UNIVERSITY OF SOUTHAMPTON
INSTITUTE OF SOUND AND VIBRATION RESEARCH

CLINICAL APPLICATIONS OF DIGITISED VIDEOFLUOROSCOPY IN THE LUMBAR SPINE

BY

MICHAEL KONDRACKI

A Thesis Submitted for the Degree of

DOCTOR OF PHILOSOPHY

JUNE 2001

UNIVERSITY OF SOUTHAMPTON

ABSTRACT

INSTITUTE OF SOUND AND VIBRATION RESEARCH

DOCTOR OF PHILOSOPHY

CLINICAL APPLICATIONS OF DIGITISED FLUOROSCOPY IN THE LUMBAR SPINE

by

MICHAEL KONDRACKI

Chronic low back pain is, in the developed world at least, a costly problem. Costly to the individual in terms of the personal misery that accompanies it and costly to society in terms of working days lost, Sickness and Invalidity Benefits and healthcare provision. It has been estimated that during any 1-month period, 66% of patients who will ever have low back pain are symptomatic (Papageorgiou et al1995).

In spite of its prevalence, however,up to 85% of back pain patients cannot be given an accurate diagnosis (Moffett and Richardson 1995). Addressing this diagnostic problem relies, to some extent, on improving our understanding of the mechanics of the spine and how disorders might reveal themselves during spinal motion.

Part 1 of this thesis considers the various methods of measuring spinal movements especially those concerned with dynamic imaging. The basis of some commonly used kinematic indices is also discussed before reviewing key studies, both *in vitro* and *in vivo*, of intervertebral motion. In addition, Part 1 includes a review of the concept of lumbar segmental instability. Part 2 considers the possible applications of digitised fluoroscopy including comparison to other spinal measures and a suggested role in improving selection for spinal surgery. Finally, Part 3 looks at recent developments in the evolution of digitised videoflouroscopy and discusses results from a study of lumbar spinal motion in a group of asymptomatic volunteers under a new passive motion protocol.

List of abbreviations

3-D	Three-dimensional
aICRs	Averaged ICRs
AP	Antero-posterior
CP(s)	Centre point(s)
CT	Computerised tomography
DAP	Dose-area product
DCRA	Distortion-compensated roentgen analysis
DF	Digitised fluoroscopy
DVF	Digitised videofluoroscopy
EMG	Electromyogram
EZ	Elastic zone
FSU	Functional spinal unit
Gy	Gray
HAM	Helical axis of motion
IAP	Intra-abdominal pressure
IAR(s)	Instantaneous axis(es) of rotation
ICA(s)	Incremental angle(s)
ICCs	Intraclass correlation coefficients
ICR(s)	Instantaneous centre(s) of rotation
ICRP	International Commission on Radiological Protection
IVA(s)	Intervertebral angle(s)
IVD	Intervertebral disc
LLF	Left lateral flexion
LZ	Lax zone
MR	Magnetic resonance
MRI	Magnetic resonance imaging
mSv	MilliSievert
NRPB	National Radiological Protection Board
NZ	Neutral zone
PMT	Passive motion table
RLF	Right lateral flexion
ROM	Range of motion
RSA	Roentgen stereophotogrammetric analysis
SLR	Straight leg raising (Test)
stICRs	Standard ICRs

ACKNOWLEDGEMENTS

I would like to express profound gratitude and thanks to my supervisor Prof Bob Allen and to Dr Jen Muggleton for all their guidance and help throughout this work. I would also gratefully acknowledge the assistance of Dr Alan Breen who, together with Prof Allen, pioneered the techniques developed in this thesis and was personally involved in most of the experimental stages. Thanks also to Jenny Langworthy for her input and for putting up with Alan and I during this period.

My appreciation is long overdue to the AECC for funding my candidature and their unstinting support for this work and to all my colleagues for the encouragement and advice I have gratefully received from them over the years. I am indebted to the Henry Smith Charity for the generous funding awarded to this project and to the many AECC students who allowed themselves to be irradiated in the name science. Thanks also to Dr David Antrobus and Atlas Tables Ltd for their excellent contribution to the development of DVF research and to the Radiology Unit of Salisbury District Hospital for allowing us the use of their busy department. A special thank you is also extended to Dr Chris Howls from the Department of Mathematics, University of Southampton for his invaluable help in the development of the matrix used in this thesis for calculating vertebral displacements.
Lastly, I would like to thank Kristine for her enduring patience, tolerance and support throughout the latter stages of this dissertation.

CONTENTS

Part I **12**

Background Studies

Introduction 13

Research Questions 14

Chapter One 20

Spinal Measurements

1.1 Introduction 20

1.2 Surface Measurement 20

1.3 Radiographic Measurement 21

1.4 Fluoroscopy 22

1.5 Digitized Videofluoroscopy 23

Chapter Two 29

Kinematic Indices

2.1 Introduction 29

2.2 Instantaneous Centre of Rotation (ICR) 30

2.2.1 Engineering Considerations 30

2.2.2 Analysis and Treatment of Error 33

2.2.3 The Minimization of Error 41

2.3 Clinical Kinematic Measurements 42

2.4 Summary 56

Chapter Three 57

Biomechanical Investigations

3.1 Introduction 57

3.2 The Functional Spinal Unit 57

3.2.1 Definition 57

3.2.1 The FSU in Use 58

3.3 Sequential Destruction Studies 59

3.4 Intervertebral Disc Studies 61

3.5 The Neutral Zone 66

3.6 Coupling Studies 71

3.7 Axial Rotation Studies 77

Chapter Four 78

Segmental Instability

4.1 Introduction 78

4.2 Radiographic Instability 78

4.3 A New Approach to Instability 82

4.4 Degenerative Instability 85

4.5 Clinical Instability 90

4.6 Biomechanical Instability 91

4.6.1 Active Factors 93

4.6.2 Axial Translation 96

4.6.3 Hysteresis 97

4.7 Multifidus 99

4.8 Intra-Abdominal Pressure Changes 110

4.9 Summary 111

4.10 Conclusion 114

Part II **115**

Application of DVF

Introduction 116

Chapter Five 117

Comparison Study Between DVF and 3Space Isotrak

5.1 Introduction 117

5.2 Methodology 117

5.3 Results 120

5.4 Discussion 124

5.5 Conclusion 127

Chapter Six 131

Detection of Failed Lumbar Fusion by Digital Videofluoroscopy

6.1 Introduction 131

6.2 Methods 132

6.3 Results 133

6.4 Discussion 133

6.5 Conclusion 134

Part III **136**

New Developments

Chapter Seven 137

Passive Motion Analysis of Lumbar Spine Motion

7.1 Introduction 137

7.2 Design 138

7.3 Implementation 143

Chapter Eight 144

The Passive Intervertebral Motion of the Lumbar Spine in Asymptomatic Subjects: A Preliminary Study (Methodology)

8.1 Passive Motion Table Protocol 144

8.2 Data Acquisition 148

8.3 Manual Marking 148

8.4 Data Analysis 150

8.5 Determination of Neutral Position 152

8.6 Estimation of Measurement Error (Accuracy) 153

8.7 Estimation of Measurement Error (Repeatability) 159

8.8 IVA Measurement Variation 160

8.9 AP Translation Measurement Variation 169

8.10 Axial Translation Measurement Variation 177

8.11 Radiation Dosage 189

Chapter Nine 192

The Passive Intervertebral Motion of the Lumbar Spine in Asymptomatic Studies: A Prelimary Study (Results)

9.1 Introduction 192

9.2 IVA Ranges (Flexion/Extension) 192

9.3 IVA Ranges (Lateral Flexion) 194

9.4 ICA Ranges (Flexion/Extension) 195

9.5 ICA Ranges (Lateral Flexion) 197

9.6 Sagittal Translation 198

9.7 Discussion 200

9.7.1 Defining the Neutral Position 200

9.7.2 IVA Ranges 204

9.7.3 ICA Ranges 216

9.7.4 Paradoxical Rotations 217

9.7.5 Sagittal Translation 220

9.7.5.1 AP Translation 220

9.7.5.2 Axial Translation 225

9.8 Conclusion 232

9.9 Future Work 233

Conclusions and Summary 235

Appendix I 238

References 242

CONTENTS OF FIGURES

Part I

Chapter One

Figure 1.1: Image processing environment 27

Chapter Two

Figure 2.1: Diagram showing three-dimensional co-ordinate system applied to vertebrae 31
Figure 2.2: Diagram showing input co-ordinates for determination of ICR 37
Figure 2.3: Diagram showing error zone associated with ICR determination 38
Figure 2.4: Diagram of George's line 46
Figure 2.5: Diagram showing method of measuring translation (Morgan & King 1957) 47
Figure 2.6: Diagram showing method of measuring translation (van Akkerveeken et al 1979) 48
Figure 2.7: Diagram showing method of measuring translation (Posner et al 1982) 49
Figure 2.8: Diagram showing method of measuring translation (Dupuis et al 1985) 50
Figure 2.9: Diagram showing centrode patterns for mild, moderate and severe degeneration 54

Chapter Three

Figure 3.1: Diagram showing the response of the disc to wedging 66
Figure 3.2: Diagram showing definition of the neutral zone 67
Figure 3.3: Diagram showing the effect of the bending moment across the lumbar spine 69
Figure 3.4: Diagram showing IARs for axial rotation 76

Chapter Four

Figure 4.1: Diagram showing Panjabi's model for spinal stability 83
Figure 4.2: Graph of velocity change in the FSU 92
Figure 4.3: Diagram showing force vectors of the lumbar multifidus 101

Part II

Chapter Five

Figure 5.1:	Isotrak mounted to subject's back	118
Figure 5.2:	Diagram showing Isotrak and DVF measurements	119
Figure 5.3:	Isotrak subject in position	121
Figure 5.4:	Optimas image processing	122
Figure 5.5:	Graphs showing Isotrak and DVF results	123
Figure 5.6:	Diagram showing skin stretch hypothesis	128

Chapter Six

Figure 6.1:	Graphs depicting pseudarthrosis	135

Part III

Chapter Seven

Figure 7.1:	Fluoroscope plinth with passive motion table attached	141
Figure 7.2:	Passive motion table with patient	142

Chapter Eight

Figure 8.1:	Subject in lateral position ready for flexion/extension screening	146
Figure 8.2:	Subject being assessed for range of extension	147
Figure 8.3:	Images of lateral lumbar spine with and without templates	149
Figure 8.4:	Graph showing an example of the change in vertebral body angle from neutral (L1-L5)	151
Figure 8.5:	Graph depicting IVAs for flexion/extension and lateral bending	152
Figure 8.6:	Aluminium calibration grid	155
Figure 8.7:	Diagram showing measurement of spine-to-table distances used to assess the position of the lumbar spine relative to the image intensifier	156
Figure 8.8:	Image of calibration grid with marked angle used to assess accuracy	158
Figure 8.9:	Diagram showing method of measuring AP translation attributed to Frobin and colleagues (1996)	170

Figure 8.10: Scatterplot showing intra-observer variation (observer 1) for AP translation (neutral position) 171
Figure 8.11: Scatterplot showing intra-observer variation (observer 1) for AP translation (full flexion position) 172
Figure 8.12: Scatterplot showing intra-observer variation (observer 1) for AP translation (full extension position) 173
Figure 8.13: Scatterplot showing inter-observer variation (observers 1,2,3) for AP translation (neutral position) 174
Figure 8.14: Scatterplot showing inter-observer variation (observers 1,2,3) for AP translation (full flexion position) 175
Figure 8.15: Scatterplot showing inter-observer variation (observers 1,2,3) for AP translation (full extension position) 176
Figure 8.16: Diagram showing points used in the calculation of mean IVD height employed as a measure of axial translation 178
Figure 8.17: Diagram showing points used in the calculation of mean vertebral body height 179
Figure 8.18: Scatterplot showing intra-observer variation (observer 1) for axial translation/IVD height (neutral position) 180
Figure 8.19: Scatterplot showing intra-observer variation (observer 1) for axial translation/IVD height (full flexion position) 181
Figure 8.20: Scatterplot showing intra-observer variation (observer 1) for axial translation/IVD height (full extension position) 182
Figure 8.21: Scatterplot showing inter-observer variation (observers 1,2,3) for axial translation/IVD height (neutral position) 183
Figure 8.22: Scatterplot showing inter-observer variation (observers 1,2,3) for axial translation/IVD height (full flexion position) 184
Figure 8.23: Scatterplot showing inter-observer variation (observers 1,2,3) for axial translation/IVD height (full extension position) 185
Figure 8.24: Diagram showing comparison of assessment of axial translation by measurement of the change in mean IVD height and change in distance between centre points 188

Chapter Nine

Figure 9.1: Example of a typical flexion/extension ICA graph 196
Figure 9.2: Example of a typical lateral flexion ICA graph 197
Figure 9.3: Examples of variation in the location of the mid-sequence neutral position 202
Figure 9.4: Examples of the initial "true" neutral and estimated mid-sequence neutral 203
Figure 9.5: Examples of the location of maximum and minimum values for IVAs throughout the motion sequence (sagittal plane) 214
Figure 9.6: Examples of the location of maximum and minimum values for IVAs throughout the motion sequence (coronal plane) 215

Figure 9.7: Examples of change in IVD height across the motion sequence 226
Figure 9.8: Diagram showing disparity between measurement of "true" IVD height and distance between vertebral templates 228
Figure 9.9: Examples of the location of maximum and minimum values for IVD height throughout the motion sequence (sagittal plane) 231

CONTENTS OF TABLES

Part I

Chapter One

Table 1: Absorbed radiation dose 25

Chapter Four

Table 4.1: Checklist for diagnosis of clinical instability in the lumbar spine (White & Bernhardt 1999) 80

Part II

Chapter Five

Table 5.1: Example of time differences between Isotrak and DVF measurements 125

Table 5.2: Agreement between DVF measurements 1 and 2 of lumbar flexion angle for each subject 129

