your comfortable limit”. A 3 second count was given before returning to upright standing. The examiner walked to the front of the patient to demonstrate (again) extension with added LSF (EwLSF) and to the right (EwRSF). Pilot studies showed that this movement can be easily misunderstood. With the examiner standing behind the patient, the following instruction was given, “lean backwards as far as you can, allow your low back to bend within your comfortable limit”, once this position was achieved the remainder of the instruction was given “now, run your left hand down the back of your leg as far as you can, within your comfortable limits”. After a count of 3 seconds in EwLSF, the patient was instructed to return to upright standing for a minimum of 3 seconds. The procedure was repeated for EwRSF, and the patient returned to upright standing for 5 seconds before data collection was stopped.

3.6. Data acquisition and processing
The method employed for CME data acquisition and processing is described in detail in Appendix VI.

3.7. Testing the MotionStar system for consistency over time
The MotionStar tracking system was used to collect data over a time period of 3.5 years, and was moved several times between The University of Western Australia, SCGH’s pain management department and outpatient neurosurgery department, and to a local private physiotherapy practice. To investigate the system’s consistency of valid
measurements over time and between examination venues, the tri-planar goniometer used in Pilot Study I was used to test the MotionStar system at each location at various time points throughout the data collection period. For details of the validation methods see Appendix III (Figure 3.2).

Figure 3.2. Re-validating the MotionStar 3-D tracking system, using the tri-planar goniometer, in the examination room used on the outpatient neurosurgery ward, SCGH.

3.8. Data analysis

3.8.1. Missing data
In the case of missing data for self-report questionnaires or CME, or if the patient was not available for questioning over the telephone or for CME retest, the patient was removed from the study cohort. Nine patients were lost to follow-up and/or missing data. A further eight patients who attended pre-surgery CME did not proceed to surgical intervention and were excluded from further investigation.
3.8.2. Data treatment
Advice on data interpretation was provided by the study coordinators. The use of ‘R’ statistical package for data analysis was guided by statisticians in the Maths and Statistics Department, The University of Western Australia.

According to the recommendation of Ostelo et al. (2008) a MCID of \( \geq 30\% \) was applied to all outcome data. The validated self-report questionnaires are recommended for use in LBP research, and were adopted in these studies in an effort to investigate the multiple facets of LBP and standardise outcome measures for comparison with future CME research.

For the VASp and VASs, the patient placed a mark on a 100mm long line to report the magnitude of the greatest pain and stiffness experienced during CME, and also reported the direction in which both symptoms were experienced. The mark is measured from the zero point and expressed as a score out of 10. For example, if during pre-intervention CME, a patient reports that lumbar extension was their most painful CME movement direction and marks the VAS at 46mm, they receive a VASp score of 4.6 for the extension movement. If for example, at post-intervention the score for lumbar extension is reduced to 18mm, giving a VASp score of 1.8, the total change score, post minus pre, of 2.8 was converted to a percentage: \( [(1.8-4.6)/10] \times 100 = -28\% \). For VAS scores a negative score indicated an improvement (Carlsson, 1983, Johnson, 2005). The RMDQ was scored out of 24 (Ostelo et al., 2004), and the SF-12 was scored using an online auto-scoring template (SF-36.org, 2012). Both RMDQ and SF-12 were also converted to percentage change scores.

Data reduction using mean, percentage, SD, CV, inter-quartile data, conversion to z-scores and illustration of movement patterns for comparison through radial plots, was performed by the examiner using Microsoft Office, Excel 2010. Graphical
representation of CME data facilitated comparison with a patient’s age- and gender-matched NRR (Figure 3.3A).

![Figure 3.3. Radial plots of pre and post-surgery CME data, from a 44 year old female. With the matched average NRR plot (A), sagittal and axial MRI, (B, C, respectively).](image)

In all cases, the examiner attempted to remain blind to radiological imaging and reports, specialist diagnosis and intervention, until after the final data were collected. These were viewed post-data analysis to compare with CME data. In all patient cohort cases, either computerised tomography (CT), or magnetic resonance imaging (MRI) were accessed (Figure 3.3B & C).

More complex calculations, including ICC and 95% confidence interval (CI), LSC and cluster analysis were performed using R statistical package, version 3.3.0. Cluster analysis in R used Euclidean distance measures and hierarchical clustering to produce dendrograms for interpretation (Blashfield, 1980).

### 3.9. Summary

This chapter, with Appendices II, III, IV, and VI, has described the methods implemented in the research design – posing of hypotheses, subject recruitment, CME procedure, data acquisition and processing, and data analysis. For all outcome data, a MCID of $\geq 30\%$ was used. For all statistical tests, probability of significant change was
set at <0.05. A significance level of 0.05 indicates a 95% chance of concluding that a real difference exists (true positive), and a 5% risk of concluding a difference exists when there is no actual difference (false positive).
4. **Study 1 results: Reliability, normal reference range and clinical utility of CME**

This chapter is based on the publication by Monie et al., (2015), Appendix V.

4.1. **Abstract**

**Objectives:** To report the development and validation of a low back computer-aided CME protocol in normal individuals and record treatment outcomes of cases with symptomatic degenerative lumbar spondylosis.

**Design:** Test-retest, following intervention.

**Background:** Self-report assessments and CME was used to record composite spinal motion, before and following neurosurgical and pain medicine interventions.

**Methods:** 151 normal individuals aged from 20-69 years were assessed using CME between L1 and S1 spinal levels to establish a reference range. Cases with degenerative LBP and sciatica were assessed before and after therapeutic interventions with CME and a battery of self-report pain and disability questionnaires. Change scores for CME and all outcome measures were derived.

**Findings:** Computer-aided CME validation and ICC with 95% CI and LSC scores indicated acceptable reliability of CME when recording lumbar movement in normal subjects. In both clinical cases lumbar spine movement restrictions corresponded with self-report scores for pain and disability. Post-intervention outcomes all showed significant improvement, particularly in the most restricted CME direction.

**Interpretation:** This study provides normative reference data for CME that may inform future clinical studies of the technique as a convenient objective surrogate for important clinical outcomes in lumbar degenerative spondylosis. It can be used with good
reliability, may be well tolerated by individuals in pain, and appears to change in
concert with validated measures of lumbar spinal pain, functional limitation and quality
of life.

4.2. Introduction
Pragmatic treatment of patients with LBP and associated movement dysfunction is
based upon an appreciation of the history of the patient’s complaint and interpretation of
the examination findings (Maitland, 1997). Assessing lumbar spine movement in the
clinical setting to investigate dysfunction and to monitor changes in spinal movement
characteristics of individuals over time is routine clinical practice (Maitland, 1997, Lyle
et al., 2005, Ha et al., 2013, Laird et al., 2014). Single plane movements are often
unrepresentative of the actual movements of the lumbar spine, so have limited value in
assessing lumbar function (Pearcy and Hindle, 1989). However, one examination
method, originally described by Edwards (1979), which assesses both planar and
combined plane (physiological) positions is the CME. The CME sequentially examines
the patient’s ability to actively side-flex the lumbar spine while in a flexed, neutral and
extended position (Figure 4.1).

Edwards (1979) and Maitland (1997) suggested that CME may be more informative
than the standard planar spinal assessment, which was confirmed by Barrett et al. (1999)
who reported acceptable CME intra-examiner reliability, as well as preliminary
evidence concerning the effectiveness of CME in identifying reduced lumbar mobility
in LBP subjects. In their work Barrett et al. (1999) used 3-D tracking to map the
characteristics of the lumbar CME. To our knowledge, no other study has reported the
putative use of lumbar CME in clinical practice using a computer-aided methodology to
quantify movement patterns. Figure 4.1 illustrates the eight low back positions of a
lumbar CME and an example radial plot of a healthy volunteer, showing the symmetrical end-points (maximal angular movement) achieved.

Figure 4.1. Example of a healthy volunteer’s CME radial plot (in degrees of angular movement). Photographs in this figure illustrate the movement directions and end-points.
The purpose of this paper is to report the intra- and inter-session reliability of lumbar CME using a validated MotionStar 3-D motion tracking system (Figure 4.2A) using custom software LabVIEW V5.0. Secondly, to describe the development of a NRR and subsequently to report proof of concept of CME as a tool to assess specific spinal pathology and monitor changes post-intervention. A CME NRR was developed to identify abnormal patterns, observe normalisation of CME post-intervention and compare the age- and gender-matched functional outcomes of two cases with different lumbar spine pathologies. It is not the intention of this paper to report clinical studies which did not use an objective quantification of lumbar spine movement.

4.3. Methods
A MotionStar 3-D motion tracking system was tested in our laboratory against a custom made triaxial goniometer to test consistency over time (Figure 4.2A). Angular displacement of the sensors was calculated by the system, using Euler’s method. Results demonstrated that the MotionStar is capable of reliably measuring angular movement with an intrinsic precision of 0.6°. The details of the validation process for the triaxial goniometer and MotionStar system are described in Appendix II and III, respectively.

After obtaining written consent and familiarisation of equipment and testing sequence, two skin mounted MotionStar sensors were placed over the volunteer’s S1 and L1 spinous process, respectively. Skin marking and sensor mounting over the L1 landmark was performed while the patient maintained a partially flexed lumbar spine position in standing, with their hands on their knees. This made palpation of the L1 level easier and pre-stretched the skin under the sensor’s double-sided tape, improving adhesion. In a relaxed standing position, all volunteers had their lumbar lordosis (angle between L1 and S1) recorded (Figure 4.2B). This became their ‘zeroed’ starting position (centre of radial plot) (Figure 4.1). The details of sensor placement and fixation are described in
Appendix IV. The patient was then instructed to move within their comfortable limits during the CME while the MotionStar system acquired data and stored to disc at a rate of 50Hz. Trial data were batch processed using a Butterworth 4th order, low pass (cf 4Hz) filter, to remove high frequency (non-biological) noise. The details of the data processing are described in Appendix VI. Maximum data values for each of the eight CME movement directions were recorded according to a pre-defined sequence – flexion, flexion with left side-flexion (FwLSF), flexion with right side-flexion (FwRSF), left side-flexion (LSF), right side-flexion (RSF), extension, extension with left side-flexion (EwLSF) and extension with right side-flexion (EwRSF).

A pilot study comparing various CME sequences was trialled, with the current format producing the most consistent data with the least discomfort. Intra-session reliability studies involving ten normal volunteers indicated there was no warm-up or fatigue effect over five repeated trials. For this reason, after a familiarisation trial, a single data collection was used on each subsequent test session.
CME reliability data were assessed with ICC and 95% CIs for the five intra-session (n=10) and five inter-session (n=10) trials. In addition, the LSC method (Bonnick and Lewis, 2013) was used to represent variance of the CME outcomes. Data confirmed acceptable reliability for all eight CME movement directions (Table 4.1).

Table 4.1. ICC and 95% CI ICC values for each movement direction of lumbar spine CME.
Data was examined using 10 cases with 5 trials performed for both intra-session and inter-session cohorts.

<table>
<thead>
<tr>
<th>CME position</th>
<th>Intra-session</th>
<th>Inter-session</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICC, 95% CI</td>
<td>ICC, 95% CI</td>
</tr>
<tr>
<td>Flexion</td>
<td>0.92 (0.85-0.98)</td>
<td>0.92 (0.83-0.98)</td>
</tr>
<tr>
<td>FwRSF</td>
<td>0.93 (0.85-0.98)</td>
<td>0.86 (0.70-0.95)</td>
</tr>
<tr>
<td>RSF</td>
<td>0.94 (0.86-0.98)</td>
<td>0.90 (0.78-0.97)</td>
</tr>
<tr>
<td>EwRSF</td>
<td>0.95 (0.88-0.98)</td>
<td>0.71 (0.47-0.90)</td>
</tr>
<tr>
<td>Extension</td>
<td>0.95 (0.88-0.98)</td>
<td>0.78 (0.57-0.93)</td>
</tr>
<tr>
<td>EwLSF</td>
<td>0.95 (0.89-0.99)</td>
<td>0.74 (0.51-0.91)</td>
</tr>
<tr>
<td>LSF</td>
<td>0.92 (0.83-0.98)</td>
<td>0.82 (0.63-0.94)</td>
</tr>
<tr>
<td>FwLSF</td>
<td>0.94 (0.86-0.98)</td>
<td>0.93 (0.85-0.98)</td>
</tr>
</tbody>
</table>

Development of a NRR: MotionStar derived CME data was captured to develop a NRR (n≥7 for each decade of life and gender) for which a convenience sample of 151 asymptomatic participants was used. Volunteers were included in this study if they were aged between 20-69 years, had no previous spinal intervention, had no significant episode of LBP requiring treatment in the previous 6 months, were able to follow verbal instructions and had a BMI ≤30. The NRR used in this study is displayed in Table 4.2. The final NRR, acquired by the end of this thesis (n=192), is shown in Appendix VII.
Table 4.2. CME NRR for males and females aged 20-69 years of age.
The mean, SD and range for standing lordosis, BMI and each movement direction for males and females.

<table>
<thead>
<tr>
<th>Gender</th>
<th>Age</th>
<th>n=</th>
<th>Statistic</th>
<th>BMI Mean (SD)</th>
<th>Lordosis Mean (SD)</th>
<th>Flexion Mean (SD)</th>
<th>FwRSF Mean (SD)</th>
<th>RSF Mean (SD)</th>
<th>EwRSF Mean (SD)</th>
<th>Extension Mean (SD)</th>
<th>EwLSF Mean (SD)</th>
<th>LSF Mean (SD)</th>
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Lumbar lordosis and angular movement values are in degrees [°]. For explanation of acronyms, refer to Figure 1.
Two cases of LBP were examined. Case A was a 53 year old male who presented with an antalgic gait, mild LBP and severe, acute anterolateral hip pain (VAS 9.7/10). MRI demonstrated a large left disc extrusion at the L2-3 level, with inferior sequestration of disc material resulting in impingement on the left L3 nerve (Figure 4.3A & B). A discectomy was performed and post-operative assessments (self-reports and CME) recorded.

Case B was a 62 year old female who presented with chronic, intermittent medial shin pain aggravated by lumbar extension. Computerised tomography (CT) identified a hypertrophic L4-5 facet joint impinging on the L4 nerve root with associated L4 exit foramen stenosis (Figure 4.4A & B).

Figure 4.3. Case A.
(A) T1 sagittal MR image of a 22mm sequestration of L2-3 disc material. (B) T2 axial MR image of the posterolateral position of the extruded disc material. (C) Patient’s standing position 16 days pre-operatively compared with (D) 24 weeks post-operatively.
Figure 4.4. Case B.
(A) Transverse CT image of the left L4 nerve root. (B) Sagittal CT image of the stenotic L4 exit foramen. (C) Patient’s active extension 1 week prior to L4-5 epidural injection and (D) 12 weeks post-injection.

The core battery of outcome measures were used to assess the patients pre-intervention and at three intervals, post-intervention (Deyo et al., 1998): VAS, the RMDQ and a SF-12. The two cases participated in a CME trial on each of four assessment days with their CME values compared to our age- and gender-matched NRR. A change of ≥ 30% in all measures was considered clinically significant (Ostelo et al., 2008). For the eight CME directions (Figure 4.1) the maximum values were displayed in a radial plot and change scores calculated between trials.

4.4. Results
The maximum values for each of the patient’s CME movements were plotted to observe changes over time. Figure 4.5A illustrates the pre- and final post-operative trials for Case A, plus a comparison age- (50-59) and gender-matched normal plot. Figure 4.5B illustrates the initial and final test values (total change) collected from CME of Case B. The age- (60-69) and gender-matched NRR data is plotted for comparison.
Figure 4.5. Case A and B plotted CME movements.
(A) The first and last trial for Case A and the average normal male CME data for 50-59 years of age. Case B shows pre-injection and final post-injection CME. (B) The average normal female CME data for 60-69 years of age. Data are in degrees of angular movement.

The natural standing lumbar lordosis and data values for each of the CME directions (Table 4.3) were compared with the matched CME NRR. Total change scores (%) in
angular movement for each CME direction, between pre-intervention (trial 1) and post-intervention (trial 4) assessments are reported in Table 4.3.

Self-report outcome data for the two cases, for the index assessments are presented in Table 4.4. Clinically important improvements (≥ 30%) are evident in the VAS, RMDQ and SF-12 domains.
Table 4.3. Maximum angular movement for each of the patient’s CME movement directions, plus standing lordosis values at each trial with total change scores (%).

<table>
<thead>
<tr>
<th>Case A CME trials</th>
<th>Flexion</th>
<th>FwRSF</th>
<th>RSF</th>
<th>EwRSF</th>
<th>Extension</th>
<th>EwLSF</th>
<th>LSF</th>
<th>FwLSF</th>
<th>Lordosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 weeks pre-op</td>
<td>38.9</td>
<td>36.8</td>
<td>11.2</td>
<td>3.2</td>
<td>-2.5</td>
<td>-3.2</td>
<td>-5</td>
<td>31.1</td>
<td>23.1</td>
</tr>
<tr>
<td>24 weeks post-op</td>
<td>45.2</td>
<td>42.6</td>
<td>20</td>
<td>4.9</td>
<td>7.6</td>
<td>7.6</td>
<td>17.1</td>
<td>38.5</td>
<td>36.9</td>
</tr>
<tr>
<td>Total change (%)</td>
<td>16.2%</td>
<td>15.8%</td>
<td>78.6%</td>
<td>53.1%</td>
<td>404.0%</td>
<td>337.5%</td>
<td>442.0%</td>
<td>23.8%</td>
<td>59.7%</td>
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</table>

<table>
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<th>Case B CME trials</th>
<th>Flexion</th>
<th>FwRSF</th>
<th>RSF</th>
<th>EwRSF</th>
<th>Extension</th>
<th>EwLSF</th>
<th>LSF</th>
<th>FwLSF</th>
<th>Lordosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 week pre-injection</td>
<td>39.8</td>
<td>37.9</td>
<td>18.3</td>
<td>3.9</td>
<td>4.9</td>
<td>4.6</td>
<td>13.6</td>
<td>38.1</td>
<td>34.7</td>
</tr>
<tr>
<td>12 weeks post-injection</td>
<td>40.0</td>
<td>36.7</td>
<td>17.4</td>
<td>4.9</td>
<td>7.1</td>
<td>5.1</td>
<td>16.8</td>
<td>30.8</td>
<td>34.1</td>
</tr>
<tr>
<td>Total change (%)</td>
<td>0.5%</td>
<td>-3.2%</td>
<td>-5.0%</td>
<td>23.4%</td>
<td>45.8%</td>
<td>11.6%</td>
<td>23.2%</td>
<td>-19.1%</td>
<td>-1.8%</td>
</tr>
</tbody>
</table>

All angular movement values are in degrees (°). For explanation of acronyms, please refer to Figure 4.2.

Table 4.4. Change in self-report instruments (pre- and post-intervention) for VAS, SF-12 PCS, SF-12 MCS and the RMDQ.

<table>
<thead>
<tr>
<th>Case A</th>
<th>VAS LBP</th>
<th>VAS hip</th>
<th>SF-12 PCS*</th>
<th>SF-12 MCS*</th>
<th>RMDQ</th>
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<td>2 weeks pre-op</td>
<td>0.8</td>
<td>9.7</td>
<td>26.8</td>
<td>32.9</td>
<td>20</td>
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<tr>
<td>24 weeks post-op</td>
<td>0.2</td>
<td>0.2</td>
<td>55.5</td>
<td>57.8</td>
<td>1</td>
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<tr>
<td>Improvement (%)</td>
<td>75.0%</td>
<td>97.9%</td>
<td>50.7%</td>
<td>41.0%</td>
<td>79.2%</td>
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</table>

<table>
<thead>
<tr>
<th>Case B</th>
<th>VAS shin</th>
<th>SF-12 PCS*</th>
<th>SF-12 MCS*</th>
<th>RMDQ</th>
</tr>
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<tbody>
<tr>
<td>1 week pre-epidural</td>
<td>2.3</td>
<td>39.9</td>
<td>47.0</td>
<td>10</td>
</tr>
<tr>
<td>12 weeks post-epidural</td>
<td>0.0</td>
<td>59.9</td>
<td>43.6</td>
<td>0</td>
</tr>
<tr>
<td>Improvement (%)</td>
<td>100.0%</td>
<td>35.3%</td>
<td>-5.6%</td>
<td>41.7%</td>
</tr>
</tbody>
</table>

*Normal SF-12 health survey: mean=50, SD=10
4.5. Discussion

Outcome assessment for LBP is complex and typically involves multiple dimensions. Self-report surveys and lumbar kinematics provide insight into the response of low back conditions to management (Deyo et al., 1998, Williams et al., 2013). Measures should be reliable, valid, practical and, for convenience, brief where possible. According to Deyo et al. (1998), assessments of severity and frequency of symptoms such as the RMDQ and SF-12 outcome measures for low back dysfunction are particularly useful for research purposes. However, outcome measures placing emphasis on pain, function and quality of life do not provide the clinician with feedback on the direction and magnitude of movement pattern disturbance (Lyle et al., 2005), the potential structure(s) at fault or departure from normal movement according to age and gender.

In this report we describe the development of a CME assessment model for the lumbar spine. Intra-session and inter-session trials showed CME to be very reliable for all movement directions. This is consistent with the study by Mieritz et al. (2012) reporting a systematic review on the reliability of 3-D measures of the lumbar spine. Further, we have established a preliminary NRR for lumbar CME with which to contrast cases of specific lumbar dysfunction.

To date, only Barrett et al. (1999) have reported preliminary evidence for use of the CME to identify reduced spinal mobility in LBP patients. They did not report NRR nor test for directional movement restrictions and attribute these to specific diagnoses. The present report investigated the novel application of CME to assess change to both the magnitude and direction of dysfunction and consequently to demonstrate a tendency towards age- and gender-matched normalisation of low back movement after intervention. Spinal 3-D motion behaviour was described by Ha et al. (2013) as a useful
assessment in monitoring changes in spinal movement in an individual over time. As spinal movement is not isolated to pure cardinal planes, 3-D information may confer greater insight into the clinical analysis of aberrant spinal mechanics.

The ability for CME to detect specific directions of restricted movement and to predict biomechanical causes has been hypothesised though not previously examined (Brown, 1988). In this investigation the CME had sufficient sensitivity to detect the greatest restriction and subsequent improvement, in the direction of the confirmed structural spinal abnormality and response to composite loading. Interestingly, passive spinal structures make up the majority of the common pathologies in the lumbar spine (Press, 2007). According to Cunningham et al. (2007) and Little et al. (2007) osseoligamentous tissues and the disc anulus are the primary contributors to spinal stiffness. This may direct a clinician to consider specific structures causing movement restrictions in specific CME directions. Pearcy and Hindle (1989) discuss the potential diagnostic value of 3-D lumbar movement assessment; however, no studies have substantiated this claim in pathoanatomical terms.

An age- and gender-matched CME NRR was used to guide provisional outcome goals. When observing Case A’s final CME radial plot (Figure 4.5A), it is clear that the original movement restriction was normalised, resulting in a symmetrical pattern, comparable to the age- and gender-matched CME plot. Figure 4.5B highlights that the extension range of Case B, after 12 weeks, though no longer painful or causing reported disability, is still well below the age- and gender-matched average. Facet joint hypertrophy at the L4-5 level (Figure 4.4A) would contribute to the patient’s movement impairment. Patients, who demonstrate little change in their CME pattern, yet report marked clinical improvement in their pain and function, as in Case B, may reflect a sub-group with structural pathology for whom initial pain management is the appropriate
intervention prior to a surgical opinion if symptoms persist. In both cases, the self-report results highlight clinically important improvements in pain, disability and health outcomes. In Case B, her pain score decreased by 23% (Table 4.4), to zero (VAS 0/10), at the 12-week final assessment.

Several previous studies have reported planar lumbar motion measures (Pearcy and Hindle, 1989, Madson et al., 1999, Troke et al., 2005), yet very few have described combined or coupled lumbar movement (Russell et al., 1993, Ha et al., 2013) and no report to our knowledge uses CME as an outcome measure when comparing symptomatic cases to an age- and gender-matched NRR. Furthermore, there seems to be a lack of normative data which can be used to inform outcomes from intervention to manage spinal pain. A systematic review and meta-analysis by Laird et al. (2014), comparing lumbar kinematics in people with and without LBP, concluded that their results do not improve the understanding of the relationship between movement and pain in individuals. They also noted the difficulty in attempting a meaningful interpretation of the data due to the varied methodologies, samples and symptoms reported by the different studies.

Further studies are currently underway with larger clinical cohorts of cases diagnosed with lumbar stenosis, confirmed facet joint dysfunction or disc herniation. We will test the hypotheses that CME can assist in the provisional diagnostic sub-grouping of mechanical back pain syndromes and, with the use of a NRR, predict the extent, rate and pattern of recovery from specific neurosurgery or pain management interventions.

The present study provides normative data for CME that may inform future clinical studies of this technique as a convenient objective surrogate for important clinical outcomes in lumbar degenerative spondylosis. It can be used with good reliability, may
be well tolerated by individuals in pain and appears to change in concert with validated measures of lumbar spinal pain, functional limitation and quality of life.

Limitations

This study used a lumbar CME NRR of 151 cases, which may be considered low to claim normative data. Additional asymptomatic cases were examined during this thesis examination, leading to the final NRR of 192 cases (Appendix VII).

Conclusion

The CME is a reliable movement examination and may be a useful outcome measure for individuals with low back movement dysfunction. A NRR CME provides an expected movement outcome matched to age and gender, although care must be taken to consider individual anatomical variations and clinical presentations. These case studies provide initial evidence that CME may possess sufficient sensitivity to detect the nature of spinal dysfunction and the natural history following intervention.
5. **Study 2 results: Evidence of structure specific movement patterns using lumbar CME in cases receiving pain management interventions**

This chapter is based on the manuscript by Monie et al., (2016c) accepted for publication in the Journal of Manipulative and Physiological Therapeutics.

5.1. **Abstract**

**Objectives:** A test-retest, cohort study was conducted to assess the use of a novel computer-aided CME to measure change in low back movement following pain management intervention in 17 cases of lumbar spondylosis. Additionally to use a CME NRR to compare and contrast movement patterns identified from three specific structural pathologies – intervertebral disc (IVD), facet joint and nerve root compression cases. Finally, to test if specific CME patterns for these three structures exist.

**Methods:** Computer-aided CME was used before and after intervention, in a cohort study design, to record lumbar ROM along with pain, disability and health self-report questionnaires, in 17 cases who received image guided facet, epidural and/or rhizotomy intervention. In the majority of cases, CME was reassessed post-injection, together with two serial self-reports after an average of 2 and 14 weeks. A MCID of 30% was used to interpret meaningful change in all self-reports. A CME NRR (n=159) was used for comparison with the 17 cases. Z-score data for each movement direction were used to assess the size of CME ROM change. A significant change in ROM was set at ≥ 1 z-score. Post-hoc observation of key outcome measures were used from 16 cases to compare and contrast pathoanatomy and CME and facilitated sub-grouping cases into three discrete pathologies – IVD, facet dysfunction and nerve root compression.

**Findings:** Seven of the 17 cases stated that a ‘combined’ movement was their most painful CME direction. Self-report outcome data showed four cases experienced
significant improvement in health survey, five improved by ≥30% on low back function and eight cases reported that LBP was more bothersome than stiffness, six of which achieved the MCID for self-reported pain. Sub-grouping of cases using the core-set of outcome measures recommended by international consensus working parties on LBP (Ostelo et al., 2008), into structure specific groups provided insight to CME movement patterns for IVD, facet and nerve root compression. For symptomatic degenerative IVD a restriction in all CME movement directions was observed, for facet or nerve root compression a CME movement restriction was seen in movement directions towards the pathoanatomy.

**Interpretation:** The use of CME assists in identifying atypical lumbar movement relative to an age and gender NRR. Data from this study, exemplified by representative case studies, provide preliminary evidence for distinct IVD, facet joint, and nerve root compression CME movement patterns in cases of chronic lumbar spondylosis.

**Keywords:** Lumbar spine; combined movement examination; facet joint injection; epidural injection; rhizotomy; pain management

### 5.2. Introduction

LBP is a major public health problem in the western world. The lifetime prevalence is as high as 85% and the reported annual incidence in adults is 22-65% (Hoy et al., 2012), with 40-70% of those experiencing LBP seeking health care (Joud et al., 2012). Despite increased efforts to understand LBP, knowledge of the underlying pathology and insights into optimising clinical outcomes have advanced little in the last 2 decades (Hancock et al., 2011).

It is assumed that a large portion of LBP is caused or influenced by biomechanical factors (Mieritz et al. (2012). As all spinal structures are potentially a source of LBP
(Biyani & Andersson, 2004; Laplante et al., 2012), an accurate diagnosis is often difficult to make (Germon and Hobart (2015). Authors of a retrospective study of 170 patients undergoing diagnostic procedures for LBP suggested the IVD and facet joints are the two most likely sources of pain, with prevalence of 42% and 31%, respectively (DePalma et al., 2011). Improved diagnostic accuracy would confer obvious cost advantages to the health system for enabling treatment to focus on particular sources of pain but more than this would enable pathology-specific interventions to be grouped for clinical research.

During a structured clinical examination of the lumbar spine, a key component includes assessing the ROM (Littlewood and May, 2007), indicating spinal function, painful movement directions, response to intervention or even permanent impairment. The literature reports various movement assessments including functional activities of daily living (Bible et al., 2010), planar movements (Burton, 1986; Lee et al., 2011; Ha et al., 2013) and CME (Edwards (1979). Sánchez-Zuriaga et al. (2015) reported subtle alterations in lumbopelvic motion, in asymptomatic patients with recurrent LBP. However, their study only tested two planar movements (flexion and extension). A lumbar CME is considered more informative than a planar movement examination (Maitland, 1997) as this approach matches functional movements to the patient’s presenting complaint and may reproduce symptoms that could in future help with diagnosis (Haswell et al., 2004; Monie et al., 2105).

The purpose of the present study was to use a validated, reliable CME testing procedure (Monie et al., 2105) to determine if structure specific movement patterns exist in cases of chronic lumbar spine dysfunction. To examine this, CME and self-report data in 17 patients who underwent pain management intervention for confirmed lumbar spondylosis, was collected and compared to a relevant NRR. Normalising of CME post-
intervention was attributed to the structure treated and provided insight into structure specific CME movement patterns. For example, if a case had reduced LSF due to LBP, and treating the left L4-5 facet joint normalised LSF, we attributed the reduced LSF CME pattern to the left L4-5 facet joint.

5.3. Methods
This study was approved by the HRECs at The University of Western Australia and SCGH (Perth, Western Australia, Australia). Patient information was provided, and consent was obtained in all cases.

A 3-D motion tracking system MotionStar (Monie et al., 2015) with custom software LabVIEW V5.0 was used to measure a standardised eight direction CME (Figure 5.1). Proof of concept for the use of computer-aided CME and acceptable intra-session and inter-session reliability has been reported elsewhere Monie et al., 2105.
Figure 5.1. Example of an asymptomatic CME radial plot in degrees of angular movement. Photographs illustrate the movement directions and end-points – flexion, FwLSF, FwRSF, LSF, RSF, extension, EwLSF and EwRSF.

5.3.1. Recruitment and CME data collection
Thirty three patients with LBP and/or leg pain diagnosed by pain specialists as originating from low back structures were recruited and attended a pre-intervention CME trial. Of these, 17 individuals received pain management intervention and completed post-intervention examination (Figure 5.2). Patients were recruited from a private physiotherapy practice (n=8) and a pain management clinic in a tertiary hospital (n=9) – the sample comprised eight males: age 53±12 years, and nine females: age 60±13 years.
Following familiarisation with test protocol, two skin mounted MotionStar sensors [Ascension Technology, VT, USA] were placed over the volunteer’s S1 and L1 spinous process. Data acquisition and post-processing is described in detail elsewhere (Monie et al., 2015). The patient was asked to remember their most painful and most stiff CME movement direction, followed by instruction and guidance into each of the eight CME movement directions (Figure 5.1). Maximal data values for ROM were recorded according to a predefined sequence – flexion, flexion with left side-flexion (FwLSF), flexion with right side-flexion (FwRSF), left side-flexion (LSF), right side-flexion (RSF), extension, extension with left side-flexion (EwLSF) and extension with right side-flexion (EwRSF). All 17 patients were tested prior to intervention and retested at approximately 14 weeks after intervention.

![Flow chart of participation.](image)

**Figure 5.2.** Flow chart of participation.

### 5.3.2. Outcome measures

A battery of self-report outcome measures were used to assess cases at each examination visit (Chiarotto et al., 2015) – VASp and low back stiffness (denoted as
VASs), the RMDQ and a SF-12. A VASs was included as an outcome measure, since clinical measures often do not seek information regarding the effect of lumbar stiffness on function (Johnson, 2005; Hart et al., 2013). A MCID of 30% was used for all self-report data (Deyo et al., 1998). CME data was also collected and expressed using z-scores (standard scores for normally distributed data), after being compared with an age and gender matched NRR (n=159). A variable can be converted to a z-score if the distribution of normal range for that variable is Gaussian. In this study, z-scores expressed each individual’s ROM relative to their age and gender-matched NRR, indicating the magnitude of each movement direction, in SD (+ or -) from the NRR mean (Bonnick and Lewis, 2013). For the eight CME directions the maximum values were displayed in a radial plot and z-scores calculated for each direction and trial. For ease of comparison, it is noted that 68% of the distribution lies within plus or minus one (+1) SD of the mean (-1 ≤ z ≤ 1) and 95% lies within ±2 SD of the mean (-2 ≤ z ≤ +2).

Total change scores and z-scores are reported and a significant improvement was considered if the change was of magnitude ≥ 1 z-score. Each case’s CME was evaluated alongside the pain specialist’s diagnosis, treatment response, lumbar computer axial CT imaging or MRI and matched NRR, in an effort to compare CME with identified pathologies. A normal NRR (n=159) was used to aid in comparing and contrasting each case’s movement patterns (Monie et al., 2105).

A sample of convenience was derived from a tertiary hospital and private practice setting. Z-scores were used to assess the clinical CME. This representation facilitates comparison with a NRR in each of the eight CME movement directions, with reference to age and gender of each case.
5.4. Results
Change scores (%) were derived for VASp and VASs in relation to their low back condition, SF-12 physical component survey (PCS) and MCS and RMDQ. VASs scores were higher than VASp scores in the majority (9/17) of cases. Figure 5.3 illustrates total change scores (%) for self-reports in all 17 cases. The movement directions showing significant changes in z-scores were compared with specialist diagnosis, intervention and radiological imaging. A histogram plot (Figure 5.4) illustrates pre- and post-CME z-scores for three representative case examples (A, K and L) selected for their different, single structure diagnoses, in the four most informative CME movement directions (flexion, extension, EwLSF and EwRSF).

Of the 17 patients, seven were most symptomatic in a combined lumbar position (FwRSF, FwLSF, EwRSF or EwLSF), suggesting that CME is informative and useful as an outcome measure when compared to a simple planar movement examination; by identifying the patient’s worst functional movement. Total change scores for VASp, SF-12 PCS and RMDQ were clinically significant in 43%, 24% and 33% of all cases (where there was no floor or ceiling effect) at final retest. Of the 17 cases, all had pre-intervention z-scores of <-1.5 in at least one CME direction, indicating that movement was in the lower end of its range as defined by the equivalent NRR distribution, even with consideration of multiple comparisons of the CME variables. Four out of 5 cases with IVD degeneration showed ≥ 1 z-score of increased ROM in both flexion and extension directions. Four of the 5 facet joint pathology cases showed ≥ 1 z-score of increased ROM in an extended direction and not flexion. The single case of nerve root compression showed ≥ 1 z-score of increased ROM in EwSF to the ipsilateral side.
Table 5.1 provides a summary of each case, showing age, key imaging features, specialist diagnosis, intervention and outcomes with >30% change in self report and/or obvious ROM change in CME movement directions.

![Figure 5.3. Total change scores (%). Boxplot illustrating total change scores (Δ) for all patient’s self-report questionnaires, with minimum, median, 25th & 75th percentiles, and maximum values. In each series, post-intervention score minus pre-intervention score was used as the total change, expressed as a percentage. Arrows indicate the direction of improvement from baseline (0%). A MCID of 30% was used in this study. Self-report acronyms are defined in section 2.2.](image-url)
A radial plot and z-scores were derived for each trial in all 17 cases, in order to illustrate inter-session changes. Key outcome measures were considered for 16 cases to compare and contrast pathoanatomy and CME (Table 5.1). Case G was not used because there were no self-report outcome measures > 30%, suggesting the symptomatic structure was not identified and treated effectively, and CME did not migrate towards the NRR. The clinical presentation and specific CME patterns of cases A, K and L are discussed in detail as they comprised single structure pathology, receiving single structure intervention. Cases with multi-structure and multi-level pathologies (O, P and Q), who received multi-level interventions, were more difficult to assess and the changes more difficult to interpret.

**Figure 5.4. Histogram plot.**
Pre- (blue) and post-intervention z-scores (red) for example cases A, K and L, in the four most informative CME directions – flexion (a), extension (b), EwLSF (c) and EwRSF (d).
Table 5.1. Summary table for Study 2.
Table reports each case’s age, key imaging features, diagnosis, pain management intervention and total change scores for self-reports.

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Imaging</th>
<th>Specialist diagnosis</th>
<th>Intervention</th>
<th>Key outcome measures and CME directions</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>48</td>
<td>L4-5 DDD and spondylolisthesis</td>
<td>disc pain</td>
<td>epidural L4-5</td>
<td>VA Sp 83%, CME Flexion, FwRSF, FwLSF, Extension, FwLSF</td>
</tr>
<tr>
<td>B</td>
<td>36</td>
<td>L4-5 DDD and left side L5 nerve abutment</td>
<td>disc pain</td>
<td>epidural L4-5</td>
<td>VA Sp 72%, SF-12 PCS 37%, CME LSF, Extension</td>
</tr>
<tr>
<td>C</td>
<td>30</td>
<td>L5-S1 DDD</td>
<td>disc pain</td>
<td>epidural L5-S1</td>
<td>VA Sp 75%, VA Sp 58%, CME FwLSF</td>
</tr>
<tr>
<td>D</td>
<td>39</td>
<td>L4-5 disc protrusion</td>
<td>disc pain</td>
<td>epidural injection</td>
<td>SF-12 PCS 44%, RMDQ 34%, CME FwRSF</td>
</tr>
<tr>
<td>E</td>
<td>62</td>
<td>L4-1 DDD and degenerative facets</td>
<td>disc pain</td>
<td>epidural L4-5</td>
<td>CME Flexion, FwLSF, FwRSF, LSF</td>
</tr>
<tr>
<td>F</td>
<td>54</td>
<td>L3-4 disc protrusion abutting left L3 nerve, right L5-S1 IVF stenosis</td>
<td>bilateral facet pain</td>
<td>bilateral L3-S1 rhizotomy</td>
<td>VA Sp 54%, CME Flexion, FwLS, FwLSF</td>
</tr>
<tr>
<td>G</td>
<td>68</td>
<td>L4-5 DDD with R-L IVF narrowing and ankylosed facets</td>
<td>bilateral facet pain</td>
<td>bilateral L3-S1 rhizotomy</td>
<td>Nil</td>
</tr>
<tr>
<td>H</td>
<td>61</td>
<td>L3-5 canal stenosis, hypertrophic bilateral L4-5 facets</td>
<td>bilateral facet pain</td>
<td>bilateral L3-S1 facet injections</td>
<td>SF-12 PCS 30%, RMDQ 50%, CME LSF</td>
</tr>
<tr>
<td>I</td>
<td>68</td>
<td>L1-5 canal stenosis</td>
<td>bilateral facet pain</td>
<td>bilateral L5-S1 facet injections</td>
<td>VA Sp 39%, CME Extension, FwRSF, FwLS</td>
</tr>
<tr>
<td>J</td>
<td>68</td>
<td>L1-5 canal stenosis</td>
<td>right L4-5 facet pain</td>
<td>facet injection</td>
<td>VA Sp 58%, VA Sp 39%, RMDQ 40%, CME FwRSF, LSF</td>
</tr>
<tr>
<td>K</td>
<td>55</td>
<td>L4-5 disc bulge</td>
<td>right L4-5 facet pain</td>
<td>L4 nerve root sleeve injection</td>
<td>CME extension</td>
</tr>
<tr>
<td>L</td>
<td>62</td>
<td>L4-5 foraminal stenosis and nerve compression</td>
<td>left radiculopathy</td>
<td>epidural L4-5</td>
<td>VA Sp 30%, RMDQ 75%, CME FwLSF, FwRSF</td>
</tr>
<tr>
<td>M</td>
<td>70</td>
<td>L4-5 and L5-S1 anterolisthesis due to facet degeneration</td>
<td>right radiculopathy</td>
<td>epidural L4-5</td>
<td>VA Sp 52%, CME Flexion, FwLS, FwRSF</td>
</tr>
<tr>
<td>N</td>
<td>64</td>
<td>L4-S1 DDD and facet degeneration with L5-S1 spondylolisthesis</td>
<td>right radiculopathy</td>
<td>epidural L4-S</td>
<td>CME Extension, LSF</td>
</tr>
<tr>
<td>O</td>
<td>50</td>
<td>L3-5 canal stenosis</td>
<td>L4 structure</td>
<td>epidural L4-5, bilateral L3-S1 rhizotomy</td>
<td>CME Flexion, LSF</td>
</tr>
<tr>
<td>P</td>
<td>69</td>
<td>L4-5 severe stenosis and bilateral nerve root abutment</td>
<td>L4 structure</td>
<td>epidural L4-5</td>
<td>CME LSF, FwLSF, FwRSF</td>
</tr>
<tr>
<td>Q</td>
<td>54</td>
<td>L3-4 bilateral facets, L4-5 canal stenosis with right L4 nerve abutment</td>
<td>L4 structure</td>
<td>epidural L4-5, bilateral L3-S1 rhizotomy</td>
<td>VA Sp 30%</td>
</tr>
</tbody>
</table>

MCID = Minimal clinically important difference, DDD = degenerative disc disease, IVF = intervertebral foramen. For all outcome measure acronyms refer to the methods section.
5.4.1. Case A: A CME pattern for a painful degenerative disc.
A 48 year old female presented with severe, diffuse, bilateral (R>L) LBP. Prior to participating in this study, the patient received a right L4-5 facet injection with minimal benefit. The L4-5 level was implicated by the radiologist on MRI and was concordant with the patient’s right side low lumbar region pain experienced during clinical assessment.