Table 5.3: Comparison of lumbar flexion range as measured by DVF and Isotrak 130

Chapter Six

Table 6.1: Comparison between DVF and surgical assessment of fusion 133

Part III
Chapter Eight

Table 8.1:	Results of calibration study showing mean discrepancy and SD of angle measurements for each of the five selected regions of the grid and all regions combined	159
Table 8.2:	Intra-observer variation for five repeated measurements of vertebral movement relative to neutral (observer 1) in the sagittal plane	162
Table 8.3:	Intra-observer variation for five repeated measurements of vertebral movement relative to neutral (observer 1) in the coronal plane	163
Table 8.4:	Inter-observer variation between 4 observers for two images (full flexion/full extension) in the sagittal plane	165
Table 8.5:	Inter-observer variation between 3 observers for two images (full flexion/full extension) in the sagittal plane	166
Table 8.6:	Inter-observer variation between 4 observers for two images (RLF/LLF) in the coronal plane	167
Table 8.7:	Inter-observer variation between 3 observers for two images (RLF/LLF) in the coronal plane	168
Table 8.8:	Mean disc and vertebral body dimensions measured from 20-35 year old male cadavers (Bogduk 1997)	187
Table 8.9:	Correlation between measurement of change in mean IVD height and change in centre point distances for levels L1-L5 for all subjects (sagittal plane only)	188
Table 8.10:	Radiation dosage and screening times for all ten subjects	190
Table 8.11:	Distribution of individual dose-area products (DAPs) for adult patients at a random sample of 20 English hospitals with NRPB reference values of DAP (Based on Shrimpton et al 1986 and Shrimpton et al 1999)	191

Chapter Nine

Table 9.1:	Summarised IVA data for flexion/extension. Mean ranges for all ten subjects across both screenings	193
Table 9.2:	Summarised IVA data for lateral flexion. Mean ranges for all ten subjects across both screenings	195
Table 9.3:	Summarised translation data for flexion/extension. Mean values for all ten subjects across both screenings	199
Table 9.4:	Comparison of representative *in vivo* angles for combined intervertebral flexion/extension	207
Table 9.5:	Comparison of representative *in vivo* angles for intervertebral flexion	208
Table 9.6:	Comparison of representative *in vivo* angles for intervertebral extension	209
Table 9.7:	Comparison of representative *in vivo* angles for intervertebral RLF	210
Table 9.8:	Comparison of representative *in vivo* angles for intervertebral LLF	211
Table 9.9:	Total numbers of paradoxical ICAs greater than 3 degrees. Data are for flexion/extension, RLF and LLF combined	219
Table 9.10:	Percentage of maximum and minimum values for translation with respect to their location within the motion sequence (L1-L4)	222
Table 9.11:	Percentage of maximum and minimum values for translation with respect to their location within the motion sequence (mean values across all levels)	223
Table 9.12:	Mean translation displacement values for each segment (as a percentage of the average superior vertebral body depth) between full extension and full flexion	224
Table 9.13:	Mean IVD heights for all subjects and lumbar levels across the entire motion sequence (as a percentage of the superior vertebral body height)	227
Table 9.14:	Comparison of maximum range of IVD height with flexion/extension range	229

PART I

BACKGROUND STUDIES

INTRODUCTION

Chronic back pain is, in terms of numbers, a problem of immense proportions. The Department of Social Security have shown that Sickness and Invalidity Benefits paid for back pain incapacity's had risen to 106 million days for the year 1993-94. In economic terms this amounts to around £1.4 billion. The cost to the NHS in services for back pain during 1993 was approximately £480 million, and in terms of lost production to industry, the cost was estimated at £3.8 billion for 1993 (CSAG, 1994). Despite the widespread nature of the problem, however, it is still estimated that up to 85% of back pain patients cannot be given an accurate diagnosis (Moffett & Richardson, 1995). Addressing this diagnostic problem relies, to some extent, on improving our understanding of the mechanics of the spine and how disorders might reveal themselves during spinal motion. Part 1 of this thesis will begin by considering the issues and research questions underpinning the study of spinal motion. There then follows a review of the various methods of measuring spinal movements, especially those concerned with dynamic imaging. The basis of some commonly used kinematic indices will also be discussed before considering key studies, both *in vitro* and *in vivo*, of intervertebral motion.

RESEARCH QUESTIONS

The universal question at the heart of almost all work on spinal studies and that, which, it would seem, has yet to be adequately answered, is the following:

Is there any relationship between spinal mechanics and back pain?

If, as most workers in this field would intuitively agree, there is a relationship, then the logical extension of this argument would be to establish if a basis exists for mechanical assumptions about the causes of back pain. To take this notion a step further into the realms of practicality, the more pertinent question to ask might be:

Is there a foundation for relating the symptomatology of spinal disorders to measurable intersegmental kinematics?

In order to answer this question, however, we must first define what constitutes "normal" kinematic variation. In this sense is meant the differences that can be seen in kinematic parameters not associated with symptoms. An "abnormality" not directly related to the production of symptoms makes little clinical sense, unless it represents a profound time-dependent change in function likely to result in symptoms at a later date. Thus by having a clinical starting point we are forced into the assumption that measurable changes in individuals not causally associated with pain or other symptomatology must designate mechanical normality. As an example, the advent of magnetic resonance (MR) technology has shown the presence, in the lumbar spine, of herniated nucleus pulposus (HNP) in up to 70% of asymptomatic subjects (Boos et al., 1995). This condition was previously thought to be invariably symptomatic. These findings have stimulated a more thorough investigation of the mechanisms of pain production (Bogduk, 1991; Olmarker & Myers, 1998; Skouen et al., 1993). In light of this it becomes even more important to establish kinematic variability in the asymptomatic population before attempting to relate changes to those with symptoms. It is this inherent biological variability, which hampers the verification of indices of motion. It is also vitally important that, for clinical use, experimental data of this kind should be shown to have practical value beyond that of research interest (Adams, 1999). The clinical arena requires a more pragmatic approach, especially when experimental data may be relied upon to determine surgical intervention. As a result of these demands clinicians tend to ask questions along the lines of:

What is the "normal" motion of the spine and how does this differ from the spinal motion achieved by those in pain?

The inference here is that any differences might help establish the cause of the pain and/or, perhaps more importantly, suggest possible interventions to reduce or abolish it. In other words: the resolution of a diagnosis. Unfortunately we have no way of knowing if any kinematic changes capable of being demonstrated are related to the cause of back pain or to its effects. Back pain is a symptom not a disease (Waddell, 1998) and there are many patients with back pain in whom no anatomical or, as far as we know, mechanical abnormality exists.

Applying sophisticated methods of dynamic motion analysis to a homogenous back pain population is more likely to generate confusion than understanding. Pearcy and colleagues (Pearcy et al., 1985), using biplanar radiography, considered two groups of back pain patients in comparison with asymptomatic controls. One group had low back and/or buttock pain and the other low back pain and associated sciatica with nerve tension signs. These authors were able to determine the three-dimensional intervertebral ranges of motion in the lumbar spine for both primary and coupled movements. They concluded that patients with low back pain alone had reduced primary motion in the lower lumbar segments together with an increase in coupled movements. This was attributed to asymmetrical muscle splinting. Pearcy's group was forced to concede that their biplanar radiographic technique was "not capable of providing clinically useful information for individuals with undiagnosed back pain of non specific origin". This study, however, examined only intervertebral ranges of rotational motion from static plain-film X-rays. Furthermore, the protocol used was one of erect voluntary bending in the sagittal plane. Pearcy's investigation had the disadvantage of having no clear hypothesis behind it, other than to obtain comparative data from a group of chronic low back pain sufferers. A more recent but similar study applied videofluoroscopy to a comparable cohort of back pain patients and asymptomatics (Okawa et al., 1998). Again, using an active, standing protocol these authors attempted to obtain data on intervertebral movement characteristics between patients and subjects. Their hypothesis was that abnormal movement should be present throughout the lumbar spine in the presence of an unstable segment and that segmental motion may be affected by pain. Once again, a lack of clarity in the development of the research question leads to serious flaws in design. In the first instance,

instability, in this study, was defined as degenerative spondylolisthesis a premise which, to say the least, is controversial (Bogduk, 1997; Kauppila et al., 1998; Mullholland, 1999; Pearcy & Shepherd, 1985; Penning & Blickman, 1980; van Akkerveeken, 1999). Since the patient group in Okawa's study (Okawa et al., 1998) was subdivided into a cohort with undiagnosed chronic low back pain and a cohort with degenerative spondylolisthesis, it was not made clear if the spondylolisthetic group were even symptomatic or not.

In addition to the patient/subject selection problems discussed above, the voluntary motion protocol adopted by Okawa's group (Okawa et al., 1998) was also flawed. Their investigation allowed movement from neutral standing to a point in forward flexion where the upper lumbar spine disappeared from view and back to neutral. Given these restrictions, only submaximal flexion motion could be considered with no useful data on true lumbar extension. The second part of the hypothesis, that pain might affect segmental motion, was also ill conceived. It is well known that pain alters active spinal movement and that, generally, it reduces the range of motion. As with the Pearcy study (Pearcy et al., 1985), without some knowledge regarding the origin of the pain, however, it is unlikely that useful conclusions can be drawn from changes in movement patterns. Not surprisingly, these authors were unable to show significant differences between the chronic pain patients and asymptomatic volunteers. Nevertheless, this paper does represent a growing trend towards *in vivo* spinal measurement throughout the motion sequence.

In direct comparison with both the Pearcy (Pearcy et al., 1985) and Okawa groups (Okawa et al., 1998), the recent work of Kaigle and colleagues (Kaigle et al., 1995; Kaigle et al., 1997) employed an intervertebral motion device on anaesthetised pigs with surgically induced disruption to the disc, facet joints, facet capsules and transverse processes. Using a controlled, passive, flexion/extension protocol these authors were able to demonstrate subtle changes in kinematic behaviour beyond that of changes in range. The differences between these approaches are, of course, quite marked. Pearcy's work applied an uncontrolled clinical protocol to human individuals with no established structural abnormalities of the spine or commonality other than that of perceived pain. Kaigle's approach was one of a controlled experimental nature involving known structural abnormalities common to all groups of subjects. Needless to say, this experimental protocol on a porcine model could never be achieved using live human subjects. The

challenge in acquiring clinically relevant data from human *in vivo* studies is, therefore, in the ethical control of subject/patient variables.

Biomechanical research involving non-specific back pain is, as Pearcy (Pearcy et al., 1985) pointed out, unlikely to yield significant information. Given this, it is perhaps more valuable to limit biomechanical investigations to those patients whose clinical presentations are suggestive of loss of holding element integrity. In this manner, the appropriate application of kinematic measurement techniques is, in some ways, co-dependant on improvements in back pain classification. The indiscriminate use of *in vivo* biomechanical investigations, as with most medical ones, is not only *bad medicine* but likely to dilute the clinical effectiveness of the assessment itself.

Under these circumstances a more focused approach is necessary. The study of back movement itself is too comprehensive an issue and its characteristics dependent on many factors other than the integrity of passive elements. Voluntary motion, by necessity, requires an integrated neuromuscular system. The neural control of muscular contraction is well known for its high level of plasticity. The variability in muscle coactivation of the trunk, for example, under similar loading conditions aptly illustrates this point (Cholewicki et al., 1997). It would more useful, perhaps, to concentrate our attention on the integrity of those restraining tissues that contribute significantly to intersegmental displacements. Effort should be directed at establishing methods which can challenge these structures *in vivo* and defining kinematic parameters sufficiently sensitive to demonstrate any loss of stiffness or dysfunction arising. A useful refinement of the clinical question might then be to ask:

How do the passive tissues, on which much of spinal integrity depends, behave under controlled conditions in both symptomatics and asymptomatics?

The neutral zone (NZ) concept proposed by Panjabi (Panjabi, 1992b) is one such notion that, theoretically, has an application in spinal kinematics. Unfortunately the NZ is, at present, defined only for *in vitro* specimens. However, Kanayama and colleagues (Kanayama et al., 1996), using a cineradiographic protocol of lumbar spine flexion/extension, have suggested that the transition between the NZ and the elastic zone (EZ) might be approximated by the phase-lag seen between segments during motion. This was defined as an intersegmental motion delay occurring at adjacent vertebrae. The

authors hypothesised that voluntary flexion/extension from the erect posture will exert bending moments on each segment sequentially from the upper to lower lumbars. Since the NZ is a region of low resistance it follows that the bending moment through the superior segment is unlikely to be transmitted to the inferior segment until its own EZ has been reached. Thus the intersegmental motion lag should, by this reckoning, approximate the magnitude of the *in vitro* NZ. These values were taken from the original cadaveric studies of Yamamoto, Panjabi and others (Yamamoto et al., 1989) and were typically around 2 degrees for the L4/5 segment and 3 degrees for the L5/S1 region. In contrast, Kanayama's findings suggested a 6-degree and 8 degree phase lag for L4/5 and L5/S1 respectively. This, therefore, represents a considerable delay in moment transmission. Motion of the inferior segment does not occur until well after the value for the *in vitro* EZ has been reached. The authors conclude, quite logically, that intersegmental motion is determined more by *in vivo* factors such as trunk musculature, than by the mechanical properties of the isolated FSU. Compression and the effect of surrounding muscles must stiffen the disc such that, even when the upper segment has entered the EZ, resistance to motion is sufficient to prevent the lower segment from starting to move. Also, as pointed out by Ogon and colleagues (Ogon et al., 1997b), the disc and other tissues are viscoelastic and thus their resistance increases with the rate of loading. As a result, fast dynamic intervertebral movements, even within the neutral zone, will be met with considerable opposition. These factors, perhaps, argue for passive procedures carried out in a steady, controlled and unloaded manner, to allow restraining tissues to show their "true colours". A recent paper on the radiographic evaluation of spondylolisthesis also provides evidence for protocols' which reduce pre-load to a minimum (Wood et al., 1994). This study used lumbar flexion/extension films in both the standing and lateral recumbent positions. These procedures were carried out on 50 spondylolisthesis patients, 6 of whom were asymptomatic, and 10 age-matched controls. The "spondylolisthesis" group were made up of isthmic, dysplastic, degenerative and post-laminectomy types and the authors reported no statistical difference in motion between the degenerative and isthmic/dysplastic varieties. Thirty-one individuals (62%) displayed abnormal translation suggestive of instability (Boden & Wiesel, 1990). Of this group, 18 (58%) demonstrated abnormal motion only when in the lateral recumbent position.

These findings support the view that in order to challenge the mechanical integrity of the living spine, the individual should be unloaded in compression and under as little

voluntary muscular control as practically possible. Thus screening lumbar spine motion under passive, recumbent conditions would appear to be a logical approach for the thorough investigation of the mechanical behaviour of these tissues *in vivo*.

CHAPTER 1

SPINAL MEASUREMENTS

1.1 INTRODUCTION

Information regarding the detailed movements of the living human spine is, without question, important (Goel et al., 1985; Yamamoto et al., 1989). The way this information is obtained is governed, largely, by the anatomical relationship of the spinal column to the body surface. The greatest part of the spine lies deep to the integument, virtually inaccessible to direct measurement during life. Related research is broadly divided, therefore, into back surface measurements, which attempt to infer spinal motion, and those techniques that endeavour to directly measure vertebral displacements. Direct measurement of segmental motion has been successfully achieved by the attachment of various devices, such as Steinmann pins and Kirschner wires, directly to the bony substance of the vertebrae (Gunzburg et al., 1991; Kaigle et al., 1992b; Liu et al., 1997; Pope et al., 1986; Steffen et al., 1997). As expected, however, these methods have not been widely used and, in the clinical setting, have serious limitations.

1.2 SURFACE MEASUREMENT

Although spinal movements have interested researchers for over a century (Weber & Weber, 1836) the practice of measuring lumbar spine mobility first gained prominence in the assessment of ankylosing spondylitis (Bennett & Burch, 1967; Dunham, 1949; Macrae & Wright, 1969). Initially these were simple one-dimensional techniques such as the skin-distraction of Schober (Macrae & Wright, 1969; Schober, 1937) or plumb-line methods of the type employed by Moll and Wright (Moll & Wright, 1971). As interest in back mobility in relation to pain has advanced, so too has the sophistication of the methods (Stokes & Frymoyer, 1987). The use of inclinometers and goniometers has allowed measurement in two dimensions providing gross range of motion data (Anderson & Sweetman, 1975). They have proved useful in providing reference values and demonstrating range of motion changes associated with age and sex (Burton & Tillotson, 1988). Skin-surface techniques such as these however, have been limited chiefly to lumbar sagittal movements, do not assess dynamic mobility and cannot address translatory motion or rotations in other planes (Pearcy, 1986). Furthermore, using radiography to

assess surface measures has exposed the shortcomings of these indirect techniques. In a cross comparison study of several clinical measures of lumbar mobility with biplanar radiography, Portek and colleagues (Portek et al., 1983), could show little correlation between either the surface techniques (inclinometer, skin distraction and plumb line) or between surface techniques and radiographic measurement. In conclusion the authors suggest that current clinical methods of assessing lumbar motion "give indices of back movement which are affected by factors such as thoracic movement, hip flexibility and skin extensibility and do not reflect true spinal movement".

Recently several authors have used a three-dimensional surface measurement device, the electromagnetic 3SPACE ISOTRAK, to study lumbar spine mobility and commend it as an acceptable and effective clinical tool (Dolan & Adams, 1993; Hindle et al., 1990; McGill & Brown, 1992; Pearcy & Hindle, 1989).

In light of the preceding discussion it should not be surprising, therefore, that radiographic methods have been, since the early part of this century, the mainstay of kinematic data gathering on the human spine (Gianturco, 1944; Tanz, 1953; Todd & Pyle, 1928).

1.3 RADIOGRAPHIC MEASUREMENT

To this day imaging methods employing X-rays are still generally acknowledged as the definitive approach to obtaining detailed information on spinal motion (Adams & Dolan, 1995; Pearcy et al., 1984; Portek et al., 1983). In particular bi-planar or stereo radiography is suggested as the only accurate non-invasive means of measuring three-dimensional vertebral motion (Pearcy et al., 1984). Roentgen stereophotogrammetric analysis (RSA) has been generally regarded as an accurate method for the measurement of spinal kinematics (Axelsson et al., 1992; Leivseth et al., 1998; Selvik, 1989; Selvik, 1990). This technique involves using two angled X-ray tubes to perform simultaneous exposures of the spine on two uniplanar radiographs. Measurements from these films allow the assessment of three-dimensional translatory and rotational motions by tracing the trajectories of implanted metal markers. The accuracy of RSA is generally accepted but its invasive nature has limited its spinal use to the post-surgical assessment of fusion (Johnsson et al., 1990; Johnsson et al., 1992).

The majority of techniques employing plain-film X-rays attempt to measure displacements of each vertebral segment with respect to its inferior neighbour. This is achieved either

graphically, using superimposed serial radiographs of the type used by Penning (Penning et al., 1984), or by digitisation of points marked on these radiographs and their subsequent computer-based computation, as employed by Pearcy (Pearcy et al., 1984), for example. Radiography, however, is not without risk. This risk was not, perhaps, fully appreciated by those involved in the first attempts to quantify spinal motion. As evidence of the deleterious effects of X-rays began to grow, however, their use became more selective. The radiation dosages associated with static radiographs restrict the technique to clinical situations where the risk\benefit ratio is usually self-evident. In terms of the clinical investigation of low back pain, plain-film radiography, especially, is thought to offer very little in exchange for significant risk (Davies et al., 1993; Lewis, 1991; Quinnell & Stockdale, 1983). This concern for radiation exposure also severely limits the study of incremental spinal motion. Given these constraints, the most that can be measured from static radiographs are limits and ranges of movement. This information, though valuable, is incomplete and may not be sufficient to characterise any deviation from normal motion that might be associated with spinal disorders (Hindle et al., 1990).

1.4 FLUOROSCOPY

In the early 1950s, the image intensifier was developed and, coupled with cinephotography, allowed the capture of spinal movement onto cine film (Teves, 1955). The "primitive" technology of the time was associated with large doses of radiation and this constrained the spinal applications to assessment of cervical spine motion (Fielding, 1957; Jones, 1960), where lower penetrations were required compared with, for example, the lumbar spine. With the substitution of cineradiography for videofluoroscopy, the possibilities for investigation widened. This was partly due to the more immediate nature of the video medium compared to cinephotography, but, probably more importantly, to the much greater versatility and robustness of magnetic-tape storage.

Radiography, videofluoroscopy and other forms of medical imaging were developed not solely as research tools for objective measurement, but as a means to obtain qualitative clinical information via simple visual inspection of the images. Their use in the field of kinematics has been limited by the appropriateness of the technique as well as by ethical considerations. As the spine is a complex structure some authors have expressed concern about using measuring techniques that are on a much lower level of sophistication than the structures they purport to investigate (Aspden, 1992; Hindle et al., 1990). In other words,

these authors have touched on the dilemma of studying a three-dimensional dynamic system using two-dimensional static methods. Radiographs, and indeed fluoroscopy, are essentially two-dimensional and are therefore of limited use in the study of three-dimensional motion. Although the work of Pearcy et al (Pearcy et al., 1984), in utilising biplanar radiography, has been successful in representing three-dimensional displacements, the method is cumbersome and radiation intensive.

Medical imaging has not been idle since the advent of fluoroscopy in the 1950s. In fact the development of magnetic resonance (MRI), ultrasound and stereophotogrammetry have largely superseded the radiograph and the fluoroscope in terms of clinico-pathological data collection, as well as being less hazardous to the patient. Advances in the technology of computed tomography (CT) and MRI have brought unrivalled image quality to both clinician and bioengineer and have provided a high degree of flexibility in image enhancement and subsequent computational power. Nevertheless, from an engineering viewpoint, projected images of an entire structure, such as those obtained by fluoroscopy, are better suited to dynamic analysis than the isolated slices or surfaces of structures seen on CT, MRI and ultrasound scans. Quite apart from this is the apparent reluctance of these modalities to provide the real-time imaging in both the upright, weight-bearing, and recumbent conditions required for the full investigation of spinal motion. It is anticipated, however, that future versions of these devices will be capable of rapid imaging within flexible, open-access environments.

What becomes clear from this discussion is the need for an imaging method that combines the real-time projected images of a fluoroscope, at minimum radiation risk, with the computational power of computer-assisted imaging. A logical approach to this problem suggests the use of computer enhancement and analysis of digitised fluoroscopic images. Image processing is a rapidly expanding field and one that provides many powerful tools for the storage and analysis of X-ray based data (Moores, 1987).

1.5 DIGITIZED VIDEOFLUOROSCOPY

With the growth of computer-based image processing the study of lumbar spine motion using videofluoroscopy was at hand. In an effort to embrace this technology Breen and colleagues (Breen et al., 1989) described a method for the acquisition and processing of fluoroscopic images in the study of spine kinematics. These authors were able to

demonstrate the feasibility of obtaining useful lumbar motion sequences with less dosage than that associated with a single plain-film X-ray of the same region. The technique became known as digitised videofluoroscopy (DVF) and was an attempt to provide quantitative analysis of, primarily lumbar, intervertebral motion throughout the bending range of the trunk (Breen et al., 1988; Breen et al., 1989). It involves the digitisation of low-dose fluoroscopic images of the moving lumbar spine and their subsequent analysis. Patients and subjects flex, extend and laterally flex the trunk during imaging. This can be achieved actively in the upright positions or, more recently, passively in the recumbent position.

Once captured the images are then amenable to computer-assisted measurement techniques. This largely entails the marking of co-ordinates on the vertebral corners and end-plates of sequential images thus providing graphical and numerical data on intervertebral rotations and translations over time (Figure 1.1).

Equipment used at the inception of DVF included a Thompson CGR X-ray machine with a 9" diameter image intensifier. This system allowed the capture of fluoroscopic sequences of lumbar spine motion from subjects in a vertically aligned seated position. Minimum stabilisation was employed to allow natural active motion. A wooden seat frame, however, was provided to stabilise the sacrum during lumbar extension. The unavoidable use of ionising radiation in this method has, of course, raised issues of patient safety. In answer to this Breen undertook a dosage study and determined absorbed radiation dosage values for a typical patient screening sequence (Breen, 1991). These values and, by way of comparison, the dosage associated with plain-film X-rays are shown in Table 1. This reveals the much-reduced X-ray exposure associated with DVF when contrasted with a standard plain-film view of the same region.

TABLE 1

ABSORBED RADIATION DOSAGE

TYPICAL DVF SCREENING (Approx. 10 seconds per view)		**TYPICAL PLAIN-FILM**	
VIEW	ABSORBED DOSE (mGy)	VIEW	ABSORBED DOSE (mGy)
Lumbar A/P	2.87	Lumbar A/P	20
Lumbar Lateral	12.61	Lumbar Lateral	50

A/P = anterio-posterior

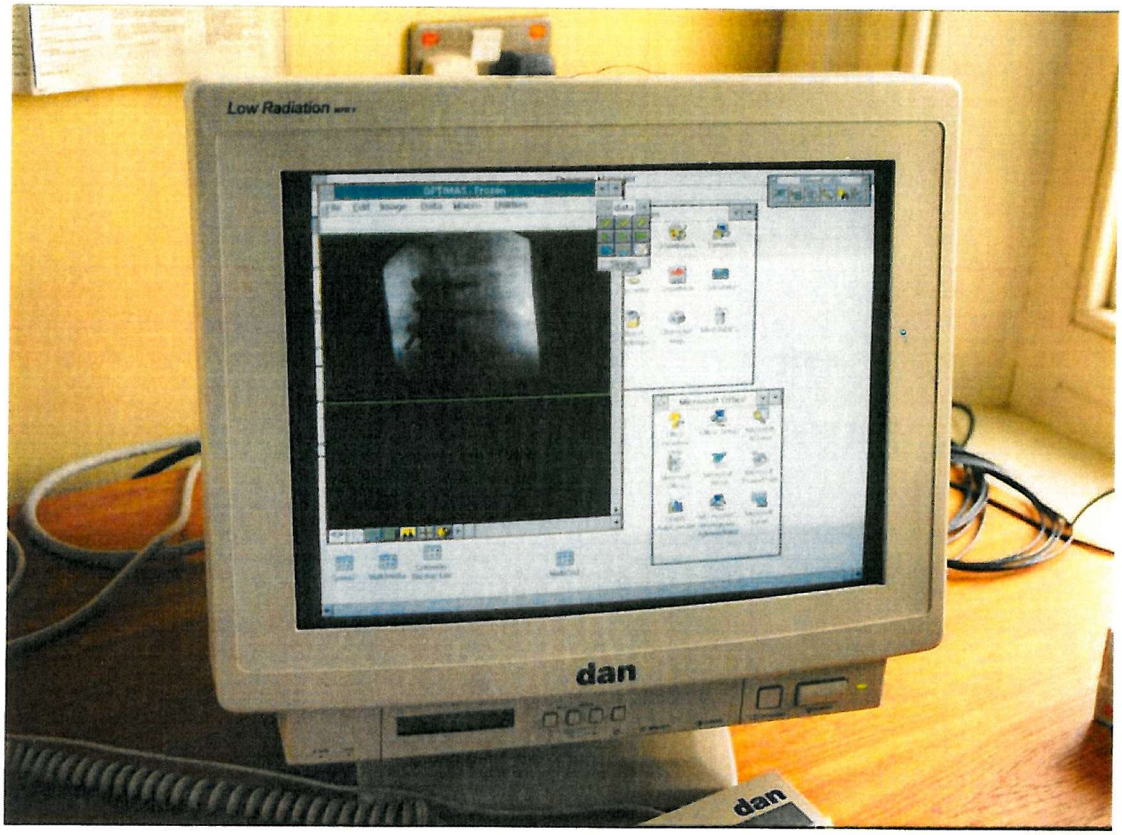
Images from this system were recorded on low-band U-Matic videotape and subsequently digitised onto a computer. The hardware used for this early work comprised a PDP11 mini-computer with 80Mb hard disc and a high-resolution colour monitor. Image processing was carried out using commercially available software capable of automatic frame grabbing, contrast enhancement and 2D-geometric manipulation of screen co-ordinates. With this prototype system Breen and colleagues established accuracy and reliability by means of a calibration model (Breen et al., 1989). Errors in determining intervertebral angles (IVAs) were found to be of the order of +/- 1 degree and for instantaneous centres of rotation (ICRs) +/- 5mm. The concept of ICRs will be discussed in much greater depth in Chapter 2 when dealing with kinematic indices.

One of the major benefits of DVF is in the provision of information over the whole range of movement and not merely at the extremes of range. It has long been supposed that the initial and final position plain-film radiographs of trunk bending also represent the extremes of intervertebral motion. Using DVF, however, this has been shown not to be the case (Breen & Allen, 1996). It is quite possible for vertebral segments to undergo their largest rotations within the trunk range and not simply mirror trunk motion. Hence excessive or aberrant intervertebral motion may be missed if only extreme positions are evaluated and DVF is eminently suited to demonstrate such phenomena.

The technique of DVF also formed the basis of work carried out by other researchers into the application of videofluoroscopy in studying vertebral motion (Cholewicki et al., 1991). The authors of this paper concerned themselves largely with the minimisation of optical distortions and digitising errors but did undertake a small, *in vivo*, study of angular motion in the lumbar spine. A similar, more recent, study concerning optical distortion correction in videofluoroscopy looked at its application in the measurement of knee-joint kinematics (Baltzopoulos, 1995). Currently, a group from Japan have attempted to characterise the pattern of lumbar spine motion in asymptomatics and patients with chronic low back pain using videofluoroscopy (Okawa et al., 1998).