CME showed a decreased ROM in all directions (global restriction) compared to the relevant NRR (Figure 5.5d). Considering this globally reduced CME pattern and the patient’s diffuse bilateral LBP symptoms without neural signs or symptoms, the IVD was implicated. The patient was referred for consideration of an epidural cortisone injection and received almost instant pain relief, attributable to the local anaesthetic. Total change score for decrease in pain at the final test was 83%. CME pre- and post-injection ROM data is illustrated in Figure 5.5d. Z-scores at 2 weeks post-injection show a large global shift in CME towards mean values of the relevant matched NRR. Figure 5.4 illustrates the change in z-score for the four most informative movement directions – flexion, extension, EwLSF and EwRSF.

![Figure 5.5. Case A’s pain diagram.](image)
Diagram illustrates the area of LBP (red) (a), MR images showing degenerative facet joints and disc at L4-5 (b) and dehydration of discs from L1 to L5 with bony oedema adjacent to L4-5 disc (arrow in yellow) (c) and CME radial plot illustrating increased movement in all directions at 1 week post-epidural cortisone injection (first retest). A decrease in SF movement is noticed in the
Case K: A CME pattern for facet joint dysfunction

A 55-year-old male complained of an 18-month history of right side LBP (Figure 5.6a). Axial CT demonstrated a single level right side facet narrowing and right posterolateral IVD protrusion at L4-5 (Figure 5.6b & c). CME showed a movement restriction in the directions of RSF, EwRSF and extension (Figure 5.6d). Clinical presentation consisting of unilateral, localised LBP with RSF or EwRSF was consistent with facet joint dysfunction (Brown, 1988). Lumbar flexion ROM was asymptomatic and greater than the NRR mean (Figures 5.4a and 5.6d). Manipulation of the right L4-5 facet provided temporary relief of CME symptoms and a provisional diagnosis of right L4-5 facet joint disease was made. The patient was referred for consideration of cortisone injection.

Z-scores for this patient showed change towards zero (0), with ROM in directions for RSF, extension and EwRSF approximating the mean value of this case’s NRR (Figure 5.6d). Total change data (%) for self-reports showed clinically significant improvements (≥ 30%) in VASp, VASs, SF-12 MCS and RMDQ.

Figure 5.6. Case K’s pain diagram.
Figure illustrates the area of right side, intermittent LBP (red) (a), axial CT showed a L4-5 right side facet joint narrowing (b) and posterolateral disc protrusion (c) (arrows in yellow) and computer-aided CME radial plot illustrating a restricted movement in the direction of lumbar extension, EwRSF and RSF, marked increase movement at final retest and an age- (50-59) and gender-matched NRR (d). Legend for CME: — Pre-injection, — First retest, — Final retest, — 40-49YO female NRR. CME acronyms are defined in section 5.3.1.
5.4.3. Case L: A CME pattern for unilateral radiculopathy

A 62 year old female presented with intermittent left medial shin pain (Figure 5.7a) and a lengthy history of acute LBP. Outcome measures showed that movement in the flexion directions – FwLSF, flexion and FwRSF – was greater than the reference average and pain free, and the low back was hypomobile in all extended CME directions – EwLSF, extension and EwRSF (Figure 5.4b-d and 5.7d). Lumbar extension was also the movement which reproduced the patient’s medial shin symptoms. The extended direction is believed to be consistent with IVF narrowing (Inufusa et al., 1976). A provisional diagnosis of left L4 radiculopathy was made.

Following CT imaging, this case was diagnosed with a left L4 radiculopathy secondary to degenerative, IVF stenosis (Figure 5.7b,c) and the patient was referred for nerve root sleeve injection.

CME for the LSF and extension directions at the first retest, converged towards their NRR values (Figure 5.7d). Total change data (%) for self-reports confirmed lasting clinically significant improvements (≥ 30%) in VASp, SF-12 PCS and RMDQ at each retest.

Figure 5.7. Case L’s pain diagram.

Figure illustrates the area of left medial shin pain (red) (a), CT images showing left side L4 radiculopathy and oedema (b) and para-sagittal view (c) and CME radial plot with increased movement in extension and LSF at first retest post-injection (d). Legend for CME: — Pre-
5.5. Discussion

In this preliminary study, we sought to investigate structure specific movement patterns for IVD, facet joint dysfunction, and nerve root compression using CME. Finally, we sought to determine if CME would converge towards the age- and gender-matched NRR, following pain management intervention for LBP.

Validated self-report questionnaires were used as additional outcome measures to identify change in pain, stiffness, health and function. This also served as a measure of successful intervention, allowing improvements to be putatively attributed to specific structures and consideration of CME patterns.

In order to investigate a hypothetical structure specific movement pattern, we focused on CME from those cases with imaging showing a single involved level, and where possible single structure changes. Nine of the 17 cases were selected for this purpose, including four IVD (cases A, B, C and D), two bilateral facet joint (cases I and J), one unilateral facet joint (case K) and two nerve root impingement cases (L and N).

All four of the IVD cases complained that a flexed direction (FwLSF, flexion or FwRSF) was their most symptomatic direction, with all cases having a flexion z-score of $\leq -1.7$. Lumbar discs are loaded and deformed in multiple movement directions (Zou et al., 2009). Certain of the eight cardinal directions of CME may trigger nociceptors within a sensitised, degenerative IVD (Coppes et al., 1997). This global reduction in CME (Figure 5.5d), often with $z$-scores $<-1.5$ in multiple directions, with complaints of diffuse LBP has been identified as a common presentation during our observation of patients receiving epidural injection for related IVD pathology. Further studies are
needed to test this initial observation. Figure 5.8a illustrates a typical CME pattern for a symptomatic lumbar disc.

In case K’s unilateral facet pain and restriction, attention is drawn to the fact that other CME directions were unrestricted (Figure 5.6d). This is consistent with facet biomechanics, since flexion causes facet load of less than 1/3 extension loading (Niosi et al., 2008) and in this case flexion ROM was greater than the mean from the respective NRR. A comparison between cases A and K CME plots (Figure 5.5d and 5.6d respectively), while considering facet loading patterns described, may explain why the L4-5 facet injection did not help case A’s condition.

**Figure 5.8. Example radial plots of four common CME presentations identified in this study.**

DDD (a), Right side facet joint disease causing right side LBP or right side radiculopathy due to inter-vertebral foraminal (IVF) stenosis (b), bilateral facet disease and/or central canal or bilateral IVF stenosis, causing bilateral lower limb symptoms (c), multi-structure pathology such as DDD and left side facet disease causing localised left LBP (d).
Cases L and N complained of lower limb pain consistent with radiculopathy. Both cases were restricted on CME with extension combined with SF to the affected side. This is consistent with narrowing of the IVF and compression of the exiting nerve roots (Inufusa et al., 1996). This can be explained, in part, by the location of the radiculopathy (posterior) and an asymptomatic IVD (anterior) relative to the lumbar segment’s sagittal axis of rotation (Monie, et al., 2015; Pearcy & Bogduk, 1988). Case L’s data shows little change in the extension directions post-injection (Figure 5.4b-d and 5.7d). With advanced bilateral L4-5 facet osteoarthritis identified on CT (Figure 5.7B) this mechanical restriction will persist. However, LBP or lumbar stiffness was not the presenting complaint. This patient’s symptom was left medial shin pain and after CME reliably identified significant reduction in ROM, and reproduced medial shin pain, a provisional diagnosis of left L4 radiculopathy was proposed and successfully treated. CME sensitivity was demonstrated in this case by the ability to monitor small changes in movement (<4°).

Figure 5.8b illustrates a typical CME pattern for unilateral lumbar facet dysfunction or unilateral radiculopathy. The difference between these two conditions, for provisional diagnosis, may lie in the location of the patient’s pain (low back vs. lower limb). Again, a larger series of cases with these presentations would need to be studied to elaborate on this prediction.

The two cases of bilateral facet joint dysfunction reported pain and demonstrated markedly reduced CME on all three of the extended directions (EwLSF, extension or EwRSF), with average Z-scores \( \leq 1.5 \). Interestingly, in both cases the ROM in flexed directions (FwLSF, flexion and FwRSF) was reduced compared to normal, yet asymptomatic. This was consistent with our description of the pain pattern of degenerative disc movement, and was noted in their imaging reports. It is likely the IVD
was not inflamed and sensitised at the time of testing. Figure 5.8c illustrates a typical CME pattern for bilateral facet dysfunction causing LBP or bilateral radiculopathy.

The three complex cases (O, P and Q) with multi-level (L3-4, L4-5 and L5-S1) and multi-structure (facet, IVD, vertebral body and nerve impingement pathology) changes on imaging received single session, bilateral, multilevel rhizotomy procedures and caudal epidural cortisone injection, for advanced posterior element related pain, which made CME interpretations difficult. Figure 5.8d illustrates an example multi-structure CME pattern.

5.5.1. Limitations and future studies
These preliminary results must be reviewed within the limitations of the study. The first limitation is that CME is not a level specific movement analysis rather CME is a global indication of L1 to S1 movement. Additional sensors would be required to measure intersegmental movement (Alqhtani et al., 2015). The second is the sample size. This cohort investigation was designed to generate hypotheses based upon the possibility of specific movement signatures related to specific pathologies and as such, no formal power calculations were performed. Thirdly, this study was an observational study, which examined routine pain management intervention, therefore was not designed to assess the efficacy of the pain interventions planned for each case.

This study used a lumbar CME NRR of 159 cases, which may be considered low to claim normative data. Subsequent testing resulted in a NRR of 192 cases (Appendix VII). A comparison of the data in both NRRs, for both genders and all age groups using an unpaired t-test showed insignificant differences between the two NRRs (p>0.05). However, we suggest using the NRR (n=192) for future CME studies due to the putative benefit of increased numbers.
Further studies with larger sample sizes of single-level, single structure cases are required to investigate CME structure specific movement signatures and MCID outcome parameters. Further investigation is also warranted in cases which show no change with CME post-intervention. The reasons for no immediate or short-term change may include multi-level pathology, movement adaptations and pain being confounded by dominant psychosocial issues (Deyo, 2015) or comorbidities.

5.6. Conclusion
This preliminary study reports the utility in using CME data in comparison with an age- and gender-matched NRR to investigate mechanical LBP. Discrete CME movement signatures for symptomatic IVD, unilateral facet joint, bilateral facet joint and nerve root compression, are proposed. In cases which improved on pre-intervention self-reports (>MCID), there was migration of CME movement towards the age- and gender-matched NRR.

Funding sources and potential conflicts of interest
There were no sources of funding or conflicts of interest associated with this research.
6. Study 3 results: Evidence of structure specific movement patterns using lumbar CME in cases receiving neurosurgical interventions

This chapter is based on the manuscript by Monie et al., (2016d), submitted for publication in Manual Therapy.

6.1. Abstract

Objectives: To report the use of a novel computer-aided CME to measure change in low back kinematics, in 18 cases of lumbar spondylosis. A CME NRR was used to compare and contrast movement patterns identified from specific cases whose primary pathology involved either; IVD disease, unilateral posterolateral disc protrusion with or without nerve root compression, and central disc protrusion with or without bilateral nerve root compression. Finally, to test if specific CME patterns for these three pathologies exist.

Design: Test-retest study design, following neurosurgery intervention.

Methods: Computer-aided CME was used in a short-term follow-up, test-retest study, to record lumbar ROM along with pain, disability and health self-report questionnaires, in 18 cases who received neurosurgery intervention. In each case, CME and self-reports were reassessed 14 weeks after surgery. A MCID of 30% was used as the criterion for meaningful change in all self-reports. A CME NRR (n=159) was used for comparison with the 18 cases. Z-scores for each movement direction were used to assess the size of CME ROM change. A significant change in ROM was set at ≥ 1 z-score. Post-hoc observation of all cases’ key outcome measures were used from to compare and contrast pathoanatomy and CME and facilitated sub-grouping cases into discrete pathologies: IVD degeneration without protrusion, unilateral posterolateral disc protrusion with or
without nerve compression, and central posterior disc protrusion with or without nerve compression, in order to test for evidence of inter-group differences.

**Findings:** Ten of the 18 cases stated that a ‘combined’ movement position was their most painful movement direction. Self-report outcome data showed 11 and seven cases improved by ≥30% in pain and stiffness, respectively. Three cases experienced clinically significant improvement in health survey and 10 on low back function self-report. Sub-grouping cases into structure specific groups provided insight to different CME movement patterns. Seventeen of the 18 cases showed ROM changes ≥ 1 z-score. A CME pattern reduced in ROM in all directions was suggestive of IVD disease, unilaterally restricted movement in the side-flexed or extended directions, and was suggestive of posterolateral disc protrusion with or without ipsilateral nerve root compression. Bilateral restrictions in extension suggested central or bilateral posterior disc pathology with or without nerve root compression.

**Interpretation:** The use of CME assists in identifying atypical lumbar movement relative to an age- and gender-specific NRR. Data from this short-term post-surgery study provide preliminary evidence for CME movement patterns in cases with IVD degenerative disease, unilateral posterolateral disc protrusion and centrally protruding discs, with or without nerve root compression. In eleven of the 18 cases, CME converged towards the NRR after surgery.

**Keywords:** Lumbar spine; combined movement examination; intervertebral disc; radiculopathy; neurosurgery

**6.2. Introduction**

LBP is a major public health problem in the Western world. The lifetime prevalence is as high as 85% and the reported annual incidence in adults is 22-65% (Hoy et al., 2012),
with 40-70% of those experiencing LBP seeking health care (Joud et al., 2012). Despite increased efforts to understand LBP, knowledge of the underlying pathology and insights into optimising clinical outcomes have advanced little in the last 2 decades (Hancock et al., 2011).

It is assumed that a large portion of LBP is caused or influenced by biomechanical factors (Bogduk et al., 2013). As all spinal structures are potentially a source of LBP (Laplante et al., 2012) an accurate diagnosis is often difficult to make (Germon and Hobart, 2015). From a retrospective study of 170 patients undergoing diagnostic procedures the IVD and facet joints are the two most likely sources of LBP, with prevalence of 42% and 31%, respectively (DePalma et al., 2011). With age, degenerative changes such as osteophytes and arthritic changes or IVD prolapses can compress spinal nerves. The prevalence of lumbosacral radiculopathy is estimated to be 3-5%, distributed equally in men and women (Tarulli and Raynor, 2007). Improved diagnostic accuracy would confer cost advantages to the health system by enabling treatment to focus on particular sources of pain, and also would enable pathology-specific interventions to be grouped for clinical research.

During a structured clinical examination of the lumbar spine, a key component includes assessing the ROM (Littlewood and May, 2007) indicating spinal function, painful movement directions, response to intervention or even permanent impairment. The literature reports various movement assessments including functional activities of daily living (Bible et al., 2010), planar movements (Lee et al., 2011, Ha et al., 2013) and CMEs (Edwards, 1979, Brown, 1988, Monie et al., 2015). A lumbar CME is considered more informative than a planar movement examination (Edwards, 1979, Maitland, 1997) as this approach matches functional movements to the patient’s presenting
complaint and may reproduce symptoms that could in future help with sub-grouping and diagnosis (Hidalgo, 2015, Monie et al., 2016a).

Measurement of ROM has not been standardised in the past and this introduces difficulty in comparing outcomes. The present study used a validated, accurate 3-D motion tracking system MotionStar (Monie et al., 2015) with custom software LabVIEW V5.0 to measure a standardised eight direction CME (Figure 6.1). Proof-of-concept for the use of computer-aided CME and acceptable intra-session and inter-session reliability has been reported elsewhere (Monie et al., 2015).
Figure 6.1. Example of an asymptomatic CME radial plot in degrees of angular movement. Photographs illustrate the movement directions and end-points – flexion, FwLSF, FwRSF, LSF, RSF, extension, EwLSF and EwRSF.

Common lumbar pathologies include degenerative disc, disc protrusion (unilateral or centrally) and radiculopathy (Moisa et al., 2014, Goldberg et al., 2015); however, there does not seem to be any non-invasive movement-based clinical test which assists in discriminating these pathology sub-groups. It is understood that more than one structure can contribute to a patient’s low back presentation; however, an effort to understand the effects of individual structures seems a logical approach, before attempting to understand more complex multi-structure presentations.

The purpose of the present study was to determine the merit of computer-aided CME and change in self-report data for 18 patients who underwent neurosurgical intervention for confirmed lumbar spondylosis. Imaging, expert diagnosis and surgery were considered when comparing pre- and post-surgery CME data. Total change scores and z-scores (standard scores for normally distributed data) are reported. Each case’s CME was evaluated alongside their lumbar axial CT or MRI and matched NRR, in order to speculate on structural causes for atypical CME. A complete table of NRR data for males and females, 20-70 years of age, has been reported in Monie et al. (2015). Interpretation of CME data was expected to be more difficult in individuals with multiple, symptomatic structures, such as disc pain coexisting with facet pain and nerve root compression.

The hypothesis tested was the notion that specific CME patterns exist for symptomatic IVD (without protrusion), unilateral posterolateral disc protrusion and/or unilateral nerve root compression, and central posterior disc protrusion and/or bilateral nerve root compression. A second hypothesis predicted that all post-surgery CME patterns would
converge to the NRR. With the relatively novel method of using computer-aided CME to measure lumbar spine movement, and at this early stage of investigating CME, this study was performed to provide preliminary evidence, to test hypotheses, and more importantly, to generate hypotheses and inform future research on lumbar CME.

6.3. Methods

6.3.1. Recruitment and CME data collection
Thirty nine patients with LBP and/or leg pain diagnosed by neurosurgeons as originating from the low back were recruited and attended a CME review prior to surgery. Of these, 18 individuals received neurosurgical intervention and completed post-intervention examination at 14 weeks (Figure 6.2). Patients were recruited from a private physiotherapy practice (n=2) and a neurosurgery clinic in a tertiary hospital (n=16) – the sample comprised six males aged 49±14 years and 12 females aged 50±11 years.

After obtaining institutional ethics approvals, consent, and following familiarisation with test protocol, two skin mounted MotionStar sensors were placed over the volunteer’s S1 and L1 spinous processes. Data acquisition and post-processing is described in detail elsewhere (Monie et al., 2015). While standing, in an attempted neutral standing position, where lumbar lordosis was measured by calculating the difference in sagittal angle between the L1 and S1 sensors, the patient was asked to remember their most painful and most stiff CME movement direction, followed by instruction and guidance into each of the eight CME movement directions (refer to Figure 6.1). Maximal data values for ROM were recorded according to a predefined sequence – flexion, flexion with left side-flexion (FwLSF), flexion with right side-flexion (FwRSF), left side-flexion (LSF), right side-flexion (RSF), extension, extension with left side-flexion (EwLSF) and extension with right side-flexion (EwRSF).
All patients were tested prior to surgery and at an average of 14 weeks after surgery.

![Flow chart of participation.](image)

**6.3.2. Outcome measures**

A battery of self-report outcome measures were used to assess cases at each examination visit (Chiarotto et al., 2015) – VASp and low back stiffness (denoted as VASs), the RMDQ and a SF-12. A VASs was included as a non-validated outcome measure, since clinical measures often do not seek information regarding the effect of lumbar stiffness on function (Hart et al., 2013). A MCID of 30% was used for all self-report data (Deyo et al., 1998). CME data was also collected and expressed using z-scores. A variable can be converted to a z-score if the distribution of normal range for that variable is Gaussian. In this study, z-scores express each individual’s ROM relative to their age- and gender-matched NRR, indicating the magnitude of each movement direction, in SD (+ or -) from the NRR mean (Bonnick and Lewis, 2013). For the eight CME directions the maximum values were displayed in a radial plot and z-scores calculated for each direction and trial. For ease of comparison, it is noted that 68% of the distribution lies within plus and minus one (±1) SD of the mean (-1 ≤z ≤+1) and 95% lies within ±2 SD of the mean (-2 ≤z ≤+2). Total change scores in ROM and z-
scores are reported and a significant improvement was considered if the change was ≥ 1 z-score.

### 6.4. Results

Change scores (%) were derived for VASp and VASs in relation to their low back condition, SF-12 PCS and MCS, and RMDQ. Of the 18 patients, 10 were most symptomatic in a combined lumbar position (FwRSF, FwLSF, EwRSF or EwLSF). VASs scores were higher than VASp scores in 11 of the 18 cases. Total change scores for VASp, SF-12 PCS and RMDQ were clinically significant in 61%, 16% and 56% of all cases, respectively, (where there was no floor or ceiling effect) at the retest. Total change scores (%) for self-reports and the direction of change in outcomes are depicted in Figure 6.3.

The movement directions showing significant changes in z-scores were compared with specialist diagnosis, intervention and radiological imaging. Of the 18 cases, 17 had pre-intervention z-scores of < -1.5 in at least one CME direction, indicating that movement was in the lower end of its range as defined by the equivalent NRR distribution, even with consideration of multiple comparisons of the CME variables. Six out of 7 cases that received unilateral discectomy for posterolateral disc protrusion with or without nerve root compression showed ≥ 1 z-score of increased ROM in extension, 5 of which also increased ROM in EwSF. Only 2 of these 7 cases improved in flexion. Of the 7 cases that received intersegmental fusion surgery, there were inconsistent directional changes, however all reduced in ROM in flexion by at least 1.2 z-scores and all reduced in extension by at least 0.4 z-scores.

Preliminary results illustrated in Figure 6.8a-d, confirmed the hypothesis that specific CME patterns exist for symptomatic IVD, posterolateral disc protrusion with or without
unilateral nerve root compression, and central disc protrusion with or without bilateral nerve root compression. Eleven of the 18 cases demonstrated convergence of post-surgery CME towards their respective NRR values, supporting the hypothesis that for single pathology sub-groups, post-surgery CME patterns approximate towards the NRR. In contrast, both hypotheses were rejected in cases with multi-level and/or multi-structure pathology because it was not possible to report a single pathoanatomical CME pattern, and the CME ROM did not approximate towards the NRR. Summary data of each case are reported in Table 6.1.

Figure 6.4 illustrates pre- and post-CME z-scores for three different cases (A, H and L), selected due to their different diagnosis and structure/s surgically addressed – IVD, unilateral posterolateral disc protrusion, and central posterior disc protrusion, with or without nerve compression, in the six most informative CME movement directions – flexion, extension, LSF, EwLSF, RSF and EwRSF).

A radial plot and z-scores were derived for both CME trials in all 18 cases, in order to illustrate inter-session changes. The specific CME patterns of cases A, H and L are discussed in detail.
Figure 6.3. Total change scores (%).
Boxplot illustrating total change scores (Δ) for all patient’s self-report questionnaires, with minimum, median, 25th & 75th percentiles, and maximum values. In each series, post-intervention score minus pre-intervention score was used as the total change, expressed as a percentage. Arrows indicate the direction of improvement from baseline (0%). A MCID of 30% was used in this study. Self-report acronyms are defined in section 6.3.1.
Figure 6.4. Pre- and post-CME z-scores for three different cases
Pre- (blue) and post-intervention z-scores (red) for example cases: A (left discectomy for LBP and left hip pain)), H (bilateral discectomy for bilateral radiculopathy) and L (fusion for DDD), in the six most informative CME directions – flexion (a), extension (b), LSF (c), RSF (d), EwLSF (e) and EwRSF (f). CME acronyms are defined in section 6.3.1.
Table 6.1. Summary table for Study 3.
Tables reports each case’s age, key imaging features, diagnosis, neurosurgical intervention and total change scores for self-reports.

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Imaging</th>
<th>Specialist diagnosis</th>
<th>Intervention</th>
<th>Key outcome measures and CME directions</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>56</td>
<td>L2-3 left posterolateral IVD protrusion</td>
<td>Radiculopathy</td>
<td>left L2-3 discectomy</td>
<td>VASp 95%, RMDQ 80%, LSF, EwLSF, Extension</td>
</tr>
<tr>
<td>B</td>
<td>61</td>
<td>L4-5 left IVD protrusion compressing the exiting L4 nerve</td>
<td>Radiculopathy</td>
<td>far lateral left L4-5 microdiscectomy</td>
<td>VASp 41%, 10° increase to LL</td>
</tr>
<tr>
<td>C</td>
<td>28</td>
<td>L5-S1 left IVD protrusion</td>
<td>Radiculopathy</td>
<td>left L5-S1 discectomy</td>
<td>VASp 100%, VASs 76%, Flexion</td>
</tr>
<tr>
<td>D</td>
<td>44</td>
<td>L5-S1 bilateral facet and foraminal narrowing. Probable impingement</td>
<td>Radiculopathy</td>
<td>L1-2 laminectomy and discectomy</td>
<td>VASs 31%</td>
</tr>
<tr>
<td>E</td>
<td>57</td>
<td>L3-4 left IVD protrusion and right L4-5 IVD protrusion</td>
<td>Radiculopathy</td>
<td>right L4-5 microdiscectomy</td>
<td>VASp 83%, VASs 44%, RMDQ 75%, Flexion, LSF, RSF</td>
</tr>
<tr>
<td>F</td>
<td>58</td>
<td>L4-5 facets and left L5-S1 facet degeneration and L2-3 right IVD protrusion</td>
<td>Radiculopathy and canal stenosis</td>
<td>left L4-5 laminectomy and facetectomy</td>
<td>VASp 37%, VASs 44%, FwLSF</td>
</tr>
<tr>
<td>G</td>
<td>41</td>
<td>L5-S1 right posterolateral IVD prolapse</td>
<td>Radiculopathy</td>
<td>right L5-S1 microdiscectomy</td>
<td>VASp 83%, VASs 44%, RMDQ 79%, RSF, LSF</td>
</tr>
<tr>
<td>H</td>
<td>42</td>
<td>L4-5 IVD prolapse</td>
<td>Radiculopathy</td>
<td>L4-5 laminectomy and bilateral microdiscectomy</td>
<td>VASp 54%, LSF</td>
</tr>
<tr>
<td>I</td>
<td>58</td>
<td>L5-S1 spondylolisthesis and bilateral L5 nerve root compression</td>
<td>Radiculopathy</td>
<td>bilateral L5-S1 lateral recess decompression</td>
<td>VASp 35%, SF-12 MCS 32%, Extension, EwLSF, FwLSF</td>
</tr>
<tr>
<td>J</td>
<td>67</td>
<td>L4-5 facet arthropathy, DDD and lateral recess stenosis</td>
<td>Radiculopathy</td>
<td>bilateral L4-5 hemilaminectomy</td>
<td>SF-12 MCS 30%, LL 42°, Extension, EwLSF, FwLSF</td>
</tr>
<tr>
<td>K</td>
<td>31</td>
<td>L4-5 disc protrusion and bilateral neural compression</td>
<td>LBP and radiculopathy</td>
<td>L4-5 laminectomy and bilateral microdiscectomy</td>
<td>VASp 50%, SF-12 MCS 32%, LSF</td>
</tr>
<tr>
<td>L</td>
<td>49</td>
<td>L4-5 DDD</td>
<td>DDD</td>
<td>L4-5 fusion</td>
<td>VASp 78%, VASs 83%, RMDQ 33%, Flexion, LSF, RSF</td>
</tr>
<tr>
<td>M</td>
<td>44</td>
<td>L5-S1 spondylolisthesis with left nerve compression</td>
<td>DDD and radiculopathy</td>
<td>L5-S1 decompression and fusion</td>
<td>RMDQ 46%, 8° decrease in LL, Flexion, Extension</td>
</tr>
<tr>
<td>N</td>
<td>45</td>
<td>L5-S1 DDD and right neural foramen stenosis</td>
<td>DDD and radiculopathy</td>
<td>L5-S1 fusion</td>
<td>Extension, EwRSF, LSF, RSF</td>
</tr>
<tr>
<td>O</td>
<td>70</td>
<td>L4-5 degenerative spinal stenosis</td>
<td>Radiculopathy</td>
<td>L4-5 fusion</td>
<td>RMDQ 40%, 12° decrease in LL, Flexion</td>
</tr>
<tr>
<td>P</td>
<td>51</td>
<td>L4-5 spondylolisthesis and severe bilateral facet degeneration</td>
<td>LBP and radiculopathy</td>
<td>L4-5 fusion</td>
<td>VASp 41%, 12° increase in LL, extended directions</td>
</tr>
<tr>
<td>Q</td>
<td>40</td>
<td>L4-5 IVD prolapse</td>
<td>LBP and radiculopathy</td>
<td>L4-5 decompression and fusion</td>
<td>VASp 67%, VASs 68%, RMDQ 75%, 7° decrease in LL</td>
</tr>
<tr>
<td>R</td>
<td>64</td>
<td>L3-4 R-L radiculopathy</td>
<td>Radiculopathy</td>
<td>L3-4 laminectomy</td>
<td>VASp 95%, LSF, EwLSF, FwLSF, Extension</td>
</tr>
</tbody>
</table>

MCID = Minimal clinically important difference, DDD = degenerative disc disease, IVF = intervertebral foramen, LL = lumbar lordosis. For all outcome measure acronyms refer to the methods section.
6.4.1. **Case A: Unilateral discectomy for posterolateral disc protrusion**

A 56 year old male presented with severe left side anterolateral hip pain, mild LBP, antalgic gait with flexed low back and hips and decreased skin sensation over the medial knee and medial calf muscle (Figure 6.5a). MRI demonstrated a large left side posterolateral disc herniation with sequestration (Figure 6.5b,c).

CME showed a decreased ROM in extension, EwLSF and LSF compared to the relevant NRR (Figure 6.5d). The patient was referred for surgery. The patient had received almost instant relief of pain in the hip and low back, and total change score for decrease in pain in the hip at the final test was 95%. CME pre- and post-surgery ROM is illustrated in Figure 6.5d. Z-scores at final retest show an increase in ROM in the effected CME directions, towards mean values of the relevant matched NRR. Figure 6.4b, c & e illustrates the change in z-score for extension, EwLSF and LSF, respectively.
Figure 6.5. Case A’s pain diagram.
Figure illustrates the area of hip and LBP (red) (a), MR images showing sequestrated disc material (green arrow) (b) and left posterolateral disc protrusion (green arrow) (c) and CME radial plot illustrating decreased LSF, EwLSF and extension pre-surgery, with ROM increases in LSF, EwLSF and extension, post-surgery (d). CME acronyms are defined in section 6.3.1.
6.4.2. Case H: Bilateral decompression for LBP and R>L radiculopathy

A 42 year old female presented with a 24-month history of intermittent, severe LBP. This case stood with a left side lateral shift (Laslett, 2009) and complained of intermittent lower limb pain (R>>L) (Figure 6.6a). The patient was unable to stand due to pain in the lower limbs, and had almost complete pain relief in sitting or flexed lumbar positions. Conservative attempts to control pain and improve function, including over the counter medication, physiotherapy, epidural and nerve root sleeve injections and an exercise program, were refractory. MRI identified a marked right sided disc protrusion at L4-5 (Figure 6.6b,c). CME showed the greatest movement restriction in the directions of LSF and RSF, with EwLSF, extension and EwRSF also reduced (Figure 6.6d).

The patient was reviewed by a neurosurgeon and underwent bilateral laminectomy and discectomy with good effect. At reassessment 12 weeks post-surgery, lumbar spine movement had normalised to within 1 SD of her age- and gender-matched NRR (Figure 6.6d) and standing lumbar lordosis increased by 8° towards the NRR mean. Total change scores for pain (VAS), RMDQ and SF-12 PCS were significantly improved (≥30%). Z-scores for this case showed improvements towards zero (0), with the largest changes seen in the directions of FwLSF, LSF and RSF. The most symptomatic movement directions – extension causing LBP, and RSF causing right lower limb pain – were both asymptomatic at the 14-week post-surgery retest.
Figure 6.6. Case H’s pain diagram.
Figure illustrates the area of LBP, intermittent left lower limb pain (red) (a), sagittal section MRI showing posterior disc protrusion (green arrow) (b) axial L4-5, left paracentral disc protrusion (green arrow) (c) and computer-aided CME radial plot illustrating most restricted movement at pre-surgery in the direction of RSF, EwRSF, extension, EwLSF, LSF and FwLSF, marked change in FwLSF, LSF and RSF at final retest, and an age- (40-49) and gender-matched NRR (d). CME acronyms are defined in section 6.3.1.
6.4.3. Case L: Fusion for DDD causing CLBP

A 44 year old female presented with CLBP and intermittent lower limb pain (L>R) (Figure 6.7a). Lumbar flexion reproduced her LBP (VASp 4.3) and extension was reported as being very stiff (VASs 9.3). CME outcome measures showed that movement in the flexed directions – FwLSF, flexion and FwRSF – and extended directions – EwLSF, extension and EwRSF – were all significantly reduced ($z \leq -2.0$) (Figure 6.4 and 6.7d).

MRI demonstrated bilateral L5-S1 pars defect, grade I (10mm) spondylolisthesis, severe stenosis of the bilateral L5-S1 IVF and left L5 nerve root impingement (Figure 6.7b,c). The patient underwent L5-S1 posterior decompression, discectomy and interbody fusion.

Reassessment at 13 weeks showed improvements in all flexed and extended CME directions (Figure 6.7d). Total change data (%) for self-reports at 13 weeks, confirmed clinically significant improvements ($\geq 30\%$) in RMDQ. Pain score in flexion was significantly improved; however, pain was reported in EwLSF, causing isolated left side LBP. Left lower limb pain had improved by 70\%.
Figure 6.7. Case L’s pain diagram.
Figure illustrates the area of LBP (red) (a), sagittal MR images showing L5-S1 spondylolisthesis (b) narrowing at the left L2-3 IVF (c) and CME radial plot with global increased movement after L5-S1 fusion surgery (d). Note there is no change to LSF post-surgery. This is attributed to the unchanged L2-3 pathology. CME acronyms are defined in section 6.3.1.
6.5. Discussion
In this study, we sought to examine whether there were discrete CME patterns for IVD, posterolateral disc protrusion or unilateral nerve root compression, and central disc protrusion or bilateral nerve root compression, in cases of lumbar spondylosis presenting for neurosurgery. We hypothesised that structure specific CME patterns would exist. Further, we tested whether neurosurgery intervention for LBP resulted in CME converging towards NRR values.

The examiners were aware that patients can present with symptoms from more than one pathoanatomical structure; however, in order to investigate a putative structure specific movement signature, we focussed on CME from those cases in which imaging displayed single level and where possible single structure pathology. This was done to control variables and movement changes due to other structures. Two unilateral discectomies cases (cases A and B), two bilateral discectomy cases (H, J) and two fusion cases (L, M) were used to illustrate this speculation. It was not the aim of this study to report lumbar CME patterns for multi-structure or multi-level cases.

The two unilateral discectomy cases presented prior to surgery, with a lumbar lordosis of 16° and 29° less than their respective NRR mean. This measure is not depicted in the CME radial plots, and should be considered when interpreting ROM changes, as the starting position will always present as the centre of a radial plot [0,0] (Figure 6.1), despite differences in lumbar lordosis between patients and if lordosis changes due to surgical intervention. Prior to surgery, z-scores for case A, in extension, EwLSF and LSF, were all <-2.0. All other CME directions were within 1 SD of the NRR mean. At 14 weeks post-surgery extension, EwLSF and LSF ROM had increased and were 1 SD closer to the NRR mean. In case B, the CME radial plot appeared relatively normal in
the extended directions, compared to the NRR; however, prior to surgery the patient’s lumbar lordosis was 3°, compared to the respective NRR mean of 32°. With an additional 10° recorded during CME extension total lumbar extension prior to surgery was 13°. In this case, standing lumbar lordosis increased by 10° at the 14-week retest, with an additional 21° of active ROM extension, resulting in total lumbar extension of 31°.

In case A, the reduced ROM on the left side, illustrated in the CME plot (Figure 6.5d) was putatively attributed to left side L2-3 posterolateral disc protrusion. The majority of the patient’s pain was in the anterolateral hip. Removal of the disc material provided reportedly immediate relief of hip symptoms and the ability to stand straight. Pre- vs. post-surgery CME demonstrates a 14° increase in standing lordosis, with a post-surgery lordosis equal to the NRR mean lordosis value of 36°.

Further studies with larger numbers of surgical cases would be required to test for clinically meaningful changes in lordosis, for comparison with CME radial plots. Figure 6.8a illustrates a proposed CME pattern for painful posterolateral disc protrusion and/or radiculopathy, which may cause LBP and/or ipsilateral lower limb symptoms, respectively.

In the two bilateral posterior decompression patients (Cases H and J), pre-surgery lordosis was less than 1/3\textsuperscript{rd} that of their NRR mean, with increases in lordosis of 8 and 30° at the 14-week retest, respectively. Once again, the CME plot did not demonstrate the effects of the lordosis change on ROM; however, when factoring in the increased lordosis between trials, the total amount of lumbar extension increased in both cases.

In case H, a large central posterior disc protrusion had resulted in mild LBP, severe lower limb pain, and decreased ROM in all extended CME movement directions, with
pain relief in the flexed directions. Pre-surgery CME radial plot demonstrated large restrictions in side flexed and extended CME directions, which were also the most symptomatic movement directions. Post-surgery, there was no lower limb pain and LBP was low (VASp=0.6) during CME.

Figure 6.8b illustrates a proposed CME movement pattern for a painful central disc protrusion with or without bilateral nerve root compression, causing LBP and/or bilateral lower limb symptoms, respectively.

Modest improvements were observed in the two fusion cases (L and M) and two factors are likely to contribute to this observation. Segmental fusion results in a global reduction in available range. Secondly, pain was decreased, therefore allowing the patient to move easier in all directions, resulting in a more symmetrical CME radial plot post-surgery (Figure 6.7d). In case M, the post-surgery trial demonstrated a 5° increase in lordosis and increased ROM by greater than 1 SD of the NRR mean in EwLSF and EwRSF. Post-surgery CME for Case L demonstrated larger increases in ROM, with an average z-score increase, towards their respective NRR mean of 1.1, in the flexed and extended directions.

In case L, the L5-S1 IVD is loaded and deformed in multiple movement directions (Zou et al., 2009). In certain of the eight cardinal directions of CME, this may trigger mechanical nociceptors within a sensitised anulus fibrous (Coppes et al., 1997, Brisby, 2006). This global reduction in CME, with z-scores ≤ -2.5 in flexion and extension, has been identified as a common presentation during our observation of cases receiving pain specialist epidural injection or fusion surgery for related IVD pathology (unpublished data).
In the majority of the fusion cases, CME showed globally reduced ROM. Figure 6.8c illustrates a proposed CME pattern for a painful degenerative disc which shows a global reduction of movement in all CME directions post-procedure.

While preliminary evidence for CME pain patterns is presented, additional surgical cases are required to verify these initial trends which reflect the sub-grouping described. These preliminary data could be used to derive effect sizes. The ideal cases would have single level disease of a specific structure as multi-segment disease and multi-segment interventions add complications to interpreting the CME assessment. The inclusion criteria for this study did not specify single level, single structure patients. As a preliminary investigation of lumbar CME in cases with mechanical LBP, all presentations satisfying our limited inclusion criteria were investigated for the purpose of informing future hypotheses and lumbar CME research.

The hypothesis that all cases’ CME would converge towards their respective NRR values was rejected for seven of the 18 cases. The clinically meaningful change in ROM (>30%) was not evident in five cases of discectomy and two cases of fusion. The reasons for this include the patient’s condition regressed, post-surgery complications, large changes in lordosis between trials, multiple segment pathologies and/or untreated structures at the level of discectomy, such as degenerative facet joints, decreased ROM due to fusion surgery, fear avoidance and hypervigilance. However, it should be noted that almost 2/3 of cases improved towards their respective NRR values for CME. A longer-term follow-up would assist in testing whether CME patterns improve further, apart from fusion cases.