FIGURE 1.1

IMAGE PROCESSING ENVIRONMENT



The use of video and TV technology has not, however, rendered the technique of cineradiography obsolete. The recording of X-ray motion sequences onto photographic film is, although perhaps less versatile, as equally valid a method as that of electronic storage. Dutch researchers, from the University of Limburg, have successfully employed cineradiography in the study of cervical spine motion (van Mameren et al., 1992). By projecting images onto drawing paper and using an X-ray digitising tablet the authors were able to record co-ordinates of various anatomical landmarks into computer memory. These co-ordinates were then used in the calculation of ICRs and IVAs. The method so described is meticulous and rigorous and capable of providing reproducible data of known variability. Nevertheless it is both laborious and unwieldy and, in its present form, unlikely to fulfil the need for rapid clinical data gathering.

The combination of low-dose fluoroscopy and computer-based image processing would appear to be, at present, the most solid foundation on which to build the investigation of *in vivo* joint kinematics.

Advancements in radiographic science, such as pulsed X-ray in synchrony with image acquisition, should ensure a progressive reduction in absorbed radiation dose. Improvements in the sensitivity of image intensifiers and shortening of their persistence times will enhance image quality and further safeguard patients from X-ray hazards. Expansion into the digital domain and, by means of bi-planar fluoroscopy, into three-dimensional analysis together with sophisticated image processors should greatly improve accuracy. These evolutionary changes, although by no means a reality as yet, should, together with improved knowledge of spinal function, ensure the future of digitised fluoroscopy for many years to come. In its present form, however, digitised fluoroscopy is still capable of generating much valuable data concerning normal and abnormal spinal motion, existing kinematic parameters and aid in the development of new indices of aberrant motion.

CHAPTER 2

KINEMATIC INDICES

2.1 INTRODUCTION

Spinal kinematics concerns itself, chiefly, with the description of motion of vertebrae with respect to each other and their linkages (disc, ligaments and joints) (Stokes et al., 1981). Although strictly speaking vertebrae are not rigid bodies, for the purposes of gross displacements they can readily be assumed to be. By and large we are not too concerned about the vertebral bodies themselves but rather what their motion tells us about their linkages or holding elements. In this respect spinal kinematics must rely, to some degree, on knowledge of the mechanical behaviour of the structures in question. In other words, although kinematics is strictly the study of motion, the causal relationship between the structural properties of the tissues and spinal dynamics must always be borne in mind. This aspect will be discussed further when considering the relative merits of *in vivo* and *in vitro* studies in chapter 3.

In 1973 Panjabi proposed a general mathematical model of the spine, which related the known properties of ligaments, disc and the effects of ribs and muscles to the expected motion of vertebrae (Panjabi, 1973). This was followed up, a few years later, by an exhaustive review of the kinematics of the spine including a comprehensive summary of intervertebral ranges of motion (White & Panjabi, 1978). Later still, Panjabi and colleagues (Panjabi et al., 1981) further refined their model for defining the three-dimensional orientation of vertebral bodies. These authors proffered a simple co-ordinate system for labelling the spatial relationships of vertebral segments that has now become the established nomenclature for spinal kinematics (Figure 2.1). Since this time several procedures and techniques have been described to determine the kinematic parameters of body parts, including the spine (Dietrich et al., 1991; Dimnet, 1980; Pittman et al., 1992). However complex these theoretical models have become, the practical measurement of intervertebral movements *in vivo* is nevertheless required in order to validate these models and fully describe spinal motion. For all intents and purposes actual *in vivo* measurements of vertebral bodies are limited to the basic elemental motions of rotations and translations. In terms of describing these motions, no parameter in spinal kinematics has received more attention than the instantaneous centre of rotation (ICR).

2.2 INSTANTANEOUS CENTRE OF ROTATION (ICR):

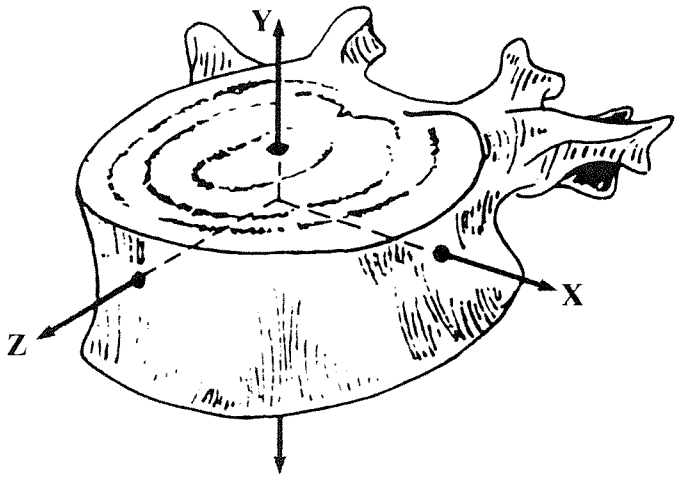
2.2.1 ENGINEERING CONSIDERATIONS

The ICR is a kinematic parameter, which together with the magnitude of rotation fully defines the planar motion of a rigid body. In engineering terms the motion of a body, at any instant in time, irrespective of the overall complexity of the entire motion sequence, can be described by a rotation about a certain point called the instantaneous centre (Tao, 1967). White and Panjabi have described it as follows: "When a rigid body moves in a plane, at every instant there is a point in the body or some hypothetical extension of it that does not move. An axis perpendicular to the plane of motion and passing through that point is the instantaneous axis (centre) of rotation (IAR) for that motion at that instant" (White & Panjabi, 1990). The use of ICRs in the investigation of joint kinematics is not new (Fick, 1904). Many authors have utilised this concept in studying human joint movements over such diverse areas as the knee, elbow and temporomandibular joints (Frankel et al., 1971; Grant, 1973; Meek et al., 1975; Morrey & Chao, 1976). Its first application in the field of vertebral kinematics appears to be that of Rosenberg (Rosenberg, 1955), who applied it to serial lumbar radiographs of thirty subjects in a preliminary attempt to establish its "normal" locations. Since then it has gained much favour as a kinematic parameter in the study of spinal motion, especially in regard to the cervical and lumbar regions.

The ICR has attracted particular interest as a means to conceptualise observed intervertebral motion. In this respect the ICR is considered as a parameter of quality of motion containing information on the rotary as well as the translatory component between two adjacent vertebrae (van Mameren et al., 1992). In biomechanical terms, ICRs can also be considered as axes about which, the spinal musculature exert their moments in bringing about movement (Gracovetsky, 1988; Haher et al., 1992). Furthermore, "abnormally" displaced ICRs may reflect mechanical derangement or instability. Therefore, defining the locations of ICRs could provide valuable diagnostic information (Pearcy & Bogduk, 1988).

FIGURE 2.1

THREE-DIMENSIONAL CO-ORDINATE SYSTEM APPLIED TO VERTEBRAE



The term instantaneous axis of rotation (IAR), reflecting the three-dimensional nature of the joint, is preferred by some authors (Amevo et al., 1991a; Amevo et al., 1991b; Amevo et al., 1991c; Haher et al., 1992; Pearcy & Bogduk, 1988). For others, the "ICR" has become the term of choice (Cossette et al., 1971; Soudan et al., 1979; van Mameren et al., 1992). The controversy over terminology would seem to be one, largely, of personal preference. Given that most of the analyses are two-dimensional, the term lAR, with its three-dimensional connotations, would appear inappropriate. In those studies where the three-dimensional information is known, (Pearcy's 1985 work with bi-planar radiography for example (Pearcy, 1985)), the use of the term IAR perhaps is more appropriate. Whatever term is employed it cannot be overemphasised that, in this case, the index is being applied to projections or images of the spine and thus can provide only inferential data regarding the actual structures. It is no bad thing, then, that terminology, as applied to the image, should acknowledge and remind us of the true three-dimensional nature of the original. Therefore, since the IAR or ICR applies only to uniplanar motion, both terms are essentially redundant as vertebral, or any other joint, motion is never truly uniplanar.

There does, however, exist a relationship between the two concepts. The ICR is the point at which the IAR crosses the plane of motion (Soudan et al., 1979). In planar motion the axis will, of course, be perpendicular to the plane. For practical reasons it must be accepted then that, in the application of ICRs, a potentially complex motion is described in a simplified way (White & Panjabi, 1990). It must also be borne in mind that the IAR or ICR is a hypothetical concept not an absolute measure. Its location represents an axis or a point about which a vertebra, or other body, could be rotated to produce the displacement demonstrated on the initial and final radiographs, at a given instant of time. The actual motion between the extremes may be at great variance to that represented by the ICR (Rosenberg, 1955).

Where there is sufficient data to fully describe the complex three-dimensional motion of human joints, a more appropriate index, such as the helical axis of motion (HAM), might be preferred (Dimnet & Guinguand, 1984; Woltring et al., 1986; Woltring et al., 1985). This is achieved by describing the motion of a rigid body in terms of helical or screw motion. Helical motion is where a body rotates around and simultaneously translates along the same axis (Maxwell, 1960). The three-dimensional motion of any rigid body from one defined position in space to another can be precisely described using the helical axis of motion. This axis is the three-dimensional counterpart to the two-dimensional ICR (Panjabi et al., 1981). The precision of the HAM index, though, is far outweighed by its conceptual complexity, which at the present time prevents its use with regard to spinal motion, in an *in vivo* or clinical setting (White & Panjabi, 1990). Quite apart from this, of course, is the significant limitation imposed by the difficulties in obtaining three-dimensional data. For the time being, at least, it would appear that the ICR, despite its shortcomings, continues to be a widely used parameter in joint kinematics. Its popularity, perhaps, is due to its inherent potential for addressing rotational and translational motion together. Clinicians have long been suspicious that translational movements of vertebral segments are an important feature for determining spinal instability (van Akkerveeken et al., 1979; Weiler et al., 1990). Hence the ability to represent translation and rotation in one measurement makes the ICR an attractive index for kinematic analysis. It follows, therefore, that the location of the ICR *in vivo* might help differentiate "abnormal" or excessive motion from that which is considered normal. By plotting ICRs incrementally throughout a range of motion it is possible to describe a path of ICRs, or centrode, for flexion/extension or sidebending. How far apart these ICRs are from each other and

where, in relation to the anatomy, they are located is thought to tell us something about the mechanical behaviour of each segment. These centrodes, or loci, have been used by several authors on both cadaveric specimens and *in vivo* subjects (Gertzbein et al., 1984; Gertzbein et al., 1985; Ogston et al., 1986; Seligman et al., 1984). Pure sagittal plane rotation, for example, would result in a single centre of rotation, (i.e. giving a centrode length of zero). Ogston (Ogston et al., 1986), realised that the greater the translation, the greater the centrode length. They further suggested that an inconsistent distribution of the proportional amounts of translation and rotation, corresponding to mechanical irregularity of the joint, would also result in a lengthened centrode. Thus, the change in centrode length as a result of disproportional translation may illustrate clinical instability.

2.2.2 ANALYSIS AND TREATMENT OF ERROR

The question of error in the application of ICRs to joint kinematics has long plagued its use. The locations of these centres, for all joints studied, have been associated with large variations (Panjabi, 1979). In the early days of joint centre analysis the ICRs were derived by graphical means (Fick, 1904; Hall, 1929). These studies employed a method adapted from Reuleaux (Reuleaux, 1876), where the perpendicular bisectors of the displacement vectors, linking particular anatomical landmarks, determined the location of the ICR. The error associated with this type of analysis has now been shown to be unacceptably large (Dimnet et al., 1976; Spiegelman & Woo, 1987). More recently, computer-based methods have been used. The majority of these methods require the use of digitisers to optically transform the anatomical landmarks into co-ordinates. Although considerably more reliable, these techniques are not themselves without error and much work has been, and continues to be, invested into its minimisation (Amevo et al., 1991a; Amevo et al., 1991b; Amevo et al., 1991c; Dimnet et al., 1976; Panjabi et al., 1992; Panjabi & White, 1971; Pearcy & Bogduk, 1988).

Almost all authors studying spine kinematics use anatomical landmarks to define vertebral positions. In order to study the motion of vertebral segments from radiographs or fluoroscopy, it is necessary to track the movement of the segment through all, or part of, the range concerned. In cadaveric studies, radio-opaque markers or Steinmann pins can be inserted into the substance of the bone and easily digitised, thus potentially improving accuracy (Gregersen & Lucas, 1967; Lumsden & Morris, 1968). *In vivo* studies, of course, need to be much less invasive. The combination of lower X-ray dosages and the

marking of anatomical points associated with studies on live subjects renders the process more liable to error.

In his seminal work on the ICR and error, Panjabi (Panjabi, 1979), took a theoretical view of the way the ICR was determined and at the possible errors involved. If we assume that two points A and B are selected on a body in plane motion and that the points are measured over a time interval. Thus the points A, B and their images A´, B´ form a total of eight input co-ordinates (X and Y at each increment of time), each of which are subject to errors in measurement (Figure 2.2). The points may have been obtained from photographs, radiographs, fluoroscopic or cineradiographic images or directly using a travelling microscope, for example. The points may then have been recorded graphically, using a grid system, or digitised. Panjabi analysed the input-output relationship by the systematic introduction of discrete errors, both positive and negative, into each of the input co-ordinates. By this method, 256 possible locations for the ICR and values for the angle of rotation were produced (Figure 2.3). As can be seen from the diagram of input error zones and output error zone, small input errors produce considerably larger output errors. These errors are dependent not only on the errors associated with the input co-ordinates but also on the values of the co-ordinates themselves. By analysing the errors in the ICR as a function of the angle of rotation, Panjabi demonstrated clear guidelines for choosing the optimum input co-ordinates and hence improving experimental method. He showed that if the two marker points subtend an angle of approximately 90 degrees at the estimated location of the ICR, the error in ICR location would be minimised. Similarly, for reducing error in the determination of rotation angle, the optimum marker angle again appeared to be 90 degrees.

The rotation itself is also a major source of error in locating the ICR. Previous researchers had observed the effect of small rotations on ICR location (Dimnet et al., 1976). Panjabi established this as a simple inverse function. That is, errors in the location of the ICR increase with a decrease in the angle of rotation. The greatest increase in error occurred when the angle was less than 5 degrees.

The following year Dimnet (Dimnet, 1980) attempted a similar error analysis, specifically for *in vivo* kinematic studies. By the application of the least squares method to the reading of input co-ordinates, Dimnet demonstrated a "zone of uncertainty", around the ICR location, comparable to the rhomboidal error zone described by Panjabi (Panjabi, 1979).

Bryant et al (Bryant et al., 1984) further developed the analysis of Panjabi and Dimnet by the substitution of discrete input error for stochastic error. Bryant and co-workers felt the simplistic error zones of Panjabi and Dimnet sacrificed accuracy for clarity. On the other hand the use of stochastic input error produced much more complex distributions of ICRs, which were consequently more difficult to describe. In an effort to bridge the gap between these approaches, Bryant et al applied the probability density function to the formulation of a single parameter, the "ICR error, e". This parameter was intended to provide the Panjabi/Dimnet error zone with the dimensions of size and probability density. Hence in the design of experimental method, or in the interpretation and analysis of experimental data, the "ICR error" could be used to describe the distribution of ICRs.