Spinopelvic alignment, including lumbar lordosis and pelvic incidence, is an important factor to consider when attempting achieving sagittal balance, and should be considered when comparing pre- and post-surgery CME radial plots. Small changes in alignment
have been shown to result in large increases in facet compressive loads and large stress peaks in the posterior anulus of the IVD (Adams and Hutton, 1980). Mehta et al. (2015) reported that reduced lumbar lordosis is associated with pain, sagittal imbalance and poor surgery outcomes, whereas Sorensen et al. (2015) reported lumbar lordosis was greater in cases of LBP, compared to asymptomatic cases, further confirmation that spinopelvic alignment is a balancing act. Changes in lumbar lordosis are a recognised, important factor to consider when measuring lumbar CME given the influence of lordosis on sagittal balance, spinal kinematics and lumbar spine pathology.

### 6.5.1. Limitations

CME is not a level specific movement analysis providing only a global assessment of the L1 to S1 lumbar region. This observational study was designed to test two hypotheses, and more importantly generate additional hypotheses for future research based upon the possibility of specific movement signatures related to specific pathologies.

This study used a lumbar CME NRR of 159 cases, which may be considered low to claim normative data. Subsequent testing resulted in a NRR of 192 cases (Appendix VII). A comparison of the data in both NRRs, for both genders and all age groups using an unpaired t-test showed insignificant differences between the two NRRs (p>0.05). However, we suggest using the NRR (n=192) for future CME studies due to the putative benefit of increased numbers.
Figure 6.8. Example radial plots of 4 common CME presentations identified in this study.
Right side posterolateral disc or nerve root disease causing right side LBP or right side radiculopathy, due to inter-vertebral foraminal (IVF) stenosis (a), central posterior disc protrusion with or without central canal or bilateral IVF stenosis, causing LBP with or without bilateral lower limb symptoms, respectively (b), symptomatic DDD without protrusion (c), multi-structure pathology such as DDD and right side posterolateral disc protrusion (d).

6.6. Conclusion
This preliminary study reports the novel utility in using CME and self-report data in comparison with an age- and gender-matched NRR to investigate mechanical LBP requiring neurosurgical intervention. Specific CME movement signatures for: IVD pain, unilateral posterolateral disc protrusion with or without unilateral nerve root compression, and central disc protrusion with or without bilateral nerve root compression are proposed. In almost 2/3 of cases post-surgery CME converged towards the NRR. The remaining cases all showed complications which may have mitigated this outcome.

Conflicts of interest None.
7. **Study 4 results: Clinical reasoning for CME of mechanical LBP**

7.1. **Abstract**

**Objectives:** The assessment and diagnosis of LBP is a clinical challenge, with up to 90% of patients being labelled non-specific (Carlsson and Rasmussen-Barr, 2013) meaning no identifiable structure has been implicated (Smith et al., 2014). NSLBP leaves the clinician speculating the pathoanatomical diagnosis in order to inform an appropriate treatment and management plan. Clinical reasoning is a cognitive process required when processing information obtained from a patient in order to speculate on the pathoanatomical diagnosis of the patient’s symptoms. Clinical reasoning is facilitated by all aspects of a patient’s presentation, including age, clinical history, posture, and movement presentations. Improving the clinicians’ ability to assess a patient with LBP, clinically reason the possible diagnoses, and provide a provisional diagnosis for the benefit of triaging into suitable clinical pathways, is considered a priority area in the management of LBP (Stynes et al., 2016). This study hypothesised that physiotherapy clinicians will be able to interpret CME photographs of a model patient demonstrating atypical movement due to mechanical LBP, and formulate diagnoses which align with the two most common lumbar pathoanatomical conditions, namely IVD and facet joint dysfunction, and nerve root compression after learning about specific CME patterns which have been proposed to be suggestive of these structures.

**Methods:** To assess for developing clinical reasoning, 13 post-graduate physiotherapy students were presented with montage images of three different idealised, abnormal lumbar CME movement patterns and asked to hypothesise the pathoanatomical source(s) in each case, based purely on the CME pattern, and assuming the subject in
the images experienced mechanical LBP (Test 1). A retest was conducted after general principles of CME were introduced (Test 2) and after the students were introduced to the concept of radial plot CME movement patterns and how these changed when loading specific abnormal lumbar spine structure(s) (Test 3). The abnormal CME pattern for select structures provided the students with specific CME movement signatures suggestive of pathoanatomical structure(s). Correct student answers were considered as those structures which were consistent with our preliminary CME patterns described in Studies 2 and 3. Associations between learning occasions were examined using the Student t-test.

**Results:** This study showed that after obtaining baseline scores, the scores received after teaching general principles of CME did not show a statistically significant change (Test 1 vs. Test 2, p >0.21). However, there was statistically significant change in mean scores between Test 1 and 2 occasions, and mean scores after describing the specific CME movement patterns and proposed structures (Test 3)(p<0.00002 and p<0.00006, respectively).

**Findings:** Teaching general principles of CME alone did not improve the ability for post-graduate physiotherapy students to hypothesise potential structure(s) causing atypical mechanical LBP. However, students’ responses showed a significant alignment with the most frequent reasons for LBP (DePalma et al., 2011) and the specific CME patterns and pathoanatomical structure(s), after receiving instruction based on preliminary evidence reported in Studies 2 and 3.

**7.2. Introduction**

Consensus guidelines for the management of LBP recommend a physical assessment, which includes a movement-based examination (NHMRC, 2004a, SAH, 2011). A basic
active movement examination involves asking the patient to move their low back in the three cardinal planes – sagittal, coronal and transverse. Edwards (1979) first described lumbar CME and proposed it may be more informative than planar movement examinations because the CME movements closely resemble functional movement of the spine. However, until our recent studies (Chapters 4-6) there is no evidence of attempting to develop a CME NRR, using a NRR to identify atypical CME movement patterns, using atypical CME movement patterns to hypothesise the source(s) of symptoms, and using CME with a NRR as an outcome measure post-intervention. In this study, the phrase ‘specific CME movement pattern, suggestive of structure(s)’ refers to an abnormal CME patterns suggestive of facet, disc, or nerve root compression.

A large proportion of LBP is influenced by painful structure in the lumbar spine (Mieritz et al., 2012, Wu et al., 2014). The most common causes of LBP include the IVD and facet joints, with prevalence rates estimated at 42% and 31%, respectively (DePalma et al., 2011). As facet joint pain is the most readily treated condition, it is worthwhile to consider this in the initial assessment of a patient presenting with LBP, especially if the patient is elderly (Laplante et al., 2012, Hutson and Ward, 2016). The gold standard for diagnosis, of two or three anaesthetic blocks, is not practical in the clinical setting due to the number of attempts required, time and cost (Hutson and Ward, 2016).

In the clinical setting, after taking a detailed history and performing a structured physical assessment, the clinician may have sufficient information to make an informed provisional diagnosis (Monie et al., 2016b). Clinical reasoning is the cognitive process, or ‘thinking’ used in the evaluation and management of a patient (Jones, 1992). Clinical reasoning errors, which may lead to misdiagnosis or lack of diagnosis, continue to
account for significant morbidity despite evidence-based guidelines for the treatment of LBP (Pinnock and Welch, 2014, Monie et al., 2016b). Competence in clinical reasoning is acquired by supervision with effective feedback, and trainees can learn clinical reasoning effectively if teachers provide guidance in making diagnostic decisions (Pinnock and Welch, 2014). Therefore, a structured CME-based assessment of a patient presenting with LBP, specific movement patterns with proposed pathoanatomical diagnosis, and logical clinical reasoning processes, has the potential to identify the source of symptoms and inform intervention and planning decisions.

Brown (1988) speculated that lumbar CME gives a greater appreciation of the likely source structure resulting in LBP from its anatomical location in the motion segment. Preliminary specific CME movement patterns suggestive of pathoanatomical structure(s) for mechanical LBP have been proposed (Chapters 5 and 6). These CME patterns with matched structures, for example within the context of a clinical assessment, may aid the clinical reasoning process and expedite a provisional diagnosis.

In the clinical setting, even with new knowledge, a clinician is encouraged to analyse and consider all of the patient’s assessment findings (Monie et al., 2016b). Two common models for clinical reasoning include hypothetico-deductive and pattern recognition. Hypothetico-deductive reasoning starts by hypothesising the cause of the presenting condition, followed by a process of elimination through assessment. Pattern recognition requires the clinician to make decisions based on previous, similar presentations. This involves rapid, non-verbal, intuitive cognition, a process used by the experienced clinician (Elstein, 2009, Langridge et al., 2015). In complex patients, vague assessment findings, or rare presentations, an experienced practitioner may use both models (Elstein, 2009).
In the musculoskeletal setting, clinical reasoning uses observation, evidence-based practice and/or theory, patient interaction, safety and accountability, and clinical intuition to develop an understanding of a patient’s presentation. It is a skill that begins during formative training and is refined through learning and experience (Langridge et al.). Understanding the cognitive components of clinical reasoning, assists critical evaluation of decision making, and aids in design of teaching methods to improve clinical reasoning (Jones, 1992). It is reported that hypothesis generation during clinical assessment is affected by several variables such as patient information, thoroughness, information analysis and time, yet these variables are not significantly different for hypothesis generation between novice and expert clinicians. The best indicator of correct diagnosis and improved patient management is the quality of the differential diagnosis concerning the cause or nature of the patient’s condition (Jones, 1992) and the response to treatment.

Therefore, the first aim of this study was to determine if clinicians could hypothesise pathoanatomical structure(s) contributing to mechanical LBP, by viewing a montage of three abnormal CME (Figures 7.1A-C). The second aim was to measure if clinician’s hypotheses changed after teaching general principles of CME, and following a discussion about the proposed specific CME movement patterns and structures implicated. In this study, pathoanatomical structures causing atypical CME representing the pattern in Figure 7.1A are the left facet joint, left posterolateral disc protrusion with or without compression of the left nerve root. The structure causing atypical CME representing the pattern in Figure 7.1B is the IVD, and the structures causing atypical CME representing the pattern in Figure 7.1C are bilateral facet joints, central posterior disc protrusion, or bilateral nerve root compression. It was hypothesised that by outlining the preliminary atypical CME patterns and structures implicated, reported in
our previous studies (Chapters 5 and 6), and then evaluating the clinical reasoning for three atypical CME movement patterns, students would not only consider the evidence-based, most common sources of mechanical LBP, but will then be able to match specific CME patterns to the pathoanatomical structure. Student responses were considered correct if the structures they hypothesised correlated with those specific CME patterns proposed in our two studies, and the recognised common causes of LBP – facet joints and IVD (Laplante et al., 2012), or atypical lumbar movement due to nerve root compression.
Figure 7.1. Specific CME patterns and the proposed pathoanatomical structures which result in these three atypical CME patterns.
Pattern A: left facet joint pathology (i), left posterolateral IVD (ii) and left nerve root compression (iii). Pattern B: sensitised IVD. Pattern C: bilateral facet joint pathology (i), posterior IVD (ii), bilateral nerve root sleeve compression (iii).
7.3. Methods

Thirteen post-graduate physiotherapy students volunteered to participate in this study (mean age 26 (3.2) years, with a mean of 8 years’ experience in musculoskeletal physiotherapy). All students were presented with montage images of a model performing three CME movement patterns (Figure 7.2-7.4). The three patterns represented common atypical CME patterns amongst 35 patients diagnosed with lumbar dysfunction, by pain specialists or neurosurgeons in a tertiary hospital. The three CME patterns and matched pathoanatomical structures are illustrated with radial plots in Figure 7.1A-C. For Figure 7.2, a mark was awarded for left facet joint, left nerve root compression, or left posterolateral disc pathology. For Figure 7.3 a mark was awarded for IVD pathology (typically degenerative), and for Figure 7.4 a mark was awarded for bilateral facet, bilateral nerve root compression, or central posterior disc pathology.
Figure 7.2. Montage of lumbar CME: restricted LSF, EwLSF and extension.
Figure taken in neutral standing (centre of each montage) and at the end-of-range of the eight CME directions, with reduced ROM in LSF, EwLSF and extension, was provided to the students (left). Radial plot illustrating the restriction relative to the age- and gender-matched NRR (right) was not provided to the students.
Figure 7.3. Montage of lumbar CME: restricted in all CME movement directions.
Figure taken in neutral standing (centre of each montage) and at the end-of-range of the eight CME directions, with reduced ROM in all of the eight CME directions, was provided to the students (left). Radial plot illustrating the restriction relative to the age- and gender-matched NRR (right) was not provided to the students.
Figure 7.4. Montage of lumbar CME: restricted in extension and side-flexion directions.
Figure taken in neutral standing (centre of each montage) and at the end-of-range of the eight CME directions, with reduced ROM in the extended movement directions EwLSF, extension and EwRSF was provided to the students (left). Radial plot illustrating the restriction relative to the age- and gender-matched NRR (right) was not provided to the students.
After analysing the images, the students were asked to speculate on structures causing the abnormal CME in each of the three movement patterns in priority order. Images one and three were weighted two marks – one for a single correct answer and a second mark for providing more than one structure consistent with our studies’ findings. Question two was weighted one mark because only one structure, a painful degenerative disc, produced this CME pattern in our studies. Therefore, a maximum attainable score for each test was five marks.

Test 1 was used to assess the first hypothesis for this study, that students were able to view three montage images of atypical CME, and propose pathoanatomical structures responsible for the atypical movement pattern, and obtain the students’ baseline score (BS). Test 2 was issued after the students were taught general concepts of CME in a lecture. This education included how to assess a patient using CME, how to record outcomes, how to recognise regular compression, regular stretch, and irregular CME patterns without speculating structure (Edwards, 1979, Maitland, 1997) and MT treatment approaches. Test 3 was issued after a one hour lecture, summarising the preliminary specific CME patterns reported in this thesis. The Student’s t-test statistic was used to test the second hypothesis, that students’ responses would align with the preliminary evidence and proposed specific CME patterns described in Studies 2 and 3.

The groups’ average scores for each test were used to calculate differences between occasions with P<0.05 used as the criterion to detect a meaningful change in outcome scores.

7.4. Results
All 13 students were able to comprehend and provide at least one answer for Test 1, 2 and 3, which agreed with our studies’ proposed specific CME patterns and structure(s). There was no statistically significant improvement in the ability for the students to
hypothesise the pathoanatomical structure(s) between baseline and general concepts scores (p=0.21); however, improvements in test 3, after teaching specific CME patterns and structures implicated, were statistically significant (p=0.0002). Overall group mean scores were 1.5, 1.8, and 3.0, for baseline, general concepts, and specific CME patterns scores, respectively (Table 7.1) (Figure 7.5).

Table 7.1. Student’s test scores and mean scores. Student scores at base line, after general concepts were taught and after specific CME patterns were taught. The mean test score of the whole group is also given.

<table>
<thead>
<tr>
<th>Student</th>
<th>Baseline</th>
<th>General concepts</th>
<th>Specific patterns</th>
</tr>
</thead>
<tbody>
<tr>
<td>Student 1</td>
<td>1</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Student 2</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>Student 3</td>
<td>1.5</td>
<td>1.5</td>
<td>2.5</td>
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<tr>
<td>Student 4</td>
<td>1</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Student 5</td>
<td>2</td>
<td>2</td>
<td>2</td>
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<tr>
<td>Student 6</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Student 7</td>
<td>2.5</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Student 8</td>
<td>2</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Student 9</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Student 10</td>
<td>2</td>
<td>2</td>
<td>4</td>
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<td>Student 11</td>
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<td>3</td>
</tr>
<tr>
<td>Student 12</td>
<td>1</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Student 13</td>
<td>2</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

| mean score | 1.5 | 1.8 | 3.0 |
Figure 7.5. **Bar graph showing the individual change between test scores.**
The overall group trend is illustrated (black) from Baseline score, after being taught General concepts and after being taught specific CME patterns.

### 7.5. Discussion
For the purpose of this study, the students were only taught CME movement patterns for these three structures. There may be other structures, such as painful bone, dura, ligaments and muscle, not investigated in our studies, which result in CME movement patterns. Additionally, the preliminary categorisation proposed in this thesis does not rule out occult pathologies such as tumour, co-existing sacroiliac joint or hip pathology, which may masquerade as atypical lumbar CME.

Although more possibilities for pathoanatomical structures causing atypical CME could have been proposed by the clinicians in this study, it is reported that between three and five diagnostic hypotheses are common, and this may be linked to various factors,
including short-term memory (Elstein, 2009). Further validation, with independent studies are required as no gold standard exists for specific CME movement patterns for structure(s). Elstein (2009) states that both number of cases and clinicians have to be adequately sampled to test clinicians’ knowledge and clinical reasoning.

After considering the prevalence of anatomical structures implicated in CLBP, Hutson and Ward (2016) state that, for every 10 patients seen clinically with CLBP, on average four will have discogenic pain and four will have facet pain. Prevalence figures are important, and can be used to guide clinical decision making (Hutson and Ward, 2016). Knowledge of the two most common causes of mechanical LBP, facet and disc (Laplante et al., 2012), and how facet, disc, and nerve root compression affect CME patterns, within the context of a structured clinical assessment, putatively improves the clinician’s ability to hypothesise the structures contributing to mechanical LBP.

This study did not demonstrate improved clinical hypotheses after learning general concepts of CME. One limitation of the general concepts lecture, was that ROM, specifically restricted ROM, was represented using a planar diagram (a cross configuration for flexion, extension and side-flexion) without human illustrations or models. Additionally, specific directional restrictions and implicated pathoanatomical structures were not discussed. Instead, the concept of stretch or compression patterns was taught. However, general concepts, taught by an experienced manual therapist, followed by teaching specific CME movement patterns and structures implicated, may have been required in such order, to trigger a learning response. Learning the general concepts will also be useful for the difficult presentation, where pattern recognition is not possible, and deductive reasoning is required.

Limitations in this study include the small sample size, and the method of delivering new knowledge. A cooperative learning environment, where students can discuss and
formulate a response as a team, generally outperforms individual efforts (Qin et al., 1995). Additionally, active participation during physiotherapy professional development, in the form of a workshop, tends to elicit greater improvements in practice behaviours compared to passive approaches such as viewing instructional material (Menon et al., 2009). The three tests in this study were completed without discussion between peers and information was presented in a lecture format. No feedback (grade or supporting comments) were given between tests, a component of learning which is known to enhance performance (Ladyshewsky).

By slowing down and verbalising clinical reasoning, experts can teach learners. When learners do the same, their reasoning can be assessed and improved with feedback (Pinnock and Welch, 2014). However, where individuals attach a personal significance to the task, intrinsic motivation is generally higher, which may drive willingness to achieve (Vedder and Veendrick, 2003). This may apply for the self-motivated clinician.

The results of this study with experienced graduate students show that clinicians are able to hypothesise the pathoanatomical diagnosis of mechanical LBP, by considering CME images. The results also demonstrate that teaching specific CME patterns suggestive of structure(s), through lectures and CME practice, improves the clinician’s ability to hypothesise structures commonly contributing to mechanical LBP, an ability recognised as being the best indicator of correct diagnosis and improved patient management (Jones, 1992).

7.6. Conclusion
Further studies, with more clinicians with varying levels of experience, and more specific CME patterns suggestive of additional structures, are required with contemporary teaching methods to evaluate the full potential of specific CME
movement patterns, suggestive of pathoanatomical structure(s) and the ability for clinicians to learn, clinically reason, and hypothesise lumbar spine structures causing mechanical LBP. The major caveat in this model is the recognition that a definitive diagnosis is not able to be made on the basis of CME alone. The objectivity of an idealised future study would be considerably enhanced with the independent access to imaging, diagnostic blocks and expert opinion.
8. Study 5 results: Cluster analysis of pooled data from Studies 2 and 3

8.1. Abstract

Objectives: NSLBP is described as LBP without an identifiable pathoanatomical source (Smith et al., 2014). Approximately 90% of those experiencing LBP are grouped in the NSLBP classification (Carlsson and Rasmussen-Barr, 2013). Germon and Hobart (2015) argue that management of LBP has lost sight of the basic clinical tenet of making a diagnosis, perhaps because it is very difficult. They state “…in the United Kingdom, and many other countries, some guidelines recommend care pathways that do not promote (and even discourage) attempts to make a diagnosis.” (Germon and Hobart, 2015:S5). Unfortunately, the consequence of not seeking a diagnosis, discouraged use of imaging, and labelling a patient’s condition as NSLBP, is that non-specific advice and non-specific treatments are offered (Germon and Hobart, 2015). Improving the clinicians’ ability to assess a patient with LBP, clinically reason, provide a provisional diagnosis, and potentially sub-group patients for the benefit of triaging into suitable clinical pathways is considered a priority area in the management of LBP (Stynes et al., 2016). Identifying clusters within cases of LBP can be used to guide future research and investigate the benefits of specific therapies (Fanciullo et al., 2003). This study aims to identify clusters using pooled data from studies 2 and 3, using relevant criteria, to answer the five secondary hypotheses. Additionally, pooled data is used to assess the third primary hypothesis, namely; that specialist intervention alters indices of lumbar CME and that CME ROM moves towards the NRR.

Method: Outcome data from Studies 2 (Chapter 5) and 3 (Chapter 6) were pooled (n=35) and used to test the five secondary hypotheses using cluster analysis. The third primary hypothesis of this thesis investigation, namely that specialist intervention alters
indices of lumbar CME and that CME ROM moves towards the NRR, was also tested using the pooled data and displayed using a pre- and post-intervention CME radial plot and box and whisker plot.

**Findings:** The results of the study in this chapter show that the third primary hypothesis can be accepted. On average the 35 cases of symptomatic mechanical LBP had post-intervention lumbar CME ROM values closer to the age- and gender-matched NRR, compared to the pre-intervention ROM.

**Interpretation:** The results of cluster analysis using multi-variate outcome data, pooled from Studies 2 and 3 were illustrated using Hierarchical cluster dendrograms. Comparison of clusters by specific variables allowed the five secondary hypotheses in this thesis to be tested. Three of the hypotheses were accepted and two rejected. These hypotheses, variables analysed, cluster dendrograms and results are described.

**8.2. Introduction**

Classification systems and sub-grouping LBP patients have been emphasised in the literature since the mid-1980s, with grouping of patients focusing on assessment, treatment or prognostic factors (Riddle, 1998). The identification of clinically relevant sub-groups of LBP is considered the number one LBP research priority in primary care (Stynes et al., 2016). Indeed, identifying specific populations based on age, CME movement patterns, personality, and method of intervention in LBP cases, may aid in selecting interventions based on the relative likelihood of treatment benefit. Such an approach has the potential for individual and population-level benefits, including improved patient outcomes and efficiencies in resource consumption (Foster et al., 2014). Of these approaches, cluster analysis of outcome data has the ability to identify potential LBP sub-groups.
The aim of this thesis investigation was to test fundamental hypotheses, consisting of four primary and five secondary hypotheses regarding lumbar CME in normal and symptomatic cases, for the benefit of future research investigating lumbar CME (Chapter 1). This chapter reports tests of the third primary hypothesis and the five secondary hypotheses.

The first aim of this study was to use pooled outcome data from Studies 2 and 3 (n=35), to answer the third primary hypothesis (Chapter 1), namely that CME changes after specialist intervention, approaching the NRR. It was assumed; that a case’s CME ROM would improve post-intervention if the pathoanatomical structure was identified and treated by the pain management or neurosurgery specialist.

The second aim was to test five secondary hypotheses using pooled outcome data from Studies 2 and 3 (n=35), using hierarchical cluster analysis. The secondary hypotheses and rationale for each were as follows,

8.2.1. Secondary hypotheses and rationale

**Hypothesis 1:** Analysis of cases presenting with mechanical LBP reflecting different pathoanatomical diagnoses will result in diagnostic clusters when analysing data for the flexion movement directions, depending on the location of the lumbar pathology relative to the sagittal axis of rotation (Appendix VII, Figure 5). The flexion movement directions for lumbar CME are flexion in the sagittal plane, FwLSF, and FwRSF.

Analysis of the ROM data using pre-intervention $z$-scores from the flexion directions for cases with DDD will cluster together, because flexion at a sensitised anterior disc will reduce ROM. Cases with lumbar dysfunction from posterior pathoanatomical structures, facet or nerve root compression, will form a separate cluster as they will be unloaded, allowing relatively asymptomatic flexion ROM. Justification for this
hypothesis includes the summary of literature provided by Adams et al. (2002), who reported resistance to lumbar flexion. In normal lumbar segments, during the first few degrees of lumbar flexion, the IVD and ligamentum flavum provide slight resistance. At half of full flexion, the IVD resists more than the posterior intervertebral ligaments, with the anterior IVD being compressed and the posterior, outer layers of the IVD tensioned. One of the fundamental factors of CME is that the patient ceases active movement (AROM) when symptoms are reproduced. It was proposed, that patients with symptomatic DDD will cease AROM relatively early, before reaching end-of-range flexion, whereas facet joint resistance and pain from tension of a symptomatic facet joint capsule is greater towards end-of-range flexion. Further justification based on the location of the IVD and facet joints in the spinal segment, is described in Appendix VII.

**Hypothesis 2:** Surgery cases will result in greater improvements in self-report outcome measures, compared with cases receiving pain management intervention. This was hypothesised due to surgery correcting a biomechanical fault and removing pathological material such as a prolapsed disc and/or bone, rather than efforts aimed solely at managing the symptom of pain.

**Hypothesis 3:** Surgery cases will achieve greater improvements in lumbar CME ROM, compared to pain management intervention. Once again, this was hypothesised because neurosurgical intervention addresses lumbar segmental instability, and removes pathological material, thereby enhancing movement. Pain management interventions are aimed at addressing pain through the control of inflammation and/or blocking pain without affecting the spinal architecture. Protruding disc material, bony spurs, and stenotic foramen are not changed with pain management intervention.

**Hypothesis 4:** The psychological state of patients in the pain management and neurosurgery cohorts will affect self-report outcome data. A review of the literature led
to the hypothesis that, those cases with abnormally low SF-12 MCS (<40), will report poor outcomes compared to those cases with normal SF-12 MCS scores (≥40), across the other self-report outcomes measured – VASp, VASs, RMDQ and SF-12 PCS, in the pain management and neurosurgery cohorts. Normal SF-12 MCS scores are 50 with a SD of 10 (SF-36.org, 2012). Abnormal SF-12 MCS scores are <40.

**Hypothesis 5:** Patients who are 50-70 years of age will make up the majority of the radiculopathy cases out of the pooled cohorts (age 20-70 years). This hypothesis was made due to the increased likelihood of age-related DDD and facet degeneration, leading to degenerative canal or IVF stenosis. It is hypothesised that cases aged 20-39 will predominantly present with disc pain, and only have radiculopathies secondary to large unilateral disc protrusions, due to acute prolapse of a more hydrated, gelatinous nucleus pulposis, and cytokines sensitising the spinal nerve root, including the most potent pro-inflammatory cytokine, tumour necrosis factor alpha (TNF-α) (Weiler et al., 2005).

### 8.3. Methods
Outcome data was pooled from the pain management cohort (n=17) in Study 2 (Chapter 5) and the neurosurgery cohort (n=18) in Study 3 (Chapter 6) (Appendix X).

To test the third primary hypothesis, the average z-score values from all 35 cases, pre- and post-intervention, in each of the CME movement directions, were calculated and plotted using a radial plot. Interquartile data – minimum, maximum, 25th and 75th percentiles, and mean values – were also calculated for z-score values from all 35 cases, pre- and post-intervention, and displayed in boxplots for each CME movement direction. Cluster analysis was performed using the R statistic software package, version
3.3.0. Euclidian distances were used to compare the spread of data between variables, and hierarchical design was used to display cluster dendrograms.

Multivariate data were used to test each of the secondary hypotheses: cluster analysis to test hypothesis 1, compared all pre-intervention flexion direction z-score data for CME – FwLSF, flexion and FwRSF – and displayed the data by pathoanatomy, typically facet, disc, and nerve root compression. For hypothesis 2, analysis compared all self-report outcome data – VASp and VASs, SF-12 PCS, and SF-12 MCS, and the RMDQ, – and displayed the data by intervention, pain management or neurosurgery. To test hypothesis 3, cluster analysis was performed using the z-scores from all eight lumbar CME movement directions, and displayed the data by intervention – pain management or neurosurgery. Hypothesis 4 was tested by analysing all pre-intervention self-report outcome data – VASp, VASs, SF-12 PCS, SF-12 MCS, and RMDQ – and displayed these by cases scoring less than normal, <40, or within normal, 50±10, for SF-12 MCS scores. Cluster analysis to test hypothesis 5, compared all cases by age and pathoanatomical diagnosis. The structure(s) treated in cases from the youngest 2 decades, 20-40 years, was compared with the structure(s) treated in the oldest 2 decades, 50-70 years.

8.4. Results
Radial plot of the average z-score values for all 35 cases, pre- and post-intervention, in each of the CME movement directions, illustrated that the mean values moved closer to the NRR (Figure 8.1A). When examined cases-by-case 70% of cases, 11 from the pain management cohort and 14 cases from the neurosurgical cohort demonstrated that the z-score for the worst CME movement direction, which matched the pathoanatomical diagnosis, moved closer to the NRR. Interquartile ranges for the same data, displayed as boxplots, show that in the majority of CME movement directions, the post-intervention
z-scores’ maximum, minimum, 25th and 75th percentiles, also moved closer to the NRR (Figure 8.1B). The third primary hypothesis, that lumbar CME approximates the NRR post-intervention, is accepted on a group level, but not on an individual case level.

Figure 8.1. **Average values for z-scores pre- and post-intervention.**
Figure shows data from all 35 cases and the NRR (A) and boxplots for each CME direction (B), show that on average the 35 cases’ CME migrated towards the NRR post-intervention – 70% of cases improved on CME in the direction consistent with the diagnosed pathology.

To test the five secondary hypotheses in this study, cluster analysis of pooled outcome data are presented in cluster dendrograms (Figures 8.2-8.6).

For hypothesis 1, cluster analysis of lumbar CME z-scores for all flexion movement directions – flexion, FwLSF, and FwRSF – produced two large clusters of 14 and 16 cases, respectively, one relatively small cluster of four, and one radiculopathy case as a suspected outlier. The cluster of 14 cases contained predominantly radiculopathy cases and no cases of only DDD or diagnosed disc pain. The cluster of 16 was a mix of all
pathoanatomical diagnoses with four of the six facet joint cases. The third cluster, of four cases included three cases where the LBP was attributed to pathological disc (Figure 8.2).

For hypothesis 2, cluster analysis compared total change (%Δ) outcome data from all self-report variables – VASp, VASs, SF-12 and RMDQ – and displayed the data by intervention. The dendrogram highlighted four clusters (Figure 8.3). Three clusters contained an approximately equal number of neurosurgery and pain management cases, and a fourth cluster of eleven cases contained seven neurosurgery cases.

Hypothesis 3 was rejected, since cluster analysis using total change (%Δ) in ROM data for all eight CME movement directions failed to demonstrate an obvious difference between clusters (Figure 8.4).

For hypothesis 4, cluster analysis of all self-report outcome data, displayed by pre-intervention SF-12 MCS score, produced three clusters (Figure 8.5). The largest cluster with 16 cases contained 13 cases with pre-intervention SF-12 MCS within the normal range. The second largest cluster, containing 11 cases, had four cases of normal SF-12 MCS scores. The third cluster of eight cases contained six cases with lower than normal SF-12 MCS scores.

Hypothesis 5 was rejected, since cluster analysis did not demonstrate that cases >50 years of age were predominantly treated for radiculopathy (Figure 8.6). Analysis did, however, produce three clusters of pathoanatomical diagnoses by age group – 28-45 years (n=11) containing eight cases of radiculopathy, 48-58 years (n=11) with no obvious pathoanatomical diagnosis dominating the cluster, and 61-70 years (n=13) with seven cases of radiculopathy and four of the five cases of bilateral facet joint pain.
Figure 8.2. Cluster analysis dendrogram of the ROM data using pre-intervention z-scores from the flexion directions – FwLSF, flexion, FwRSF – displayed by pathoanatomical diagnosis.

Two large clusters of 14 and 16 cases are evident. The cluster of 14 cases (right) contains predominantly radiculopathy cases, with no cases of solely DDD or diagnosed disc pain. The cluster of 16 (centre) is a mix of all pathoanatomical diagnoses, containing four of the six facet joint cases. The third cluster, of four cases (left) included three cases where the LBP was attributed to pathological disc.
Figure 8.3. Cluster analysis dendrogram displaying percentage change (%) self-report outcome data, by intervention – pain management and neurosurgery.

Three clusters contained a relatively equal number of neurosurgery and pain management cases, and a fourth cluster of eleven cases contained seven neurosurgery cases. Reason for these clusters is not evident from the dendrogram alone. Investigating elements within each cluster may provide insight as to why these clusters exist, such as the details of the interventions – rhizotomy, cortisone injection, fusion, or discectomy, or severity of the condition prior to intervention.
Figure 8.4. Cluster analysis dendrogram displaying total change in ROM data (%Δ) for all eight CME movement directions, by intervention – pain management and neurosurgery.

Other than two outliers (left), change in ROM data alone does not separate the 35 cases by intervention – pain management and neurosurgery. Percentage change does not indicate where on a scale of ROM the changes were made. Further analysis would be required to investigate if ROM changes exist between interventions, such as early ROM versus late ROM, relative to the NRR.
Figure 8.5. Cluster analysis dendrogram displaying all total change (%Δ) self-report outcome data, by pre-intervention SF-12 MCS scores. The largest cluster with 16 cases contains 13 cases with pre-intervention SF-12 MCS within the normal range (≥40). The second largest cluster, containing 11 cases, has four cases of normal SF-12 MCS scores. The third cluster of eight cases contains six cases with lower than normal SF-12 MCS scores (<40). The dendrogram shows that total change in self-report outcomes – pain, stiffness, physical health and disability, are effected by abnormal mental health status.
Figure 8.6. Cluster analysis dendrogram displaying subjects by age and pathology (diagnosis).
The cluster containing cases of age 28-45 years (left) contains a majority of radiculopathy cases. The cluster containing the cases of age 61 to 70 (middle) contains all but one of the bilateral facet joint cases. The cluster containing cases of age 45 to 58 has mixed pathologies.
8.5. Discussion
This chapter sought to test the third primary hypothesis in this thesis investigation, and to raise five secondary hypotheses to be tested using cluster analysis of pooled outcome data from cases receiving specialist intervention for mechanical LBP (Chapters 5 and 6). The outcome data were collected from pre- and post-intervention self-report questionnaires – VASs, VASs, SF-12 and RMDQ, and lumbar CME ROM data.

Hypothesis three, of the primary four hypotheses, stated that CME changes after specialist intervention will approach the NRR. Although reporting the effectiveness of intervention was not the aim of this thesis investigation, there is evidence to suggest that on average, lumbar CME normalises towards the age- and gender-matched NRR at 14 weeks post pain management or neurosurgical interventions.

Interestingly, only one of the post-intervention maximum values (FwLSF) was from the neurosurgery cohort. All other maximum values in the post-intervention boxplots came from four cases in the pain management cohort, with two of these cases contributing more than one maximal value, suggesting these outliers affected the z-score ranges (Figure 8.1B).

8.5.1. Secondary hypotheses
Hypothesis 1 was accepted, with results showing preliminary evidence for differences in lumbar CME pattern and structure, relative to the location of pathology in a lumbar motion segment. These results suggest that mechanical LBP originating from disc, facet, or nerve root compression can be sub-divided into three groups based on the CME restriction in the flexed movement directions. The disc pain cases and nerve root compression cases formed individual clusters, with a third, diagnostically heterogeneous group.
Hypothesis 2 was rejected because clusters analysis of self-report outcome measures failed to demonstrate an obvious difference between pain management and neurosurgery cases. Several factors may contribute to the heterogeneous clusters by intervention, including the fact that 17 of the 18 neurosurgery patients were treated for structural conditions resulting in radiculopathy, requiring more invasive intervention, including six cases of single segment, lumbar interbody fusion. Additionally, nerve root pathology secondary to compression may take longer to recover, and may not completely recover, compared to facet joint or disc pathology treated for inflammation alone, or ablation of pain pathways with rhizotomy in the pain management cohort. The pain management cohort of 17 cases contained 11 cases of diagnosed disc or facet pathology without diagnosed neural pathology.

It is likely that this analysis, with short-term outcome data, is not representative of long-term outcome. As patients returned to their normal lifestyle activities, pain management cases could have a relapse of symptoms from the same pathological structure(s). In cases treated with rhizotomy, axons within the medial branch of the dorsal ramus regrow and reunite, potentially resulting in a return of afferent pain impulses (Bogduk, 2008). Therefore, assuming patients did not receive further intervention, outcome data at 1 or 2 years post-intervention may produce different clusters, with the neurosurgery cohort reporting greater improvement on self-reports compared to those who receive pain management intervention. Interestingly, Parker et al. (2015) report that 3-month self-report outcome data, in cases receiving spinal surgery, correlates with 12-month measures overall in aggregate, but does not reliably predict 12-month outcome at a patient level.

Hypothesis 3 was rejected because analysis using total change in ROM data for all CME movement directions failed to show any obvious differences between clusters.
The reason for this may be due to the differences in pathoanatomical diagnosis treated in each cohort. The pain medicine cohort was predominantly treated for inflammation or putative facet pain, whereas the neurosurgery cohort was predominantly treated with the aim of decompressing nerve roots and/or stabilising hypermobile segments through fusion. Once the inflammation was treated in the pain medicine cohort, and neurosurgery cohort cases healed for 14 weeks, respectively, the differences in ROM changes between cohorts was not dissimilar. The ROM outcome data from the neurosurgery cohort was affected by the six cases who received fusion surgery in an effort to stabilise a vertebral segment. These six cases had reduced post-intervention lumbar CME ROM. These cases made up one third of the neurosurgery cohort, and may have negated any obvious differences between the pain management cohort and those cases receiving other neurosurgery interventions. Further sub-grouping of the pain management and neurosurgery cohorts, with larger numbers of cases per intervention, and per pathoanatomical structure, may produce additional clusters. Future studies with larger cohorts of cases receiving discectomy, fusion, and intervertebral canal decompression surgeries, and pain medicine epidural, nerve root sleeve injection, and rhizotomy procedures, are suggested.

Cluster analysis of all self-report outcome data produced three clusters when compared by SF-12 MCS scores. Two clusters contained cases predominantly scoring below average (<40) and the third cluster of 16 cases, contained a majority of normal pre-intervention SF-12 MCS scores (>40). Consequently, hypothesis 4 was accepted. The factors contributing to the SF-12 MCS scores and clusters by overall outcome suggest that low scores (<40) may require referral to a psychologist, to improve prognosis, as part of a multidisciplinary approach to address biopsychosocial factors (Kamper et al., 2015). In a study of 330 outpatients with NSLBP, of similar average age to the cases in
this thesis investigation, Coste et al. (1991) used cluster analysis to identify clinical subtypes. They found that 41% of the subjects had a psychiatric disorder and stated that psychological disturbances may account for many cases of NSLBP. The authors also state that this is controversial, and psychological disturbances could be a cause or a result of LBP. Coste et al. found that among the patients with psychiatric disorders, three clusters existed – a purely psychiatric, almost purely organic, and a mixed cluster, respectively.

Additionally, Aguerrevere (2010) used a personality inventory for 608 cases of spinal pain and reported three clusters which were not related to any spine-related organic factor. Instead, malingering status, education, ethnic background and legal status were different among pain sub-groups. The identification of patients with organic, versus non-organic LBP is an important process in clinical practice, allowing for appropriate triage and specific management. The use of lumbar CME as part of a structured clinical examination, along with consensus-based clinical reasoning, may facilitate the identification of mechanical LBP. Those patients not presenting with mechanical LBP signs and symptoms, including the proposed specific CME patterns in this thesis suggestive of pathoanatomical diagnosis, and with abnormal scores on mental health self-report could be identified in the primary care setting and triaged appropriately. The potential benefits of early identification and management of such cases include savings in time and cost.

Hypothesis 5 was rejected. There was no obvious clustering of radiculopathies for the >50 years of age cases. However, three clusters were displayed by age group. The cluster containing the youngest individuals, 28-45 years (n=11) included eight cases of radiculopathy. The cluster with cases aged 48-58 years (n=11) showed no obvious predominating pathoanatomical diagnosis, and the cluster with cases 61-70 years (n=13)
contained seven cases of radiculopathy and four of the five cases of bilateral facet joint pain. One reason why the youngest cluster group was predominantly made up of radiculopathy cases is due to IVD prolapse. Eight of the 11 cases in the 28-45 year old cluster received intervention for radiculopathy, with associated IVD prolapse (Appendix X). In these cases, despite the diagnosis being the nerve root, the IVD protrusion was the cause of the nerve compression. Disc protrusions are more common in the younger IVD which has a more hydrated, gelatinous nucleus pulposis. Additionally, TNF-α and other pro-inflammatory cytokines are present in damaged discs, which contribute to mechanical LBP and nerve sensitisation (Weiler et al., 2005). Interestingly, the cluster of cases aged 61-70 years old, contained all but one case of bilateral facet joint interventions, a sign of lumbar spine degeneration with age.

When assessing patients with LBP in clinical practice, after taking a detailed history of the presenting complaint and performing a structured physical examination with the use of CME, clinical reasoning should be informed by the patient’s age. The results of this cluster analysis, although preliminary, and using low cohort numbers, suggest that if a patient <50 years of age presents with clinical features including lumbar CME consistent with unilateral or bilateral radiculopathy (Figure 7.1A or C), the clinician should consider unilateral or central IVD prolapse, respectively. Additionally, if a patient >60 years of age presents with LBP, without radiculopathy signs and symptoms, and specific lumbar CME patterns consistent with reduced movement in the extended CME directions – extension, EwLSF and EwRSF (Figure 7.1C) º the clinician should consider bilateral facet joint pathology.