In 1988 Pearcy and Bogduk (Pearcy & Bogduk, 1988) investigated the possible practical sources of error involved in the clinical acquisition of spinal ICRs. These authors outlined the steps involved in the collection of data and quantified the variations, both within and between observers, entailed in each step. Using lateral radiographs of the lumbar spines of ten normal individuals and a digitiser, they employed a superimposition technique to determine ICRs for each lumbar segment. In doing so they outlined the process of obtaining an ICR. The authors considered that five separate stages are required for each pair of vertebrae, using this method. (i) Marking and/or tracing the upper vertebral image in the initial position; (ii) matching the images of the same vertebrae in the final position; (iii) superimposition of the lower vertebrae to demonstrate relative motion; (iv) construction of reference axes on the images; (v) an algorithm, manual or computer-based, to process the data and plot the ICR. Results of the error analysis demonstrated clearly that errors involved at each of stages 1-4 were "acceptably small". However, the summation of these small errors produced an amplification effect in the uncertainty associated with ICR location. The authors noted that, for this study, two observers may differ in the location of a given reference point by a mean of 2mm, with a standard deviation of up to 4mm. Panjabi (Panjabi, 1979) shows us that a range of this magnitude would produce unacceptably large errors. Pearcy and Bogduk (Pearcy & Bogduk, 1988), point out however, that the extreme ends of the range are statistically unlikely and that, for the most part, the observers would be around the mean difference. Much of the between and within observer variation can be explained by the small range of motion involved in some of the movements, since the greatest errors occurred where the segmental movements were smallest. To reduce error, the authors suggest that only rotations of

greater than 5 degrees are suitable for ICR determination. Although Pearcy and Bogduk normalized their data with respect to the vertebral dimensions, little attention is paid to the factor of image size. Since ICR "accuracy" is largely dependent on input error and this, in turn, is influenced by the precision of identifying anatomical landmarks, it is likely that the size of the image will play a role in ICR determination. Image resolution, of course, will dictate the range of usable image size and this may become more important as digital advances improve resolution.

FIGURE 2.2

INPUT CO-ORDINATES FOR DETERMINATION OF ICR

(ADAPTED FROM PANJABI 1979)

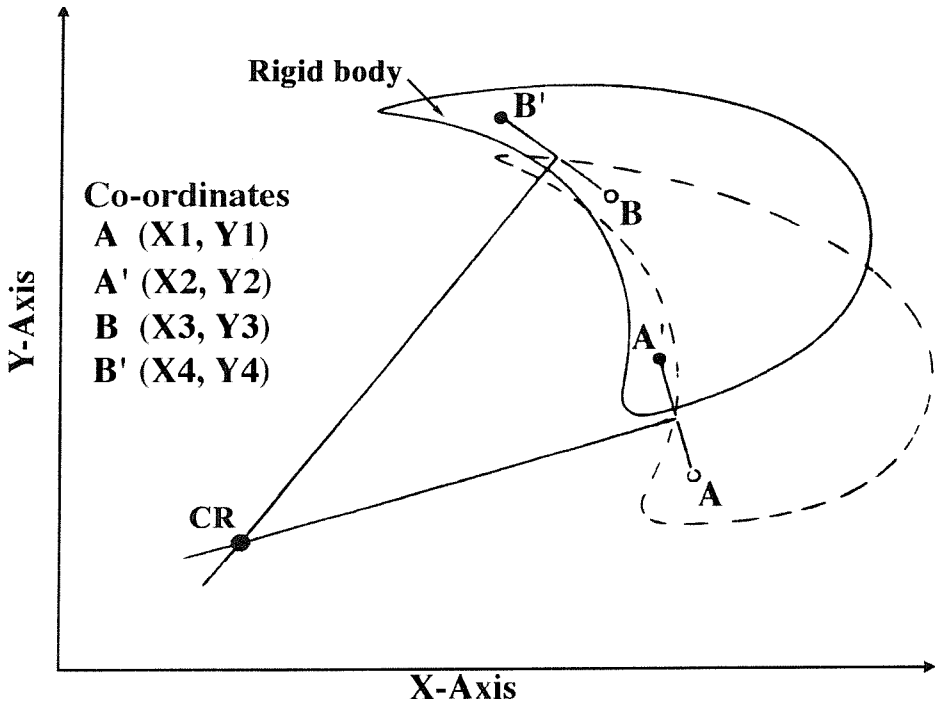
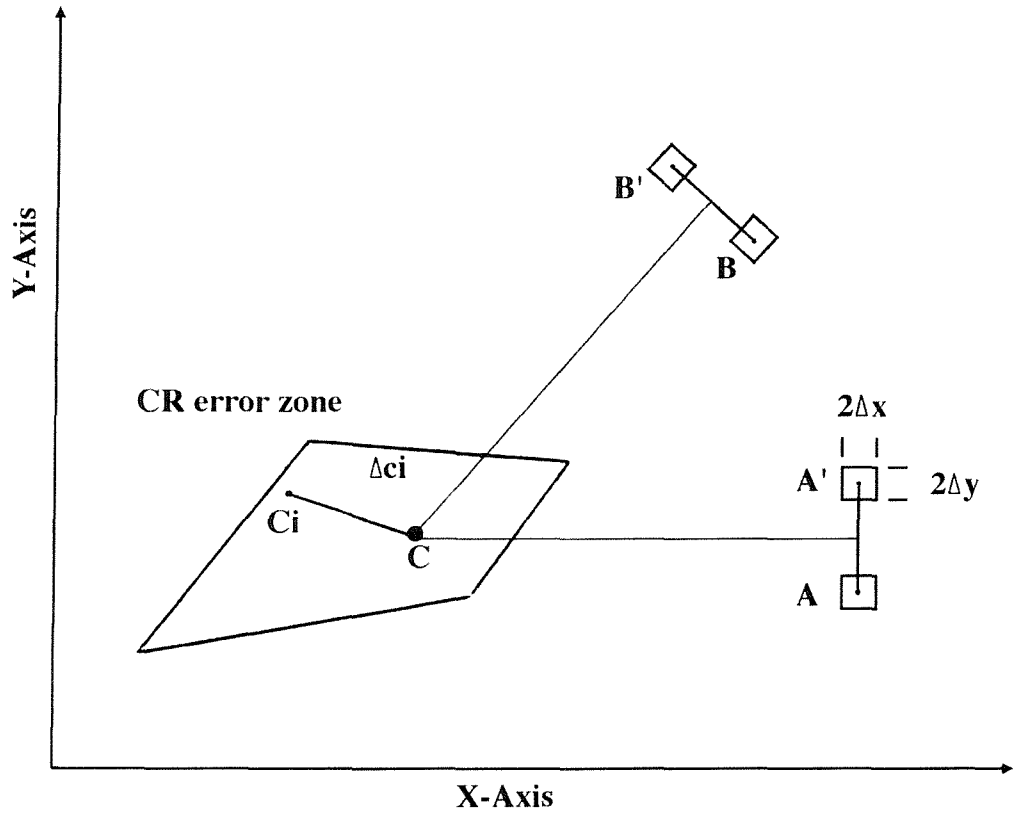


FIGURE 2.3

ERROR ZONE ASSOCIATED WITH ICR DETERMINATION
(ADAPTED FROM PANJABI 1979)



More recently Panjabi et al (Panjabi et al., 1992) attempted a similar analysis of practical errors in utilising ICRs and other kinematic parameters. The study was carried out to quantify errors in the analysis of lumbar spine lateral radiographs. The authors concentrated on the effect of errors produced as a consequence of digitiser quality, radiographic quality and vertebral level. Large errors were found in association with lumbar levels at either end, i.e. L1/2 and L4/5, compared with those in the middle. This was probably the result of deviation from the central X-ray beam. Remarking the films was also associated with large errors, whereas redigitising the same points using the same

and different digitisers was not.

As one might expect, one of the most significant sources of error found in this study was in the manual superimposition and marking of radiographs. Errors involved in remarking in this study were, the authors suggested, a result of remarking the same sets of radiographs, not radiographs of the same individuals taken at different times. The "biological" variation associated with repeated screenings of the same individual was considered beyond the scope of the study. The authors also noted that radiographic quality did affect error production.

Another example of how error might be dealt with is provided by Amevo and colleagues (Amevo et al., 1991a; Amevo et al., 1991b; Amevo et al., 1991c). This study attempted to determine the normal range of ICRs for cervical motion segments in order to provide essential normative data for its clinical application. The authors examined lateral radiographs of 40 normal subjects aged between 22 and 66 in the fully flexed and fully extended positions. Using a superimposition technique, ICRs were derived for each motion segment, in millimetres, with respect to the origin of the X-Y co-ordinate system. The origin of this Cartesian system was arbitrarily assigned to the posterior inferior corner of the lower vertebra. The ICR co-ordinates were then "normalised" by dividing the X and Y values by the corresponding width and height for the appropriate level. This allowed meaningful comparison of ICR location between individuals. The data were then displayed, graphically, as scatter points on a representative cervical spine made up of trapezoids whose dimensions reflected the mean sizes of all subjects for each segment. Once the mean value was plotted, the distribution of ICRs for each segment and their two times standard deviation (2SD) range were superimposed on to the diagram. The authors then added the 3SD range for inter-observer error to all points at the limit of the ICR distribution so producing an "envelope" of possible observer error (96%) superimposed on the 96% range of observed values. This would seem an unnecessary augmentation of error limits when, perhaps, taking the largest, or "worst-case", error would have sufficed. Despite this, the ICRs in this study were found to form smaller scatters than those described by another investigator studying normal cervical motion (Penning, 1978). This was thought to be due, in part, to the normalisation process that was not used by Penning. Amevo and colleagues also attempted qualitative analysis of the data. They concluded that identification of the radiographic images of the vertebrae and their subsequent tracing

was the greatest source of error for the modified overlay technique employed by them.

In another cervical spine study, van Mameren and co-workers (van Mameren et al., 1992) attempted to reduce the ICR error by means of an iterative process. Since significant errors are inherent in the acquisition of reference points, van Mameren et al set about optimising this stage before the ICR was calculated. Using a cineradiographic technique, the authors obtained images of cervical spine motion in the sagittal plane. In order to obtain the sharpest possible images, van Mameren and colleagues employed a 105-mm film camera at a frame rate of 4 frames per second. They then investigated the reproducibility and variability of ICRs. Two types of ICRs were calculated, averaged ICRs and standard ICRs. Averaged ICRs (aICRs) where the ICRs are derived from the displacement between 20 frames or increments, for example, frames 5 and 25, 6 and 26, 7 and 27 and so on. This method produced a cluster of ICRs from which only those derived from a segmental rotation of 7 degrees or greater were selected. The mean, or average ICR position from the selected group then constituted the "alCR". The "Standard" ICR (stICR) was simply that calculated from the two extreme frames of the sequence. Analysis was achieved by projecting the images onto drawing paper and tracing vertebral outlines. After marking each traced segment, using a needle, with five points, an average pentagon was "best fitted" with the original pentagon by means of iteration. The iteration continued until the distances between the corners of the average and original pentagons was less than 1mm. With this methodology the authors analysed the cervical spine motion of ten asymptomatic subjects. With the exception of the upper cervical complex (occiput to C2), the aICRs corresponded largely to those found by Penning (Penning, 1978). In addition the study also confirmed the notion that translation will result in a drop, or inferior shift, of the ICR. This relationship between translation and ICR was also shown by Kondracki (Kondracki, 1991). In this study it was suggested that this held true only if the rotation and translation were in the same direction, for example, anterior translation with anterior rotation producing an inferior displacement of the ICR. In terms of clinical application, van Mameren and colleagues submit that the ratio between translation and rotation may be important in the detection of early degeneration (instability), rheumatoid arthritis and ligamentous injuries (van Mameren et al., 1992).

The work of van Mameren et al (van Mameren et al., 1992) represents a thorough and painstaking approach to the problem of error minimisation in kinematic analysis.

Nevertheless the choice to use manual, and indeed graphical, methods in the marking and collection of data points seems an odd one in light of available alternatives. The electronic capture and storage of images and their subsequent digital manipulation, provides a potentially less error-prone environment for analysis than the method embraced by van Mameren and team. The introduction of a graphical technique into an otherwise elegant method would appear to contradict the literature. In spite of this the study does contribute valuable information for further research. The formation of a geometric shape from the reference points and the subsequent "best-fit" approach may help to simplify and optimise the determination of ICRs.

The authors found also that a combination of an interval or increment of 20 frames and rotations of 7 degrees and greater was optimal in terms of error minimisation. This too is useful for future experimental design and confirmation of previous work. The authors conclude that, based on this study, aICRs are a good parameter of motion quality, showing small inter- and intra-individual variability. In contrast, however, a recent study of errors in the placement of points in spinal flexion/extension kinematics has questioned the value of ICRs even for relatively large rotations (Harvey & Hukins, 1998). These authors, using images of a three-dimensional spinal model, found unacceptably large errors in ICR location despite measurements involving as much as 10 degrees of sagittal rotation.

2.2.3 THE MINIMIZATION OF ERROR

The two-dimensional analysis of a dynamic three-dimensional structure will always be associated with error. This is because information is lost, compelling the observer to make assumptions. A necessary assumption is that spinal motion is planar. Pearcy (Pearcy, 1985) has shown that for lumbar spine sagittal plane motion, this assumption is not too far from the truth. For other lumbar movements, when coupled motions occur, the hypothesis is invalid. Lateral flexion, or coronal plane sidebending, is associated with the greatest extent of coupled motion. This is true for all regions of the human spine and partly explains the scarcity of published studies investigating coronal motion. In radiographic studies the X-ray plate, or image intensifier, is usually arranged parallel to the presumed plane of motion. The real instantaneous axis may not be perpendicular to this plane and thus the bodies will describe elliptical paths around these centres, or centrodes (Soudan et al., 1979). As these authors point out, if the motion is truly planar all points on the body will move along a circle around the ICR. The degree to which these circles are

"deformed" represents the deviation from planar motion, perhaps suggesting that "sliding" is taking place. Similar discrepancies occur when the X-ray plate or beam is not wholly perpendicular to the body under scrutiny. However, provided the 90-degree protocol is adhered to, even quite substantial deviations out-of-plane can provide meaningful results (Soudan et al., 1979). In conclusion, these authors advised the three-dimensional investigation of all joints before applying two-dimensional analyses. Furthermore, precautions should be taken to avoid inclination of the X-ray source or target and care taken in the selection of anatomical landmarks.

2.3 CLINICAL KINEMATIC MEASUREMENTS

Clinicians have long suspected linear displacements of vertebral bodies, particularly in the sagittal plane, to be related to segmental stability. Direct measurement of translational movement has, therefore, been in clinical use for some considerable time (Morgan & King, 1957) and examples of excessive translation include retro- and spondylolisthesis. Retrolisthesis is thought to be consequence of disc degeneration and the normal posterior orientation of the facet joint planes and is, perhaps, more appropriately termed retrodisplacement, since listhesis tends to suggest forward or downward slippage (Giles & Singer, 1997) (Grobler & Wiltse, 1997). Spondylolisthesis is a term used to represent a mixed group of disorders associated with the anterior displacement or slippage of one vertebra on another (Grobler & Wiltse, 1997). The most widely accepted classification remains that proposed by Wiltse and colleagues in 1976 (Wiltse et al., 1976) that includes congenital, degenerative, traumatic and pathological causes. The most common form, however, is the type II or isthmic spondylolisthesis which follows from a fracture or defect in the pars interarticularis (spondylolysis), resulting in a separation from the posterior elements, or an intact but elongated pars interarticularis caused by repeated fracture and healing (Giles & Singer, 1997). Under these circumstances the spondylolisthesis follows the spondylolysis only when the defect is bilateral and most commonly involves the lumbosacral junction as a consequence of the considerable anterior shear forces experienced at this level (Floman, 2000; Giles & Singer, 1997). The shear forces producing anterolisthesis in these patients, are now resisted solely by the stiffness in the intervertebral disc.

Since there is complete seperation between the anterior and posterior elements, the vertebral body slips forward with no tension on neural structures and thus no neurological

damage. Translation in these cases is usually quite obvious on lateral radiographs of the lumbar spine and complex methods of measureing displacement are rarely required. The severity of the slippage is assessed by the degree of translation with respect to the inferior vertebral body. A slip of 25% or less of the lower segment is Grade I, whilst translation of 75% or more is Grade IV.

The isthmic spondylolysis is generally seen in the adolescent when, it is thought, a genetic predisposition or weakness at this anatomical site is combined with a growth spurt and large forces from sporting activities (Floman, 2000; Giles & Singer, 1997). This type of lesion, in the main, has been commonly accepted as a stable deformity with few progressing beyond adolescence (Axelsson et al., 2000; Fredrickson et al., 1984; Grobler & Wiltse, 1997; Pearcy & Shepherd, 1985). The degenerative types of spondylolisthesis and whether they represent true instability, is a contentious topic and will be discussed further in Chapter 4.

There are a variety of methods for directly measuring linear displacements of the lumbar spine, principally using lateral radiographs. These methods generally express the translation as a displacement, along an axis, in millimetres or as a percentage of mean vertebral diameter. An exception to this is the simplest radiological assessment of translation, "George's line" (Figure 2.4) (Yochum & Rowe, 1996). This line is formed by the posterior vertebral bodies as viewed on a lateral X-ray and involves no quantification, being simply an eyeball technique. Normally, the line should be smooth and unbroken with any deviation suggesting excessive translation. One of the earliest attempts to actually quantify translatory displacements was conducted by Morgan and King (Morgan & King, 1957). In this study the radiological appearance of excessive lumbar sagittal plane translation, or "primary instability", was discussed with regard to the clinical features. The authors provided a method for obtaining the necessary radiographs and estimating "instability" (Figure 2.5). It attempts to measure sagittal plane translation by drawing a line adjacent to the anterior edge of the lower vertebral body (L). A line is then drawn perpendicular to L to the inferio-anterior vertebral corner of the upper body (I). Translation is thus estimated by the magnitude of line I. The technique described is one of the few employing the anterior borders of the lumbar vertebrae. Stokes and Frymoyer (Stokes & Frymoyer, 1987) later embellished this simple measure and used it together with superimposed lateral lumbar films. This method was considered to give a more

accurate measure of translation by reducing the artefact produced by angular motion between segments. In a sequential destruction study, van Akkerveeken and colleagues (van Akkerveeken et al., 1979) developed a measure for translation, which incorporated any sagittal plane rotations (Figure 2.6). The method involved drawing lines along the superior end-plate of the lower vertebra (S), and the inferior end-plate of the upper vertebra (I). Extension will place the intersection of these lines posteriorly (a, d) and flexion will cause the intersection to be located anteriorly. If points b and e are the inferio-posterior corners of the upper vertebrae and points c and f the superio-posterior corners of the lower vertebrae, then translation is defined as the difference between line segments ab and ac and between line segments de and df. This approach was later adapted by Posner and co-workers (Posner et al., 1982) in a similar study, which used the percentage translation as an index of stability (Figure 2.7). This method entails drawing lines along the superior end-plate of the lower vertebra and, perpendicular to this, passing through the inferio-posterior corner of the same vertebra, lines X and Y respectively. If point I is the inferio-posterior corner of the upper vertebral body, then translation is defined as the length of the line segment IY. For normalization purposes the translation was expressed as the ratio of absolute translation to vertebral body width (W). By this method the authors were able to directly compare their data with other studies without the need to account for magnification or distortion of radiographs. A further modification of this measurement technique was developed by Dupuis and colleagues (Dupuis et al., 1985) in a study on the radiological diagnosis of degenerative instability in the lumbar spine (Figure 2.8). In this method lines are drawn connecting the two posterior vertebral corners to each other on both the upper (U) and lower (L) segments. A line through the inferior end-plate of the superior body (I) is also drawn. At the point of intersection between I and U, at the posterio-inferior vertebral corner, a fourth line (R) is drawn parallel to line L. Translation is then defined as the perpendicular distance between lines R and L. This method can also express translation as a percentage of vertebral body width (W). Much of the recent work on segmental instability has utilised the "Dupuis" method, or modified versions of it, for establishing the diagnosis in patient groups (Bram et al., 1998; Fujiwara et al., 2000a; Fujiwara et al., 2000b; Murata et al., 1994).

In 1990 several of the methods used to measure translation were assessed using an experimental model, which allowed precise manipulation of sagittal translation (Shaffer et al., 1990). Other factors, such as radiographic quality and coupled motion were also

considered. With regard to consistency and accuracy, Schaffer and colleagues found the method described by Morgan and King, where the anterior vertebral margins were clearly visible on the radiographs, to be superior to the other methods tested. It is interesting to note that these reviewers favoured the earliest and simplest measure as opposed to later, more elaborate, efforts.

FIGURE 2.4

GEORGE'S LINE

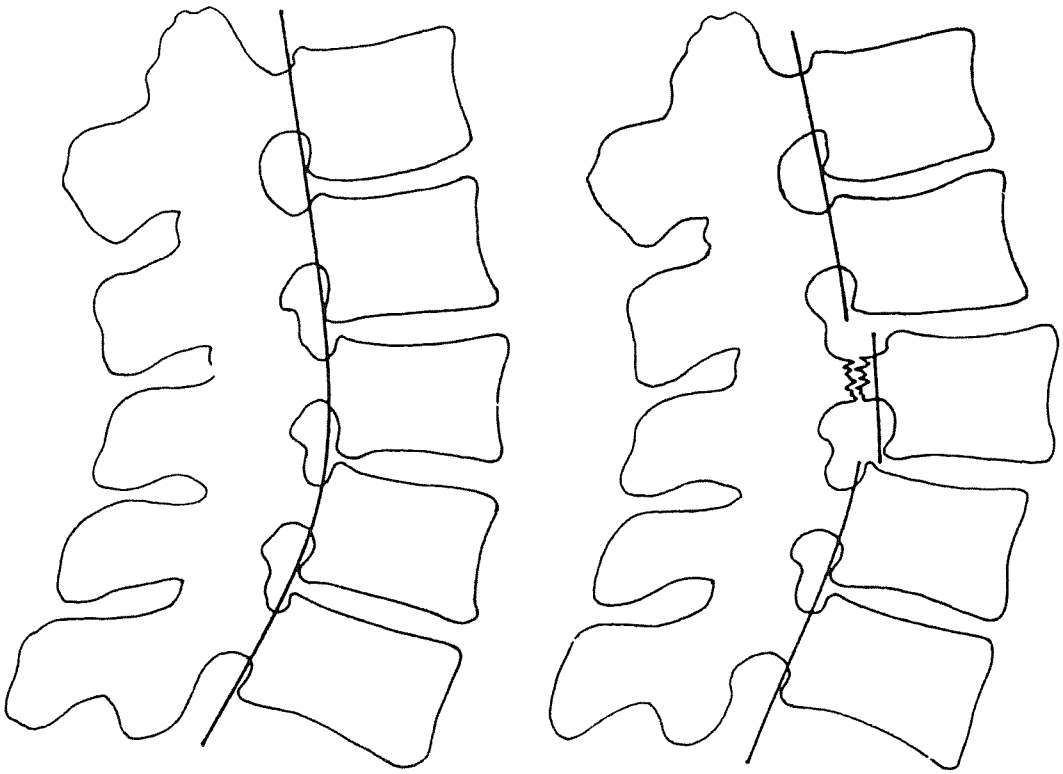


FIGURE 2.5

METHOD OF MEASURING TRANSLATION
(MORGAN & KING 1957)

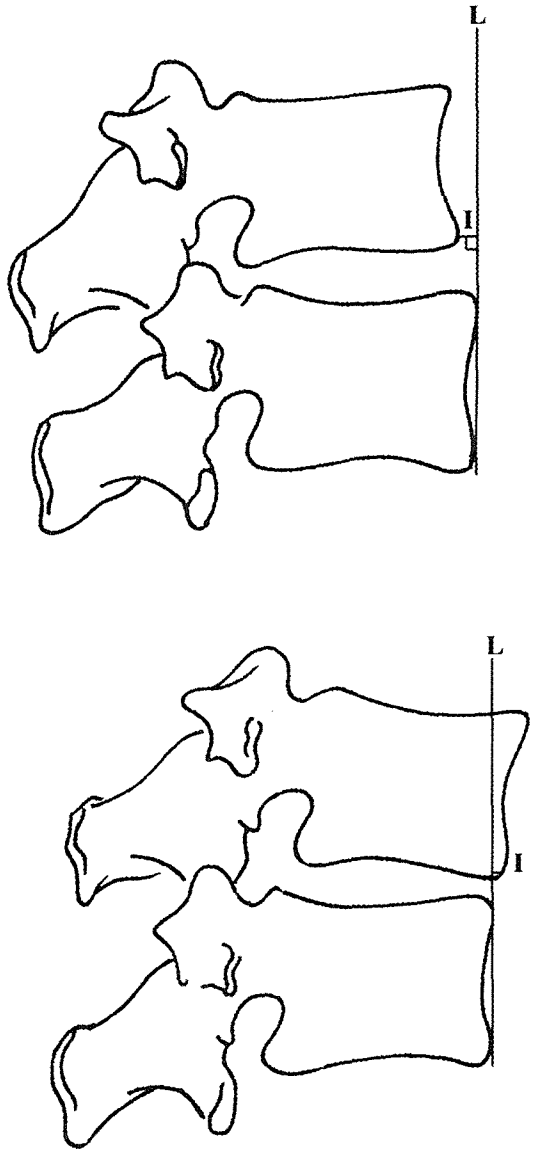
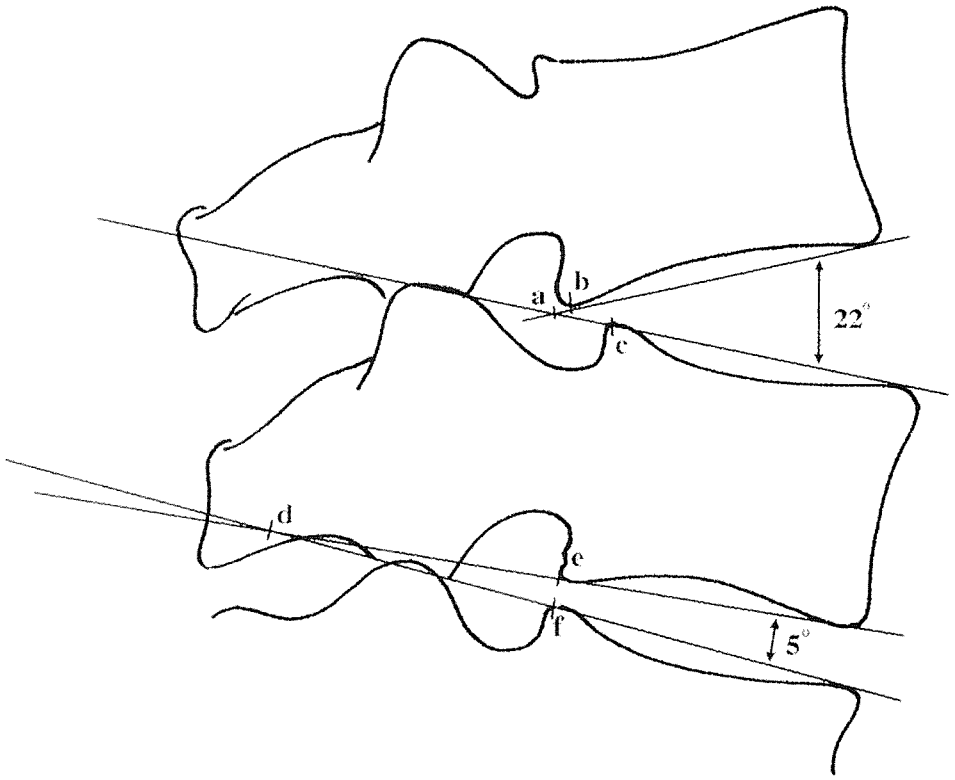


FIGURE 2.6

METHOD OF MEASURING TRANSLATION
(VAN AKKERVEEKEN ET AL. 1979)



The lower segment is stable de=df.
The upper segment shows radiologic instability.
The line ab is shorter than the line ac by 3mm.