8.5.2. Limitations
Limitations for cluster analysis in this chapter include the small sample size of 35, which is further divided by intervention into 17 cases in the pain management cohort.
and 18 cases in the neurosurgery cohort. Additionally, each cohort included a heterogeneous sample of pathoanatomical diagnoses, with cases who received different interventions for the same structure, such as rhizotomy or cortisone injection for facet dysfunction. A limitation is recognised by the inability to control variables related to a patient’s psychosocial status, and beliefs and behaviours related to pain. These factors are known to play an important role in a patients’ experience and management of LBP (Deyo, 2015). Appropriate power testing on this sample of convenience was not performed. The preliminary evidence in this chapter was intended for use by future researchers investigating lumbar CME and/or clinical assessment of mechanical LBP. Future studies investigating clinical cases of LBP, should take efforts to select homogeneous samples of pathoanatomical diagnoses, and perform appropriate tests for power.

8.6. Conclusion
Outcome data from Chapters 5 and 6 have provided preliminary evidence to show that on average, in a combined group, CME ROM migrated towards the age and gender NRR following pain management and neurosurgery interventions, respectively.

Cluster analysis of lumbar CME z-scores for all flexion movement directions – flexion, FwLSF and FwRSF – produced three clusters by pathoanatomical structure, with one predominantly containing radiculopathy cases, the second a mixed cluster, and the third a small cluster of cases with IVD pain. Analysis of total change in outcome data from all self-reports displayed four clusters by intervention, with one cluster comprising predominantly neurosurgery cases, and the other three clusters containing mixed interventions. Cluster analysis using total change in ROM data for all eight CME movement directions did not produce any clusters by intervention. Analysis of pooled self-report outcome data produced three clusters by SF-12 MCS, with predominantly
normal scores, lower than normal scores, and a mixed cluster, respectively. Cluster analysis of pathoanatomy by age produced three clusters. A cluster of the younger cases contained a majority of radiculopathies, and the oldest cases contained the majority of the bilateral facet joint pathologies.

Cluster analysis of larger numbers of cases with specific pathoanatomical source structures is required to strengthen evidence that sub-groups exist within the LBP population. Identification of LBP sub-groups facilitates aspects of assessment triage, clinical reasoning, and appropriate patient management, and creates an informed avenue for further research.
9. Discussion

9.1. Background to this research

LBP is a major public health problem, and one of the most common globally (Hoy et al., 2012). The lifetime prevalence is as high as 85% and the reported annual incidence in adults is 22-65% (Hoy et al., 2012), with 40-70% of those experiencing LBP seeking health care (Joud et al., 2012). According to data from the Australian Institute of Health and Welfare, the cost of LBP in Australia in 2008-09, the most recent year for which data are available, was A$1.2 billion. Of this amount, A$464 million was attributed to out-of-hospital costs. The exact expenditure is not captured due to lack of comprehensive data, such as allied health costs (AIHW, 2016b). Despite efforts to understand LBP, knowledge of the underlying pathology and insights into optimising clinical outcomes have advanced little in the last 2 decades (Hancock et al., 2011). Germon and Hobart (2015) reported the often illogical approaches used when managing patients presenting with LBP, commented on the lack of effort to diagnose, readiness to label cases as NSLBP, and resulting non-specific advice and treatment offered. “…it is clear that primary research is required to determine the pathophysiological causes of back pain…” (Germon and Hobart, 2015).

Improved diagnostic accuracy confers cost advantages to the health system by enabling treatment to focus on particular sources of pain, and also would enable pathology-specific interventions to be grouped to facilitate studies into comparative clinical outcomes and cost efficacy.

During a structured clinical examination of the lumbar spine, a key component includes assessing the ROM (Littlewood and May, 2007) indicating spinal function, painful movement directions, response to intervention or even permanent impairment. The literature reports various ROM assessments including functional activities of daily
living (Bible et al., 2010), planar movements (Lee et al., 2011, Ha et al., 2013) and CME (Edwards, 1979, Brown, 1988). A lumbar CME is considered more informative than a planar movement examination (Edwards, 1979, Maitland, 1997) as CME has the potential to match functional movements to the patient’s presenting complaint and may reproduce symptoms that could in future help with sub-grouping and diagnosis (Brown, 1988, Barrett et al., 1999, Hidalgo, 2015).

Several questions naturally arise from a review of the literature on LBP, its assessment and diagnosis. Is CME a reliable and clinically efficacious examination? What is a normal CME? Is there a difference in normal CME between genders and across age groups? Are there structure specific CME movement patterns? Does effective treatment of the structure(s) normalise CME?

9.1.1. Thesis evolution

CME of the lumbar spine was first described and recommended for use by the late, Brian Edwards (Edwards, 1979). Lumbar CME was investigated by Barrett (1995), who identified reduced movement ROM in subjects with LBP compared to asymptomatic subjects. Barrett et al. (1999) state that due to their LBP cohort of 23 cases with mixed pathologies, diagnostic patterns were not recognised. They speculated that patterns may exist if patient sub-groups could be assembled with the same pathology or clinical presentation. More recently, Hidalgo (2015) recommended the use of CME, and reported reduced speed of movement (°/s) in cases with non-specific mechanical LBP. He concluded that the effects of MT treatment may be more objectively addressed using kinematic analysis.

The purpose of this thesis was to test fundamental hypotheses for the use of lumbar CME, and to stimulate questions and additional hypotheses for future investigation.
Four primary hypotheses were proposed and tested in this thesis, with an additional five secondary hypotheses for testing using cluster analysis of outcome data.

Two pilot studies and five major studies were conducted in sequential order, to test the primary and secondary hypotheses. These studies are discussed below, before addressing each of the four primary hypotheses and five secondary hypotheses, described in Chapter 1.

9.2. The studies in this thesis

9.2.1. Pilot studies
Pilot study 1: To test for asymptomatic and symptomatic lumbar CME movement patterns during this thesis investigation, a non-invasive 3-D measuring device was used. The first pilot study in this thesis examined the accuracy and validity of the MotionStar 3-D motion tracking system by using a tri-planar goniometer as a validated, accurate testing device. The details of this study are reported in Appendix III.

Pilot study 2: A second pilot study reported the reliability of using the MotionStar system to collect angular movement data, in degrees, with sensors placed at L1 and S1 spinal levels, during lumbar CME. Both intra-session and inter-session reliability were tested using 10 asymptomatic volunteers in each cohort. The details of this study are reported in Appendix V.

9.2.2. Chapter studies
Following the two pilot studies, five major studies were conducted. These are described in Chapters 4-8, respectively.
9.2.2.1. Study 1

Chapter 4 describes the first major study, which reports a CME NRR (n=151) for males and females, aged 20-69 years, and Appendix VII displays the final lumbar CME NRR (n=192). This age group was selected, because it captures the most prevalent years for LBP for both genders (Hoy et al., 2012). Volunteers were recruited from a local university (n=71), within a tertiary hospital (n=64) and community residents (n=57). During the collection of normal data, the SD of ROM across the CME movement directions, lordosis, BMI, genders and age groups were monitored. A stable SD with cohort populations of five versus ten subjects was observed for several CME ROM directions, indicating stability in the data estimate of biological variability. To further improve statistical power, subject recruitment continued to the final cohort population average of 19 subjects per age and gender decade (n=192) (Appendix VII). The latter is recommended for future use, due to the putative benefit of increased numbers in a NRR.

The NRR is used for comparison purposes in virtually all fields of science and is available for almost all joints in the body (Jahn, 1979). There are many asymptomatic reference ranges for planar movement of the lumbar spine (Burton, 1986, Chiou et al., 1996, Bible et al., 2010, Svedmark et al., 2012, Dreischarf et al., 2014); however, these vary due to different metrics, methods and materials. The NRR data from Chapter 2 was compared with data from other studies reporting asymptomatic lumbar ROM, using 3-D tracking devices (Pearcy, 1985, Barrett et al., 1999, Ha et al., 2013). It was difficult to make an informed comparison of these data, because of different examination methods, including lumbar movement directions and sequences; therapist handling and cueing active, passive, or active-assisted movement; commands, type of sensors and method of sensor fixation; and data reduction.
Establishing a lumbar CME NRR was necessary for the later studies reported in this thesis to allow comparisons leading to identification of atypical CME movement patterns from symptomatic cases, for both genders and ages 20-69 years. Without a sample of normal CME specific to age and gender, it would be impossible to discern abnormal CME and identify clinically meaningful change towards the normal range, particularly considering reports on the natural decrease in lumbar ROM with age (Dvořák et al., 1995, Dreischarf et al., 2014, McCarthy et al., 2015). The NRR also allowed for z-score calculations to be used, expressing symptomatic cases’ data relative to the age- and gender-matched NRR using SD.

Plotting mean values of the NRR data, in each CME movement direction for both genders and each decade of life, from 20-69 years of age, facilitated comparisons. Normal CME closely resembles a symmetrical pattern, with LSF and RSF being within 5° of difference and with the magnitude of flexion ROM at least 2.5 that of the ROM into extension (Appendix VII).

Study 1 (Chapter 4) also reported two cases treated with laminectomy and discectomy, and epidural cortisone injection, respectively. Pre- and post-intervention self-report questionnaires and lumbar CME were conducted to test for proof of concept – that lumbar CME is sensitive in identifying change post-intervention. In both cases, atypical CME was apparent when compared with the age- and gender-matched NRR. Cross-sectional imaging was used to diagnose the lumbar level and putative pathoanatomical structure contributing to symptoms. Both cases showed significant improvement in outcome measures for pain and disability, which correlated with their post-intervention CME data showing increased ROM, approximating the NRR.
9.2.2.2. Studies 2 & 3

Studies 2 and 3 (Chapters 5 and 6, respectively) tested more cases with symptomatic mechanical LBP, in a short-term test-retest study design, before and after pain medicine (n=17) and neurosurgery (n=18) interventions, respectively. A CME NRR of 159 cases was used in these studies. Additional data collection resulted in a final NRR of 192 cases. Comparison of the NRR (n=159) and NRR (n=192) showed no statistically significant difference (p>0.05). Due to the increased power with the additional 33 cases, the NRR with 192 asymptomatic cases is recommended for future use.

Before examining symptomatic cases in Studies 2 and 3, there was an expectation of testing predominantly patients with facet and/or disc pathology, consistent with the two most likely structures to generate LBP (DePalma et al., 2011). After the pilot studies and reporting proof of concept (Chapter 4), it became apparent that nerve root compression secondary to IVD prolapse or degenerative changes, was another common diagnosis. The studies in Chapters 5 and 6 were improved by investigating three common pathological structures – facet joint, IVD and spinal nerve root.

The preliminary evidence from these studies was used to test the third and fourth primary hypotheses – that CME changes after specialist intervention, resulting in a migration of CME towards the NRR, and that preliminary CME patterns would be identified, that were specific to facet pathology, IVD pathology, and nerve root compression, respectively.

Collectively, the cases’ atypical movement patterns from Chapters 5 and 6 were compared and contrasted. Cases demonstrating significant change in CME (>1 SD) and change in self-report outcome measures (>30%) were considered as those who had their pathoanatomical structures effectively treated. These cases were used when considering abnormal CME movement patterns, to test the possibility of specific lumbar
structure(s). All 35 cases were subsequently entered into a cluster analysis to determine whether CME could be used to predict clinical outcomes.

The proposed CME movement patterns were based on interventions that targeted three structures – the facet joints, IVD and lumbar spine nerve roots. These three structures were chosen, due to their reported likelihood of being the sources of LBP (Tarulli and Raynor, 2007, DePalma et al., 2011) and the higher frequency of interventions targeting these structures in the available cohorts.

9.2.2.3. Study 4

A fourth study (Chapter 7) was conducted to assess a clinical application of lumbar CME, by testing the effects of presenting the preliminary specific CME movement patterns and implicated pathoanatomical structure(s), to clinicians to stimulate clinical reasoning.

Low back problems are commonly treated in primary care settings, with the most important symptoms being pain and disability (Koes et al., 2006). Diagnosis of LBP is mainly focused on the triage of patients into specific or NSLBP. Approximately 90% of those experiencing LBP are grouped in the NSLBP classification, which in essence is based on excluding specific identifiable pathology (Carlsson and Rasmussen-Barr, 2013). Improved diagnostic accuracy would reduce costs to the health system by enabling treatment to focus on particular pathoanatomical structures.

Study 4 used the preliminary evidence from the pain management and neurosurgery cohort studies (Studies 2 and 3). The specific CME patterns (Figure 8.1 A-C) were shown to post-graduate physiotherapy students in montage images, before and after they received further post-graduate training, which included general concepts in CME, and finally after presenting the proposed pathoanatomical structure(s) in a lecture format.
On each occasion they were asked to nominate the likely structure(s) resulting in the atypical CME montages. The purpose of this study was to measure the clinician’s ability to recognise the three different specific CME patterns, and to use this to clinically reason and inform a provisional diagnosis.

Numerous randomised control trials, systematic reviews and guidelines are available for the diagnosis and treatment of LBP. However, the approximately 90% of all patients who are categorised as having ‘NSLBP’ have resisted further sub-categorisation that are reliable or clinically useful (Koes et al., 2006). This inability to diagnose, ultimately receiving non-specific intervention, is a concern. Therefore, it is worthwhile to consider if knowledge of lumbar CME in such patients can elucidate this problem.

CME is recommended as a clinical assessment for LBP (Edwards, 1979, Brown, 1988, Maitland, 1997). A complete assessment of the patient presenting with LBP, including the collection of a detailed history of the presenting problem, active movements, manual assessment, and if indicated, neurological screening and radiological imaging, is recommended (Chou et al., 2007, SAH, 2011, Deyo, 2015). Information gained from all components of a clinical assessment should be considered during a clinical reasoning process, before a provisional diagnosis is made. Recognising the specific CME patterns, with preliminary evidence to suggest pathoanatomical structure(s), provides additional information for the clinician to consider when examining a patient presenting with mechanical LBP. This added clinical information potentially facilitates a provisional diagnosis and intervention, instead of imposing the label of ‘NSLBP’ and providing pragmatic non-specific interventions.

9.2.2.4. Study 5

A final, fifth study, used pooled outcome data from Studies 2 and 3 (Chapters 5 and 6), to test the five secondary hypotheses of this thesis. Hierarchical cluster dendrograms
were used to display relationships between multiple variables and intervention, self-report surveys, and CME ROM.

The results of Studies 1-3, providing the intra- and inter-session reliability of lumbar CME, a CME NRR, and CME movement patterns for the most common structures contributing to mechanical LBP, have not previously been reported in the literature. Additionally, the results of Study 4 (Chapter 7) provide evidence that there is potential to improve clinical reasoning during the assessment of mechanical LBP, and facilitate a provisional diagnosis using lumbar CME. Such an examination of lumbar CME through the progression described above, contributes an original insight to the existing literature on CME of the lumbar spine, and the clinical implications.

9.3. Hypotheses

9.3.1. Primary hypotheses and major findings

9.3.1.1. Primary hypothesis 1
Computer-aided CME is a valid and reliable test for measuring lumbar ROM in single and combined planes of movement.

To test validity, repeated measures recorded while testing the MotionStar 3-D tracking device against a tri-axial goniometer of known accuracy, produced an average error of 0.6°. The CV for all data samples when the MotionStar was tested 10 times at three separate angles (20°, 40° and 60°), in each cardinal plane of movement (x, y, z) was ≤ 1%.

To test reliability, lumbar CME data from 10 asymptomatic subjects, in five intra-session trials, and five inter-session trials were recorded. ICC and 95% CI values suggest high to moderate reliability for each of the eight lumbar CME movement directions. The ICC(3,1) values for planar movement directions were ≥0.8, and for CME
directions \( \geq 0.6 \). CME of 10 asymptomatic subjects also demonstrated there was no warm-up or fatigue effect over five intra-session trials. This study’s reliability data were consistent with findings in the study by Mieritz et al. (2012) who systematically reviewed the reliability of various 3-D measurement systems used to assess the lumbar spine.

Computer-aided CME is a valid and reliable test for measuring lumbar ROM in single and combined planes of movement, and therefore a candidate technique for biomechanical measurements of the lumbar spine. The system was retested at each of the four testing locations, and demonstrated consistency over time (Appendix III, Table III.2). It was concluded that there was acceptable intrinsic reliability for the purpose of using the non-invasive MotionStar 3-D tracking system to measure lumbar CME.

9.3.1.2. Primary hypothesis 2

The second primary hypothesis was that CME ROM for normal subjects will decrease with age, and females will have greater ROM compared to males, per decade of life. The results of the study showed that for both genders, the magnitude of movement in all directions reduced with age (Figure 9.1). However, females did not have clinically significantly greater ROM. The average decrease in ROM in each CME movement direction, for both genders between the age of 20-69 years was 7.2° for flexion, 5.8° for flexion with SF, 10.0° for SF, 6.3° for extension, and 5.4° for extension with SF (Appendix VII, Table 2). Dreischarf et al. (2014) reported a significant difference in range of lumbar extension between genders in normal subjects, with females extending further than males between the ages of 20-49 years, and less so after the age of 50. The CME NRR data (Appendix VII, Table 1) does not support this finding in any of the CME directions. The results of Study 1 (Chapter 4) are consistent with the conclusion
by Dvořák et al. (1995) who reported no difference in 104 subjects between genders, for normal planar ROM.
Figure 9.1. The decline in ROM in all planes with age, observed when using the CME of the lumbar spine.
9.3.1.3. **Primary hypothesis 3**
The hypothesis that CME changes after specialist intervention, approaching the NRR, was accepted. In the majority of cases, lumbar CME did change after specialist intervention, approaching the NRR. Interestingly, not all patients’ CME improved in ROM, despite significant improvements in self-report questionnaires. The reasons for this in the pain medicine cohort may include the structure(s), facet or IVD, being inflamed and degenerative, all conditions which respond favourably to anti-inflammatory injection procedures. However, the restricting structure(s) and/or hypomobile joint remains, and thus only modest changes to CME were recorded. Examples of this include cases C, M, N and L from the pain medicine cohort (Chapter 5). A similar outcome was evident in cases O and R in the neurosurgery cohort, who received lumbar fusion surgery (Chapter 6). In cases O and R, the painful segment was stabilised, resulting in multi-directional decrease in CME ROM and decreasing mechanical stresses at the IVD and facet joints.

9.3.1.4. **Primary hypothesis 4**
The hypothesis that preliminary lumbar CME patterns will be identified, which are specific to facet pathology, IVD pathology and nerve root compression, was rejected. Preliminary CME patterns were indeed identified; however, these were not specific to a single pathoanatomical diagnosis. Instead, the preliminary evidence supports three specific CME movement patterns, with two of these patterns suggesting more than one pathoanatomy. Figure 9.2 shows that for patterns A and C, the pathoanatomy could be facet, disc and/or nerve root compression. Each of the three CME patterns proposed in this thesis was derived from cases who presented with similar signs and symptoms, were diagnosed with the same pathoanatomical structure, and responded favourably to intervention. Four cases who demonstrated unique CME patterns or received multi-
structure or multi-level interventions, were not used when considering specific CME patterns for single pathoanatomical diagnoses. Interpretation of multi-structure and multi-level cases’ data made it difficult to differentiate which direction(s) of a CME pattern was affected by each intervention. In particular, cases in the surgery cohort, with data demonstrating large changes in spinopelvic alignment between test occasions, were similarly difficult to interpret.

The preliminary evidence from the pain management cases, and neurosurgery cases led to three proposed specific CME movement patterns, which are suggestive of unilateral or bilateral facet, disc, or nerve root compression (Figure 9.2 A-C).
Figure 9.2. Specific CME patterns suggestive of pathoanatomical source structure(s).

The specific CME patterns are: (A) unilateral, posterior movement restrictions, suggestive of ipsilateral facet pathology (A(i)), ipsilateral posterolateral disc pathology (A(iii)) and ipsilateral nerve root compression (A(iii)). (B), Reduced CME movement in all directions, suggestive of DDD (B(i)). (C), Bilateral posterior movement restriction with CME, suggestive of bilateral facet pathology (C(i)), bilateral posterior disc pathology (C(ii)) and bilateral nerve root compression (A(iii)).
Decreased ROM, in all planar movement directions for subjects with lumbar stenosis, disc prolapse and DDD, was reported by McGregor et al. (1997). They also concluded that lumbar motion characteristics are not sensitive enough in categorising individual patients, limiting the usefulness of this aspect of the clinical assessment. In contrast, the results from the present thesis study suggest that lumbar CME using a NRR has sufficient sensitivity, on an individual patient basis, to provide valuable information during clinical assessment and monitoring change post-intervention. Additionally, the preliminary evidence shows that lumbar CME can be used to identify sub-groups of mechanical LBP by pathoanatomical region in the lumbar segment.

According to Brown (1988) a CME NRR may have clinical assessment and treatment benefits, although his hypotheses were never tested and reported. The results of this thesis investigation confirm two of Brown’s statements – CME patterns do change when pathology is present, and CME adds value to the process of identifying a potential pathoanatomical structure. However, this study does not agree with Brown’s prediction that each unique pathoanatomical structure has a different CME pattern. It is worth mentioning that the text by Maitland (1997) and articles by Barret et al (1999) and Brown (1988), describe an alternate method of expressing CME restriction, without specifying potential structure, by naming restriction as a stretch or compressive pattern. For example, left side LBP experienced during right side-flexion and also during flexion would be considered a stretch pattern on the left. This general interpretation of CME patterns is consistent with our findings, in that they are all compressive. By examining a greater number of cases with different pathoanatomy, stretch patterns may also exist.

A study by Dewitte et al. (2015) stated that there is no persuasive scientific evidence to underline the discriminative value of clinical tests for LBP. In contrast, the results in this thesis provide support for the use of lumbar CME in the clinical setting. There is
preliminary evidence for the proposed three specific CME patterns suggestive of three pathoanatomical structures. Facet, IVD and nerve root compression, combined with knowledge of pain patterns for these structures, and a structured clinical assessment, has the potential for discriminative value and improved provisional diagnosis.

9.3.2. Five secondary hypotheses and post-hoc examination findings
These following five secondary hypotheses were generated to test pooled outcomes data from Studies 2 and 3 using cluster analysis:

1. Patients presenting with low back dysfunction from different pathoanatomical structures will result in clusters by pathoanatomy when analysing data for flexion, depending on the location of the lumbar pathology relative to the sagittal axis of rotation (Appendix VI, Figure 5).

2. Surgery cases will result in greater improvements in self-report outcome measures, compared with cases receiving pain management intervention.

3. Surgery cases will achieve greater improvements in ROM, compared to pain management intervention, primarily because the restricting anatomical structure is removed.

4. A review of the literature led to the hypothesis that the psychological state of patients will have an effect on self-report outcome data. Those with low SF-12 MCS will score lower than those with average SF-12 MCS scores on the self-report outcome measures.

5. Older subjects, >50 years of age, will make up the majority of the radiculopathies due to the increased likelihood of age-related DDD and facet degeneration, leading to degenerative canal or IVF stenosis.
The concept of studying and understanding the similarities and differences between subgroups of patients with LBP, for the benefit of triaging into suitable clinical pathways, is considered a priority area in the management of LBP (Stynes et al., 2016).

Cluster analysis of short-term outcomes data from this thesis, reported in Chapter 8, provided further insight into – mechanical LBP; how lumbar CME can differentiate pathoanatomical source structures; self-report and lumbar CME ROM outcome arising from pain management and neurosurgery intervention; how psychological differences affect self-report outcomes, and how pathoanatomical facet, disc, and nerve root structures change with age.

These factors were investigated for the benefit of informing future research. It is conceded that larger cohorts of patients with single source structure pathologies would be required to formally test these hypotheses. Despite limitations with the available pooled data, a preliminary investigation of factors contributing to outcomes was deemed important.

Secondary hypotheses one, four and five, have primary care clinical implications, particularly for assessment, clinical reasoning and provisional diagnosis. Results from secondary hypothesis one facilitate differentiating degenerative disc pain from facet, and nerve root compression. Results from secondary hypothesis four inform our management of patients presenting with LBP and SF-12 MCS scores below average, and results from secondary hypothesis five inform clinical reasoning, and provisional diagnosis, after assessing patients of different ages, presenting with LBP and reduced lumbar CME in the extended movement directions suggestive of facet, posterior disc protrusion and nerve root compression.
Secondary hypotheses two and three essentially compare short-term self-report and CME ROM outcomes, respectively, in cases receiving pain management and neurosurgery intervention. However, care must be taken not to extrapolate the implications of these two hypotheses given the invasive nature of neurosurgery and variable post-operative recovery, healing and restoration of functional, pain-free spinal mobility.

9.4. Limitations

Identification of study limitations is important to avoid over-interpretation and inappropriate extrapolation. It also assists in identifying factors to consider for future research. The critical limitations relevant to the study series are elaborated here.

Computer-aided CME data in this thesis was collected by placing a skin mounted sensor at the L1 and S1 levels of the spine (Figure 4.2B,C). The data is therefore an indication of the resultant movement of the lumbar spine, and not inter-segmental movement. Differences in upper and lower lumbar movements and/or compensatory strategies by neighbouring lumbar segments are not captured. Additionally, in the ideal case, to compare intra-subject pre- and post-intervention CME, the starting point, the centre of the CME radial plot (Figure 9.2), should be have same lumbar lordosis angle. Interpretation of CME data was especially difficult in cases who received neurosurgery intervention which resulted in large changes in lumbar lordosis, in the magnitude of \( \geq 10^\circ \) post-surgery (Chapter 6). The reasons for large changes in lumbar lordosis may include changes in lumbar muscle activity between CME trials, the angle of fixation after lumbar segment fusion, and removal of restricting bone such as osteophytes, or as a result of facetectomy.
The studies in this thesis were performed to generate hypotheses based on the possibility of CME patterns being suggestive of pathoanatomical structures, using a convenience sample of participants. No power analysis was performed to estimate the minimum number of cases to test, as conducting a study using CME of symptomatic cases presenting with degenerative spondylosis, pre- and post-intervention had not been previously reported, and the variation in age, pathoanatomical conditions, and methods of intervention between cases’ presentations was unknown. Predicting the array of symptomatic presentations satisfying inclusion criteria was difficult, even with advice from senior consultants who were experienced in case selection. On the other hand, focussing on a specific pathology, for example unilateral facet joints, would have limited the yield of cases. Additionally a single pathology, such as arthritic facet joints, is likely to present differently between patients due to several factors including varying facet orientations between the pathological level in the lumbar spine, the patient’s activities of daily living, the chronicity of the patient’s pain, and medications at the time of testing. A single pathology could present as a painful joint, stiff joint, at any level from L1 to S1, and left or right/ dominant or non-dominant side. Therefore, data derived from each cohort reflected a variety of presentations and more than one intervention for a specific structure, which contributed to the variability within these data sets. With the relatively novel method of using computer-aided CME to measure lumbar spine movement, and at this early stage of investigating CME, these studies were performed to provide preliminary evidence, to test hypotheses, and more importantly, to inform future research on lumbar CME.

Due to hospital access hours and number of volunteers suiting inclusion criteria, the number of participants and the variety of LBP conditions and structures investigated was limited. The specific CME patterns reported in this thesis were generated from 35
symptomatic cases presenting with mechanical LBP, who were diagnosed by pain management or neurosurgery specialists and also were willing to attend a 14-week retest.

It was stated by Brown (1988), that CME reduces the risk of making a wrong MT treatment choice. The results from this thesis do not support this claim, since more than one pathoanatomical structure can produce a specific CME pattern. Incorrect diagnosis is still possible if using CME data, the NRR, and the preliminary patterns suggestive of pathoanatomical structures.

Many other potentially pain producing structures of the lumbar spine have not been investigated in the literature or this thesis to date using CME, such as pathology of the:

- pars interarticularis (stress reaction or defect)
- dura (sensitivity and/or scar tissue adhesion)
- spinal nerve or peripheral nerve (mechanosensitivity)
- vertebral body or end-plate (oedema or defect)
- vertebral ligaments
- fascia and muscle

Other factors which could contribute to atypical lumbar CME include dysfunctional hip joints, thoracolumbar junction pathology, occult pathology (Grieve, 1988), motivation and the effects of medication. Only the CME direction producing the worst pain, and worst stiffness, was recorded. Less severe symptoms in other CME movement directions were not used in the development of preliminary CME patterns.

Non-mechanical LBP was not investigated in this thesis examination. LBP which is not consistent with clinical signs of spinal pathoanatomy and/or does not produce a
symptom-response consistent with predictable mechanical stretch or loading, could be of non-organic origin. Non-mechanical LBP, comprises ≤5% of LBP conditions (Borenstein, 2013) and may require psychological assessment (Waddell et al., 1980). Psychosocial factors, such as abnormal behaviours and beliefs are recognised influences on LBP and function (Deyo, 2015). Behaviours using trunk muscles to brace the lumbar spine or beliefs that further damage can be done by moving the low back would result in reduced CME relative to the NRR.

During recruitment of cases from the hospital system it was difficult to remain blinded from provisional diagnoses and proposed interventions. The patient was selected from the department’s consultation list for that day, which provided information such as age, gender, a brief description of the presenting condition, and proposed intervention. In several cases, the intervention was cancelled or changed after the initial (pre-intervention) CME test. Therefore, the examiner never assumed at the time that the diagnosis was confirmed or the intervention was carried out. At post-intervention retests, the examiner attempted to remain blind to the intervention. After the final retest, the examiner obtained full and accurate confirmation of the final diagnosis and intervention.

The use of validated questionnaires as a self-report outcome measures in LBP research is recommended. However, questionnaires have intrinsic limitations which are well understood and reported in the literature (Deyo et al., 1998). Limitations of self-report surveys include the patient’s ability to remember pain intensity and/or their functional abilities, and surveys not being able to measure multi-dimensional aspects of pain (Carlsson, 1983). Additionally, medication changes and/or mood may affect outcome scores.
9.5. **Clinical implications**

Back pain is one of the seven existing national health priority areas in Australia (Briggs and Buchbinder, 2009), with the total burden of LBP ranked sixth in the world and first in Australia (Hoy et al., 2012, Murray et al., 2012). Direct expenditure allocated to back problems by the Australian health services in 2008-09 was A$1.18 billion (AIHW, 2016b).

An active movement examination is recommended as an important component of low back assessment. The CME is recommended for use, because it is simple, quick in experienced hands, non-invasive, inexpensive, functional, and more informative than a planar movement examination (Edwards, 1979). With preliminary evidence for three specific lumbar CME patterns suggestive of facet, IVD and nerve root compression (Chapters 5 and 6) and preliminary evidence to show that clinicians are able to identify these three abnormal CME patterns without the use of computer-aided 3-D tracking data, the use of CME in clinical examination of mechanical LBP appears justified.

The ability to propose specific structures and provide a provisional diagnosis may facilitate the use of specific therapeutic techniques such as manual therapy and/or exercise, which putatively treat the pathoanatomical structure. If ROM and/or symptoms change, the clinician should reflect (and perhaps adjust) their provisional diagnosis. The obvious limitation with using CME without the aid of 3-D computer tracking is the inability to compare CME ROM when small changes occur between interventions and treatment sessions.

Computer-aided CME described in this thesis has been reported in chapter 4 as validated outcome measure. Its use in future studies, along with the normal reference range; provide objective data from larger numbers of specific pathoanatomical cases, to
provide additional clinically useful information. Reliable, objective data is particularly useful when the time between retests is lengthy.

In this chapter, a mechanical LBP clinical algorithm by (Kim and Shin, 2007) has been adapted with the addition of lumbar CME and is illustrated in Figure 9.3. The use of lumbar CME in this clinical algorithm, with the three specific atypical movement patterns and respective pathoanatomical diagnoses proposed in this thesis, may be used with other components of a structured clinical examination. This will facilitate clinical reasoning and aid the clinician to arrive at a provisional diagnosis of facet, IVD, nerve root compression, or a combination of these structures. Additionally, if after completing a physical examination, the CME does not match any of these three specific CME patterns, other lumbar structures may be considered. If clinical presentation, including CME, is not typical of mechanical LBP, occult pathology, imaging and/or referral to a medical practitioner may be considered for other diagnostic procedures. By improving the assessment of LBP, promoting sound clinical reasoning, and facilitating a clinician’s ability to arrive at a provisional diagnosis, the chance of misdiagnosis and mismanagement of LBP decreases (Appendix IX).
A clinical algorithm for the assessment of mechanical LBP.

Figure 9.3. The adapted algorithm proposes where lumbar CME replaces X-ray imaging. Together with the patient’s history of the condition, neurological screening (when indicated) and consideration of pain-related factors, CME may facilitate clinical reasoning and provisional diagnosis of specific pathoanatomical structures – nerve root compression, IVD, facet joint or a combination of these structures. Where the CME is not consistent with the proposed patterns, and symptom reproduction is consistent with mechanical LBP, other lumbar structures should be considered. In the case that the presenting LBP is not consistent with mechanical LBP, referral to a medical practitioner and/or imaging should be considered. Figure adapted from Kim and Shin (2007).

9.6. Conclusions

This thesis investigation provides preliminary evidence for the implication of three structures of the lumbar spine which commonly cause lumbar spine dysfunction – facet joint, IVD and nerve root compression (Tarulli and Raynor, 2007, DePalma et al., 2011).

Care must be taken not to assume that increased movement in all cases is an improvement. The painful hypermobile or unstable lumbar segment may require interventions to limit movement, before a clinical improvement is achieved. This was evident in the fusion cases reported in the neurosurgery cohort (Chapter 6).
The CME is recommended for use with patients presenting with mechanical LBP and can be used as a reliable outcome measure. With the use of an age- and gender-matched NRR, CME can be used for patient feedback, assessing treatment effect, and goal setting. Where the condition is complex, not responding to treatment, or where symptoms masquerade as more sinister pathology (Greenhalgh and Selfe, 2015), appropriate imaging and/or referral to appropriate health professionals is encouraged. In the absence of a comprehensive assessment, clinical reasoning, and evolving diagnosis, there is risk of missed or misdiagnosis (Monie et al., 2016b). There is also potential use for lumbar CME at a group level in mechanical LBP research. Lumbar CME may be used as an additional outcome measure, with an age- and gender-matched NRR, with specific pathoanatomical sub-groups for comparison of interventions.

This chapter has described the evolution of this thesis investigation, and systematically discussed the results of each study by addressing the four primary *a priori* hypotheses. The results from each study were compared with results, hypotheses and theories previously reported in the literature. Study limitations are described, and clinical implications proposed. Additional hypotheses, using summary data, were tested using cluster analysis. Where clusters were identified, speculations have been made along with supporting or contradictory evidence from the literature.
10. Conclusion and recommendations

10.1. Introduction
The primary purpose of this thesis investigation was to test the diagnostic accuracy of
the CME. Additionally, a NRR for both genders, ages 20-69 was developed to help
discriminate clinical change in patient cohorts. The NRR data and movement patterns,
illustrated using radial plots, served as a template for comparing and contrasting
symptomatic cases. The principal hypothesis was that there would be structure specific
CME movement patterns for facet, IVD and nerve root compression. These structures
were selected, because they are common sources of mechanical LBP (Tarulli and
Raynor, 2007, DePalma et al., 2011). The method of collecting lumbar CME data was
shown to have acceptable reliability for human biomechanical measurements, both
within a session and over time.

10.2. Pilot studies

10.2.1. Pilot study 1
A pilot study consisted of assessing the MotionStar 3-D motion tracking system for
validity and reliability. The details of these two assessments are reported in Appendix
III. The system was retested, against the triaxial goniometer every 6 months and
whenever the system was transported to one of the four testing locations, with these
results also reported in Appendix III. There was acceptable validity, with the
MotionStar accurately measuring angles in each of the three cardinal planes (x, y, and z)
within 0.6° and a CV of <1%.

10.2.2. Pilot study 2
A second pilot study tested the reliability of using the MotionStar tracking system to
collect CME data in 10 asymptomatic volunteers, for both intra-session and inter-
session conditions. ICC with 95% CIs, and SC was used to report accuracy and precision, respectively. The outcome of this study showed that computer-aided lumbar CME is a valid measure and can be performed with acceptable reliability. ICC values for intra-session testing was >0.9 and for inter-session testing >0.7, for all CME movement directions. The average inter-session LSC value for the eight CME directions was 7.7°. The details of this study are reported in Chapter 4 and Appendix III.

10.3. Chapter studies

10.3.1. Study 1
The object of the first major study was to instigate an original NRR for CME for males and females aged 20-69 years. The NRR data from 151 asymptomatic volunteers, allowed the following conclusions to be made:

1. Normal CME is symmetrical, with average SF values for both genders and across the age groups being within 5° (Monie et al., 2015).

2. In asymptomatic cases, the angular movement of flexion is at least two and a half times that of extension. The factor increases to almost three times in the over 50 age groups. Decreasing extension ROM is likely a result of natural aging and spondylosis (McCarthy et al., 2015).

The results from this study served as a template and allowed for comparison and contrasting of CME data and movement patterns in the following studies.

10.3.2. Study 2
This study was designed to test the hypothesis that pathoanatomical structures – facet joints, IVD and nerve roots – would result in atypical CME. A second hypothesis was that CME ROM will approximate the age- and gender-matched NRR, following pain management intervention for LBP. Additionally, this study sought to compare and
contrast the atypical CME movement patterns with specialist diagnosis and pain medicine interventions.

The following conclusions were drawn:

1. The use of CME assisted in identifying atypical lumbar movement relative to an age- and gender-specific NRR (n=159). In the majority of cases, pain and/or joint degeneration resulted in decreased ROM. Some cases who complained of pain during CME moved beyond the NRR in the painful movement direction.

2. There is preliminary evidence of discrete atypical CME movement patterns suggestive of symptomatic IVD (n=4), unilateral facet joint (n=1), bilateral facet joint (n=2) and nerve root compression (n=2).

3. In cases which improved on pre-intervention self-reports (>MCID) there was migration of CME ROM towards the age- and gender-matched NRR.

4. CME patterns from cases with multi-structure and/or multi-level pathology are more difficult to interpret.

10.3.3. Study 3
This study was designed to test the hypothesis that pathoanatomical structures – IVD disease, unilateral posterolateral disc protrusion and/or nerve root compression, and central disc protrusion and/or bilateral nerve root compression – would result in atypical CME. A second hypothesis was that CME ROM would migrate towards the age- and gender-matched NRR, following neurosurgical intervention for LBP. Additionally, this study sought to compare and contrast the atypical CME movement patterns with specialist diagnosis and neurosurgical interventions.

The following conclusions were drawn:
1. The use of CME assisted in identifying atypical lumbar movement relative to an age- and gender-specific NRR (n=159).

2. There was preliminary evidence in six cases to support the concept of specific CME movement patterns suggestive of IVD degenerative disease (n=2), unilateral posterolateral disc protrusion (n=2) and centrally protruding discs (n=2), with or without nerve root compression.

3. In 11 of the 18 cases, the CME movement patterns converged towards the NRR after surgery.

4. CME patterns from cases with large post-surgical changes in sagittal or coronal plane lumbopelvic alignment are difficult to interpret.

10.3.4. Study 4
This study was designed to report change in the ability of post-graduate physiotherapy students to speculate on the pathoanatomical diagnoses in three semi-discrete CME movement patterns, by observing a montage of the eight CME positions (Figures 7.2-7.4).

The following conclusions were drawn:

1. Clinicians are able to speculate pathoanatomical diagnoses by viewing a montage of CME images depicting the three proposed specific CME patterns for mechanical LBP.

2. Providing the preliminary evidence of specific CME patterns and the proposed pathoanatomical diagnosis indicated for each pattern may aid in clinical decision making in cases presenting with mechanical LBP by encouraging consideration of likely source(s).
10.3.5. Study 5
This study used pooled outcome data from cases in the pain management cohort (n=17) and neurosurgery cohort (n=18) to test the third primary hypothesis, – that CME ROM will approximate the NRR post-intervention – and also to perform cluster analysis to test five secondary hypotheses.

The following conclusions were drawn:

1. On a group level, the average post-intervention CME ROM data, for all movement directions, approximated the NRR post-intervention. However, this was not observed on an individual case level. For example, four of the seven single-segment fusion cases had decreased ROM post-surgery. In the majority of cases, the post-intervention z-scores showed the greatest amount of migration towards the age- and gender-matched NRR in the direction consistent with the case’s pathoanatomical diagnosis.

2. Pre-intervention CME z-scores in the flexed directions (flexion, FwLSF and FwRSF) facilitates sub-grouping of cases with mechanical LBP. Cases with pathoanatomical diagnoses of radiculopathy and symptomatic DDD were separated after cluster analysis using pre-intervention CME z-scores.

3. Cluster analysis of total change in outcome using VASp, VASs, SF-12 and RMDQ places into sub-groups a portion of cases receiving neurosurgery from cases receiving pain medicine or neurosurgery intervention.