FIGURE 2.7

METHOD OF MEASURING TRANSLATION

(POSNER ET AL. 1982)

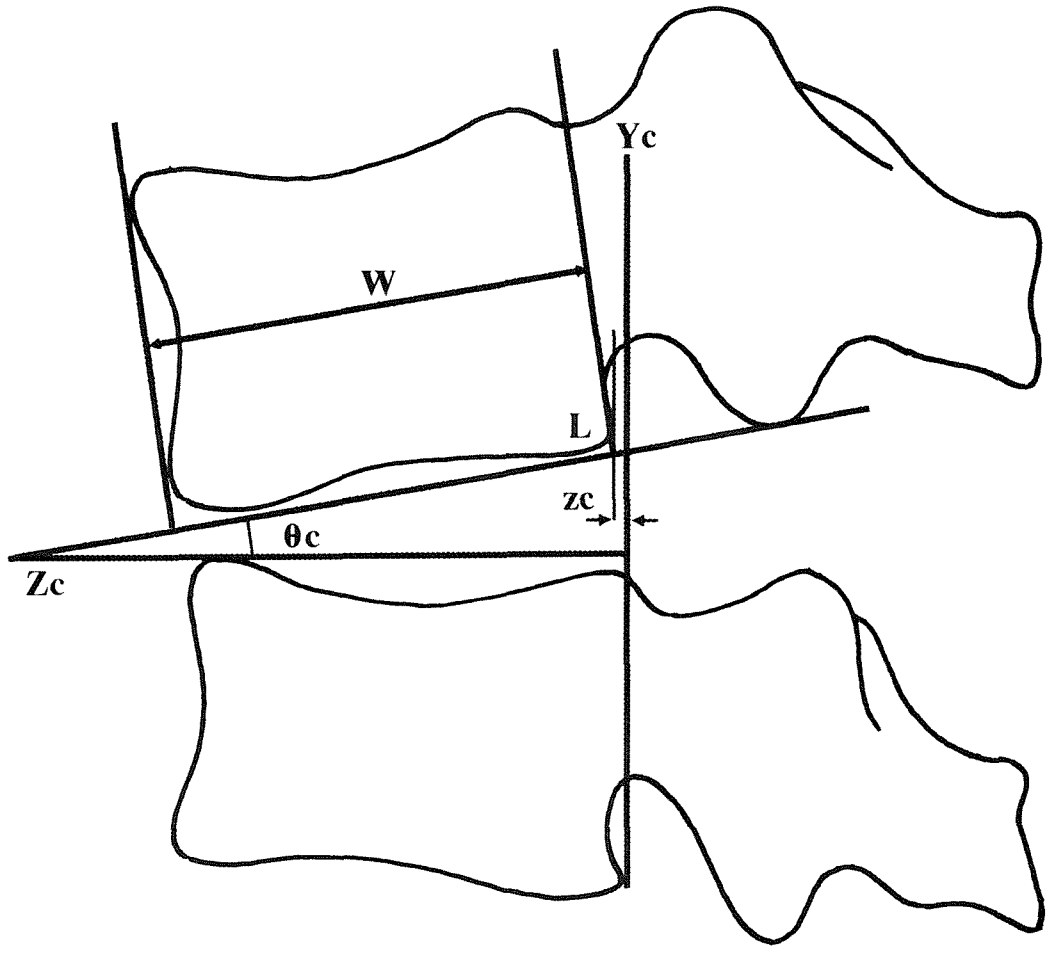
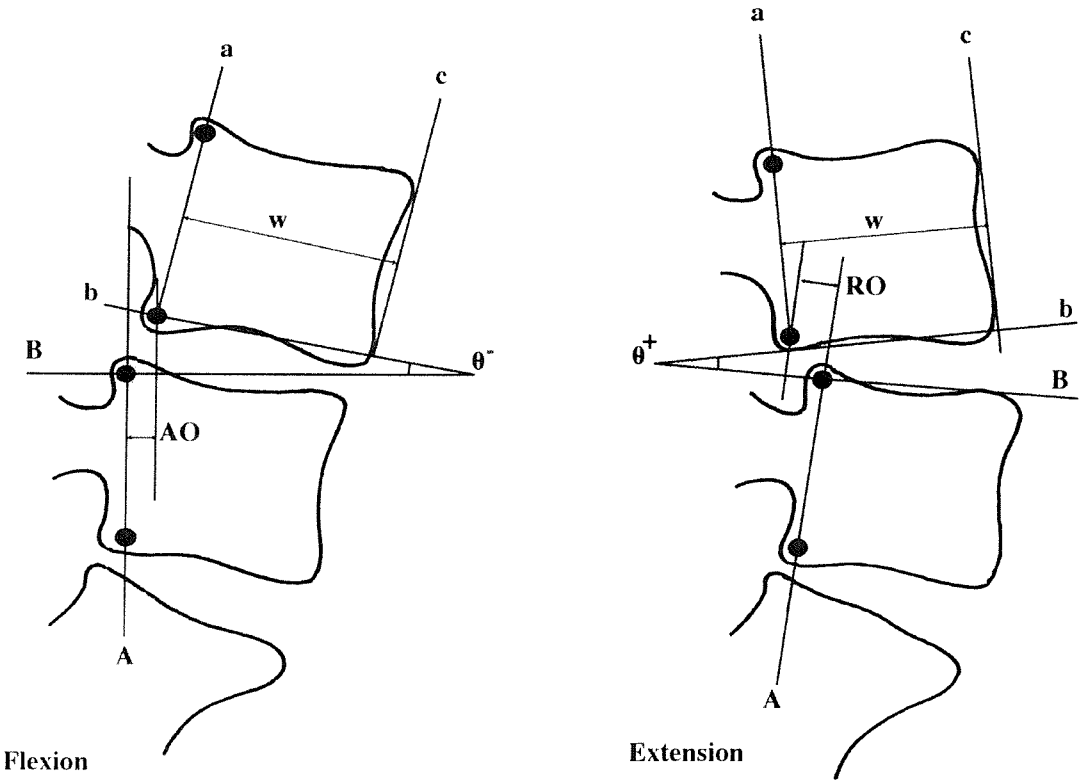


FIGURE 2.8

METHOD OF MEASURING TRANSLATION
(DUPUIS ET AL. 1985)



Percentage horizontal displacement is
HD% = (AO or RO/W) x 100

The major drawback with most of these direct techniques, however, is that they tell us little about the rotational motion that may, or may not, be associated with any translation. It is for this reason that the ICR has largely superseded the direct measurement of linear displacements. Interestingly, however, a recent paper has proposed a new method for the direct measurement of disc height, vertebral height and sagittal plane displacements of the lumbar spine (Frobin et al., 1997). These authors claim that their results have much less associated error than previous techniques and are virtually independent of distortion produced by patient/radiographic tube misalignment. Frobin and co-workers have also related translation to the degree of rotational motion. Using these methods, a recent study has proposed a new protocol for measuring sagittal plane segmental motion from conventional lateral radiographs (Leivseth et al., 1998). This work compares the new protocol, distortion-compensated Roentgen analysis (DCRA), with the accepted accuracy of Roentgen stereophotogrammetric analysis (RSA). RSA involves tracing the trajectories of implanted tantalum balls and has been confined to post-surgical assessments of spinal motion (Johnsson et al., 1990; Johnsson et al., 1992). The authors claim that, in terms of accuracy, DCRA provides slightly inferior but comparable results with a much less invasive protocol. If these claims are substantiated they may stimulate renewed interest in more direct measures of displacement.

Considering translational or linear movements of vertebrae, the authors White and Panjabi (White & Panjabi, 1990) noted that shear stiffness in the horizontal plane has a high value in normal intervertebral discs. This finding suggests that considerable force is required to overcome this resistance and produce increased translation. The corollary of this is that if there were evidence of excessive translation of an intervertebral segment, this would strongly suggest loss of integrity of the restraining tissues. Rolander (Rolander, 1966) demonstrated that, in general, there is only 1 to 2mm of translation along the frontal or sagittal axes. This finding is supported by Pearcy (Pearcy, 1985) using stereoradiography in living subjects. In this study it was noted that normal linear movements of the lumbar segments rarely exceeded 2mm. How these motions might alter with degenerative changes, became the subject of an *in vitro* study in the same year (Gertzbein et al., 1985).

Applying sequential ICRs (or centrodes) to radiographs, Gertzbein et al (Gertzbein et al., 1985), using cadaveric spines, attempted to relate change in ICR location with spinal degeneration. This work followed on from a study, similar in both methodology and

results, the previous year (Seligman et al., 1984). The specimens employed represented a range of degenerative disc disease from "normal" to "severe". Confining the study to the L4/5 segment in sagittal plane motion, the authors suggested that, in the presence of degenerative disc disease, the ICRs are displaced away from the "normal" position. The authors used plain X-rays of the specimens in 3-degree increments from full extension to full flexion. An ICR was determined for each 3-degree rotation, thus forming a centrode (i.e. path of ICRs). By measuring the distances between each ICR, in order, a "length of locus" was found by summation. The normal controls had short loci (<30mm). The loci of specimens showing minor degenerative change were longer, although the overall position of the loci was found to be essentially the same as the control group. Those specimens with moderate degenerative change demonstrated displacement of loci inferiorly, with length of loci similar to that of the mild and minor categories. The severely degenerated specimens, on the other hand, were found to have a reduction in the length of their loci but, due to the small number of specimens in this group, the authors were unable to determine a trend in the position of the loci (Figure 2.9). The greatest change in the pattern of centrodes is thus seen in the earliest stages of degeneration when the radiographic changes are minimal. It is interesting to note that, in this group, only one-third of the specimens were found to have an increased range of motion.

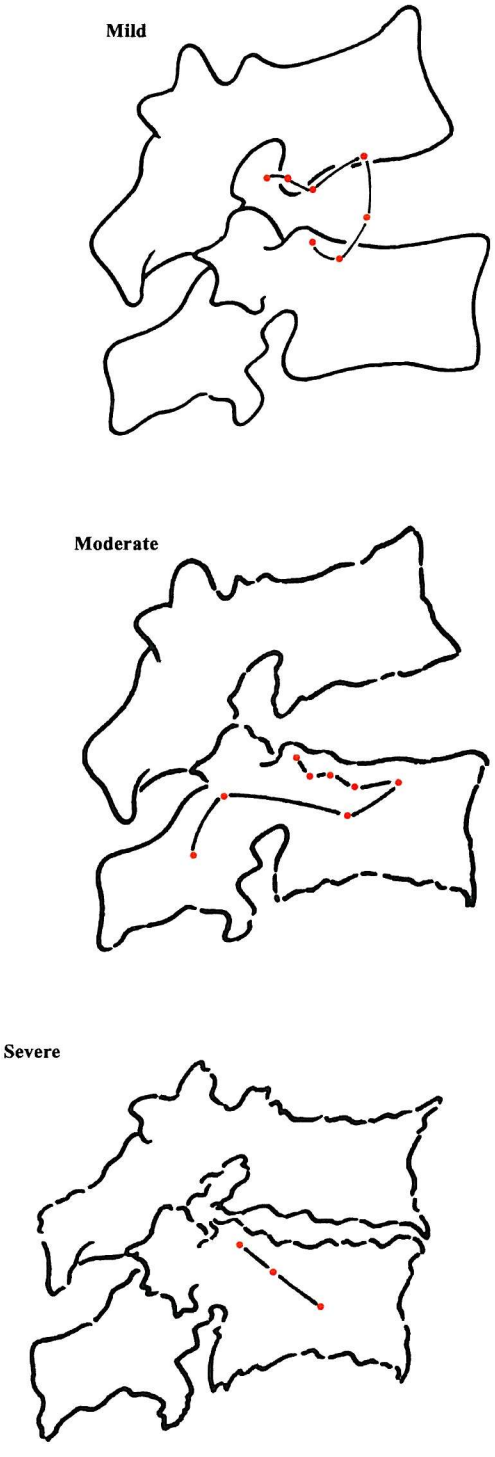
This aspect of ICRs was examined in an *in vivo* setting, by another study (Ogston et al., 1986). Radiographs of 21 normal males were taken at six intervals throughout the movement of flexion from extension. The films were then analysed to determine centrodes in both location and length. The average location of the L4/5 centrode was found to be in the posterior half of the L5 vertebral body and just below the vertebral end-plate. This location was more inferior than that determined for cadaveric specimens at the same level (Seligman et al., 1984) but similar to those found in an earlier *in vivo* study (Pennal et al., 1972). The average location of the L5/S1 centrode was found in the posterior half of the L5 intervertebral disc. This paralleled other cadaveric studies of normal lumbar motion segments (Gertzbein et al., 1984; Gertzbein et al., 1985) and the *in vivo* study by Pennal and colleagues (Pennal et al., 1972). The difference in the centrode length (L4/5) between the cadaveric study reported by Seligman et al (Seligman et al., 1984), i.e. 20.9mm, and this *in vivo* study, 43.7mm, is discussed by Ogston and co-workers. They proposed that the larger centrode length of the *in vivo* subjects could be attributed to; (i) an increase in shear stress across the disc and ligaments, producing a

greater magnitude of translation as a result of the more vertical posture adopted by the lumbar spine on flexion of the trunk; (ii) unaccounted muscle action; (iii) age differences between the two groups. In conclusion, the authors advocated centrode analysis as a worthwhile clinical test of the lumbar spine.

FIGURE 2.9

CENTRODE PATTERN FOR MILD, MODERATE AND SEVERE DEGENERATION

(GERTZBEIN ET AL. 1984)



A more recent *in vivo* study attempted to determine the motion characteristics of the normal lumbar spine in over 600 subjects. Using lateral lumbar radiographs in full extension, neutral and full flexion, Yoshioka and colleagues (Yoshioka et al., 1990) plotted, amongst other parameters, the ICR location for each segment (although the authors of this paper favoured the use of the term IAR). Interestingly their findings were in agreement with both earlier *in vivo* studies (Ogston et al., 1986; Pennal et al., 1972), in that the L5/S1 ICR was located around the posterior disc space, whereas all other lumbar ICRs were found below their respective disc levels. This was thought to reflect the greater translation occurring at levels above L5. The reduced translation, noted at the L5/S1 level, was attributed to the restraining effects of the iliolumbar ligaments, which act to anchor the L5 segment to the pelvis. The L4 vertebrae, on the other hand, were shown to exhibit considerable translation with only moderate rotation. This finding has received current support from another *in vivo* study employing cineradiography (Harada et al., 2000). These authors performed lumbar segmental motion analysis (L3-S1) on ten asymptomatic male volunteers during both flexion from extension and extension from flexion. They concluded that, at L5/S1, angular motion predominates over horizontal displacement and that this may be a function of the iliolumbar ligament, shape of the IVD and orientation of the facet joints. Another interesting finding from this study was that at L5/S1 a difference in motion characteristics was noted between flexion and extension. Using simple linear regression on the coordinates of one point (anterior-inferior corner of the upper segment) it was found that the relationship between the xy coordinates for this point remained relatively constant in both directions for L3/4 and L4/5. For L5/S1 however, the relationship changed depending on the direction of motion. Harada and colleagues (Harada et al., 2000), speculate that this might be explained by the functional anatomy of the iliolumbar ligament. The morphology of this structure is complex with anterior, posterior, superior and inferior divisions subserving slightly different functions (Leong et al., 1987). This subdivision of the lumbosacral segment from the rest of the lumbar spine, on the basis of function, will be further discussed in chapter 3.