4. Total change scores (%) in CME ROM does not sub-group cases which receive pain management or neurosurgery intervention. Four of the seven cases receiving lumbar fusion surgery which resulted in decreased ROM post-intervention, reduced the ability for this analysis to identify difference between the pain management and neurosurgery cohorts. Cases receiving lumbar fusion
surgery should be considered separately as a sub-group of neurosurgery cases when examining change of CME ROM post-intervention.

5. Cluster analysis of self-report outcome data from VASp, VASs, SF-12 and RMDQ analysed by pre-intervention SF-12 MCS score, aids in sub-grouping cases into normal SF-12 MCS scores (≥40) and abnormal scores (<40). The identification of patients with abnormal SF-12 MCS scores will aid in triage of patients presenting with LBP who would benefit from further assessment for psychosocial factors.

6. Cases with mechanical LBP over 50 years of age do not exhibit a predominance of nerve root compression pathology. Cluster analysis showed that in cases aged less than 46 years there was a predominance of nerve root compression. In cases of age 48-58 there was no predominant pathoanatomy. The majority of cases diagnosed with bilateral facet pathology were over 60 years of age.

10.4. Recommendations for future studies
The series of studies presented in this thesis were designed to test the primary and secondary hypotheses, and inform hypotheses for future studies investigating CME of the spine, in particular CME for mechanical LBP. Apart from recommending lumbar computer-aided CME being used as a validated outcome measure, an age- and gender-matched NRR, and proposed specific CME movement patterns suggestive of pathoanatomical diagnosis, studies such as the ones described below would provide insight to the clinical potential of lumbar CME.

10.4.1. Consider larger cohorts of specific presentations
Future studies with larger cohorts of specific pathologies for each of the pathoanatomical structures would help to improve the power of the results for specific
CME patterns by pathology, and decrease the dilution of outcome measures from heterogeneous cohorts by comparing data in cohorts with the same pathoanatomy. For example, a cohort of degenerative facet joints which are asymptomatic, a cohort of acute facet joints in the younger age groups (20-30 years), cases with discitis, cases with DDD without symptoms, a cohort of radiculopathy with and without evidence of stenosis on imaging, and hypermobile cases experiencing LBP.

10.4.2. **Blind the examiner from presentation and intervention**

Testing the diagnostic value of the proposed specific CME patterns, and the pathoanatomical structures they suggest, would be of value to the clinical reasoning process when assessing patients with mechanical LBP. To test this, the CME examiner would test patients who have been diagnosed by specialists, as having symptoms which originate from the lumbar spine. The CME examiner could test CME and be asked to speculate the potential source structure(s) contributing to their CME pattern. This would be best tested if the examiner is completely blinded from the patient’s history and specialist diagnosis. Adding asymptomatic volunteers, dispersed amongst the symptomatic line-up, would further test the specificity and sensitivity of CME to detect symptomatic mechanical LBP. The examiner’s speculation can be tested for accuracy by comparing it with specialist diagnosis, imaging, pain provocation or nerve blocks for facet joint (Bogduk, 2008), IVD (Ni et al., 2015) or nerve pathology (Hayek and Shah, 2014) and outcome data including CME data and pain and function self-reports.

10.4.3. **Place additional motion tracking sensors over the lumbar spinous processes**

A future study with additional sensors along the lumbar spine will provide data for ROM comparison between individual segments or by region, such as L1-4 and L4-S1. The lower segments of the lumbar spine are of particular interest because these segments are usually more degenerative than the upper and mid lumbar segments, and
degenerative changes often, when not unstable, result in reduced ROM (Muriuki et al., 2016). A recent study by Christe et al. (reported reduced sagittal angular movement during sit-to-stand movements, in cases of CLBP, using multi-segment analysis of spinal kinematics. Acceptable reliability for assessing regional spinal ROM using 3-D, skin mounted motion tracking sensors, has been reported by (Alqhtani et al., 2015). With a NRR for CME per segment, the ability to identify hypomobile, normal, and hypermobile segments, and the ability to identify compensatory mechanisms, such as extending the lumbar spine during CME directions which require flexion, may be possible. Additional sensors could also be used to compare movement between genders, across the age groups, and compare lumbar pathologies.

10.4.4. Compare velocity of CME in asymptomatic and symptomatic cases
The aim of this study would be to compare the speed at which cases performed each movement of the CME. A patient with a symptomatic left lumbar facet joint may move towards proposed painful CME directions, namely LSF, EwLSF and extension (Figure 9.2Ai), slower than subjects from a CME NRR for speed. A comparison of movement speed could be made in patients of different pathologies, or similar pathologies at different stages of recovery, such as acute and CLBP, or with different presentations, such as a primary complaint of low back stiffness versus a LBP. The use of both NRR, CME for ROM and speed, would serve as objective rehabilitation goals.

10.4.5. Consider whether CME patterns with the location of symptoms could improve diagnostic accuracy
This study could be performed in the same study design and format as the blinded study recommended earlier (10.4.2). However, in this study the examiner would collect CME data as well as information from the patient regarding the location of the symptoms. Using innervation patterns for facets and IVD, described in the literature (Bogduk, 1985, Groen et al., 1987, Groen and Stolker, 2000) and known lumbar radicular patterns
(Grant, 1999, Taylor et al., 2013), a speculation can be made as to the pathoanatomical structure(s). The matching of symptom location with specific CME patterns may allow further differentiation of patients presenting with the CME patterns illustrated in Figures 9.2A and C. For example, a patient demonstrating a CME pattern consistent with 9.2A, with left side L5 region pain may have left side low lumbar facet joint pathology. However, a patient presenting with the same CME pattern, restricted due to left lower limb pain consistent with S1 dermatome, may have nerve root compression.
Bibliography


**Introduction:** We seek permission to measure the movement in your low back. Please read this information carefully, as it will tell you all about the research, procedures, risks and benefits. Ask questions about anything you are not sure about, and feel free to talk about the research with a relative or friend before you make a decision. If you need help reading, or English is not your first language, please tell the research team so they can get you some assistance.

This study has been reviewed by the University of Western Australia Human Research Ethics Committee whose primary concerns are the safety, welfare and rights of participants in this research. The ethics committee members are independent of the study sponsor and study team.

Participation is voluntary and you are free to withdraw your permission at any time and without prejudice to any future treatment.

**Background:** Measuring the amount of movement in the low back region provides therapists with information regarding what is ‘usual’ and what is ‘restricted’ movement. This helps Physiotherapists assess the cause of low back pain and select appropriate treatment approaches. One method of assessing low back movement response to pain in a clinical setting is to use a Combined Movement Examination [CME].

**Purpose of this study:** We would like to measure the movement in persons without low back conditions using the CME approach to establish a ‘normal’ reference range for comparison with persons who have low back pain. We would like to use this information to determine if the CME is useful in detecting specific low back problems.

**Methods:** We are recruiting subjects [by word of mouth and poster advertisement] with ‘normal’ low backs to perform the CME. The CME is a series of low back movements which we would like you to perform in a standing position, with our guidance. Before starting, you would watch a short video of the examination, so that you can familiarise yourself with the procedure. When you are ready to be measured we will place two sensors on your low back. Your low back will need to be exposed for the attachment of the 2 sensors. We will then ask you to perform the set of movements within your abilities. The movements are essentially ‘every-day’ movements – forward bend, sideways bend, backwards and in diagonal directions. You should not experience any discomfort associated with the CME testing. The tracking device will record your low back movement. The CME should take no longer than 2 minutes.

**Risk and inconvenience:** The tracking device is safe, non-invasive and involves no risk. There are no obvious inconveniences other than the need to attend the laboratory at the Centre for Musculoskeletal Science building at the University of Western Australia.
The testing session will be supervised by Mr Aubrey Monie, an experienced research physiotherapist. Your participation in this study does not prejudice any right to compensation, which you may have under statute or common law for any injuries arising through negligence of any of the investigators.

**Benefits:** Whilst there are no specific benefits to you for participation there is the potential benefit to the community of contributing to an investigation of low back movement and pain evaluation.

**Dissemination of Results:** The information sought from this study will be summarised for a research thesis being undertaken at UWA and may also be used in publications. However, all recorded material would be de-identified so that the collected data would be anonymous.

Should you have any questions or request further information about this study please contact me at UWA on (08) 6488 7078.

Professor Kevin Singer - Chief Investigator, Centre for Musculoskeletal Studies, School of Surgery. The University of Western Australia. Corner Park and Crawley Avenue, CRAWLEY.

Approval to conduct this research has been provided by The University of Western Australia, in accordance with its ethics review and approval procedures. Any person considering participation in this research project, or agreeing to participate, may raise any questions or issues with the researchers at any time.

In addition, any person not satisfied with the response of researchers may raise ethics issues or concerns, and may make any complaints about this research project by contacting the Human Research Ethics Office at The University of Western Australia on (08) 6488 3703 or by emailing to hreo-research@uwa.edu.au

All research participants are entitled to retain a copy of any Participant Information For and/or Participant Consent Form relating to this research project.
Appendix I.2

**Introduction:** Your pain specialist doctor has invited you to participate in this study because he/she feels that you are suitable to volunteer. We seek permission to measure the movement in your low back before and after your pain specialist has treated you. We also seek permission to view your low back scan results [after testing] and communicate with your specialist regarding the treatment he/she used and the treatment outcome. This will help to cross-check our study findings.

Please read this information carefully, as it will tell you all about the research, procedures, risks and benefits. Ask questions about anything you are not sure about, and feel free to talk about the research with a relative or friend before you make a decision. If you need help reading, or English is not your first language, please tell the research team so they can get you some assistance.

This study has been reviewed by the University of Western Australia Human Research Ethics Committee whose primary concerns are the safety, welfare and rights of participants in this research. The ethics committee members are independent of the study sponsor and study team.

Participation is voluntary and you are free to withdraw your permission at any time and without prejudice to any future treatment.

**Background:** Measuring the amount of movement in the low back region provides information regarding what is ‘usual’ and what is ‘restricted’ movement. This helps Physiotherapists assess the cause of low back pain and select appropriate treatment approaches. One method of assessing low back movement response to pain in a clinical setting is to use a Combined Movement Examination [CME].

**Purpose of this study:** We would like to measure the movement in persons with low back conditions, for comparison with persons who have no low back conditions. We would like to use this information to determine if the CME is useful in detecting specific low back problems.

**Methods:** The CME is a series of set low back movements which we would like you to perform in a standing position. Before starting you would watch a short video of the examination, so that you can familiarise yourself with the procedure. When you are ready to be measured we will place 2 sensors on your low back. Your low back will need to be exposed for the attachment of the sensors. We will then ask you to perform the set of movements within your abilities. The movements are essentially ‘every-day’ movements – forward bend, sideways bend, backwards and in diagonal directions. You should not experience any unusual discomfort during the testing. The tracking device will record your low back movement. The CME should take no longer than 2 minutes.
Risk and inconvenience: The tracking device is safe, non-invasive and involves no risk. There are no obvious inconveniences other than the need to attend the laboratory at the Centre for Musculoskeletal Science building at the University of Western Australia. The testing session will be supervised by Mr Aubrey Monie, an experienced research physiotherapist. Your participation in this study does not prejudice any right to compensation, which you may have under statute or common law for any injuries arising through negligence of any of the investigators.

Benefits: Whilst there are no specific benefits to you for participation there is the potential benefit to the community of contributing to an investigation of low back movement and pain evaluation.

Dissemination of Results: The information sought from this study will be summarised for a research thesis being undertaken at UWA and may also be used in publications. However, all recorded material would be de-identified so that the collected data would be anonymous.

Should you have any questions or request further information about this study please contact me at UWA on (08) 6488 7078.

Professor Kevin Singer - Chief Investigator, Centre for Musculoskeletal Studies, School of Surgery. The University of Western Australia. Corner Park and Crawley Avenue, CRAWLEY.
PARTICIPANT CONSENT FORM [A]
Measuring low back movement using the Combined Movement Examination

Appendix I.3

Principal Investigator: Professor Kevin Singer –
Centre for Musculoskeletal Studies, School of Surgery. The University of Western Australia.
Corner Park and Crawley Avenue, CRAWLEY

I ___________________________ have read the information provided in the participant sheet A and any questions I have asked have been answered to my satisfaction. I agree to participate in this activity, realising that I may withdraw at any time without reason and without prejudice to my future treatment.

I understand that all identifiable information I provide is treated as strictly confidential and will not be released by the investigator in any form that may identify me. The only exception to this principle of confidentiality is if documents are required by law.

I have been advised as to what data is being collected, what the purpose is, and what will be done with the data upon completion of the research.

I agree that research data gathered for the study may be published provided my name or other identifying information is not used.

_________________________  ______________________
Signature                        Date

Should you have any questions or request further information about this study please contact me at UWA on (08) 6488 7078.

Approval to conduct this research has been provided by The University of Western Australia, in accordance with its ethics review and approval procedures. Any person considering participation in this research project, or agreeing to participate, may raise any questions or issues with the researchers at any time.

In addition, any person not satisfied with the response of researchers may raise ethics issues or concerns, and may make any complaints about this research project by contacting the Human Research Ethics Office at The University of Western Australia on (08) 6488 3703 or by emailing hreo-research@uwa.edu.au.

All research participants are entitled to retain a copy of any Participant Information For and/or Participant Consent Form relating to this research project.
PARTICIPANT CONSENT FORM [B]
Measuring low back movement using the Combined Movement Examination

Principal Investigator: Professor Kevin Singer –
Centre for Musculoskeletal Studies, School of Surgery. The University of Western Australia.
Corner Park and Crawley Avenue, CRAWLEY

I ________________________________ have read the information provided in the participant sheet B and any questions I have asked have been answered to my satisfaction. I agree to participate in this activity, realising that I may withdraw at any time without reason and without prejudice to my future treatment.

I understand that all identifiable information that I provide is treated as strictly confidential and will not be released by the investigator in any form that may identify me. The only exception to this principle of confidentiality is if documents are required by law.

I agree that research data gathered for the study may be published provided my name or other identifying information is not used.

_________________________  __________________________
Signature                   Date

Should you have any questions or request further information about this study please contact me at UWA on (08) 6488 7078.

Approval to conduct this research has been provided by The University of Western Australia, in accordance with its ethics review and approval procedures. Any person considering participation in this research project, or agreeing to participate, may raise any questions or issues with the researchers at any time.

In addition, any person not satisfied with the response of researchers may raise ethics issues or concerns, and may make any complaints about this research project by contacting the Human Research Ethics Office at The University of Western Australia on (08) 6488 3703 or by emailing to hreo-research@uwa.edu.au

All research participants are entitled to retain a copy of any Participant Information For and/or Participant Consent Form relating to this research project.
PARTICIPANT INFORMATION SHEET A

Measuring low back movement in participants with a normal low back

Combined Movement Examination of the Human Lumbar Spine

Researchers: Winthrop Professor Kevin Singer, Professor Chris Lind, Professor Roger Price, Aubrey Monie

Please take time to read the following information carefully and to discuss it with your family, friends and general practitioner if you so wish. If any part of the information is not clear to you, or if you would like more information do not hesitate to ask us to explain it more fully. Make certain you do this before you sign the consent form to participate in this study.

Introduction: We seek permission to measure the movement in your low back. Please read this information carefully, as it will tell you all about the research, procedures, risks and benefits. Ask questions about anything you are not sure about, and feel free to talk about the research with a relative or friend before you make a decision. If you need help reading, or English is not your first language, please tell the research team so they can get you some assistance.

This study has been reviewed by the Sir Charles Gairdner Group Human Research Ethics Committee whose primary concerns are the safety, welfare and rights of participants in this research. The ethics committee members are independent of the study sponsor and study team.

Participation is voluntary and you are free to withdraw your permission at any time and without prejudice to any future treatment. Your choice not to participate will have no impact on your care.

Background: Measuring the amount of movement in the low back region provides therapists with information regarding what is ‘usual’ and what is ‘restricted’ movement. This helps Physiotherapists assess the cause of low back pain and select appropriate treatment approaches. One method of assessing low back movement response to pain in a clinical setting is to use a Combined Movement Examination [CME]. This requires the participant to stand while the examiner guides them through eight low back movements including forwards, side-ways [left and right], and backwards directions. All movements are to be done slowly and within the participant’s comfortable limits.

Purpose of this study: We would like to measure the movement in persons without low back conditions using the CME approach to establish a ‘normal’ reference range for
comparison with persons who have low back pain. We would like to use this information to determine if the CME is useful in detecting specific low back problems.

**Methods:** We are recruiting subjects with ‘normal’ low backs to perform the CME. The CME is a series of low back movements which we would like you to perform in a standing position, with our guidance. Before starting, you would watch a short video of the examination, so that you can familiarise yourself with the procedure. When you are ready to be measured we will place two sensors on your low back. Your low back will need to be exposed for the attachment of the 2 sensors. We will then ask you to perform the set of movements within your abilities. The movements are essentially ‘every-day’ movements. You should not experience any discomfort associated with the CME testing. The tracking device will record your low back movement. The test should take no longer than 2 minutes.

**Risk and inconvenience:** The tracking device is safe, non-invasive and low risk. There are no obvious inconveniences other than the need to attend the testing session at Sir Charles Gairdner Hospital. Hopefully this can be done during scheduled follow-up appointments with your specialist. The testing session will be supervised by Mr Aubrey Monie, an experienced research physiotherapist. Your participation in this study does not prejudice any right to compensation, which you may have under statute or common law for any injuries arising through negligence of any of the investigators.

**Benefits:** Whilst there are no specific benefits to you for participation there is the potential benefit to the community of contributing to an investigation of low back movement and pain evaluation. Costs would include travel to and from Sir Charles Gairdner Group and a parking fee. Efforts will be made by the examiner, to test your low back if/when you attend the site for other reasons.

**Dissemination of Results:** The information sought from this study will be summarised for a research thesis being undertaken at UWA and may also be used in publications. However, you will not be identifiable in any publications arising from the study.

Should you have any questions or request further information about this study please contact the site Investigator, Aubrey Monie at UWA on (08) 6488 7078.

Professor Kevin Singer - Chief Investigator, Centre for Musculoskeletal Studies, School of Surgery. The University of Western Australia.

The Sir Charles Gairdner Group Human Research Ethics Committee has reviewed this study and has given its approval for the conduct of this research study. In doing so, this research conforms to the principles set out by the National Statement on Ethical Conduct in Human Research and abides by the Good Clinical Practice Guidelines.
Measuring low back movement in participants with a normal low back

Combined Movement Examination of the Human Lumbar Spine

Researchers: Winthrop Professor Kevin Singer, Professor Chris Lind, Associate Professor Roger Price, Aubrey Monie

Please take time to read the following information carefully and to discuss it with your family, friends and general practitioner if you so wish. If any part of the information is not clear to you, or if you would like more information do not hesitate to ask us to explain it more fully. Make certain you do this before you sign the consent form to participate in this study.

Introduction: Your pain specialist doctor has invited you to participate in this study because he/she feels that you may be suitable to volunteer. We seek permission to measure the movement in your low back before and after your pain specialist has treated you. Please read this information carefully, as it will tell you all about the research, procedures, risks and benefits. Ask questions about anything you are not sure about, and feel free to talk about the research with a relative or friend before you make a decision. If you need help reading, or English is not your first language, please tell the research team so they can get you some assistance.

This study has been reviewed by the Sir Charles Gairdner Group Human Research Ethics Committee whose primary concerns are the safety, welfare and rights of participants in this research. The ethics committee members are independent of the study sponsor and study team.

Participation is voluntary and you are free to withdraw your permission at any time and without prejudice to any future treatment. Your choice not to participate will have no impact on your care.

Background: Measuring the amount of movement in the low back region provides information regarding what is ‘usual’ and what is ‘restricted’ movement. This helps Physiotherapists assess the cause of low back pain and select appropriate treatment approaches. One method of assessing low back movement response to pain in a clinical setting is to use a Combined Movement Examination [CME]. This requires the participant to stand while the examiner guides them through eight low back movements including forwards, side-ways [left and right], and backwards directions. All movements are to be done slowly and within the participant’s comfortable limits.
Purpose of this study: We would like to measure the movement in persons with low back conditions, for comparison with persons who have no low back conditions. We would like to use this information to determine if the CME is useful in detecting specific low back problems.

Methods: The CME is a series of set low back movements which we would like you to perform in a standing position. Before starting you would watch a short video of the examination, so that you can familiarise yourself with the procedure. When you are ready to be measured we will place 2 sensors on your low back. Your low back will need to be exposed for the attachment of the sensors. We will then ask you to perform the set of movements within your abilities. The movements are essentially ‘every-day’ movements. You should not experience any unusual discomfort during the testing. The tracking device will record your low back movement. The test should take no longer than 2 minutes.

Risk and inconvenience: The tracking device is safe, non-invasive and low risk. You will be observed throughout the procedure and encouraged to move within your comfortable limits. There are no obvious inconveniences other than the need to attend the testing session at Sir Charles Gairdner Hospital. Hopefully this can be done during scheduled follow-up appointments with your specialist. The testing session will be supervised by Mr Aubrey Monie, an experienced research physiotherapist. Your participation in this study does not prejudice any right to compensation, which you may have under statute or common law for any injuries arising through negligence of any of the investigators.

Benefits: Whilst there are no specific benefits to you for participation there is the potential benefit to the community of contributing to an investigation of low back movement and pain evaluation. Costs would include travel to and from Sir Charles Gairdner Group and a parking fee. Efforts will be made to test your low back immediately before or after your specialist appointment.

Dissemination of Results: The information sought from this study will be summarised for a research thesis being undertaken at UWA and may also be used in publications. However, all recorded material would be de-identified so that the collected data would be anonymous.

Should you have any questions or request further information about this study please contact the site investigator, Aubrey Monie at UWA on (08) 6488 7078.

Professor Kevin Singer - Chief Investigator, Centre for Musculoskeletal Studies, School of Surgery. The University of Western Australia.

The Sir Charles Gairdner Group Human Research Ethics Committee has reviewed this study and has given its approval for the conduct of this research study. In doing so, this research conforms to the principles set out by the National Statement on Ethical Conduct in Human Research and abides by the Good Clinical Practice Guidelines.
Participant Consent Sheet

Sir Charles Gairdner Hospital

Measuring low back movement in participants with a normal low back
Combined Movement Examination of the Human Lumbar Spine

Researchers: Winthrop Professor Kevin Singer, Professor Chris Lind, Associate
Professor Roger Price, Aubrey Monie

Participant Name: ___________________________ Date of Birth: __________

NOTE: If you are still unclear about anything you have read in the Participant
Information Sheet and Consent Form, please speak to your doctor before signing
this Consent.

1. I have been given information, both verbally and in writing, about this study and
having had time to consider it, am now able to make an informed decision to
participate.

2. I have been told about the potential benefits and known risks of taking part in this
study and I understand what this means to me.

3. I have been given the opportunity to have a member of my family or a friend with
me when this study was being explained to me. I have been able to ask questions
and have had all my questions answered.

4. I know that I do not have to take part in this study, and that my decision to take part
is voluntary. I understand that I can withdraw from this study at any time without
this decision affecting my medical care.

5. I understand that participating in this study does not affect any right to
compensation, which I may have under statute or common law.

6. I accept that by taking part in this research, that any information obtained about me
during the study may be published, provided that my name and other identifying
information are not used.

Name of Participant: ___________________________ Signature of Participant: ___________________________
Date: __________

Name of Researcher: ___________________________ Signature of Researcher: ___________________________
Date: __________

The Sir Charles Gairdner Group Human Research Ethics Committee has granted
approval for the conduct of this study. If you have any concerns about the ethics or
code of practice of the study, please contact the Executive Officer of the Sir Charles
Gairdner Group Human Research Ethics Committee on (08) 9346 2999.

Study participants are to receive a copy of the Participant Information Sheet and
Consent Form for their personal record.
Our Ref: RA/4/1/6020

09 April 2013

Winthrop Professor Kevin Singer
School of Surgery
MBDP: M424

Dear Professor Singer

HUMAN RESEARCH ETHICS APPROVAL - THE UNIVERSITY OF WESTERN AUSTRALIA

Combined Movement Examination of the Human Lumbar Spine

Student(s): Aubrey Monie - PhD - 19319938

Ethics approval for the above project has been granted in accordance with the requirements of the National Statement on Ethical Conduct in Human Research (National Statement) and the policies and procedures of The University of Western Australia. Please note that the period of ethics approval for this project is five (5) years from the date of this notification. However, ethics approval is conditional upon the submission of satisfactory progress reports by the designated renewal date. Therefore initial approval has been granted from 09 April 2013 to 01 April 2014.

You are reminded of the following requirements:

1. The application and all supporting documentation form the basis of the ethics approval and you must not depart from the research protocol that has been approved.
2. The Human Research Ethics Office must be approached for approval in advance for any requested amendments to the approved research protocol.
3. The Chief Investigator is required to report immediately to the Human Research Ethics Office any adverse or unexpected event or any other event that may impact on the ethics approval for the project.
4. The Chief Investigator must inform the Human Research Ethics Office as soon as practicable if a research project is discontinued before the expected date of completion, providing reasons.

Any conditions of ethics approval that have been imposed are listed below:

Special Conditions

None specified

The University of Western Australia is bound by the National Statement to monitor the progress of all approved projects until completion to ensure continued compliance with ethical standards and requirements.

The Human Research Ethics Office will forward a request for a Progress Report approximately 60 days before the due date. A further reminder will be forwarded approximately 30 days before the due date.

If your progress report is not received by the due date for renewal of ethics approval, your ethics approval will expire, requiring that all research activities involving human participants cease immediately.

If you have any queries please contact the HREO at hreo-research@uwa.edu.au.

Please ensure that you quote the file reference – RA/4/1/6020 – and the associated project title in all future correspondence.

Yours sincerely

Dr Mark Dixon
Associate Director, Research Ethics and Biosafety
Appendix I.9

Professor Kevin Singer
School of Surgery
FMDHS M424
The University of Western Australia
CRAWLEY WA 6009

Dear Professor Singer

HREC No: 2014-009
Project Title: Combined Movement Examination of the Human Lumbar Spine

The ethics application for the project referenced above was reviewed by the Sir Charles Gairdner Group (SCGG) Human Research Ethics Committee (HREC) at its meeting on 17 July 2014. It has been approved and the following documents have been approved for use in this project.

<table>
<thead>
<tr>
<th>Documents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Research Proposal, version dated July 2013</td>
</tr>
<tr>
<td>SCGH Participant Information Sheet A, version 1 dated 24 June 2014</td>
</tr>
<tr>
<td>SCGH Participant Information Sheet B, version 1 dated 24 June 2014</td>
</tr>
<tr>
<td>Consent Form, version 1 dated 24 June 2014</td>
</tr>
<tr>
<td>The Roland-Morris Low Back Pain and Disability Questionnaire</td>
</tr>
<tr>
<td>SF-12 Questionnaire</td>
</tr>
</tbody>
</table>

Approval of this project from the Sir Charles Gairdner Group Human Research Ethics Committee EC00271 is valid to 3 July 2017 and on the basis of compliance with the 'Conditions of HREC Approval for a Research Project' (attached).

The nominated participating site/s in this project is/are:

Sir Charles Gairdner Hospital

[Note: If additional sites are recruited prior to the commencement of, or during the research project, the Coordinating Principal Investigator is required to notify the HREC. Notification of withdrawn sites should also be provided to the HREC in a timely fashion.]

A copy of this ethical approval letter must be submitted by all site Principal Investigators to the Research Governance Office or equivalent body or individual at each participating institution in a timely manner to enable the institution to authorise the commencement of the project at its site/s.

This letter constitutes ethical approval only. This project cannot proceed at any site until separate site authorisation has been obtained from the CE, or delegate, of the site under whose auspices the research will be conducted at that site.
CTN Scheme (Devices): Acknowledgement of New Trial

Your notification to conduct a clinical trial under the Clinical Trial Notification (CTN) Scheme, pursuant to Regulation 7.1 of Schedule 4 of the Therapeutic Goods (Medical Devices) Regulations 2002, has been received by the Office of Scientific Evaluation (OSE).

Trial Number: 2014/0625
Protocol Number: 2014-009

Therapeutic Good(s):

<table>
<thead>
<tr>
<th>Name of Device</th>
<th>Trade Name</th>
<th>Code Name</th>
<th>Strength</th>
</tr>
</thead>
<tbody>
<tr>
<td>MotionStar System</td>
<td>MotionStar</td>
<td>N/A</td>
<td>N/A</td>
</tr>
</tbody>
</table>

It is noted that:

i. the approval of the goods for this trial was given in accordance with Item 2.3 of Schedule 4 of the Therapeutic Goods (Medical Devices) Regulations 2002 by the body or organisation conducting the trial at each additional site.

ii. the representative of the Ethics Committee for each additional site has certified that the Committee is constituted and operates in accordance with the NHMRC “National Statement on Ethical Conduct in Human Research” has considered this clinical trial, and has provided advice to the body or organisation conducting the trial.

The Therapeutic Goods Administration has not carried out an assessment of the quality, safety or efficacy of any drug product in relation to this notification.

Please note that, in the event that the Secretary of the Commonwealth Department of Health becomes aware that to undertake or continue the clinical trial would be contrary to the public interest, the Secretary has the authority to direct that use of the drug product(s) for this clinical trial must cease.

A form “CTN and CTX Trial Completion Advice” is enclosed. Please fill out and return this form after the Clinical Trial has completed.

Christina Kotowski
Experimental Products Section
Office of Scientific Evaluation
05 September 2014
RECRUITMENT ADVERTISEMENT (E-MAIL)

Combined Movement Examination of the Human Lumbar Spine [Ref No RA/4/1/6020]

Seeking participants for an investigation examining 'NORMAL' low back movement

Professor Kevin Singer is supervising a PhD research project being undertaken by
physiotherapist Aubrey Monie (PhD candidate). Research participants, with no history of low
back pain [requiring treatment in the last 12 months] are invited to volunteer for an assessment
of their back mobility. All movements of your lower back are to be completed within your
comfortable range. A non-invasive 3-D tracking device will record the low back movement.

The data obtained will be used to develop a 'NORMAL' reference of males and females, for 20
to 70 years of age. Future studies on individuals with low back pain will be compared to this
'NORMAL' range.

Participants will be asked to attend 1 session (15 mins). The study is being held in Room G14 of
the Park Avenue Building [Corner of Crawley and Park Ave]. Sessions are available from early
May, 2013.

If you are interested in this study or have any questions, please contact Aubrey Monie at
19319938@student.uwa.edu.au to arrange to receive a Participant Information Form that will
explain the details of the study. After reading the Participant Information Form, if you choose to
participate in this study, please contact Aubrey to arrange an appointment for one of the session
times. When you attend one of the session times, you will be asked to sign a Participant Consent
Form to take part in the study.

This study has been approved by the Human Research Ethics Committee of the University of
Western Australia.
Normal volunteers required

Assessing low back mobility

The Centre for Musculoskeletal Studies [CMS] at UWA invites pain free volunteers to attend a 5-10 minute assessment of their lower back mobility. The information will be used to report ‘normal low back movement’ values.

Testing will take place at the CMS laboratory, Room G12, Corner Park & Crawley Ave.

Volunteers required:

- Must be between the ages 20 and 70 years
- Have no low back problem over the last 12 months requiring treatment
- Be able to stand and perform lower back movements for approximately 2 minutes

If you would like to volunteer or would like more information please contact Aubrey Monie [Physiotherapist] by telephone on 0421 717 932 or by e-mail at aubreymonie@gmail.com

The Centre for Musculoskeletal Studies
The University of Western Australia
SF-12

This survey asks for your views about your health. This information will help you keep track of how you feel and how well you are able to do your usual activities.

Answer every question by selecting the answer as indicated. If you are unsure about how to answer a question, please give the best answer you can.

1. In general, would you say your health is:

<table>
<thead>
<tr>
<th>Excellent</th>
<th>Very good</th>
<th>Good</th>
<th>Fair</th>
<th>Poor</th>
</tr>
</thead>
<tbody>
<tr>
<td>○</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2. The following questions are about activities you might do during a typical day. Does your health now limit you in these activities? If so, how much?

<table>
<thead>
<tr>
<th>Yes, limited a lot</th>
<th>Yes, limited a little</th>
<th>No, not limited at all</th>
</tr>
</thead>
<tbody>
<tr>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
</tbody>
</table>

   a. Moderate activities, such as moving a table, pushing a vacuum cleaner, bowling, or playing golf
   b. Climbing several flights of stairs

3. During the past 4 weeks, have you had any of the following problems with your work or other regular daily activities as a result of your physical health?

<table>
<thead>
<tr>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>○</td>
<td>○</td>
</tr>
</tbody>
</table>

   a. Accomplished less than you would like
   b. Were limited in the kind of work or other activities

4. During the past 4 weeks, have you had any of the following problems with your work or other regular daily activities as a result of any emotional problems (such as feeling depressed or anxious)?

<table>
<thead>
<tr>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>○</td>
<td>○</td>
</tr>
</tbody>
</table>

   a. Accomplished less than you would like
   b. Did work or other activities less carefully than usual

5. During the past 4 weeks, how much did pain interfere with your normal work (including both work outside the home and housework)?

<table>
<thead>
<tr>
<th>Not at all</th>
<th>A little bit</th>
<th>Moderately</th>
<th>Quite a bit</th>
<th>Extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
</tbody>
</table>
6. These questions are about how you feel and how things have been with you during the past 4 weeks. For each question, please give the one answer that comes closest to the way you have been feeling.

<table>
<thead>
<tr>
<th></th>
<th>All of the time</th>
<th>Most of the time</th>
<th>A good bit of the time</th>
<th>Some of the time</th>
<th>A little of the time</th>
<th>None of the time</th>
</tr>
</thead>
<tbody>
<tr>
<td>a Have you felt calm and peaceful?</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>b Did you have a lot of energy?</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
<tr>
<td>c Have you felt downhearted and blue?</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
</tbody>
</table>

7. During the past 4 weeks, how much of the time has your physical health or emotional problems interfered with your social activities (like visiting friends, relatives, etc.)?

<table>
<thead>
<tr>
<th></th>
<th>All of the time</th>
<th>Most of the time</th>
<th>Some of the time</th>
<th>A little of the time</th>
<th>None of the time</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
<td>○</td>
</tr>
</tbody>
</table>

Thank you for completing these questions!
Appendix I.14

The Roland – Morris Low Back Pain and Disability Questionnaire

Patient name: ___________________________ File # _________ Date: __________

Please read instructions: when your back hurts, you may find it difficult to do some of the things you normally do. Mark only the sentences that describe you today.

[ ] I stay at home most of the time because of my back.
[ ] I change position frequently to try to get my back comfortable.
[ ] I walk more slowly than usual because of my back.
[ ] Because of my back, I am not doing any jobs that I usually do around the house.
[ ] Because of my back, I use a handrail to get upstairs.
[ ] Because of my back, I lie down to rest more often.
[ ] Because of my back, I have to hold on to something to get out of an easy chair.
[ ] Because of my back, I try to get other people to do things for me.
[ ] I get dressed more slowly than usual because of my back.
[ ] I only stand up for short periods of time because of my back.
[ ] Because of my back, I try not to bend or kneel down.
[ ] I find it difficult to get out of a chair because of my back.
[ ] My back is painful almost all of the time.
[ ] I find it difficult to turn over in bed because of my back.
[ ] My appetite is not very good because of my back.
[ ] I have trouble putting on my sock (or stockings) because of the pain in my back.
[ ] I can only walk short distances because of my back pain.
[ ] I sleep less well because of my back.
[ ] Because of my back pain, I get dressed with the help of someone else.
[ ] I sit down for most of the day because of my back.
[ ] I avoid heavy jobs around the house because of my back.
[ ] Because of back pain, I am more irritable and bad tempered with people than usual.
[ ] Because of my back, I go upstairs more slowly than usual.
[ ] I stay in bed most of the time because of my back.

Score: __________ Improvement: __________ %
Appendix I.15

Visual analogue scale

Absence of Pain  Worst Pain Experienced

0                        10
Validation of a triaxial protractor device prior to assessment of the MotionStar 3-D motion tracker

**Purpose:** In order to test the reliability and precision of the MotionStar™ 3-D motion tracking system (Ascension Technology, VT, USA) prior to a series of clinical studies, a measuring device with inherent accuracy and precision was used to establish a baseline reference.

**Background:** The MotionStar™ system and custom software (LabVIEW V5.0, National Instruments, Austin USA) measure movement in three dimensions. The 3-D position and angular movement of the MotionStar motion sensors are collected in a designated two meter radius field. Euler angles are transposed into X, Y and Z angles in the cardinal planes. Mieritz et al. (2012) performed a systematic review and reported the reliability and error of 3-D lumbar motion measures. Their review reports inconsistent methods, and incomplete reporting in the literature, and concludes there is uncertainty with respect to the degree that repeated measurements by 3D regional motion instruments are reproducible. Therefore, before using this apparatus to measure a subject’s lumbar spine movement it was necessary to report the repeatability of the MotionStar to measure sensor angles with acceptable accuracy and precision. A custom made triaxial protractor was constructed for the purpose of a standard while the MotionStar device was under test. In a previous study Agarwal et al. (2005) used the same triaxial protractor to validate the same MotionStar apparatus proposed for use in this series of thesis investigations. Agarwal et al. (2005) stated that the triaxial protractor was accurate to within 0.5 degrees.

Barrett (1995) used a Fastrak™ 3-D tracking device to develop and report a graphical method of recording lumbar combined movements in normal subjects and control patients. In the present study the same triaxial protractor (Figure II.1) was validated and then used as a standard to calibrate the Fastrak 3-D tracking device.
Before the triaxial protractor could be used as a standard in this study, it was reassessed using Pythagorean trigonometry and compared the results of those reported by Barrett (1995), and the 0.5 degree accuracy claim by Agarwal et al. (2005).
Hypothesis: This validation investigation hypothesises that the triaxial protractor is a reliable device for use when validating the MotionStar™ 3-D recording system. It is also hypothesised that the error reported from this investigation of the triaxial protractor is similar to the \( \leq 0.1 \) degree error reported by Barrett (1995).

The method & materials: The triaxial protractor is a hard plastic device, designed and constructed by the Bioengineering Division, Medical Physics, Royal Perth Hospital, such that the X, Y and Z-axis are orthogonal and independently movable.

Figure II.1: The triaxial protractor. Constructed such that the X, Y and Z axes are capable of moving independent to each other. [X] Movable plane for measuring X-axis rotation, [Y] Movable plane for measuring Y-axis rotation and [Z] Movable plane for measuring Z-axis rotation.

Barrett (1995) used a laser pointer placed on the triaxial protractor, with reference marks projected onto a vertical wall, and Pythagorean trigonometry used to calculate the real angle error using the formula:

\[
\text{Tan } \Theta = \frac{\text{Opposite length}}{\text{Adjacent length}}
\]
Appendix II

Where the opposite length was the length along the wall and adjacent length was a fixed distance from the wall (Figure II.2). The error analysis by Barrett (1995) indicated that the real angle could be calculated to $\leq 0.1$ degrees.

![Figure II.2](image)

**Figure II.2**: Set-up used to validate the triaxial protractor. Triaxial protractor (T), vertical length on wall (O), horizontal length from wall (A), and angle ($\Theta$). Barrett (1995).

The wall was measured for vertical at various heights using a fluid (spirit) level and an electronic level with a reported accuracy of 0.1 degrees. The wall angle from true vertical ranged from 0 to 1 degree putatively due to variations in plastering. A level horizontal surface was used to register the triaxial protractor. The surface level was checked and adjusted using the same spirit level and electronic level.

The triaxial protractor was positioned with its axis of rotation 1000mm from the wall. A laser pointer was attached to the flat surface of the triaxial goniometer (Figure II.3).
Figure II.3: The triaxial protractor with laser pointer mounted (A), spirit level (B), and positioned 1000m from the vertical wall.

A mark was made on the wall with the triaxial goniometer set at 0, 20, 40 and 60 degrees. Ten measures were made at each of the three angles. The vertical distance was then measured from the 0 degree mark to the 20, 40 and 60 degree mark, giving the vertical (opposite) length. \( \tan \theta = \frac{O}{A} \) was used to calculate the angle \( \theta \) (Figure II.2).

Results: The ten measures of vertical heights for each of the triaxial protractor axes at 20°, 40° and 60° degrees were measured and recorded. The real angles were then calculated (Table II.1).

Coefficient of variation, as a measure of dispersion of data is expressed as a percentage, and is a measure of the reliability of the method. The CV (\%) is calculated by the standard deviation (SD) of the data, divided by the mean, multiplied by 100 (Table 1).

Table II.1: Ten calculated angles with the triaxial protractor set at 20°, 40° and 60° degrees in the X, Y and Z-axes, with standard deviation (SD), mean, and coefficient of variation (CV\%) values.
Appendix II

The error reported by Barrett (1995) was less than or equal to 0.1 degrees. The error in this study was ≤ 1.9% of the calculated angle, which is at greatest 1.2 degrees, with a CV of ≤ 1.1%.

These data show, as expected, an acceptable error. The subtle inconsistencies could relate to the small deviation from a vertical wall, the size of the laser dot (approximately 2mm at 1000mm), and visually positioning the protractor with 0.5 degree increments.

**Discussion:** The same triaxial protractor, in this study, was used by Argawal (2005) and Barrett (2005). Argawal (2005) states the triaxial protractor is accurate to 0.5 degrees. Barrett (2005) reported in his validation investigation that the triaxial protractor error was ≤ 0.1 degrees (Barrett, 1995). However, the protractor has its smallest marked increments as 0.5 degrees. The ability for the operator to eyeball the angular movements of the triaxial protractor is therefore to 0.5 degrees at best.

When the proposed lumbar spine movement studies are considered, it is important to consider the acceptable error. An acceptable error will still produce results which are valid. The American Medical Association considers ±10% error or a maximum of 5 degrees error (whichever is greater) for total motion is acceptable (Gelalis et al., 2009). For biological assessment of human motion, a CV < 5% would indicate an acceptable reliability and for measures derived from static images, for example x-ray, a CV of < 2.5% would indicate acceptable reliability (Singer, 2012).

<table>
<thead>
<tr>
<th>Trial</th>
<th>Axis</th>
<th>Angle</th>
<th>X 20°</th>
<th>X 40°</th>
<th>X 60°</th>
<th>Y 20°</th>
<th>Y 40°</th>
<th>Y 60°</th>
<th>Z 20°</th>
<th>Z 40°</th>
<th>Z 60°</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>X</td>
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<td>40.79</td>
<td>61.32</td>
<td>20.15</td>
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<td>4</td>
<td>X</td>
<td>20.30</td>
<td>41.09</td>
<td>61.24</td>
<td>20.00</td>
<td>40.06</td>
<td>59.93</td>
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<tr>
<td></td>
<td>SD</td>
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<td>0.21</td>
<td>0.20</td>
<td>0.10</td>
<td>0.19</td>
<td>0.10</td>
<td>0.12</td>
<td>0.11</td>
<td>0.10</td>
<td></td>
</tr>
<tr>
<td></td>
<td>mean</td>
<td>20.02</td>
<td>41.02</td>
<td>61.40</td>
<td>20.02</td>
<td>39.96</td>
<td>59.88</td>
<td>19.94</td>
<td>39.29</td>
<td>58.15</td>
<td></td>
</tr>
<tr>
<td></td>
<td>CV (%)</td>
<td>1.14</td>
<td>0.50</td>
<td>0.33</td>
<td>0.51</td>
<td>0.48</td>
<td>0.17</td>
<td>0.62</td>
<td>0.29</td>
<td>0.17</td>
<td></td>
</tr>
</tbody>
</table>
The angular movements through the symptomatic and asymptomatic lumbar spine have been reported between 15-70 degrees (Portek et al., 1983, Pearcy, 1985, Russell et al., 1993, Barrett, 1995, Palastanga, 1995, Van Herp et al., 2000, Wong and Lee, 2004, Shum et al., 2007, Yang et al., 2008, Gelalis et al., 2009). An acceptable 5% error for lumbar range of movement would therefore be 0.75-3.5 degrees depending on the magnitude of the lumbar spine movement. This study demonstrates that the triaxial protractor is accurate to ≤ 1.2 degrees.

**Conclusion:** The validation of the triaxial protractor in this study and comparison to Barrett’s validation in 2005 demonstrates that the apparatus is accurate to 1.2 degrees. This is within acceptable error for biological assessment of human movement. The investigator concludes that the triaxial protractor measures angles with sufficient accuracy and precision to use it as a reference standard. The investigator can therefore confidently use this standard testing device to test the validity of the MotionStar™ 3-D tracking apparatus.
References:


Validation of the MotionStar™ 3-D motion tracking system
(Ascension Technology, VT, USA)

**Purpose:** In order to perform a series of clinical studies using the MotionStar 3-D motion tracking system [Ascension Technology, VT, USA] and its integrated software [LabVIEW V5.0, National Instruments, Austin USA], the device was tested for reliability and precision, before during and after data collection.

**Background:** The MotionStar apparatus and software measure movement in three dimensions. The 3-D position and angular movement of the MotionStar motion sensors are collected in a designated field for the proposed human lumbar spine study. Euler angles are transposed into X, Y and Z angles in the cardinal planes. Before using this apparatus to measure a subject’s lumbar spine movement it was necessary to report the repeatability of the MotionStar to measure sensor angles with acceptable accuracy and precision. A custom made tri-axial protractor was constructed for the purpose of a standard while the MotionStar device was under test. Barrett (1995) used the tri-axial protractor to validate the same MotionStar apparatus used in this series of thesis investigations. Argawal et al (2005) stated that the tri-axial protractor was accurate to within 0.5 degrees. Barrett (1995) stated that the tri-axial protractor was accurate to within 0.1 degrees. A previous study (Appendix II) performed as part of this thesis indicated that the real angle could be calculated to within 0.6 degrees, with a CV of ≤ 0.01%.

The three studies suggest that the triaxial protractor is reliable and accurate. It was therefore seen suitable to use as a standard to validate the reliability and accuracy of the apparatus under test, the MotionStar 3-D tracking device.

**Hypothesis:** This validation investigation hypothesises that the MotionStar 3-D tracking device and its integrated purpose-designed software (Labview V5.0, National Instruments, Austin Tex) is a reliable device for measuring angle of rotation in the three cardinal planes (X, Y and Z). It is hypothesised that repeated measures will have a CV of <5%. This is an acceptable error for
biological assessment of human motion (Singer, 2012). It is also hypothesised that the MotionStar system will remain reliable throughout the course of the studies proposed for this thesis.

**The method & materials:** A triaxial protractor (designed and constructed by the Bioengineering Division, Medical Physics, Royal Perth Hospital) was validated (Appendix II) and used as a standard (Figure III.1).

![Figure III.1 The tri-axial protractor. Constructed such that the X, Y and Z axes are capable of moving independent to each other.](image)

The MotionStar electromagnetic 3-D tracking apparatus (Figure III.2 A,B) was set up in a room free of nearby ferrous material. This was done in order to minimise any noise affecting the magnet. The MotionStar manufactures (Ascension Technology) customer support was consulted regarding system set-up and ‘noise’ reduction and introducing a new direct current power source, thus eliminating the use of varying power from the battery pack.
Figure III.2 The validation testing area, The MotionStar electromagnetic device (2A), the tracking sensors mounted on the protractor (2B), and Triaxial protractor (C). Figure 2B (Right) Close up of the two MotionStar tracking sensors.

One sensor was firmly attached to the tri-axial protractor in order to sense movement in the X-axis. With the software collecting data from the sensor at 50 Hz, the tri-axial protractor was moved by hand from 0 degrees to 20 degrees and repeated at least 10 times. This process was repeated for angles 40 degrees and 60 degrees in the X-axis. The same angles were recorded in the Y-axis and Z-axis, thus collecting 10 samples for 3 different angles in each of the 3 cardinal planes.

The raw data was filtered using batch processing to remove background electromagnetic noise equally in all processed data. Using general data processing software, the following parameters were used for filtering:

- A Butterworth Second order Filter was used because impulsive movements are rarely seen in human movement (Winter, 1990)

- Low Pass Filter 4Hz

Appendix III

- Low cut frequency of 4 (usually between 2 and 5) (Winter, 1990)
- Mirrored 100, Phase lag ON

These parameters are explained in Appendix IV. The processed data was then viewed using Microsoft Excel software. A typical filtered graph is shown in figure 3.

Figure III.3 A Microsoft Excel graph of filtered data showing 12 samples taken at 20 degrees rotation in the Z-axis.

**Results:** A sample of 10 points from each of the ‘peaks’ and ‘troughs’ on the graphs were tabulated for each angle (20, 40 and 60 degrees) in each axis (X, Y and Z).

The Coefficient of variation [CV] was calculated for each sample using the following formula:

\[
CV = \frac{\text{Standard Deviation}}{\text{mean}}
\]

Coefficient of variation, as a measure of dispersion of data is expressed as a percentage and therefore of reliability of the method and is derived from the standard deviation [SD] divided by the mean.
A typical table of data is shown for the graph in figure 3, with 10 data points in a column from 10 separate ‘peaks’ of 20 degrees of angular movement in the Z-axis (Table III.1).

Table III.1: Ten data samples taken from 10 separate peaks at 20 degrees rotation in the X-axis. The overall average value and coefficient of variation [CV%] is shown.

<p>| | | | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
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<th></th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>20.11</td>
<td>19.97</td>
<td>19.58</td>
<td>19.89</td>
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<td>20.04</td>
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<td>20.09</td>
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<td>20.03</td>
<td>20.11</td>
<td>20.11</td>
<td>20.00</td>
</tr>
</tbody>
</table>

Average  19.96  
CV       0.01

The results demonstrate that the apparatus is capable of measuring angular movement with an error $\leq 1\%$ (0.3 degrees) of the total angle tested.

Coefficient of variation values for each direction at four testing locations over a period of three and a half years, are shown in Table III.2
Table III.2 Coefficient of variation (CV) values from the MotionStar tracking system validation at the four testing locations over three and a half years.

<table>
<thead>
<tr>
<th>Validation Date</th>
<th>The University of Western Australia</th>
<th>Pain Medicine Dept</th>
<th>Neurosurgery Dept</th>
<th>Private Practice</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV value (Flexion)</td>
<td>0.002</td>
<td>0.001</td>
<td>0.002</td>
<td>0.002</td>
</tr>
<tr>
<td>CV value (Side flexion)</td>
<td>0.003</td>
<td>0.002</td>
<td>0.000</td>
<td>0.008</td>
</tr>
<tr>
<td>CV value (Rotation)</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
<td>0.004</td>
</tr>
</tbody>
</table>

Each CV value is calculated from 100 data points: 10 measures, each of 10 data values collected at 50Hz. Dept = Department.
**Discussion:** The overall CV values for all samples at all angles (20, 40 and 60 degrees) were ≤ 1.0%. The highest CV, which was measured at 20 degrees, was 0.76% spread. The examiners note that the error (CV) is smaller when using the direct current power source compared to the battery power supply.

This error over a 20 degree angle of movement is clearly not clinically significant and is well below the acceptable 5% error for biological assessment involving human motion.

Retesting the MotionStar system at each of the four examination locations, over the three and a half year period, shows consistency in measurement over time. The data show ≤ 1.0% error in each of the three axes, at each location.

**Conclusion:** The MotionStar 3-D motion tracking system and its integrated software was tested for reliability and precision on several separate occasions to test consistency over time and in different testing environments (The University of Western Australia, QEII Hospital pain management and outpatient neurosurgery departments, and a private physiotherapy practice). The results demonstrate that the apparatus is capable of measuring angular movement with an error ≤ 5%. In fact, the largest variation recorded was < 1% (0.3 degrees) of the total angle tested. Gatton and Pearcy (1999) reported an error of 0.4 degrees using the same brand of 3-D motion tracker on the lumbar spine. Clearly, the error measured in our study and Gatton and Pearcy (1999) is an insignificant error for the purpose of using the MotionStar™ apparatus to test human lumbar spine angular movement.
References:


Singer, K. P. 2012. RE: Winthrop professor and director of the centre for musculoskeletal studies. School of surgery. The University of Western Australia. Crawley, WA.

**MotionStar Sensor fixation to the skin overlying the L1 and S1 spinous processes**

**Background:** The MotionStar device records the angle and position of sensors using Euler’s angles. The sensors are accurate to $\geq 0.6^\circ$ (Appendix III). However, it is important that the two sensors are fixed to the skin well enough to avoid system error. Before attempting data acquisition, several previous studies were reviewed (Pearcy, 1985, Burton, 1986, Pearcy, 1986, Cohn et al., 1989, Barrett, 1995, Barrett et al., 1999, Gatton and Pearcy, 1999, Edmondston et al., 2000, Van Herp et al., 2000, Wong et al., 2004, Cargill et al., 2007, Yang et al., 2008, Bible et al., 2010, Moga, 2010, Kilby et al., 2012). Several tests were performed in the laboratory with various methods of locating landmarks and combinations of fixing techniques. The final method, which proved to be effective by inspecting the raw data and by reassessing sensor fixation post testing, is described.

**Landmarks for sensor placement**

**S1 Sensor:** The horizontal line connecting the left and right posterior superior Iliac spines (PSIS) is at the level of S2. Therefore – positioning the bottom of the sensor on this line gives a consistent landmark over S1, with bias towards the S1-S2 rather than the L5-S1 joints. Any error from sensor placement would therefore have an insignificant affect because the sacral segments are fused, laterally, by the age of 20 years (Baker B.J., 2005). Favouring the lower portion of S1 also eliminates the possibility of placing the sensor over the often mobile L5/S1 segment.

**L1 Sensor:** L1 is located by palpating for the L5 spinous process, which is the first process felt superior to the depression superior to the Sacrum. For ease of distinguishing L5 from L4, it is useful to know that the L4 spinous process is more superficial and
Appendix IV

larger than the L5 spinous process (Kilby et al., 2012). By palpation and counting up from the L5 spinous process, L1 can be identified. Care should be taken by palpating between the relatively large spinous processes, for example, the average L4 spinous process is 20.8mm (Kilby et al., 2012) and L1 spinous process is significantly larger than that of T12 (Snider et al., 2011). Palpation and counting segments was made easier by asking the subject to bend forward slightly and resting their hands on their thighs, therefore gapping the spinous processes and pre-tensioning the skin, which aids for improved sensor fixation during the flexes CME positions.

While using palpation to identify landmarks is considered unreliable, the reliability may be related to the experience of the examiner (Kilby et al., 2012) and by using more than one method of location L4 versus L5. Cooper et al. (2013) reported that two experienced manual therapists were able to correctly identify the location of L4 spinous process 71% of the time and the average error was error was small, 2.7mm. Using a permanent marker pen also assisted with identifying the same landmark for the inter-session reliability study. Once the positions were marked, a second check was completed by visually inspecting the sensor location. The sensors should be approximately 20 cm apart and the L4 region should be approximately level with Tuffier’s line (Snider et al., 2011), an imaginary line across the low back level with the highest point of the Iliac crests.

Palpation of the lower ribs to locate spinous processes can result in error. A study by Snider et al. (2011) reported that 27% of their subjects (n=60) had lower rib anomalies.

Sensor fixation over the L1 and S1 spinous processes:
The skin was marked over the L1 and S1 spinal levels. Skin preparation ‘glue’, Tensospray® Adhesive, was sprayed over the surface markings. A paper-thin double sided tape (3M®) was cut into small squares to cover the surfaces of the sensors. The
Sensor used for measuring angular movement at the L1 level was fixed to a donut shaped semi-rigid block approximately 30mm$^2$. The donut block provided a greater surface area of contact around the, often bony, L1 spinous process. In subjects requiring repeated tests, in the intersession reliability cohort and test-retest intervention studies, a measurement was taken, using a tailor’s tape, between the markings while the patient was in the semi-flexed position with hands on their knees. This improved accuracy by using both palpation and measurement in following tests. The sensor fixation was monitored by the examiner during trials. In some cases, such as those who presented with excess low back perspiration, the fixation was corrected and the trial repeated, however, in almost all cases the sensors remained firmly adhered to the skin.
Figure 0 Equipment used to fix the sensors to the skin overlying L1 and S1 spinous processes. Marker pen (A), Tensospray® skin ‘glue’(B), tailor’s tape (C), scissors (D), double sided tape (E).

Figure 0 Postero-anterior view of sensors fixed at L1 and S1
Figure IV.3 Method of fixating the sensor to the skin overlying the L1 spinous process. Palpate and mark the spinous process (black dot), double sided tape (in red) (A), donut shaped rubber block (B), sensor (C).

**Calculating the angular movement of the Lumbar spine**

The range of motion in the lumbar spine was calculated by subtracting the angle of the S1 sensor from the angle of the sensor at L1. A resulting negative angle indicates that the lumbar spine vertebra is in extension. A resulting positive angle indicates that the lumbar spine is in flexion. For example, Figure IV.4A shows the angle of the L1 sensor is -20° and the angle of the S1 sensor is -5°. Therefore the resultant angle is -20 – [-20] = -15° or 15 degrees extension. In Figure IV.4B the L1 sensor is at an angle of +20 degrees and the angle of the S1 sensor is +10 degrees. Therefore the resultant angle between the two sensors is +20 – [+10] = +10 degrees or 10 degrees flexion.
The individual angles of each sensor and resultant angle is given for the relaxed standing position (A), and flexed position (B).

**Summary**

The method adopted in this thesis investigation, for palpating and locating the L1 and S1 spinous processes on subjects, marking and fixating the MotionStar sensors, and calculating the angle of lumbar lordosis is described.
Appendix IV

References


Appendix IV


Assessing the clinical utility of combined movement examination in symptomatic degenerative lumbar spondylosis

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b Department of Medical Technology and Physics, Sir Charles Gairdner Hospital, Perth, Western Australia 6000, Australia
c Department of Neurosurgery, Sir Charles Gairdner Hospital, Perth, Western Australia 6000, Australia

Abstract

Objectives: The aim of this study is to report the development and validation of a low back computer-aided combined movement examination protocol in normal individuals and record treatment outcomes of cases with symptomatic degenerative lumbar spondylosis.

Design: Test–retest, following intervention.

Background: Self-report assessments and combined movement examination were used to record composite spinal motion, before and following neurosurgical and pain medicine interventions.

Methods: 151 normal individuals aged from 20 years to 69 years were assessed using combined movement examination between L1 and S1 spinal levels to establish a reference range. Cases with degenerative low back pain and sciatica were assessed before and after therapeutic interventions with combined movement examination and a battery of self-report pain and disability questionnaires. Change scores for combined movement examination and all outcome measures were derived.

Findings: Computer-aided combined movement examination validation and intraclass correlation coefficient with 95% confidence interval and least significant change scores indicated acceptable reliability of combined movement examination when recording lumbar movement in normal subjects. In both clinical cases lumbar spine movement restrictions corresponded with self-report scores for pain and disability. Post-intervention outcomes all showed significant improvement, particularly in the most restricted combined movement examination direction.

Interpretation: This study provides normative reference data for combined movement examination that may inform future clinical studies of the technique as a convenient objective surrogate for important clinical outcomes in lumbar degenerative spondylosis. It can be used with good reliability, may be well tolerated by individuals in pain and appears to change in concert with validated measures of lumbar spinal pain, functional limitation and quality of life.

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1. Introduction

Pragmatic treatment of patients with low back pain (LBP) and associated movement dysfunction is based upon an appreciation of the history of the patient’s complaint and interpretation of the examination findings (Maitland, 1997). Assessing lumbar spine movement in the clinical setting to investigate dysfunction and to monitor changes in spinal movement characteristics of individuals over time, is routine clinical practice (Ha et al., 2013; Laird et al., 2014; Lyle et al., 2005; Maitland, 1997). Single plane movements are often unrepresentative of the actual movements of the lumbar spine, so have limited value in assessing lumbar function (Pearcy and Hindle, 1989). However, one examination method, originally described by Edwards (1979), which assesses both planar and combined plane (physiological) positions, is the combined movement examination (CME). The CME sequentially examines the patient’s ability to actively side-flex the lumbar spine while in a flexed, neutral and extended position (Fig. 1).

Edwards (1979) and Maitland (1997) suggested that CME may be more informative than the standard planar spinal assessment, which was confirmed by Barrett et al. (1999) who reported acceptable CME intra-examiner reliability, as well as preliminary evidence concerning the effectiveness of CME in identifying reduced lumbar mobility in low back pain (LBP) subjects. In their work Barrett et al. used 3-D tracking to map the characteristics of the lumbar CME. To our knowledge, no other study has reported the putative use of lumbar CME in clinical practice using a computer-aided methodology to quantify movement.
patterns. Fig. 1 illustrates the eight low back positions of a lumbar CME and an example radial plot of a healthy volunteer, showing the symmetrical end-points (maximal angular movement) achieved.

The purpose of this paper is to report the intra- and inter-session reliability of lumbar CME using a validated MotionStar™ 3-D motion tracking system (Ascension Technology, VT, USA) (Fig. 2A) using custom software (LabVIEW V5.0, National Instruments, Austin USA). Secondly, to describe the development of a normal reference range (NRR) and subsequently to report proof of concept of CME as a tool to assess specific spinal pathology and monitor changes post-intervention. A CME NRR was developed to identify abnormal patterns, observe normalisation of CME post-intervention and compare the age and gender matched functional outcomes of two cases with different lumbar spine pathologies. It is not the intention of this paper to report clinical studies which did not use an objective quantification of lumbar spine movement.

2. Methods

2.1. Validation of a non-invasive 3-D motion tracking system

A MotionStar™ 3-D motion tracking system was tested in our laboratory against a custom made triaxial goniometer to test consistency over time (Fig. 2A). Angular displacement of the sensors was calculated by the system, using Euler’s method. Results demonstrated that the MotionStar™ is capable of reliably measuring angular movement with an intrinsic precision of 0.6°.

2.2. CME data collection

After obtaining written consent and familiarisation of equipment and testing sequence, two skin mounted MotionStar™ sensors were placed over the volunteer’s S1 and L1 spinous process, respectively.
2.3. Reliability of computer-aided combined movement examination

Combined movement examination reliability data were assessed with intraclass correlation coefficients (ICC) and 95% confidence intervals for the five intra-session (n = 10) and five inter-session (n = 10) trials. In addition, the least significant change (LSC) method (Bonnick and Lewis, 2013) was used to represent variance of the CME outcomes. Data confirmed acceptable reliability for all eight CME movement directions (Table 1).

2.4. Development of a normal reference range

MotionStar™ derived CME data was captured to develop a NRR for a convenience sample of 151 asymptomatic participants was used. Volunteers were included in this study if they were aged between 20 and 69 years, had no previous spinal intervention, had no significant episode of low back pain requiring treatment in the previous 6 months, were able to follow verbal instructions and had a BMI ≤ 30. The NRR is displayed in Table 2.

2.5. Examination of two cases of low back pain

Case A was a 53 year old male who presented with an antalgic gait, mild LBP and severe, acute anterolateral hip pain (VAS 9.7/10). Magnetic resonance (MR) imaging demonstrated a large left disc extrusion at the L2–3 level, with inferior sequestration of disc material resulting in impingement on the left L3 nerve (Fig. 3A, B). A discectomy was performed and post-operative assessments (self-reports and CME) recorded.

Case B was a 62 year old female who presented with chronic, intermittent medial shin pain aggravated by lumbar extension. Computerised tomography (CT) identified a hypertrophic L4–5 facet joint impinging on the L4 nerve root with associated L4 exit foramen stenosis (Fig. 4A, B).

Table 1

<table>
<thead>
<tr>
<th>Position</th>
<th>Intra-session</th>
<th>Inter-session</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICC, CI</td>
<td>LSC</td>
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<tr>
<td>Flexion</td>
<td>0.92 (0.85–0.98)</td>
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<td>FwRSF</td>
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<tr>
<td>LSF</td>
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</tr>
<tr>
<td>FwLSF</td>
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</tbody>
</table>

Least significant change (LSC) in degrees calculated at p < 0.05 and 1 measurement per inter-session visit.
Table 2
Combined movement examination normal reference range for males and females aged 20 to 69 years of age. The mean, standard deviation and range for standing lordosis, BMI and each movement direction for males and females.

<table>
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<tr>
<th>Gender</th>
<th>Age</th>
<th>n</th>
<th>Statistic</th>
<th>BMI</th>
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<th>Flexion</th>
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<th>RSF</th>
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<tr>
<td></td>
<td>30–39</td>
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<td>Mean (SD)</td>
<td>25.7 (2.9)</td>
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<td>46.4 (5.5)</td>
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<td>22.7</td>
<td>16.4</td>
<td>17.0</td>
<td>19.0</td>
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</tbody>
</table>

Lumbar lordosis and angular movement values are in degrees [°]. For explanation of acronyms, refer to Fig. 1.

Fig. 3. T1 sagittal MR image of a 22 mm sequestration of L2–3 disc material (A). T2 axial MR image of the posterolateral position of the extruded disc material (B). Patient’s standing position 16 days pre-operatively (C) compared with 24 weeks post-operatively (D).

Fig. 4. Transverse CT image of the left L4 nerve root (A). Sagittal CT image of the stenotic L4 exit foramen (B). Patient’s active extension 1 week prior to L4–5 epidural injection (C) and 12 weeks post injection (D).
2.6. Outcome measures

The core battery of outcome measures were used to assess the patients pre-intervention and at three intervals, post-intervention (Deyo et al, 1998): Visual Analogue Scale (VAS), Roland Morris Low Back Pain and Disability Questionnaire (RMDQ) and a Short Form health survey (SF-12). The two cases participated in a CME trial on each of four assessment days with their CME values compared to our age and gender matched NRR. A change of $\geq 30\%$ in all measures was considered clinically significant (Ostelo et al., 2008).

For the eight CME directions (Fig. 1) the maximum values were displayed in a radial plot and change scores calculated between trials.

3. Results

The maximum values for each of the patient’s CME movements were plotted to observe changes over time. Fig. 5A illustrates the pre- and final post-operative trials for Case A, plus a comparison age (50–59) and gender matched normal plot. Fig. 5B illustrates the initial and final test values (total change) collected from CME of Case B. The age (60–69) and gender matched NRR data is plotted for comparison.

The natural standing lumbar lordosis and data values for each of the CME directions (Table 3) were compared with the matched CME NRR. Total change scores ($\%$) in angular movement for each CME direction, between pre-intervention (trial 1) and post-intervention (trial 4) assessments are reported in Table 3.

Fig. 5. The first and last trial for Case A and the average normal male CME data for 50–59 years of age (A). Case B shows pre-injection and final post-injection CME. The average normal female CME data for 60–69 years of age (B). Data are in degrees of angular movement.
Self-report outcome data for the two cases, for the index assessments are presented in Table 4. Clinically important improvements (≥30%) are evident in the VAS, RMDQ, and SF-12 domains.

4. Discussion

Outcome assessment for low back pain is complex and typically involves multiple dimensions. Self-report surveys and lumbar kinematics provide insight into the response of low back conditions to management (Deyo et al., 1998; Williams et al., 2013). Measures should be reliable, valid, practical, and for convenience, brief where possible. According to Deyo et al. (1998), assessments of severity and frequency of symptoms such as the RMDQ and SF-12 outcome measures for low back dysfunction are particularly useful for research purposes. However, outcome measures placing emphasis on pain, function and quality of life do not provide the clinician with feedback on the direction and magnitude of movement pattern disturbance (Lyle et al., 2005), the potential structure(s) at fault or departure from normal movement according to age and gender.

In this report we describe the development of a CME assessment model for the lumbar spine. Intra-session and inter-session trials showed CME to be very reliable for all movement directions. This is consistent with the study by Mieritz et al. (2012) reporting a systematic review on the reliability of 3-D measures of the lumbar spine. Further, we have established a preliminary NRR for lumbar CME with which to contrast cases of specific lumbar dysfunction.

To date, only Barrett et al. (1999) have reported preliminary evidence for use of the CME to identify reduced spinal mobility in LBP patients. They did not report normal reference values or test for directional movement restrictions and attribute these to specific diagnoses. The present report investigated the novel application of CME to assess change to both the magnitude and direction of dysfunction and consequently to demonstrate a tendency towards age and gender matched normalisation of low back movement after intervention. Spinal 3-D motion behaviour was described by Ha et al. (2013) as a useful assessment in monitoring changes in spinal movement in an individual over time. As spinal movement is not isolated to pure cardinal planes, 3-D information may confer greater insight into the clinical analysis of aberrant spinal mechanics.

The ability for CME to detect specific directions of restricted movement and to predict biomechanical causes has been hypothesised though not previously examined (Brown, 1988). In this investigation the CME had sufficient sensitivity to detect the greatest restriction and subsequent improvement, in the direction of the confirmed structural spinal abnormality and response to composite loading. Interestingly, passive spinal structures make up the majority of the common pathologies in the lumbar spine (Press, 2007). According to Cunningham et al. (2007) and Little et al. (2007) osseoligamentous tissues and the disc anulus are the primary contributors to spinal stiffness. This may direct a clinician to consider specific structures causing movement restrictions in specific CME directions. Peacry and Hindle (1989) discuss the potential diagnostic value of 3-D lumbar movement assessment however no studies have substantiated this claim in pathoanatomical terms.

An age and gender matched CME NRR was used to guide provisional outcome goals. When observing Case A’s final CME radial plot (Fig. 5A), it is clear that the original movement restriction was normalised, resulting in a symmetrical pattern, comparable to the age and gender matched CME plot. Fig. 5B highlights that the extension range of Case B, after 12 weeks, though no longer painful or causing reported disability, is still well below the age and gender matched average. Facet joint hypertrophy at the L4–5 level (Fig. 4A) would contribute to the patient’s movement impairment. Patients, who demonstrate little change in their CME pattern, yet report marked clinical improvement in their pain and function—as in Case B, may reflect a sub-group with structural pathology for whom initial pain management is the appropriate intervention prior to a surgical opinion if symptoms persist. In both cases, the self-report results highlight clinically important improvements in pain, disability and health outcomes. In Case B, her pain score decreased by 23% (Table 4), to zero (VAS 0/10), at the twelve week final assessment.

Several previous studies have reported planar lumbar motion measures (Madson et al., 1999; Peacry and Hindle, 1989; Troke et al., 2005), very few have described combined or coupled lumbar movement (Ha et al., 2013; Russell et al., 1993) and no report to our knowledge uses CME as an outcome measure when comparing symptomatic cases to an age and gender matched NRR. Furthermore, there seems to be a lack of normative data which can be used to inform outcomes from intervention to manage spinal pain. A systematic review and meta-analysis by Laird et al. (2014), comparing lumbar kinematics in people with and without LBP, concluded that their results do not improve the understanding of the relationship between movement and pain in individuals. They also noted the difficulty in attempting a meaningful interpretation of the data due to the varied methodologies, samples and symptoms reported by the different studies.

Future investigations: Further studies are currently underway with larger clinical cohorts of cases diagnosed with: lumbar stenosis; confirmed facet joint dysfunction or disc herniation. We will test the hypotheses that CME can assist in the provisional diagnostic subgrouping of mechanical back pain syndromes and, with the use of a NRR, predict the extent, rate and pattern of recovery from specific neurosurgery or pain management interventions.

Table 3
Maximum angular movement for each of the patient’s CME movement directions, plus standing lordosis values at each trial with total change scores (%).

<table>
<thead>
<tr>
<th>Case A CME Trials</th>
<th>Flexion</th>
<th>FwRSF</th>
<th>RSF</th>
<th>EwRSF</th>
<th>Extension</th>
<th>EwLSF</th>
<th>LSF</th>
<th>FwLSF</th>
<th>Lordosis</th>
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</thead>
<tbody>
<tr>
<td>2 weeks pre-op</td>
<td>38.9</td>
<td>36.8</td>
<td>11.2</td>
<td>3.2</td>
<td>−2.5</td>
<td>−3.2</td>
<td>−5</td>
<td>31.1</td>
<td>23.1</td>
</tr>
<tr>
<td>24 weeks post-op</td>
<td>45.2</td>
<td>42.6</td>
<td>20</td>
<td>4.9</td>
<td>7.6</td>
<td>7.6</td>
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<td>38.5</td>
<td>36.9</td>
</tr>
<tr>
<td>Total change (%)</td>
<td>16.2%</td>
<td>15.8%</td>
<td>78.6%</td>
<td>53.1%</td>
<td>404.0%</td>
<td>337.5%</td>
<td>442.0%</td>
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<td>59.7%</td>
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<td>1 week pre-injection</td>
<td>39.8</td>
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<td>12 weeks post-injection</td>
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<td>36.7</td>
<td>17.4</td>
<td>4.9</td>
<td>7.1</td>
<td>5.1</td>
<td>16.8</td>
<td>30.8</td>
<td>34.1</td>
</tr>
<tr>
<td>Total change (%)</td>
<td>0.5%</td>
<td>−3.2%</td>
<td>−5.0%</td>
<td>23.4%</td>
<td>45.8%</td>
<td>11.6%</td>
<td>23.2%</td>
<td>−19.1%</td>
<td>−1.8%</td>
</tr>
</tbody>
</table>

All angular movement values are in degrees (°). For explanation of acronyms, please refer to Fig. 1.

Table 4
Change in self-report instruments (pre- and post-intervention) for Visual Analogue Scale (VAS), Short Form Health Survey Physical Component Score (SF-12 PCS), Short Form Health Survey Mental Component Score (SF-12 MCS) and Roland Morris Low Back Pain Disability Questionnaire (RMDQ).

<table>
<thead>
<tr>
<th>Case A</th>
<th>VAS LBP</th>
<th>VAS Hip</th>
<th>SF-12 PCS</th>
<th>SF-12 MCS</th>
<th>RMDQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 weeks pre-op</td>
<td>0.8</td>
<td>9.7</td>
<td>26.8</td>
<td>32.9</td>
<td>20</td>
</tr>
<tr>
<td>24 weeks post-op</td>
<td>0.2</td>
<td>0.2</td>
<td>55.5</td>
<td>57.8</td>
<td>1</td>
</tr>
<tr>
<td>Improvement (%)</td>
<td>75.0%</td>
<td>97.9%</td>
<td>50.7%</td>
<td>41.0%</td>
<td>79.2%</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Case B</th>
<th>VAS Shin</th>
<th>SF-12 PCS</th>
<th>SF-12 MCS</th>
<th>RMDQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 week pre-epidural</td>
<td>2.3</td>
<td>39.9</td>
<td>47.0</td>
<td>10</td>
</tr>
<tr>
<td>12 weeks post-epidural</td>
<td>0.0</td>
<td>59.9</td>
<td>43.6</td>
<td>0</td>
</tr>
<tr>
<td>Improvement (%)</td>
<td>100.0%</td>
<td>33.3%</td>
<td>−5.6%</td>
<td>41.7%</td>
</tr>
</tbody>
</table>
The present study provides normative data for CME that may inform future clinical studies of this technique as a convenient objective surrogate for important clinical outcomes in lumbar degenerative spondylosis. It can be used with good reliability, may be well tolerated by individuals in pain and appears to change in concert with validated measures of lumbar spinal pain, functional limitation and quality of life.

5. Conclusion

The CME is a reliable movement examination and may be a useful outcome measure for individuals with low back movement dysfunction. A normal CME reference range provides an expected movement outcome matched to age and gender, although care must be taken to consider individual anatomical variations and clinical presentations. These case-studies provide initial evidence that CME may possess sufficient sensitivity to detect the nature of spinal dysfunction and the natural history following intervention.

Conflict of interest statement

No authors have benefitted financially as a direct consequence of this study or publication.

References


Data acquisition and processing

Introduction

The series of studies included in this thesis used the MotionStar 3-D motion tracking system (Ascension Technology, VT, USA) and its integrated software (LabView V5.0, National Instruments, Austin Tex). The MotionStar apparatus (Appendix III, Figure 2A) created an electromagnetic field with a 2m radius. The movement of sensors (Appendix III, figure 2B) were detected within the electromagnetic field. The position and angle of the sensors were calculated by the system, using Euler angles. These are expressed as X, Y and Z position coordinates, in inches, and X, Y and Z angles, in degrees. The LabView software converted the position data to Standard International units, centimetres, for easier interpretation and comparison with existing literature. Figure V.1 shows a typical graph displayed by the software after data collection.
Figure V.1 A Y-axis graph obtained from data collected at the sensor over the L1 spinous process. A complete trial of the combined movement examination is shown.

**Data Processing**

The data was then labelled with the subjects initials and test name, for example, AM pre-surgery, and saved as “raw data” in a Microsoft Excel, comma separated value file. A typical collection over 58 milliseconds is displayed in Table V.1. This data was then time normalised with software designed to display data every 20 milliseconds, a frequency of 50Hz. Winter (1990) states that in normal gait studies, data acquired at 24Hz results in negligible error. This study has obtained data at twice this rate.

Table V.1 Fifty-eight milliseconds of data recorded by the sensors at L1 and S1.

<table>
<thead>
<tr>
<th>14 Pos X</th>
<th>14 Pos Y</th>
<th>14 Pos Z</th>
<th>14 Angle Z</th>
<th>14 Angle Y</th>
<th>14 Angle X</th>
<th>15 Pos X</th>
<th>15 Pos Y</th>
<th>15 Pos Z</th>
<th>15 Angle Z</th>
<th>15 Angle Y</th>
<th>15 Angle X</th>
<th>Time (ms)</th>
</tr>
</thead>
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<td>1.80938</td>
<td>-39.5264</td>
<td>2.2608</td>
<td>84.80113</td>
<td>3.66128</td>
<td>1.49709</td>
<td>-23.6778</td>
<td>3.63017</td>
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<tr>
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<td>2.18945</td>
<td>84.76765</td>
<td>3.72826</td>
<td>1.56512</td>
<td>-23.6167</td>
<td>3.64003</td>
<td>12</td>
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<tr>
<td>93.74226</td>
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<td>0.06697</td>
<td>1.77852</td>
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<td>3.68361</td>
<td>1.51572</td>
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<tr>
<td>93.75574</td>
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<td>1.58366</td>
<td>-23.7275</td>
<td>3.55882</td>
<td>47</td>
<td></td>
</tr>
</tbody>
</table>

**Batch Processing: Filtering, zeroing and subtracting S1 angle from L1 angle**

Batch processing software was created by a Scientific Programmer (Smith, 2013). The software performed 3 processes. These are explained below.

*Filtering of data:*

Data collected by the MotionStar electromagnetic system includes ‘noise’. Noise are components in the final signal which are not due to the process itself (Winter, 1990). An example of this is ambient interference from external electromagnetic signals, vibration and nearby metallic materials (Bronner, 2012, Smith, 2013). In general, equipment noise has a much higher frequency signal (Winter, 1990, Challis, 1999, Bronner, 2012). Removing high frequency noise from the data before further calculations prevents the noise from being magnified and possibly effecting the results (Challis, 1999, Bronner, 2012).

The parameters used to filter the data in these studies are briefly explained below:
**Butterworth 4th order Filter:** Also known as a maximally flat filter, this filter removes noise so that a plotted graph appears smooth. Biological studies of human movement are not impulsive (Winter, 1990), therefore the Butterworth 4th order filter is suitable for the CME. The order of a filter determines the amount of additional ‘smoothing’ required for the frequencies above the cut-off frequency.

**Low Pass Filter:** The Nyquist critical frequency is the basis of the sampling theorem (Bronner, 2012, Smith, 2013). This states that the sampling must be at least twice the frequency than the highest frequency of the signal to be recorded (Bronner, 2012). Since we are not expecting to see human movement at a frequency higher that 2Hz, the data was filtered to remove signals higher than 4Hz (Smith, 2013).

**Mirrored:** Mirroring is used at the front of the data, during quiet standing, to allow the filter to become stable before the actual CME data is recorded. It takes a sample of data points, reverses them and then appends them to the front of the data. The filter is then applied to the data, and then the sample points are removed.

**Zero lag:** This runs the filter forwards then backwards to remove the phase distortion of the Butterworth filter. The order is halved, in these studies two, and the cut off frequency is modified to allow for the double filtering, divided by 0.802, so that the final result would be the same as the 4th order filter but without the phase distortion.

**Zeroed start position:**

This process set the starting point of the data collection to ZERO. In ‘quiet standing’ before the subject commenced the CME, both sensors are at ‘zero’ position and angle. From this initial neutral standing position, if the Y-axis is considered, a positive (+) angle represents flexion and a negative (-) angle represents extension of the spinal segment. By zeroing the data the magnitude of movement was easily read off a graph. Figure V.2 shows a typical Y-axis graph of a CME.

**Subtracted S1 angle from L1 angle:**
The third process performed during the batch processing was the subtraction of the S1 sensor data from the L1 sensor data. This gave the resulting angle of movement of the lumbar spine, between the L1 and S1 spinous processes (Figure V.3).

Figure V.3 A graph of the L1 sensor (blue), the S1 sensor (red), and the subtracted data (green) of a complete combined movement examination. Positive values are in the flexed direction, and negative values are in the extended direction, relative to the beginning of the test, in standing.
References


Smith, R. 2013. RE: [scientific programmer. School of surgery, the university of western australia] consulted for data filtering software, motionstar and labview technical faults, data collection advice and displaying of data by graphs.

The lumbar combined movement examination normal reference range: supplementary data

Final normal reference range data

The normal reference range (NRR) reported in Monie et al. (2015) consisted of 151 participants. During the course of the studies, additional participants were examined. The final NRR of 192 subjects is displayed in Table VII.1, with the average decrease in ROM from the 20-29 year old subjects to the 60-69 year old subjects, show in Table VII.2

Combined movement examination pattern for the asymptomatic lumbar spine

The relative proportions of ROM for each CME movement direction were calculated for each gender.

Range of motion characteristics for the average asymptomatic CME movement pattern is illustrated in Figure VII.1
Table VII.1 Combined movement examination normal reference range for males and females aged 20 to 69 years. The mean, standard deviation and range for standing lordosis, BMI and each movement direction for males and females.

Lumbar lordosis and angular movement values are in degrees (°). Flexion with right side-flexion (FwRSF), flexion with left side-flexion (FwLSF), right side-flexion (RSF), extension with right side-flexion (EwRSF), extension with left side-flexion (EwLSF), left side flexion (LSF), flexion with left side-flexion (FwLSF).

<table>
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<tr>
<th>Age</th>
<th>n=</th>
<th>Statistic</th>
<th>BMI</th>
<th>Lordosis</th>
<th>Flexion</th>
<th>FwRSF</th>
<th>RSF</th>
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<th>Extension</th>
<th>EwLSF</th>
<th>LSF</th>
<th>FwLSF</th>
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<td>25.6 (7.8)</td>
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Table VII.2 The average decrease in range of motion, for each combined movement examination direction, from the 20-29 year old subjects, to the 60-69 year old subjects.

<table>
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<th></th>
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</tr>
</tbody>
</table>

Average relative range of motion for asymptomatic subjects

Figure VII.1 The relative proportions of CME movement directions, using average range of motion data for both genders.

Reference

Case report

Computer-aided combined movement examination of the lumbar spine and manual therapy implications: Case report

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a Centre for Musculoskeletal Studies, School of Surgery, The University of Western Australia, Perth, Western Australia 6009, Australia
b Department of Medical Technology and Physics, Sir Charles Gairdner Hospital, Perth, Western Australia 6009, Australia
c Department of Neurosurgery, Sir Charles Gairdner Hospital, Perth, Western Australia 6009, Australia

Abstract

Combined movement examination (CME) of the lumbar spine has been recommended for clinical examination as it confers information about mechanical pain patterns. However, little quantitative study has been undertaken to validate its use in manual therapy practice.

This study used computer aided CME to develop a normal reference range, and to guide provisional diagnosis and management. Two cases were assessed, before and after manual therapy using CME, a pain Visual Analogue Scale, the Roland Morris Low Back Pain and Disability Questionnaire and the Short Form (SF-12) Health Survey. Diagnosis and management were guided by comparing each CME pattern with the age and gender matched reference range. Self-reports data and CME total change scores were markedly improved for both cases, particularly for the most painful and restricted CME directions.

This report describes how computer-aided CME and a normal reference range may be used objectively to inform a diagnosis and as an outcome measure in cases of mechanical LBP. Future investigations of cases with specific lumbar pathologies are required to validate this concept.

1. Introduction

Assessing lumbar spine movement in the clinical setting to investigate dysfunction and to monitor changes in a patient’s spinal movement characteristics over time is routine clinical practice (Maitland, 1997; Ha et al., 2013; Laird et al., 2014). This is used, along with other assessment findings, to develop a provisional diagnosis, treatment and management plan.

According to Pearcy and Hindle (1989) single plane lumbar movements are often unrepresentative of the lumbar spine function, and as such have limited value in clinical assessment. The combined movement examination (CME), originally described by Edwards (1979), examines the patient’s ability to perform a planar movement examination as well as actively combined side-flexion of the lumbar spine while in flexed, neutral and extended positions. Edwards (1979) originally proposed that the CME may be more informative than the standard planar assessment. This approach was subsequently confirmed by Barrett et al. (1999) who reported acceptable CME intra-examiner reliability, as well as preliminary evidence concerning the effectiveness of CME in identifying reduced lumbar movement in LBP cases. To the authors’ knowledge, no study has reported the objective use of computer-aided CME to inform clinical practice.

Validation of the MotionStar™ 3-D motion tracking system (Ascension Technology, VT, USA), establishing the reliability of computer-aided CME, the development of a CME normal reference range (NRR) and proof of concept with clinical cases has previously been reported (Monie et al., 2015). This paper is to first report the use of computer-aided CME as a tool to objectively assess intra- and inter-session lumbar movement in two cases with different (non-specific) LBP presentations. Second, to report how an individual’s CME ‘signature’ (Fig. 1) may be used to guide manual therapy (MT) intervention. Finally, a comparison is made between each case’s CME data and an age and gender matched CME NRR.

1.1. Presenting concerns

Two symptomatic individuals were recruited from a convenience sample of clients at a local Physiotherapy private practice. Case A, was a 35 year old female house-wife (BMI 22) who complained of an
acute exacerbation of central, constant lumbar stiffness and intermittent pain with movement. This patient attended for CME examination and MT on two consecutive days. Case B, was a 57 year old female, school principal, (BMI 23) who presented with an acute exacerbation of right sided, intermittent, mechanical LBP. This patient attended four sessions (days 1, 4, 5 and 8).

1.2. Clinical findings

Both cases considered themselves in very good health (SF-12) with no complaint of dominant psychosocial factors, systemic disease, trauma or co-morbidities. Both individuals stated that they had experienced mild low back discomfort or tightness 1-2 times per year. However, neither had experienced the same pain location or intensity as their presenting complaint.

1.3. Diagnostic focus and assessment

Both cases were screened for ‘red flags’, questioned for symptoms of neurological involvement and assessed for myotomal strength, deep tendon reflexes and altered sensation. There was no indication of neural pathology in either patient, thus both cases were classified as predominantly mechanical musculoskeletal pathologies.

After obtaining informed written consent and familiarisation of equipment and testing sequence, both cases were examined using computer-aided CME. Skin mounted MotionStar™ sensors were placed over the volunteer’s S1 level and L1 spinous process (Fig. 2A and B). In a relaxed standing position, participants had their lumbar lordosis (angle between L1 and S1) recorded using the Motion-Star™ system (Fig. 2A). This became the ‘zeroed’ starting position (centre of radial plot) (Fig. 1).

The patient was instructed to move within their comfortable limits and then cued through the eight CME positions by the examiner. Maximum data values for each CME direction were recorded according to a pre-defined sequence: Flexion, Flexion with Left Side-Flexion (FwLSF), Flexion with Right Side-Flexion (FwRSF), Left Side-Flexion (LSF), Right Side-Flexion (RSF), Extension, Extension with Left Side-Flexion (EwLSF) and Extension with Right Side-Flexion (EwRSF). Position data were acquired by the
MotionStar™ computer system in real-time and post-processed in Labview (V5.0, National Instruments, Austin USA) to derive actual CME coordinates. In case A, the most painful CME movement was lumbar extension. In case B, the patient complained of right side LBP and the most painful CME direction was right side-flexion (RSF).

1.4. Questionnaires

The core battery of outcome measures was used to assess the patients at each MT treatment session in the laboratory (Deyo et al., 1998). Visual Analogue Scale (VAS) for pain and stiffness experienced during CME was recorded before and after each MT session. Roland Morris Low Back Pain and Disability Questionnaire (RMDQ) and a Short Form health survey (SF-12) were recorded prior to CME at each MT session. During the development of an NRR the examiners had noted average normal data (by age and gender) showed left to right symmetry to within 5° (Monie et al., 2015). In this study, the examiners considered a symmetrical ‘signature’, to ≤5 degrees of the asymptomatic side, as a realistic clinical outcome goal.

1.5. Therapeutic focus and assessment

The grade, dose and frequency of joint mobilisation was guided by the patient’s symptoms and the acute stage of the LBP (Maitland, 1997; Edwards, 1999). The atypical movement pattern observed on their CME radial plot compared to the NRR (Table 1 and Fig. 4A and B) was used, in a novel approach, to inform patient positioning on the treatment table and the direction of joint mobilisation. For an NRR of both genders and each decade of life between 20 and 70 years, see Monie et al. (2015).

In case A, the most painful CME movement was lumbar spine extension with the right postero-lateral quadrant (EwRSF) being reduced on CME (Fig. 3A). This patient was treated with a right sided postero-anterior (P-A) directed, passive spinal mobilisation technique progressed by graded increments of lumbar extension and combined right side-flexion (Fig. 3A and B). In case B, a global reduction in movement (compared to the NRR) is evident in the radial plot (Fig. 4B) and side-flexion was markedly restricted bilaterally. However, the patient complained of right side LBP and the most painful CME direction was right side-flexion (RSF). Considering case B’s CME ‘signature’ showing reduced RSF and the

Table 1

Combined Movement Examination normal reference range for females aged 30–39 and 50–59 years of age, reporting the mean and standard deviation for each movement direction, standing lordosis and BMI. This was used for comparison in case A and B, respectively.

<table>
<thead>
<tr>
<th>Age and gender</th>
<th>Flexion</th>
<th>FwRSF</th>
<th>RSF</th>
<th>EwRSF</th>
<th>Extension</th>
<th>EwLSF</th>
<th>LSF</th>
<th>FwLSF</th>
<th>Lordosis</th>
<th>BMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>30-39YO F</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>Mean</td>
<td>50.1</td>
<td>37.8</td>
<td>24.9</td>
<td>13.6</td>
<td>16.2</td>
<td>14.6</td>
<td>21.1</td>
<td>41.4</td>
<td>31.6</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>5.7</td>
<td>8.7</td>
<td>10.9</td>
<td>4.4</td>
<td>4.3</td>
<td>5.0</td>
<td>6.9</td>
<td>7.9</td>
<td>8.2</td>
</tr>
<tr>
<td>50-59YO F</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>Mean</td>
<td>40.7</td>
<td>31.7</td>
<td>20.6</td>
<td>12.3</td>
<td>14.5</td>
<td>12.2</td>
<td>15.9</td>
<td>31.3</td>
<td>33.8</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>10.7</td>
<td>11.2</td>
<td>9.7</td>
<td>4.9</td>
<td>4.5</td>
<td>4.9</td>
<td>6.4</td>
<td>11.3</td>
<td>8.8</td>
</tr>
</tbody>
</table>

All angular movement values are in degrees [°]. Lordosis angle was measured between the L1 and S1 sensor in relaxed standing. For explanation of acronyms, refer to Fig. 1.
1.6. Follow-up and outcome

In both cases the CME assessment confirmed improvement in the patient’s restricted movement pattern. Table 2 shows angular movement values for both cases in each of the CME directions and relaxed standing lumbar lordosis, before and after MT with total change scores (%).

Table 3 shows self-report measures pre- and post-MT intervention with total change scores (%) for each outcome measure.

2. Discussion

Self-report surveys and lumbar kinematics provide insight into the response of low back conditions to management (Deyo et al., 1998; Williams et al., 2013). Measures should be reliable, valid, practical, and for convenience, brief, where possible. However, outcome measures placing emphasis on pain, function and quality of life do not provide the clinician with feedback on the direction and magnitude of movement pattern disturbance (Lyle et al., 2005), the potential structure(s) at fault, nor the departure from usual movement according to normative age and gender reference ranges.

Fig. 3. Examples of manual therapy techniques applied to each case, using a model to demonstrate the positioning. Case A, manual therapy session 1: Patient in prone lying, right sided P-A mobilisation of L5 (A), session 2: Patient in lumbar extension with right side-flexion during right sided P-A mobilisation of L5 (B). Case B, manual therapy session 1: right side-flexion mobilisation with the patient in left side-lying (C), session 2: patient positioned on an inclined treatment table, in right side-flexion, while receiving right side-flexion spinal mobilisation (D).

Fig. 4. Case A’s pre-Manual Therapy (blue) and post-Manual Therapy (red) CME. The average normal female CME data for 30–39 years of age is shown in black (A). Case B’s pre-Manual Therapy (blue) and post-Manual Therapy (red) CME recording plus the average normal female CME data for 50–59 years of age is shown in black (B). Data are in degrees of angular movement. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

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The present report investigated the novel application of computer assisted CME to measure change to both the magnitude and direction of movement dysfunction and consequently to demonstrate a tendency towards age and gender matched normalisation of low back movement after MT intervention.

The ability for CME to detect specific directions of restricted movement and provide insight into the possible biomechanical causes have been hypothesised though not previously examined (Brown, 1988). Although Pearcy and Hindle (1989) discuss the potential diagnostic value of 3-D lumbar movement assessment, no studies have investigated this claim in pathoanatomical terms.

A speculative model for inferring pain may be derived clinically and CME may assist the clinician in formulating a series of hypotheses about mechanical pain origin in LBP. From clinical studies, the intervertebral disc and facet joints are the two most likely pain sources in the low back, with prevalence rates estimated to be 42% and 31%, respectively (Laplante et al., 2012). Osseo-ligamentous tissues and the disc anulus are the primary contributors to spinal stiffness (Cunningham et al., 2007; Little et al., 2007). Lumbar discs have multi-level anterior compartment innervation by direct branches which arise from the sympathetic trunk and the posterior disc from the sinu-vertebral nerve. In each case, this innervation is multi-segmental and bilateral (Fig. 5) (Groen and Stolker, 2000).

Table 2

<table>
<thead>
<tr>
<th>Case A</th>
<th>Flexion</th>
<th>FwRSF</th>
<th>RSF</th>
<th>EwRSF</th>
<th>Extension</th>
<th>EwLSF</th>
<th>LSF</th>
<th>FwLSF</th>
<th>Lordosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-MT Day 1</td>
<td>49.1</td>
<td>51.2</td>
<td>9.9</td>
<td>5.0</td>
<td>13.6</td>
<td>8.9</td>
<td>11.3</td>
<td>49.4</td>
<td>34.8</td>
</tr>
<tr>
<td>Post-MT Day 2</td>
<td>50.5</td>
<td>39.6</td>
<td>12.9</td>
<td>10.6</td>
<td>23.4</td>
<td>13.5</td>
<td>15.0</td>
<td>39.4</td>
<td>27.6</td>
</tr>
<tr>
<td>Total change [%]</td>
<td>2.8%</td>
<td>-22.7%</td>
<td>31.1%</td>
<td>112.4%</td>
<td>72.3%</td>
<td>50.6%</td>
<td>33.0%</td>
<td>-20.2%</td>
<td>-20.5%</td>
</tr>
</tbody>
</table>

Table 3

<table>
<thead>
<tr>
<th>Case A</th>
<th>Pain</th>
<th>Stiffness</th>
<th>RMDQ</th>
<th>SF-12 PCS</th>
<th>SF-12 MCS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-MT Day 1</td>
<td>5.2</td>
<td>5.1</td>
<td>7.0</td>
<td>36.5</td>
<td>60.1</td>
</tr>
<tr>
<td>Post-MT Day 2</td>
<td>0.5</td>
<td>0.3</td>
<td>4.0</td>
<td>44.0</td>
<td>60.1</td>
</tr>
<tr>
<td>Total change [%]</td>
<td>47.0%</td>
<td>48.0%</td>
<td>12.5%</td>
<td>13.3%</td>
<td>0.0%</td>
</tr>
<tr>
<td>Case B</td>
<td>Pain</td>
<td>Stiffness</td>
<td>RMDQ</td>
<td>SF-12 PCS</td>
<td>SF-12 MCS</td>
</tr>
<tr>
<td>--------</td>
<td>------</td>
<td>----------</td>
<td>------</td>
<td>-----------</td>
<td>-----------</td>
</tr>
<tr>
<td>Pre-MT Day 1</td>
<td>8.6</td>
<td>9.9</td>
<td>20.0</td>
<td>26.2</td>
<td>42.6</td>
</tr>
<tr>
<td>Post-MT Day 8</td>
<td>0.2</td>
<td>0.3</td>
<td>5.0</td>
<td>30.6</td>
<td>60.2</td>
</tr>
<tr>
<td>Total change [%]</td>
<td>84.0%</td>
<td>96.0%</td>
<td>62.5%</td>
<td>7.8%</td>
<td>28.9%</td>
</tr>
</tbody>
</table>

Note: SF-12 Normal = 50, SD = 10. MT = Manual Therapy.

Table 2

Maximum angular movement and total change score (%) for each of the patient’s CME movement directions, before and after MT intervention.

Table 3

Change in self-report instruments (pre- and post-intervention) for Visual Analogue Scale (VAS; Pain and Stiffness), Short Form Health Survey Physical Component Score (SF-12 PCS), Short Form Health Survey Mental Component Score (SF-12 MCS) and Roland Morris Low Back Pain Disability Questionnaire (RMDQ).

The present report investigated the novel application of computer assisted CME to measure change to both the magnitude and direction of movement dysfunction and consequently to demonstrate a tendency towards age and gender matched normalisation of low back movement after MT intervention.

1. Afferent branches from Sympathetic trunk
2. Sympathetic trunk ganglion
3. Ramus Communicans
4. Ventral ramus
5. Dorsal root ganglion
6. Sinu-vertebral nerve
7. Medial branch of dorsal ramus to facet joint
8. Intermediate branch of dorsal ramus to muscle
9. Lateral branch of dorsal ramus to skin

Fig. 5. A lumbar vertebra divided along two axes, defining anterior-posterior and left-right quadrants. Adapted from Brown (1988). The typical innervation pattern described by Groen and Stolker 2000 is depicted.

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innervation which have potential to cause ipsilateral multifidus muscle contraction and spasm (Bogduk, 1985; Edgar, 2007). Using Fig. 4 one could hypothesise the source of LBP in each of these cases from the patient’s description of symptoms and their CME. The provisional diagnosis of facet joint pathology was made for both cases as CME identified a unilateral, symptomatic side with specific movement restriction and reproduction of their concordant pain. The vertebral level reproducing the patient’s concordant symptoms during MT assessment (L5), was chosen as the candidate level to treat.

To inform treatment outcomes, an age and gender matched CME NRR was used to establish goals. In both cases, VAS data for pain decreased by >46%, which exceeds the minimal clinically important change value of 30%, proposed by Ostelo et al. (2008). VAS data for stiffness also decreased by >48%. While a CME NRR provides a putative movement outcome target, care must be taken to consider individual anatomical variations and clinical presentations. In both cases computer-aided CME contributed to the planning and outcome assessment of their management.

3. Conclusion

This study reports the use of computer-aided CME as both an assessment tool and as an outcome measure in two cases of non-specific, mechanical LBP managed with manual therapy intervention. Further studies, investigating a larger number of cases with specific lumbar pathologies are required.

Ethics statement and informed consent

Approval to conduct the study was obtained from The University of Western Australia Human Research Ethics Committee. Written informed consent was obtained prior to participation.

Competing interests

There were no sources of funding or conflicts of interest associated with this research.

References


Low back pain misdiagnosis or missed diagnosis: Core principles
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ABSTRACT
Consensus guidelines for the management of low back pain recommend that the clinician use contemporary best practice for assessment and treatment, consider biopsychosocial factors and, if chronic, use a multimodal and multi-disciplinary approach. Where guidelines are not followed and basic assessment is inadequate the diagnosis may be compromised and the sequelae of errors compounded. Factors such as a lack of knowledge or recognition of the common structure specific pain referral patterns, poor clinical reasoning, inappropriate referral and predilection for popular management approaches also contribute to mis-diagnosis and mis-management. This report describes two cases of chronic low back pain with lengthy histories of multiple failed interventions to highlight the consequences of focussing on a singular approach to the exclusion of evidence based pathways and the resulting risk of a missed diagnosis. The eventual management to mitigate these problems is reported with the aid of low back pain outcome measures, computer-aided combined movement examination, disability and pain questionnaires and health quality of life surveys.

1. Introduction
Low back pain (LBP) is a major public health system problem, being one of the five most common reasons for physician consultation, with a lifetime prevalence as high as 85% (Joud et al., 2012). Chronic low back pain (CLBP) often includes psychological factors, such as higher than usual levels of stress, depression and/or anxiety, with the potential for fear-avoidance and catastrophising behaviours (Besen et al., 2015; Deyo, 2015). Furthermore, social factors involving relationships, family, work and navigating the medico-legal system, may amplify or prolong pain (Deyo, 2015).

Minimum clinical assessment of LBP includes obtaining a medical history and a physical examination (NHMRC, 2004; SAH, 2011). Physical examination incorporates a movement assessment which can include a multi-planar combined movement examination (CME) (Edwards, 1979; Barrett et al., 1999), soft tissue palpation, passive movement examination and neurological screening if implicated. Together, the history and physical assessment should result in a provisional diagnosis (Grieve, 1988). Progressing to treatment without a thorough assessment or without correlating symptoms with examination findings, increases the prospect of misdiagnosis and mismanagement. Where indicated, focussed spine imaging assists with diagnosis and staging interventions (Deyo et al., 2014). Where the condition is complex, not responding to treatment, or where symptoms masquerade as more sinister pathology (Greenhalgh and Selfe, 2015), referral to appropriate health professionals is encouraged.

A wide variety of approaches are recommended for treatment of LBP, ranging from passive manual therapy (Maitland, Mulligan, manipulative therapy) to active management (general exercise, Pilates, hydrotherapy, changes to workplace and sleeping habits) and those with a focal psychosocial component (cognitive behavioural therapy) (Beck, 2011). Jull and Moore (2012) advocate finding the balance which optimises a multimodal approach and the outcome.

In recent years there has been much focus on the psychosocial component of LBP and graded movement rehabilitation (O’Sullivan and Lin, 2014; Deyo, 2015; O’Sullivan et al., 2015). This emphasis risks compromising the importance of assessing fundamental pathoanatomical sources of LBP (Hancock et al., 2011). Additionally, Moore and Jull (2000) remind clinicians to select an appropriate approach based on clinical guidelines. “With our enthusiasm for new trends to seem smart and stylish in our therapeutic practice, we must be careful that we don’t fall into the trap of not adhering to recognised and established practices” (Moore and Jull, 2000:197).
This report describes two cases, highlighting the consequences of focussing on a singular emphasis to the exclusion of a conventional systematic assessment and the resulting risk of a missed diagnosis.

2. Method

Two CLBP cases with lengthy histories of multiple failed interventions are reported. The eventual effective management is also reported along with: CME (Monie et al., 2015a), visual analogue scale (VAS) (Ogon et al., 1996), Roland Morris disability questionnaire (RMDQ) (Chapman et al., 2011) and short form health survey (SF-12) (Ware et al., 1996) outcome measures, pre- and post-intervention.

2.1. Case 1

This 55 year old male consulted his local GP for right side intermittent LBP (Fig. 1A), aggravated by walking >200 m, or any attempt to run, and when standing longer than 10 min, which resulted in a pain level of 7/10 (VAS). The GP referred the patient for three physiotherapy sessions in a tertiary hospital, to develop a rehabilitation program and requested lumbar x-ray and a gluteal region ultrasound scan. Both were reported as normal. After two months of prescribed exercises and using simple analgesics, the GP referred the patient to an orthopaedic spinal surgeon for an opinion. The specialist requested a CT investigation, which showed a L4-5 right side paracentral disc bulge with possible L4 nerve root compromise (Fig. 1B and C). The specialist referred the patient for image-guided L4-5 epidural injection, followed by a hip, trochanteric bursa, injection after four months, and finally a repeat L4-5 epidural after an additional four weeks. All three interventions failed to improve the patient’s LBP. Both GP and specialist did not offer any further management strategies.

After two years of unsuccessful LBP management, the patient consulted a Physiotherapist privately, where a structured examination was completed. This assessment reproduced his symptoms during CME in lumbar extension with added right side flexion (EwRSF); consistent with a regular compression pattern (Monie et al., in press). Passive joint assessment directed to the right side L5 level reproduced the patient’s symptoms (Maitland, 1997; Cook et al., 2015). The patient was then examined using computer-aided CME (Monie et al., 2015a) (Fig. 1D), along with RMDQ and SF-12 health survey outcome instruments. A provisional diagnosis of right side facet dysfunction was made on the basis of (a) pain location, (b) patterns of lumbar innervation (Bogduk, 1985; Groen and Stolker, 2000), (c) CME pattern being consistent with loading posterolateral vertebral structures (Brown, 1988) and (d) eliminating the disc as a source of local pain following two unsuccessful epidural injections. A corticosteroid injection into the right L4-5 facet joint was recommended by the physiotherapist, as both a diagnostic and therapeutic intervention. The patient was referred by his GP for the facet injection and reported excellent pain relief within one week (VAS for pain was 1.6 during CME in the EwRSF position). Lasting benefit was evident at an eleven week reassessment, with obvious changes to CME (Fig. 1D) and clinically significant total change scores for VAS and RMDQ of 54.0% and 45.8%, respectively (Deyo et al., 1998). SF-12 health survey scores were rated normal by the eleven week retest (refer Table 1).

2.2. Case 2

This 42 year old female presented with severe low back pain (VAS 8.5) and right lower limb pain (Fig. 2A). She described emotional issues and stated that she had occasionally consulted a psychologist.

The patient was prescribed NSAIDs and Diazepam and referred by her GP for an MRI within 6 weeks of onset. Physiotherapy consisted of four manual therapy treatments and swimming was recommended. However this did not have any reported effect. Imaging showed disc protrusions at L4-5 and L5-S1 with bilateral facet arthropathy at both segments. Five months later the GP referred her for a L5-S1 epidural cortisone injection and prescribed Pregabalin medication. However, the symptoms did not change. The patient was advised by her physiotherapist to continue exercise and recommence physiotherapy treatment. Approximately six months later, a left S1 nerve root injection was performed for an episode of left lower limb pain. This provided temporary relief only. The patient requested a repeat MRI from the GP, but was refused and told that they knew the pain was from the disc bulge and anxiety. This view was reinforced by a second physiotherapist who implemented a program comprising cognitive functional therapy (O’Sullivan et al., 2015), manual therapy, home stretches and swimming five days per week. The advice reported to the patient was that the sciatic pain was anxiety related, and she needed to relax and practice breathing exercises. Her LBP increased with now significant right lower limb pain and she developed a left side lateral shift (Laslett, 2009). After complaining that she could no longer get

Fig. 1. Case 1 Pain diagram illustrating the area of right side, intermittent low back pain (red) (A), axial CT showed a right side posterolateral disc protrusion (B) and sagittal image (C) (arrows in black) and computer aided CME radial plot illustrating a restricted movement in the direction of lumbar extension combined with right side flexion (EwRSF), marked improvement at 11 weeks and an age (50–59) and gender matched normal reference range (NRR) (D). Legend for CME: — Pre-injection, — 1/52 post-injection, — 11/52 post-injection, — 50–59YD male NRR. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
dressed or swim, the physiotherapist recommended cycling. Neither the GP nor physiotherapist referred her for specialist opinion.

When asked, the patient summarised her situation: "The second SI injection was a result of many visits to the doctor for something to help the pain as no analgesics/opiates were helping. After this was unsuccessful, I literally begged for another MRI, acknowledging I would have to pay for it etc and my doctor literally refused to refer me for one, stating that 'we' knew what was causing my pain, ie. the disc bulge and anxiety. This was backed up by the second physiotherapist who encouraged me to read a book called 'The Divided Mind' by John Sarno which outlines Sarno's diagnosis of Tension Myositis Syndrome which is basically how deep unconscious emotional issues can manifest themselves as pain so, yes, I was basically told that the pain was caused by my mind."

A friend of the patient recommended a sports physician who referred her for a repeat MRI which identified a markedly prograde right sided disc protrusion at L4-5 (Fig. 2 B and C). After a two year period since initial onset, the patient was reviewed by a neurosurgeon and underwent laminectomy and discectomy with good effect. At reassessment 12 weeks post-surgery, the lumbar spine movement had normalised to within 1 SD of her age and gender matched normal CME reference range (Fig. 2D) and total change scores for pain (VAS), RMDQ and SF-12 PCS were significantly improved (Table 1). The patient was able to return to work.

3. Discussion

Best practice guidelines to assist in managing patients with LBP are available from authoritative groups such as the United Kingdom’s NICE agency (NICE, 2009), the American College of Physicians or American Pain Society (Chou et al., 2007) and the Australian National Health and Medical Research Council (NHMRC, 2004) and systematic review (Dagenais et al., 2010), to assist in managing patients with LBP. For CLBP, brief education, advice to stay active, NSAIDs, weak opioids, exercise therapy and spinal manipulation are common direction across many guidelines. Secondary recommendations include multi-disciplinary rehabilitation, adjunctive analgesics, cognitive behavioural therapy and strong opioids. Antidepressants are suggested in some guidelines (Balague et al., 2011) as well as cognitive functional therapy (O’Keefe et al., 2015; O’Sullivan et al., 2015).

Evidence based guidelines also recommend conventional clinical assessment of patients presenting with LBP (NHMRC, 2004; Chou et al., 2007; NICE, 2009), which informs the diagnosis and initial management. Key components to a low back assessment include: taking a detailed history and conducting a physical examination, neurosurgical evaluation and psychosocial screening.

In case 1, the right side LBP was reproduced using CME and with the use of posterior—directed pressure, during passive accessory inter-vertebral movement assessment over the right side L5 level, which putatively loads the right facet joints (Niosi et al., 2008). Considering the lumbar vertebral segment’s instantaneous axes of rotation (Pearcy and Bogduk, 1988), facet joint ipsilateral and localized innervation (Groen and Stolker, 2000) and the fact that facets joints are the second most common cause of LBP (Laplante et al., 2012), this presentation is consistent with facet joint pain.

In case 2, after having LBP for over 18 months, pain had returned with greater intensity in the right lower limb and the LBP had increased to the point that it was disabling. Using MRI is considered appropriate where therapy has failed and the patient is a potential
candidate for surgery or epidural steroids (Deyo et al., 2014). Balagué et al. (2011) state that the absence of a pathoanatomical gold standard (MRI) precludes any definitive conclusions. In this case, the benefit of the MRI ordered by the sports physician converted a non-specific CLBP case into a structure specific case with an appropriate neurosurgical referral and positive outcome.

The initial management of Case 1 failed to isolate the nature of the LBP and clinical reasoning overlooked the second most common cause of LBP, the facet joint (Laplante et al., 2012). Case 2 was treated with a focus on psychosocial factors, using an emphasis of cognitive functional therapy rather than on the discipline of clinical guidelines, and the nature and history of the symptoms.

4. Summary

Where guidelines are not followed and basic assessment is inadequate the diagnosis is compromised and the sequelae of errors may compound. Factors such as lack of knowledge or recognition of the common structure specific pain referral patterns, poor clinical reasoning, inappropriate referral and predilection for popular management approaches also contribute to mis-diagnosis and mis-management.

The astute clinician is mindful of authoritative best practice guidelines, is systematic in both assessment and management, and in the event of worsening symptoms conscious of the need for timely appropriate referral.

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Computer-aided Combined Movement Examination of the lumbar spine and manual therapy implications: case report

A.P. Monie a, K.P. Singer a

a Centre for Musculoskeletal Studies, School of Surgery, The University of Western Australia, Perth, Western Australia 6009, Australia

ABSTRACT

Background: The combined movement examination (CME) of the lumbar spine has been recommended as being a useful clinical examination which confers information about mechanical pain patterns, however little quantitative study has been undertaken to validate manual therapy (MT) practice.

Objective: To use computer aided CME and a CME normal reference range (NRR), to guide provisional diagnosis and MT. Additionally, to use the CME NRR as a treatment goal.

Design: Test Retest

Method: Two cases were assessed, before and after MT using CME and a pain Visual Analogue Scale (VAS), and prior to MT treatment using the Roland Morris Low Back Pain and Disability Questionnaire (RMDQ) and Short Form (SF-12) Health Survey. Provisional diagnosis, treatment and management were guided by comparing the patient’s CME with an age and gender matched reference range.

Results: Self-reports total change scores (%) were improved for cases A and B, and CME improved for case A, particularly for the most painful and restricted CME direction. In case A, lumbar lordosis increased, and CME in the most painful and restricted direction decreased.

Conclusion: Computer-aided CME may be used both as an assessment tool and as an outcome measure in some cases of mechanical LBP. This study reports two cases of non-specific, mechanical LBP and supports the merit of CME to guide assessment and treatment. Where CME does not increase, in cases of mechanical LBP, further investigation may be warranted.

Keywords: Lumbar spine; Low Back Pain, Combined Movement Examination; Lumbar Movement, Manual Therapy
**Introduction**

Assessing lumbar spine movement in the clinical setting to investigate dysfunction and to monitor changes in a patient’s spinal movement characteristics over time is routine clinical practice (Ha et al., 2013; Laird et al., 2014; Maitland, 1997). This is used, along with other assessment findings, to develop a provisional diagnosis, treatment and management plan.

According to Pearcy & Hindle (1989) single plane lumbar movements are often unrepresentative of the lumbar spine function, so have limited value in clinical assessment. The combined movement examination (CME), originally described by Edwards (1979), examines the patient’s ability to perform a planar movement examination as well as actively combined side-flexion of the lumbar spine while in flexed, neutral and extended positions.

Brian Edwards, a specialist manipulative physiotherapist from Perth, Western Australia, originally proposed that CME may be more informative than a conventional planar assessment. This model was subsequently investigated by Barrett et al. (1999) who reported acceptable CME intra-examiner reliability, as well as preliminary evidence confirming the effectiveness of CME in identifying reduced lumbar movement in LBP cases. This investigation provided first, a normative control group and second, the opportunity to compare clinical CME patterns. It is rare in physiotherapy practice to have a normal reference range (NRR) of outcome measures upon which to assess an intervention. To our knowledge, no study has reported the objective use of computer-aided CME to inform clinical assessment and direct manual therapy (MT) practice.

Validation of the MotionStar™ 3-D motion tracking system (Ascension Technology, VT, USA), establishing the reliability of computer-aided CME, the development of a CME normal reference range (NRR) and proof of concept with clinical cases has previously been reported (Monie et al., 2014). The purpose of this study is to report the use of computer-aided CME as a tool to objectively assess intra- and inter-session lumbar movement in two cases with different (non-specific) LBP presentations. Secondly, to report how an individual’s CME ‘signature’ (Figure 1) may be used to guide MT intervention. Finally, a comparison is made between each case’s CME data and an age and gender matched CME NRR.
Figure 1: Example of an asymptomatic volunteer’s CME radial plot in degrees of angular movement. Photographs illustrate the movement directions and end-points, namely: Flexion with Left Side-Flexion (FwLSF), Flexion with Right Side-Flexion (FwRSF), Left Side-Flexion (LSF), Right Side-Flexion (RSF), Extension with Left Side-Flexion (EwLSF) and Extension with Right Side-Flexion (EwRSF).

Presenting concerns

Two symptomatic individuals were recruited from a convenience sample of clients at a local Physiotherapy private practice. Case A, was a 30 year old female office worker who complained of constant, central lumbar pain and stiffness lasting three days, with increased LBP during forward bending. This patient attended for CME examination and MT on two consecutive days. Case B, was a 61 year old male, librarian, who complained of chronic low back stiffness and sub-acute right posterior thigh, intermittent pain. This patient attended three sessions (days 1, 3 and 5).

Clinical Findings
Both cases considered themselves in very good health, with no complaint of dominant psychosocial factors, systemic disease, trauma or co-morbidities. Both individuals stated that they had experienced mild low back discomfort or tightness 1-2 times per year; however neither had experienced the same pain location or intensity as their presenting complaint.

**Diagnostic Focus and Assessment**

Both cases were screened for ‘red flags’, questioned for symptoms of neurological involvement and assessed for myotomal strength, deep tendon reflexes and altered sensation. In case B, radicular signs were not obvious, although L5 nerve root symptoms were reported, including a recent history of right lateral calf pain, which had resolved, and the presenting complaint of intermittent right hamstring pain, which may have been nerve root or somatically referred from the lumbar spine. Initial clinical reasoning in both cases, lead to a predominantly mechanical neuromusculoskeletal cause.

After obtaining informed written consent and familiarisation of equipment and testing sequence, both cases were examined using computer-aided CME. Skin mounted MotionStar™ sensors were placed over the volunteer’s S1 level and L1 spinous process. In a relaxed standing position, participants had their lumbar lordosis (angle between L1 and S1) recorded using the MotionStar™ system (Figure 2). This became their ‘zeroed’ starting position and is depicted as the centre of the radial plots (Figure 1).
The patient was instructed to move within their comfortable limits and then cued through the eight CME positions by the examiner. Maximum data values for each CME direction were recorded according to a pre-defined sequence: Flexion (Flex), Flexion with Left Side-Flexion (FwLSF), Flexion with Right Side-Flexion (FwRSF), Left Side-Flexion (LSF), Right Side-Flexion (RSF), Extension (Ext), Extension with Left Side-Flexion (EwLSF) and Extension with Right Side-Flexion (EwRSF). Position data were acquired by the MotionStar™ computer system in real-time and post-processed in LabVIEW (V5.0, National Instruments, Austin USA) to derive actual CME coordinates.

In case A, the most painful CME movement was lumbar flexion. In case B, the most symptomatic CME direction, causing right hamstring pain, was right side-flexion (RSF).

**Questionnaires**

The core battery of outcome measures was used to assess the patients at each MT treatment session in the laboratory (Deyo et al 1998). Visual Analogue Scale (VAS) for pain and stiffness experienced during CME was recorded before and after each MT session. Roland Morris Low Back Pain and Disability Questionnaire (RMDQ) and a Short Form health survey (SF-12) were recorded prior to CME at each MT session. During the development of a NRR the examiners had noted average normal data (by age and gender) showed left to right symmetry to within 5 degrees (Monie et al 2014). In this study, the examiners considered a symmetrical ‘signature’, to ≤ 5 degrees of the asymptomatic side, as a realistic clinical outcome goal.
Therapeutic Focus and Assessment

The grade, dose and frequency of joint mobilisation was guided by the patient’s symptoms and the acute stage of the LBP (Edwards, 1999; Maitland, 1997). The atypical movement pattern observed on their CME radial plot compared to their NRR (Table 1) was used, in a novel approach, to inform patient positioning on the treatment table and the direction of joint mobilisation.

In case A, the most painful CME movement was lumbar spine flexion with all flexed directions (FwLSF, Flexion, FwRSF) being reduced on CME. This patient was treated with a passive spinal flexion mobilisation technique (Maitland, 1997) with graded increments of lumbar flexion, soft-tissue mobilisation techniques, and a flexion stretch (Hunter, 1998) (Figures 3A-E). In case B, a right side-flexion (RSF) reduction in movement (compared to the NRR) was recorded. The most painful CME direction, causing thigh pain, was also RSF.

Considering case B’s CME ‘signature’ and reported symptoms, right side L5 nerve root compression was the provisional diagnosis, and a passive LSF spinal mobilisation technique was used while the patient lay in left side lying (Figure 4A) in an effort to decompress the right L5 nerve root. This was done as a precautionary initial treatment, to avoid nerve root compression. Graded increments of LSF were used over three MT sessions, by inclining the treatment table (Figure 4B). Pre- and post- MT CME plots, with reference to each case’s NRR, are illustrated in Figure 5A,B.
Table 1: Combined Movement Examination normal reference range for males and females, age 20 to 69 years, reporting the mean, standard deviation and range for body mass index, standing lordosis and each CME direction. This was used for comparison in case A and B, respectively.

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<td>18.2</td>
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All angular movement values are in degrees (°). Lordosis angle was measured between L1 and S1 sensor in relaxed standing. For explanation of acronyms refer to Fig. 1.
Figure 3: Examples of manual therapy techniques applied to each case, using a model to demonstrate the positioning. Case A, manual therapy session 1: Patient in sidelying, passive physiological flexion to within the patient’s pain limits, progressed from early flexion (A) to end-of-range flexion (B) and soft tissue techniques for hyperactive lumbar extensors in a stretched position (C). Session 2: progress to passive accessory joint mobilisation of L5, using a cephaladly directed posterior-anterior pressure in prone, with the lumbar spine flexed over two pillows. A home exercise, encouraging lumbar flexion in a relatively unloaded position was prescribed for use at home, between sessions (E).

Figure 4: Case B, manual therapy session 1: left side-flexion mobilisation with the patient in right side-lying, to gap the right side low lumbar spine (A), session 2: the table is inclined, encouraging left side-flexion at the low lumbar spine, while patient receives passive mobilisation to gap the right low lumbar spine (B).
Figure 5: Case A’s pre-Manual Therapy (blue) and post-Manual Therapy (red) CME. The average normal female CME data for 30-39 years of age is shown in black (A). Case B’s pre-Manual Therapy (blue) and post-Manual Therapy (red) CME recording plus the average normal male CME data for 60-69 years of age is shown in black (B). Note: In case B’s CME plot, the five degree change in standing lordosis between trials is not evident. Data are in degrees of angular movement.

Follow-up and Outcome

In case A the CME assessment confirmed improvement in the patient’s restricted movement pattern. In case B, there was a 5 degree increase in lumbar lordosis from the NRR mean of 32 to 37 degrees which is not evident in the CME radial plot (Figure 5), and a 7 degree decrease in RSF. Table 2 shows angular movement values for both cases in each of the CME directions and relaxed standing lumbar lordosis, before and after MT with total change scores (%).

Table 3 shows self-report measures pre- and post-MT intervention with total change scores (%) for each outcome measure.

Table 2: Maximum angular movement and total change score (%) for each of the patient’s CME movement directions, before and after MT intervention.

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<tr>
<th></th>
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<th>FwRSF</th>
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<th>EwRSF</th>
<th>Extension</th>
<th>EwLSF</th>
<th>LSF</th>
<th>FwLSF</th>
<th>Lordosis</th>
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<tr>
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<td>40.8</td>
<td>33.4</td>
<td>14.9</td>
<td>11.3</td>
<td>9.3</td>
<td>8.0</td>
<td>13.4</td>
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<td>32.1</td>
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<tr>
<td>Total change (%)</td>
<td>83.6</td>
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<td>69.0</td>
<td>-6.5</td>
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<tr>
<td>Pre-MT Day 1</td>
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<td>46.6</td>
<td>16.6</td>
<td>19.0</td>
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<td>23.3</td>
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<tr>
<td>Post-MT Day 2</td>
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<td>9.9</td>
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<td>7.0</td>
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<tr>
<td>Total change (%)</td>
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All angular movement values are in degrees [°]. For explanation of acronyms, please refer to Figure 1.
Table 3: Change in self-report instruments (pre- and post-intervention) for Visual Analogue Scale (VAS; Pain and Stiffness), Short Form Health Survey Physical Component Score (SF-12 PCS), Short Form Health Survey Mental Component Score (SF-12 MCS) and Roland Morris Low Back Pain Disability Questionnaire (RMDQ).

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<thead>
<tr>
<th>Case A</th>
<th>Pain</th>
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<th>Case B</th>
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Note: WNL = Within normal limits, SF-12 Normal = 50 (10), MT = Manual Therapy

DISCUSSION

Self-report surveys and lumbar kinematics provide insight into the response of low back conditions to management (Deyo et al., 1998; Williams et al., 2013). Measures should be reliable, valid, practical, and for convenience, brief, where possible. However, outcome measures placing emphasis on pain, function and quality of life do not provide the clinician with feedback on the direction and magnitude of movement pattern disturbance (Lyle et al., 2005), the potential structure(s) at fault, nor the departure from usual movement according to normative age and gender reference ranges.

The present report investigated the novel application of computer assisted CME to measure change to both the magnitude and direction of movement dysfunction and consequently to demonstrate a tendency towards age and gender matched normalisation of low back movement after MT intervention.

The ability for CME to detect specific directions of restricted movement and provide insight into the possible biomechanical causes have been hypothesised by Brown (1998) though not subsequently examined. Pearcy and Hindle (1989) proposed the potential diagnostic value of 3-D lumbar movement assessment, however no studies have investigated this claim in pathoanatomical terms.

It is reasonable to derive a speculative model for inferring pain clinically and CME may assist the clinician in formulating a series of hypotheses about mechanical pain origin in LBP. From clinical studies, the intervertebral disc and paired facet joints are the most likely pain sources in the low back, with prevalence rates estimated to be 42% and 31%, respectively (Laplante et al., 2012). Osseo-ligamentous tissues and the disc anulus are putatively the primary contributors to spinal stiffness (Cunningham et al., 2007; Little et al., 2007). The prevalence of lumbosacral radiculopathy is estimated to be 3-5%, distributed equally in men and women (Tarulli and
Raynor, 2007). Lumbar discs have multi-level anterior compartment innervation by direct branches which arise from the sympathetic trunk, and the posterior disc from the rami communicans. In each case, this innervation is multi-segmental and bilateral (Figure 6) (Groen and Stolker, 2000). Whereas facet joints have bi-segmental, ipsilateral, posterior compartment innervation which have potential to cause ipsilateral multifidus muscle contraction and spasm (Bogduk, 1985; Edgar, 2007). From figure 6 one could hypothesise the source of LBP in each of these cases from the patient’s description of symptoms and their CME ‘signature’. The provisional diagnosis of disc and right nerve root compression was made for case A and B, respectively. In case A, CME identified a globally reduced movement pattern and in case B, a unilateral, symptomatic side. In both cases, CME reproduced the patient’s concordant symptoms. Based upon palpation, the L5 vertebral level reproducing the patient’s symptoms during MT assessment was chosen as the candidate level to treat.
Figure 6: A lumbar vertebra divided along two axes, defining anterior-posterior and left-right quadrants. The typical innervation pattern described by Groen & Stolker (2000) is depicted. Neural structures surrounding the vertebral body (VB) and intervertebral disc (IVD) are labelled: dorsal root ganglion (G), recurrent meningeal nerves (RM), sympathetic trunk ganglion (ST), afferent branch of ST crossing the mid-line (ST Br), branch to anterior longitudinal ligament (ST Br ALL), rami communicans (RC), ventral ramus (VR), dorsal ramus (DR), with medial branch to the facet joint (DRz), intermediate branch to muscle (DR int), and lateral branch to skin (DR cut).

To inform treatment outcomes, an age and gender matched CME NRR was used to establish goals. In both cases, VAS data for pain decreased by 100%, which exceeds the minimal clinically important change value of 30%, proposed by Ostelo et al. (2008). Visual analogue scale data for stiffness decreased by 74% and 33% for case A and B, respectively. While a CME NRR provides a putative movement outcome target, care must be taken to consider individual anatomical variations and the initial clinical presentation.

In case B, the increased lumbar lordosis, and simultaneous pain-free decrease in RSF at final retest, may imply that anatomical structure(s) restrict RSF. In this case, if symptoms return, further investigation such as appropriate imaging, may be warranted in an effort to identify (a reason for) the mechanical restriction.
Case B’s condition was unchanged after a few weeks of home program, comprising: right hip-to-wall passive (gapping) stretch, a lumbar extensor stretch, postural advice, and the suggestion to decrease walking as his daily exercise and replace with cycling). However, symptoms returned over the following month. Magnetic resonance imaging showed severe central stenosis at L4-5, and moderate to moderately severe foraminal stenosis present on the right at L5/S1 with mild flattening of the right L5 nerve within the foramen. An epidural cortisone injection was administered at L4-5, with temporary improvement. He was unsure if this was due to the temporary rest recommended post-injection or the medication. This patient has since been offered neurosurgery intervention at L4-5. At this stage, the patient has refused surgery and will explore the option of a right sided L5 nerve root sleeve injection, and further conservative management. Importantly, this case highlights the natural history and the outcome of further investigation, medical intervention and the potential for surgical management. While CME highlights movement asymmetry that may be due to compressive or tissue tension related pathologies, this assessment complements conventional MT assessments of the lumbar spine and also informs the decision to refer on for further investigation and medical intervention where indicated.

In both cases computer-aided CME contributed to the planning of manual therapy interventions and outcome assessment of their management. Further studies, investigating a larger number of cases with specific lumbar pathologies are required, along with invasive pain management approaches, to further validate the clinical utility of CME.
Ethics statement and Informed Consent

Approval to conduct the study was obtained from The University of Western Australia Human Research Ethics Committee. Written informed consent was obtained prior to participation.

Competing interests

There were no sources of funding or conflicts of interest associated with this research.

References


### Pooled data (n=35)

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### NS Case

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<td>56</td>
<td>L2-3 left posterolateral IVD protrusion</td>
<td>Radioculopathy</td>
<td>A</td>
<td>Y</td>
<td>epidural L2-3</td>
<td>VAAs 39%, RMDQ 96%, CME EVLSF, Extension</td>
</tr>
<tr>
<td>B</td>
<td>61</td>
<td>L4-5 left IVD protrusion compressing the exiting L4 nerve</td>
<td>Radioculopathy</td>
<td>No pattern</td>
<td>N</td>
<td>epidural L4-5 decompression</td>
<td>VAAs 41%, 10° increase to LL</td>
</tr>
<tr>
<td>C</td>
<td>28</td>
<td>L5-S1 left IVD protrusion</td>
<td>Radioculopathy</td>
<td>A &amp; B</td>
<td>Y</td>
<td>epidural L5-S1</td>
<td>VAAs 100%, CME LSF, VAAs 76%</td>
</tr>
<tr>
<td>D</td>
<td>44</td>
<td>L5-S1 bilateral facet and foraminal narrowing, Probable impingement</td>
<td>Radioculopathy</td>
<td>Y</td>
<td>Y</td>
<td>epidural L5-S1</td>
<td>VAAs 31%</td>
</tr>
<tr>
<td>E</td>
<td>57</td>
<td>L3-4 left IVD protrusion and right L4-5 IVD protrusion</td>
<td>Radioculopathy</td>
<td>C</td>
<td>Y</td>
<td>epidural L4-5</td>
<td>Extension and EVLSF, 20° increase to LL</td>
</tr>
<tr>
<td>F</td>
<td>58</td>
<td>L4-5 facets and left L5-S1 facet degeneration and L2-3 right IVD protrusion</td>
<td>Radioculopathy and canal stenosis</td>
<td>A &amp; B</td>
<td>Y</td>
<td>epidural L4-5</td>
<td>CME Flexion, LSF, LS</td>
</tr>
<tr>
<td>G</td>
<td>41</td>
<td>L5-S1 right posterolateral IVD prolapse</td>
<td>Radioculopathy</td>
<td>C</td>
<td>N</td>
<td>epidural L5-S1</td>
<td>VAAs 37%, VAAs 44%, PVLSF</td>
</tr>
<tr>
<td>H</td>
<td>42</td>
<td>L4-5 IVD prolapse</td>
<td>Radioculopathy</td>
<td>B &amp; C</td>
<td>Y</td>
<td>epidural L4-5</td>
<td>VAAs 83%, VAAs 44%, RMDQ 79%, LSF, RSF</td>
</tr>
<tr>
<td>I</td>
<td>58</td>
<td>L5-S1 spondylolisthesis and bilateral L5 nerve root compression</td>
<td>Radioculopathy</td>
<td>A &amp; B</td>
<td>Y</td>
<td>epidural L5-S1</td>
<td>VAAs 38%, CME EvRSF, PWRSF</td>
</tr>
<tr>
<td>J</td>
<td>67</td>
<td>L4-5 facet arthropathy, DDD and lateral recess stenosis</td>
<td>Radioculopathy</td>
<td>B</td>
<td>Y for DDD, N for radiculopathy</td>
<td>epidural L5 laterality</td>
<td>SF-12 PCS 39%, LL 42°, Extension, EVLSF, PWLSF</td>
</tr>
<tr>
<td>K</td>
<td>31</td>
<td>L4-5 disc protrusion and bilateral neural compression</td>
<td>LBP and radioculopathy</td>
<td>C</td>
<td>Y</td>
<td>epidural L5 laminectomy</td>
<td>VAAs 30%, SF-12 PCS 33%, LL 52°, EVLSF, PWLSF</td>
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<td>L</td>
<td>49</td>
<td>L4-5 DDD</td>
<td>DDD</td>
<td>B</td>
<td>Y</td>
<td>epidural L5 fusion</td>
<td>VAAs 78%, VAAs 83%, RMDQ 33%, CME LSF, EVRSF, PWRSF</td>
</tr>
<tr>
<td>M</td>
<td>44</td>
<td>L5-S1 DDD with left nerve compression</td>
<td>DDD and radiculopathy</td>
<td>B &amp; C</td>
<td>Y</td>
<td>epidural L5-S1 decompression and fusion</td>
<td>RMDQ 46%, 8° decrease in LL, Flexion, Extension</td>
</tr>
<tr>
<td>N</td>
<td>45</td>
<td>L5-S1 DDD and right neural foramus stenosis</td>
<td>DDD and radiculopathy</td>
<td>A &amp; C</td>
<td>Y</td>
<td>epidural L5-S1 decompression and fusion</td>
<td>Extension, EvRSF, LSF, RSF</td>
</tr>
<tr>
<td>O</td>
<td>70</td>
<td>L4-5 degenerative spinal stenosis</td>
<td>Radioculopathy</td>
<td>A</td>
<td>N</td>
<td>epidural L5 fusion</td>
<td>RMDQ 40%, 12° decrease in LL, Flexion</td>
</tr>
<tr>
<td>P</td>
<td>51</td>
<td>L4-5 spondylolisthesis and severe bilateral facet degeneration</td>
<td>LBP and radioculopathy</td>
<td>B &amp; C</td>
<td>Y</td>
<td>epidural L5 fusion</td>
<td>VAAs 41%, 12° increase in LL, extended directions</td>
</tr>
<tr>
<td>Q</td>
<td>40</td>
<td>L4-5 IVD prolapse</td>
<td>LBP and radioculopathy</td>
<td>B &amp; C</td>
<td>Y</td>
<td>epidural L5 fusion</td>
<td>VAAs 67%, VAAs 68%, RMDQ 79%, 7° decrease in LL</td>
</tr>
<tr>
<td>R</td>
<td>64</td>
<td>L3-4 R&gt;L radiculopathy</td>
<td>Radioculopathy</td>
<td>A, B &amp; C</td>
<td>Y</td>
<td>epidural L3-L4</td>
<td>LSF, PVLSF, PWRSF, 20° increase in LL</td>
</tr>
</tbody>
</table>

**MCID** = Minimal clinically important difference, **DDD** = degenerative disc disease, **IVF** = intervertebral foramen, **LL** = lumbar lordosis. For all outcome measure acronyms refer to the methods section. CME patterns: **A** = ipsilateral posterolateral decrease in ROM, **B** = globally reduced, **C** = bilateral posterior decrease in ROM. **Pooled data** (n=35)
Appendix XIII

NON-INVASIVE LUMBAR SPINE MOVEMENT: VALIDATION OF THE MOTIONSTAR™ 3-D ELECTROMAGNETIC TRACKING SYSTEM & PRELIMINARY EVIDENCE

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Introduction

Disturbed movements of the lumbar spine can provide ‘signatures’ to underlying pathology and will usually differ from normal patterns in terms of quality and range [Fig 1]. Non-invasive spine movement assessment using 3-D motion tracking systems discriminates normal from symptomatic subjects1, and may help to triage pathology subgroups. System validation is a necessary prerequisite to ensure reliable data acquisition in clinical studies.

Figure 1. Physiological spine motion requires an intact lumbar segment with normal hydration of the IVD (left). This enables lumbar sagittal and coronal plane motion, with side-flexion inducing axial rotation (so called - coupled motion).

Methods

The MotionStar™ 3-D tracking device (Ascension Technology, VT, USA) records displacement coordinates at 50Hz in the three cardinal planes. A custom triaxial protractor with a known accuracy of 0.5° was designed for replicate trials to simulate the normal range of human lumbar movement [Fig 2]. Data from 10 trials for displacements: 0°–10°, 0°–30° and 0°–60°, in each axis (X/Y/Z), were acquired in real-time using Labview software with post-processing in Excel.

Asymptomatic volunteers were assessed to map the coupled movement patterns for: flexion (F), side-flexion (SF) and extension (E) [Fig 3], to establish reliability from repeated trials and define a preliminary normal reference range. A subset of cases were used for inter-session reliability assessment on five occasions. Normal asymptomatic volunteers involved 14 males (mean age 26, range 20-32yrs) & 27 females (mean age 26, range 20-34yrs).

Figure 2. Triaxial protractor [left] was custom designed to allow a 3-D assessment of the MotionStar system error. The protractor device had an intrinsic accuracy to ±0.5°, although smaller gradations could be discerned. The MotionStar comprised an electromagnetic source with tracking sensors in the field calibrated to detect 3-D motion displacements.

Results

The Coefficients of Variation (CVs) for each triaxial protractor trial series, across all end-points and axes, ranged between 0.001 – 0.14%, representing ±0.3° error. Preliminary data for asymptomatic volunteers showed F<0.5° and ranges equivalent to published data for non-invasive lumbar range of motion. Intra-session repeatability trials involving the same subjects produced CVs ±0.5% [Fig 4]. Inter-session reliability trials showed similar repeatability [Fig 5]. Data are depicted in 360° rotating plots recording each adopted position shown in Figure 3 above.

Figure 3. Physiological spine motion depicting the test sequences of lumbar flexion, extension, side flexion plus the coupled positions of flexion + side flexion and extension + side flexion.

Figure 5. Example of inter-session repeatability of the combined movement examination in a 360° male. Five trials per session are depicted. Normal reference ranges for asymptomatic subjects are depicted in Figure 6 and show a gender difference for extension. Other range values are not significantly different.

Figure 6. Radial plot of 360° lumbar motion assessment presenting mean data for males [blue] and females [red]. Contrasting the asymptomatic movement signatures for each gender.

Discussion & Conclusion

The MotionStar™ 3-D motion tracking system demonstrated low system error across all planes and ranges using a triaxial protractor standard with CV<0.15%, or an error ±0.5degree. Preliminary assessment of variability within normal subject data was acceptable and normal reference ranges were consistent with published data for lumbar movement relative to age. Future investigations of composite lumbar spine movements, involving flexion, side flexion and extension, which involve coupled movements, may assist pattern recognition of mechanical spine pathologies.

Acknowledgement

Ray Smith for technical and data management advice.

References

This chapter is divided into two sections. Section one describes some of the physiological mechanisms of ageing, to introduce the reader to the changes we face when dealing with the ageing musculoskeletal system. The second section details the more specific changes ageing brings to the axial spine.

AGEING OF THE MUSCULOSKELETAL SYSTEM

Ageing is the declining ability to respond to stress and by virtue of the increasing homoeostatic imbalance and incidence of pathology, death remains the ultimate consequence of ageing. There are a number of theories regarding ageing, with a quick reference list displayed in Table 12-1. For a more detailed overview of this area please consider the excellent review by Weinert and Timiras published in 2003.

Numerous hypotheses regarding the diminishing function of cells with time exist. Mechanisms that have been proposed to be life- and/or function-limiting for cells include cumulative oxidative damage to proteins, accumulation of mutations and genomic instability, glycation of proteins and telomere (the protective region of the chromosome) dysfunction. Ageing of tissues is accompanied by increases in genome rearrangements and mutations, which may cause cell senescence and/or apoptosis (programmed cell death). Cell senescence refers to the temporal decrements in the ability of cells to replicate, repair and maintain tissue, and is induced by both cell-intrinsic and cell-extrinsic mechanisms.

The cellular senescence theory of ageing (1965) describes a process where there is a limit to the number of cell divisions normal human cells can undergo in culture. This ‘limit in replicative capacity’ occurs after a characteristic number of cell divisions and results in terminally arrested cells with altered physiology. Classical descriptions of cell senescence most often refer to the loss of the ability of mitotic cells to further divide in culture after a period of 30–40 population doublings. However, cell senescence appears to be much more complex than simple cell-cycle arrest occurring after a finite number of cell divisions. More recently, attention has been drawn to other forms of cell senescence sometimes referred to as ‘extrinsic’ or ‘stress-induced’ senescence as opposed to the intrinsic senescence resulting from replication. Stress-induced senescence can occur from diverse stimuli including oxidative damage, activated oncogenes and chronic inflammation. Stress-induced senescence due to oxidative stress fits well with one of the long-standing theories of ageing that suggests that free radicals could be mediators of ageing.

Ageing Joints

Oxidative damage from the chronic production of endogenous reactive oxygen species and free radicals has been associated with ageing in various human tissues and has long been thought to play a central role in the ageing process. Increased production of reactive oxygen species leads to oxidative stress, a condition within cells where the amount of reactive oxygen species exceeds the antioxidant capacity of the cell. Human articular chondrocytes can actively produce reactive oxygen species and have been found to increase directly with age.

Some of the changes observed in ageing joints and their contribution to the development of osteoarthritis are detailed in Table 12-2.

Ageing Muscles

In both young and aged skeletal muscle, it has been shown that oxidative stress increases in response to unloading (lack of activity/immobilization) and may have an important role in mediating muscle atrophy. Decline of muscle mass is primarily due to type II fibre atrophy and loss of muscle fibre numbers. Increased variability in fibre size, accumulation of scattered and angulated fibres and expansion of extracellular matrix are characteristic to muscle atrophy. The rate of muscle loss has been estimated to range from 1–2% per year past the age of 50, 25% in persons under the age of 70 and 40% of those older than 80 years are sarcopenic. Significant loss of myofibrils results in an increased negative nitrogen balance which exacerbates reductions in strength and endurance.

With advancing age, muscles display ‘hybrid’ muscle fibre characteristics. Ageing muscle demonstrates selective loss of fast motoneurons, leading to ‘orphan’ fast twitch muscle fibres that are adopted by the relatively more abundant slow motor units. These fibres partially convert to slow twitch fibres, ending with a hybrid phenotype showing the characteristics of both fast and slow fibres. When, fast type II muscle fibres are incorporated into slow motor units (and eventually turned into a hybrid fibre), there are irregularities in the size distribution of motor units. This, in turn, affects motor accuracy, especially with low force movements, as the recruitment order does not adjust well to the previously small motor
units having grown bigger and stronger, and is one of the main reasons that motor and locomotor skills deteriorate with age.\(^\text{12}\)

**Ageing Nervous System**

In the central nervous system there are age-related reductions in the total number of brain cells and fibres and the organization of fibres within the brain’s white matter in addition to the reduction to the large diameter (A-beta proprioceptive) fibres in peripheral nerves.\(^\text{11}\) In addition to reductions in the number of fast myelinated fibres, within the nerve the speed of signal conduction within the axon also reduces with age.\(^\text{15}\) Neuromuscular junctions have been shown to demonstrate age-related reductions in size and speed of conduction, thereby reducing the efficiency of efferent transmission to the muscles.\(^\text{16}\) These widespread physiological changes have been shown to result in poorer performance on cognitive and motor tests.\(^\text{14,15}\)
Ageing, Falling and Pain

Rates of falls among community-living, generally healthy elderly people (age ≥65) are of the order of (0.3–1.6 per person annually), with about 5% of these resulting in a fracture or hospitalization. Fall rates rise steadily with age and are about doubled for persons aged >75 years. Persons living in long-term care institutions have much higher fall rates (0.6–3.6 annually). Falls among those in institutions also tend to result in more serious complications; with 10–25% of such falls resulting in fracture or laceration. It is often reported that older adults experience greater prevalence of pain, greater pain intensity and pain at more sites than younger adults.15,16

It has long been thought that the increase in the prevalence of pain among older adults is partly due to the progressive musculoskeletal degeneration that accompanies ageing. Another explanation for increased pain in older populations has been that ageing is associated with greater sensitivity to painful stimuli that results from changes in the structure and function of the nociceptive system.17

Ageing and the Beneficial Effects of Movement

It is thought that mechanical forces are important regulators of several biological functions, with mechanical signals having been shown to mediate the development of a variety of tissues (e.g. skeletal muscle, bone, cartilage).18,19 Mechanical signals can affect diverse cellular processes including cell growth, differentiation, cellular migration, gene expression, protein synthesis and apoptosis.18,19 Given the potential importance that mechanical signalling functions have in maintaining cellular homeostasis, it is likely that changes in mechanotransduction may also play a role in the pathophysiology of disease.19

Recent data strongly support this, as it is recognized that many aspects of sarcopenia may be related to alteration in cellular mechanotransduction.19 The ability of cells involved in the musculoskeletal system to sense, process and respond to mechanical stimuli deteriorates with age and that these changes may be involved in the aetiology of ageing-associated disease.20 Encouragingly, movement quantity, quality, locality and intensity are relatively modifiable influences and are certainly influences within the scope of the manual therapists. The influence of mechanotransduction on tissue health and the symptoms of ageing are exciting areas for future research, particularly for those of us involved in the provision and prescription of therapeutic movement.

THE AGEING SPINE

Most individuals achieve old age with some evidence of degenerative or pathological changes in spinal joints, which reflect the interactions between: genetics, occupation, lifestyle, nutrition, trauma and destiny. Most published reviews on the health of the spine are seen through the prism of clinical perspectives, with an absence of large-scale, long time-lapse epidemiological surveys to discern patterns and to test predictions. Post-mortem surveys and large radiological population-based studies21–23 agree that spinal degenerative and age changes have an extremely high prevalence in adult populations. Of late, subtle differences in disc degeneration patterns are being realized as genomic sequencing emerges as an investigatory tool.24 In the current era of Western society, osteoporosis is acutely studied given the cost and morbidity associated with the declining bone mineral health of the axial skeleton and its attendant fracture risk. This trend is not surprising, as over the last 100 years developed societies have evolved from physically demanding occupations to becoming increasingly urbanized and inactive. The call to add physical activity to counter the epidemic of poor lifestyle choices (i.e. inactivity, smoking, poor nutrition) has become urgent given the projected cost and negative health outcomes for society.25

Our biology uses both ageing and degeneration strategies to constrain the musculoskeletal system against further injury or damage. In the case of the spine, which serves the principal objectives of mobility, stability and protection of neural elements, overload or dynamic loading strains can induce a spectrum of either local or regional trauma and degenerative changes. Consequently, this chapter draws upon literature that represents both age-related and degeneration models, and their outcomes for the axial skeleton.

Normal physiological strains are well accommodated by each functional mobile segment, which comprises an intervertebral disc (IVD) with an anulus fibrosus and nucleus pulposus, and the vertebral end-plates (VEP). Paired synovial zygapophysial joints link both vertebrae posteriorly and articulate closely to regulate both load and movement of the segment. Applied moments from muscle actions and axial compressive loads may be coupled with shear, bending (rotations) and torsion about the long axis of the spine, which are in turn moderated by the unique geometry of the segment’s zygapophysial and ligamentous anatomy (Fig. 12-1). Inertial strains from dynamic loading, even several times body weight, may also be tolerated by the spine given its unique capacity to attenuate energy.26 The regional response to loading is reflected in different patterns of injury or degeneration.

Vertebral bone adapts to loading by the cyclic remodelling which is optimal by the third decade of life and declines variably thereafter.27 Loss of trabecular connectivity, endocortical bone trabeculation and intracortical porosity are the late stages of remodelling. VEP collapse occurs due to trabecular bone fragility, with vertebral body fracture the clinical end-point in some cases. IVD degeneration is considered a normal process of ageing, but may be precipitated by multiple factors including genetic, anatomical, mechanical (occupational, overload, torsion, vibration), cell-mediated molecular cascades, trauma, infection and toxins as major influences to disc health28–30 (Fig. 12-2).

Osteoarthritis of the spine develops as a consequence of the natural ageing process coupled with attrition, and is associated with a degenerative cascade that may involve
the discrete elements or an entire functional mobile segment, individually or regionally.\textsuperscript{31,12} The IVD is essentially aneural apart from the peripheral superficial outer third, although with injury to the disc, vascular ingrowth associated with repair may contribute vasomotor nerves.\textsuperscript{31} The disc is also avascular, apart from the peripheral annulus, with a reliance upon nutritional substances transported via diffusion across the VEPs\textsuperscript{14} or through vessels that communicate directly with the outer annular layers. Consequently, disruption to either system occurring through normal ageing, surgical intervention, spinal deformity or trauma can disrupt and lengthen the pathways of nutritional support to the disc and is presumed to contribute to subsequent disc degeneration. The consequence of either ageing or injury to the functional mobile segment may be degeneration of its elements with initial progressive increase in strain tolerance beyond the normal, which may progress to increased segmental mobility. One mechanical response to such changes, particularly affecting the stability and function of the IVD, is spondylosis, initiated through osteogenic stimulation in the junctional region between the VEP periphery and the annulus, resulting in the early formation of osteophytes.\textsuperscript{35} Experimentally induced osteoarthrosis of the paired zygapophyseal joints has been associated with anular rim lesions of the IVD.\textsuperscript{36} The posterior paired costotransverse and costovertebral zygapophyseal joints are true synovial joints invested with hyaline articular cartilage, a capsule and synovium. These joints contribute stability of the respective segment(s) and facilitate respiratory excursions of the thorax and regional mobility within the vertebral column, respectively. Each may respond to overload with degenerative patterns of synovial joints characterized by mechanical changes of the articular cartilage. Subchondral bone sclerosis, fissuring and detachment of the cartilage, and marginal joint osteophytosis may follow changes in the IVD, particularly a loss of vertical height which in turn alters the mechanical alignment of the respective superior and inferior articular processes of the posterior joints. Bumper fibrocartilage formations at the joint margin are associated with evidence of articular cartilage degeneration and fissuring, ossification of the ligamentum flavum, and reactive hyperplasia at the posterior joint margins. A further consequence of degenerative changes leading to altered morphology of the IVD and vertebral bodies is the response by the spinal ligaments. With progressive deformation of the segment, ligaments may demonstrate buckling and, in response to exaggerated segmental motion strains, subsequent hypertrophic changes may contribute to stenotic change within the vertebral and intervertebral canals.\textsuperscript{37} Ossification within the ligamentum flavum may occur as a consequence of degeneration of the articular triad, although this tends to predominate in the region of the lower thoracic and upper lumbar segments.\textsuperscript{38} Patterns of spinal degeneration and age changes become evident when merged onto a common model of the axial skeleton; the mobile cervical and lumbar segments, and their respective stiffer transitional junctions display different trends. The general pattern is for spinal motion to decline in all directions with age, and this feature is illustrated with the combined movement examination assessment for the lumbar spine (Fig. 12-3). Where segmental mobility is greatest degeneration of the disc and facet joints dominate. In the case of the bony thorax, focal changes are seen at the respective costo-vertebral joint articulations of the first and last ribs, a consequence of transferring large torques from the musculature of the neck and trunk, respectively. When one
considers the complete vertebral column as a multisegmented curved rod, with physiological inflexions that cross the neutral axis line, the literature presents evidence of different responses to stress accumulations at points of both maximum and minimum change in curve. The segments adjacent to the transitional junctions, having less relative motion, are designed more for stability and represent locations where axial compressive load is greater, the change in spinal curvature is least and where arthrosis of these synovial joints is found. In contrast, where the curvature away from the neutral axis line is maximum, as in the middle region of the lordosis and kyphosis, respectively, and where bending, torsion and shear stresses are relatively higher, the trend is for greater disc degeneration (see Fig. 12-1).

The major degenerative conditions reviewed in this chapter include osteoporosis and anomalies of spinal curvature, and changes that arise secondary to trauma. Inflammatory disease of the spine is excluded from this discussion; the interested reader is directed to the compilation by Klippel and Dieppe for a comprehensive review. Degenerative conditions that principally have a spinal manifestation may involve all elements of the functional mobile segment, either singularly as in the case of early IVD degeneration, or across the joint complex, exemplified by late zygapophysial joint arthrosis coincident with IVD degeneration.40,41

**Disc Degeneration**

Literature describing the incidence of disc degeneration throughout the vertebral column concentrates predominantly on the lumbar and cervical regions of the spine.42 From post-mortem studies, discs with altered vascularity during the second decade of life show precursor changes to early degeneration.43 The pathway of age-related degeneration change has been described as compromised nutrition, loss of viable cells, cell senescence, post-translational modification of matrix proteins, accumulation of degraded matrix molecules, a reduction in pH levels that may impede cell function and ultimately induce cell death, and finally fatigue failure of the matrix.44 The highest prevalence of disc degeneration is in the mid-cervical, mid-thoracic and mid-lumbar discs as these regions show a marked degree of reactive changes of the vertebral bodies with marginal osteophyte formation (Fig. 12-4). Early post-mortem studies by von Lushka45 demonstrated a large proportion of cervical discs with fissures and clefts. This was considered to be a normal characteristic of the region, with complete transverse clefts extending across and into the region of the uncovertebral joints found in the middle of healthy cervical discs on coronal section.46 From similar post-mortem reviews of the thoracic spine, the most severely affected discs are located predominantly within the middle segments, peaking between T6–T7, with a greater incidence in males.47 Given the tendency to axial plane segmental motion in the mid-thoracic spine, reported in the classic paper by Gregersen and Lucas,48 such degenerative changes may relate to the large rotation strains imposed upon these segments. Investigations by Farfan et al.49 into the effects of torsion on lumbar IVDs concluded that relatively small rotation strains >2° per segment induced potential injury in the anulus fibrosus. The pattern of age-related decline in anterior disc height in men typifies the disc ageing process associated with senile kyphosis whereby the cumulative effects of axial loading and torsional stresses result in degeneration of the anterior anulus and osteophytosis.21 In females, however, loading through the anterior aspect of the kyphotic curve is more likely to produce progressive change of the vertebral bodies, causing the wedge deformity commonly associated with spinal osteoporosis.50 Mechanically, the middle vertebral segments are predisposed to greater axial compressive and bending moments, due to their position within the apex of the thoracic kyphosis.51

**Osteophytosis**

Osteophyte formation and its associated IVD degeneration has been recognized as an attempt to distribute force more uniformly across the VEPs to achieve stress reduction on the segment.52 Where thoracolumbar disc degeneration is present, marginal osteophyte formation of the
vertebral body is frequently seen. This pattern of excess bone formation, commonly referred to as spondylosis deformans, is seen in approximately 60% of women and 80% of men. The degree of intervertebral space narrowing and subsequent tilting of the vertebral bodies, resulting from disc degeneration, often determines the extent and the type of marginal osteophytes. In summary, the segments that appear susceptible to osteophytes are often the most mobile regions with the higher levels of disc degeneration, or where local stress may be accumulated.

**Vertebral End-Plate Lesions and Schmorl’s Nodes**

The vertebral end-plate is a membrane of tissue comprising hyaline cartilage and a 0.5-mm-thin trabecular layer at the discovertebral junction. Its role is to mediate axial compressive load applied to the IVD and permit transfer of this energy within the subchondral and cancellous bone of the vertebral body. Physiological axial loading, as occurs with gait, acts as a ‘pump’ to assist diffusion of nutrients within the vascular vertebral body across the VEP and into/away from the disc. Abrupt or fatigue axial loading of the spine may cause localized failure of the VEP resulting in either a frank sharply demarcated vertebral intra-osseous prolapse, often termed a Schmorl’s node, or marked irregularity of the end-plate. The repair process for both lesions often results in bony sclerosis which can significantly impair the normal nutrient exchange to the IVD by reducing the effectiveness of this diffusion pathway. Schmorl’s nodes have been reported to occur during the late teens, with lesions as frequent in the young as in the older individual. Cadaveric studies of lumbar spines have indicated that Schmorl’s nodes develop at an early age and can exhibit advanced degenerative changes. Schmorl’s nodes are found most commonly in males and are considered to be related to a genetic disposition, strenuous occupations or sports involving dynamic and violent axial loading as might occur with a heavy landing in flexion. Most authors agree that the inferior end-plate is more susceptible to infraction which implies that the VEP fails under compression (Fig. 12-5).

**Zygapophysial and Costovertebral Joint Degeneration**

There appear to be specific sites within the spine where preferential degeneration of the synovial joints occur. The upper and lower segments of the thoracic region show a tendency for zygapophysial and costovertebral joint degeneration. Similar trends for osteophytic remodelling of the zygapophysial joints of the lumbo-sacral junction have been reported. This may be due to the design of these elements that provide stability and protection in contrast to the adjacent mobile segments which show a correspondingly higher frequency of disc disease (see Fig. 12-1). The development of osteophytes and eventual bony fusion of costovertebral and costo-transverse joints in aged vertebral columns was also noted by Schmorl and Junghans in their extensive survey of spinal pathology. The cervicothoracic junction and thoracolumbar junction represent transitional areas between mobile and relatively immobile regions of the spine. At the cervicothoracic junction, Boyle et al. found evident IVD and VEP changes, along with osteophytic formation that were more pronounced in the mobile segments immediately above the transition. The upper thoracic region and thoracic cage acted to impede intersegmental motion and thus safeguard these levels from marked degeneration. At the thoracolumbar junction, Malmivaara et al. demonstrated that particular pathologies tended to be concentrated at each segment. The T10–T11 segment was characterized by disc degeneration, vertebral body osteophytosis and Schmorl’s nodes; the T11–T12 segment tended to show both anterior and posterior degeneration, involving zygapophysial and costovertebral joints, while the T12–L1 joint was characterized primarily by posterior joint degeneration. A comparison of zygapophysial joint orientation with degenerative findings suggested that the posterior elements play a significant role in resisting torsional loads.
Osteoporosis and Osteoporotic Fracture

Asymmetry in the zygapophysial joint orientation tended to result in degenerative changes occurring mostly on the sagittal facing facet, an observation originally made by Farfan et al. at the lumbosacral junction.

Degenerative Spinal Curvature Anomalies

Idiopathic scoliosis involves a lateral curvature of the spine that is introduced through a disturbance in the longitudinal growth of the spine. It may occur early in the growth of the child and particularly during the early adolescent years. Four main curve patterns have been identified: thoracic, lumbar, thoracolumbar and double major curves. Each of these curvature patterns has its own characteristics and predictable end-point. It is well accepted that the severity of the scoliosis can continue to progress through the life span. Disc degeneration is known to develop due to the often extreme compression and ipsilateral tension strains experienced within wedged scoliotic IVDs. A cascade of degenerative changes occur in advanced scoliosis due to the attempt to stabilize against the increasingly asymmetric mechanical loads induced by this deformity (Fig. 12-6).

Osteoporosis is an endocrine disease characterized by decreased bone mass and micro-architectural deterioration of bone, which may lead to bone fragility and subsequently to an increased rate of fracture. Although resorption of bone follows the normal process of ageing, it may be induced through disordered metabolism and is accelerated following menopause in women. A gender difference in bone fragility emerges due to the dynamic change in relationship between the mechanics of load transfer and the margins of safety. Males accumulate more periosteal bone than females, with a corresponding increase in vertebral cross-sectional area which confers a relatively higher load-bearing capability such that reductions in bone strength are less dramatic than seen in women. During ageing, this ratio is disturbed and fracture risk increases as the stress on bone begins to approximate its strength. Twenty per cent of postmenopausal women have a stress-to-strength ratio imbalance, whereas only 2–3% of men are at risk of fracture due to the greater preservation of bone strength. The epidemiology of osteoporosis is well known whereby the risk factors of age, gender and racial contributors to bone loss and corresponding fracture risk increase exponentially with age. For the thoracic spine, one in four women over the age of 60 years will show at least one vertebral body fracture on radiographic examination, while the incidence increases to 100% in women over 80 years of age; for men, there is a decade offset before osteopenia and osteoporosis develops. The mid-thoracic segments are the most vulnerable to osteoporotic collapse or progressive wedge deformity due to the mechanical disadvantage of these segments situated within the apex of the thoracic kyphosis. The second peak for thoracic osteoporotic fracture is at the thoracolumbar junction where more rapid loading of the thoracic spine can induce a hinging of the stiffened thorax on the upper lumbar spine. These
Junction transition. Markolf occur immediately above the level of thoracolumbar coupled with localization of torsional forces that can typically greater disc height and volume at this region, commonly involved. This trend may be due to the relation the mid to lower cervical region and lower lumbar segments within the region of the thoracolumbar transition, depicted from the (FIGURE 12-6) anterior aspect. Note the remarkable osseous degeneration and remodelling. (C) A surface contour image of a marked scoliosis in an elderly woman, showing the typical rib hump appearance and asymmetry of the thoracic cage.

Intervertebral Disc Prolapse

Clinically, the regions susceptible to prolapse of the intervertebral disc and the resulting disc degeneration typically are those with higher levels of mobility within the mid to lower cervical region and lower lumbar segments. What is not often appreciated is the high frequency of macroscopic disc prolapse within the thoracic region (see Fig. 12-4) with the T11–T12 level most commonly involved. This trend may be due to the relatively greater disc height and volume at this region, coupled with localization of torsional forces that can occur immediately above the level of thoracolumbar junction transition. Markolf proposed that the eleventh and twelfth thoracic vertebrae represented a site of structural weakness for stresses in the vertebral column, due to the reduced constraint of the ribcage and the change in zygapophysial joint morphology that facilitated rotation above the transitional levels and impeded it below. The implication of disc prolapse is mechanical decomposition of the nucleus, fissuring of the annulus and the cascade of changes that follow this injury.

Summary

The human spine contributes a large proportion of the musculoskeletal presentations seen in manual therapy practice. This chapter has reviewed the effect of age on the human spine, including those degenerative processes that are secondary to metabolic disease, spinal deformity or trauma. Ageing of the spine is not merely a chronological process, as remodelling and repair follow such insults as trauma, disease, deformity or surgery and reflects a biological strategy to stabilize against further segment damage from imposed loads. While ageing is an unavoidable certainty, skeletal loading remains a critical requirement for optimal function. Loading the musculoskeletal system throughout its dynamic range, over the lifespan, is crucial for sustaining not just musculoskeletal health but health in general.

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Ref 19319938
11 December 2014

Mr A Monie
94 The Promenade
Mount Pleasant WA 6153

Dear Mr Monie

AUSTRALIAN POSTGRADUATE AWARD AND UWA SAFETY NET TOP-UP SCHOLARSHIP FOR 2015

Student No: 19319938
Scholarship: Australian Postgraduate Award
            UWA Safety Net Top-Up Scholarship
School: School of Surgery
Tenure: 1 January 2015 to 4 January 2017
Candidature: PhD
Proposed Supervisor: Winthrop Professor K Singer
Previous candidature: 22 April 2013

I am pleased to advise you that the Scholarships Committee of The University of Western Australia has awarded you an Australian Postgraduate Award (APA) in the School of Surgery. The APA provides a tax-free stipend (for full-time study) of $25,849 per annum in 2015, and will be tenable from 1 January 2015 to 4 January 2017. You have also been awarded a supplementary UWA Safety Net Top-Up Scholarship, currently valued at $3,151 per annum. The UWA Safety Net Top-Up is provided to students who receive an APA and who do not receive other top-ups of more than $5,000 per year. The combined value of the APA and UWA Safety Net Top-Up Scholarship package for 2015 is $29,000 per annum. As you have already been enrolled in the degree for which the scholarships have been awarded, the maximum tenure of the scholarship will be reduced by the length of your prior candidature.

Please note that to be eligible for payment of the UWA Safety Net Top-Up Scholarship you must not be in receipt of other top-up scholarships totalling more than $5,000 per annum. The APA stipend will be adjusted annually in line with the Commonwealth Department of Education indexation. The value of the UWA Safety Net Top-Up Scholarship is at the discretion of the UWA Scholarships Committee and will also be adjusted each year, reducing as the APA increases. The Safety-Net Top-Up program will be discontinued when the value of the APA reaches or exceeds $29,000 per year.

Conditions of Offer

This offer of an Australian Postgraduate Award and UWA Safety Net Top-Up Scholarship is conditional upon the following:

(a) The awards are for full-time research at The University of Western Australia and may not be taken up at or transferred to any other institution or deferred until a later year.

(b) Return of the signed Conditions of Award and the Acceptance Form, including confirmation that you have not previously held an Australian Postgraduate Award for more than six months.

(c) The conditions of the APA are explicitly binding on the UWA Safety Net Top-Up Scholarship.

APA Offer - PhD - PC