In an *in vivo* study involving subjects with chronic low back pain, Penning et al (Penning et al., 1984) did not find overtly abnormal patterns of motion, i.e. Instability. The inability to determine abnormal motion was attributed to; (i) patient guarding and (ii) measurement errors. In the first instance, the assumption was made, by Penning et al, that abnormal motion will produce pain and thus initiate an involuntary protection mechanism

preventing the abnormal motion and in the second, errors will mask smaller abnormalities.

2.4 SUMMARY

ICRs have been extensively investigated by many involved in spinal kinematic research. Their widespread acceptance and use in the clinical arena, on the other hand, has been virtually non-existent. Much of this reluctance is probably a result of the inherent error-prone nature of ICRs and the laborious methods associated with error minimisation. Clinicians require rapid, reliable and robust tools for patient assessment and tolerate poorly the time-consuming, necessarily repetitive constraints under which most of their research colleague's work. Until improvements in image quality and data acquisition have led, inevitably, to reduced error and robustness of use, it is likely that the ICR will remain, almost entirely, a research tool.

Direct techniques of measuring translation have the advantage of simplicity but in isolation tell us little about the nature of vertebral movement. New methods of describing segmental displacements in terms of their component parts, if sufficiently error-free, may provide the optimum means for readily distinguishing abnormal from normal spinal motion.

CHAPTER 3

BIOMECHANICAL INVESTIGATIONS

3.1 INTRODUCTION

The scientific investigation of the human spine has traditionally followed two distinct paths. Those researchers interested in observing spinal function under normal physiological conditions, and/or in pathological states, have attempted *in vivo* studies. Those more concerned with the explanation, than description, of spinal function have been compelled to employ *in vitro*, or cadaveric, methods. In the latter group two further divisions occur, although in many instances the distinctions can be less than clear, *in vitro* studies principally directed at (i) kinematic analysis and (ii) material properties of tissues. This review will concern itself, largely, with the kinematic studies of spinal function since the detailed material properties of the spine are beyond the scope of this thesis.

3.2 THE FUNCTIONAL SPINAL UNIT

3.2.1 DEFINITION

Much of the early experimental work, on cadaveric specimens, involved the smallest functional component of the spine, the motion segment. This was described by Junghanns (Junghanns, 1931) as comprising two adjacent vertebrae and all intervening soft tissue. To begin to understand the link between kinematics and dynamics the reductionist viewpoint of the day made it inevitable that researchers would commence their studies with an isolated spinal unit. The definition of a motion segment, however, may have lead to confusion in some cases, since the majority of researchers left only ligamentous tissue between segments. White and Panjabi (White & Panjabi, 1978) revised this concept and included only the disc, apophysial joints and ligaments as intervening tissues. They renamed this motion segment as the functional spinal unit (FSU) and considered it to represent the smallest mechanical unit of the spine. These initial biomechanical studies were directed, in the main, toward the cervical spine (Panjabi et al., 1975; White et al., 1975). In the 1970s and 80s, however, attention was drawn to the lumbar spine and the clinical consequences of instability in that region.

3.2.2 THE FSU IN USE

Posner and colleagues (Posner et al., 1982) undertook an exhaustive *in vitro* study of the lumbar and lumbosacral spine in an attempt to furnish clinicians with numerically based information on normal motion. The experiments were carried out on 18 FSUs taken, at autopsy, from individuals with no history of chronic back pain, spinal surgery or disease. The study was concerned with sagittal plane displacements and the effect of axial preload and flexion/extension forces. Serial transection of the ligaments was performed whilst LVDTs attached to the upper vertebrae recorded any translations or rotations. The specimens were loaded/preconditioned for 4 minutes prior to data recording. This time interval was chosen as the required period to allow for all creep in the specimen to take place (Panjabi et al., 1976). Horizontal displacements (anterior translation) were determined as a percentage of the AP diameter of the lower vertebral body and were found to be almost 3 times as large as those found by Nachemson and colleagues using intact lumbar spines (Nachemson et al., 1979). Posner and co-workers suggested that maximal anterior translation in the normal lumbar spine was no more than 2.3mm or 8% of the lower vertebral diameter. These figures are in good agreement with the *in vivo* work of Pearcy (Pearcy, 1985). Posner and colleagues were also one of the first groups to counsel the subdivision of the lumbar spine into lumbar (L3-L5) and lumbosacral (L5-S1) regions on a functional basis. This kinematic demarcation between lumbar and lumbosacral segments, particularly for flexion/extension, was first noted by Knutsson (Knutsson, 1944). Since that time it has become a recurring feature in spinal kinematics (Frobin et al., 1996; Nachemson, 1981b; Oxland et al., 1992; Pearcy, 1985). The work by Posner et al represented a good attempt to bring experimental and numerical methods to bear on clinical decision-making. In providing the clinician with numerical guidelines the authors hoped to influence the often *ad hoc* decision of when to embark on surgical intervention. Nachemson, in 1981, suggested that only translatory motion in excess of 4mm between two vertebrae could safely be described as abnormal (Nachemson, 1981a). Based on the findings of Posner et al (Posner et al., 1982) White and Panjabi (White & Panjabi, 1990) revised the figures for anterior translation and suggested 4.5mm or 15% of the adjacent vertebral body diameter as the upper limit of normal motion. It is interesting to note that even this revised figure is open to contention. In a recent *in vivo* study involving radiographic measurement of asymptomatic individuals, the determination of 5mm translational motion was so common in the L3-L5 region, as was 4mm in the L5/S1 segments, that "these values cannot be considered pathological." (Tallroth et al., 1992). It

would appear then, that segmental translation is subject to wide anatomical variation. In light of this, arbitrary measures of what constitutes normal or abnormal behaviour are unlikely, in isolation, to provide information of great clinical worth.

3.3 SEQUENTIAL DESTRUCTION STUDIES

As previously discussed, the most widely used imaging technique in the study of spinal pain is plain radiography. In the case of low back pain, however, it has long been recognised that its use is limited (Lewis, 1991). Much work has been carried out in attempting to correlate radiographic findings of degeneration with clinical pain patterns, mostly without success (Frymoyer et al., 1986; Magora & Schwartz, 1976; Quinnell & Stockdale, 1982). Clinicians investigating spinal pain have, therefore, had to deal with at least one major dilemma. Since the majority of spinal pain syndromes are thought to be soft tissue in origin, how can these tissues be identified using imaging methods traditionally concerned with bony tissue pathology? To help solve this problem researchers have attempted to combine *in vitro* methods with *in vivo* findings. One popular way to achieve this has been to cause sequential destruction of cadaveric soft tissues and observe any changes in kinematic parameters. This, it has been hoped, will provide a rationale for the identification of spinal soft tissue injury. The results of sequential destruction studies to date, however, do not appear to provide the detailed correlative findings that might be anticipated.

One of the earliest studies to involve the lumbar spine was performed by van Akkerveeken and colleagues (van Akkerveeken et al., 1979). Nine cadaveric spines were taken, at autopsy, from asymptomatic adults and included all segments between L1 and the sacrum. These were ligamentous spines, which unusually, included the bulk of the paravertebral muscles. Radiographs of the intact specimens in full sagittal flexion and extension were taken before and after experimental destructive lesions were produced. Surgical division of the posterior longitudinal ligament and adjacent annular fibres together with removal of the nucleus pulposus were performed. Translation only was measured (according to Morgan and King (Morgan & King, 1957)) and was found to increase in 20 of the specimens. The maximum translation, however, was no greater than 1.5mm. Sidebending was not studied nor were the effects of compression or distraction. These results were confirmed by Goel et al in 1985 (Goel et al., 1985). In this study significant hypermobility was found only when the disc was considerably disrupted and the nucleus

removed. This work followed on from the sequential destruction investigations carried out by Adams and colleagues (Adams et al., 1980) who discovered that, in terms of resistance to bending, the intervertebral disc and capsular ligaments provide much greater opposition than both the ligamentum flavum and the supraspinous/interspinous ligaments. Goel and colleagues (Goel et al., 1985), however, were concerned with how disc disruption directly affects the motion of lumbar segments. The purpose of this study was to examine the effect of sequential injury (laminectomy/total discectomy) on whole lumbar ligamentous spine specimens. Eight specimens were mounted at the sacrum and loads applied through a frame at T12 to produce flexion/extension, sidebending and axial torsion. The 3-D kinematics was analysed using an optoelectronic system incorporating LEDs fixed to the specimen. Partial laminectomy, partial facetectomy, subtotal discectomy and total discectomy were performed on the right side of the L4/5 level sequentially and the kinematic data normalised with respect to the intact results. The results suggest that significant increases in motion (and thus possibly instability) were present only after subtotal and total discectomies. Total discectomy was associated with significant increases in both rotation and translation, except for extension, which showed no significant increase in translation. Subtotal discectomy, however, was associated only with increases in rotations and not translations and these were witnessed only with flexion and right sidebending.

The above results are for the level of injury (L4/5); at the level above this (L3/4) a significant increase in translation is noted. This increase is present for both subtotal discectomy and total discectomy and therefore the tendency for L3 to "slip" on L4 is not dependent on the amount of nucleus removed at L4/5. This aspect of lumbar kinematics is reflected in the *in vivo* work of Tibrewal and colleagues (Tibrewal et al., 1985). This study employed biplanar radiography on fifteen patients with lumbar disc herniation. Although rotations and coupled motions during flexion and extension were studied, translational movements, however, were not addressed. The results showed significant changes in motion characteristics at the levels above the herniation. Primarily these changes were noted as an increase in coupled lateral bending and axial rotation. At the level of the herniation the normal motion of flexion/extension was reduced by approximately 50% with no alteration in coupling.

Goel et al (Goel et al., 1985) also attempted to observe the effect of sequential injury on

axial rotation. Nevertheless, axial rotation, when studied, produced "inconsistent behaviour" with regard to translations and therefore could not be recorded. Sagittal rotation was, however, quantified and an increase in motion at the level of injury was noted only after total discectomy. Rotational increases were not noted above the level of injury. The authors propose that these findings are in keeping with common clinical thinking that only the smallest amount of nuclear material should be removed to reduce instability at the level of injury. And furthermore, that extension exercises are useful since the lumbar spine would appear most stable under loads in extension (i.e. no or very little translation). However, due to an overall accuracy and repeatability of +/- 5%, any changes in motion of less than 10% were not considered significant.

In some instances bony tissues are ablated in order to establish their influence on spinal mechanics. Recent work by Haher and colleagues has focused on the role of the facet joints in lumbar spine stability (Haher et al., 1994). After facet destruction, cadaveric lumbar spines were subjected to compressive forces. The authors concluded that the facet joints of the lumbar spine were not the main supporting structures in extension. Alternative pathways of loading shift axial loads to the annulus and anterior longitudinal ligament to support the spine. This transfer of load, although conceivably contributing to accelerated disc degeneration, is unlikely to produce acute instability.

These studies, then, would seem to confirm the notion that the intervertebral disc forms the primary restraint between spinal segments. Furthermore, when damage occurs to the disc the kinematic effects extend beyond the involved FSU to adjacent levels. It is therefore appropriate to examine the kinematics of the disc and in particular the effect of nuclear herniation.

3.4 INTERVERTEBRAL DISC STUDIES

Given that the disc has been found to be so important structurally, much of the *in vitro* work on motion segments or FSUs has been directed toward the problem of intervertebral disc herniation (Adams & Hutton, 1982a; Adams & Hutton, 1982b; Adams & Hutton, 1985b; Adams et al., 2000b; Nachemson, 1981b; Wilder et al., 1988). This has often involved the effects of large axial compressive loads on lumbar spine segments. These have helped to dispel the notion that herniated discs are somehow less stiff than their intact partners. Markolf and Morris (Markolf & Morris, 1974) demonstrated

experimentally that discs with the nucleus removed, display the same compression stiffness as undisturbed discs. Some years later Ebara and colleagues (Ebara et al., 1992) discovered, whilst undertaking an *in vivo* study on patients undergoing spinal decompressive surgery, a similar finding for tensile stiffness. Using dynamic radiographic data on patients with herniated discs, they were able to show that although the radiographs demonstrated a larger range of motion, the same discs exhibited high tensile stiffness. The opposite was also true, that those motion segments with reduced range of motion did not necessarily show higher intraoperative stiffness. This has important implications for kinematics since, in conventional dynamic radiography, motion segments with diminished range of motion are thought to be stiffer and hence more stable.

Few of the *in vitro* studies, however, have concerned themselves with the associated kinematic behaviour of the spine and are thus beyond the scope of this review. Nevertheless one such study which deserves mention is the work of Wilder et al (Wilder et al., 1988) on the biomechanics of lumbar disc herniation, as it provides us with insight into the vertebral response to loading.

The purpose of this study was to examine the mechanical effects of sitting and vibration on lumbar motion segments. In particular the authors wished to determine if overload and vibration would increase the likelihood of herniation. Wilder and colleagues undertook a particularly exhaustive review of the literature on motion segment testing and concluded that "the motion segment is viscoelastic, absorbs energy, moves with six degrees of freedom, exhibits coupled motion, has limited fatigue tolerance and depends upon its bony and ligamentous components for specific mechanical tasks".

20 cadaveric spines were divided into two groups of 20 L3/4 and 20 L4/5 motion segments, half of which received a 1 hour exposure to combined flexion-compression and vibration loading (5 Hz) and the other half 1 hour of static combined flexion-compression. This work was unique, among the published *in vitro* mechanical testing studies, in its point of vertical loading on the chosen segment. In most of the previous work the geometric centre of the disc was the point over which the load was applied. In this study the vertical "balance point" was used as a loading reference. This point was defined as a functional reference area where an applied axial load produced the minimum coupled flexion and lateral bending motion. In all of these experiments the mean balance point location was found to be posterior to the geometric centre of the specimen and, more

specifically, between 4 and 13% (of the AP diameter of the end-plate) posterior to this geometric centre. This, the authors point out, is within the limits of the ICR as reported by Gertzbein et al (Gertzbein et al., 1984).

Instability, in response to a vertical load, was defined as a translation or rotation of more than 1mm or 1 degree respectively. These movements were usually noted as a "sudden, non-linear change of displacement or rotation or rotation in response to a linearly increasing load". As is the case with most spinal *in vitro* experimentation, the specimens were frozen prior to testing. This method of storage has been shown not to significantly affect the mechanical properties of the spinal tissues (Hirsch & Galante, 1967; Sedlin & Hirsch, 1966; Tkaczuk, 1968; Woo et al., 1983; Woo et al., 1984). Nevertheless, it has been shown that discs removed post-mortem have marked differences in fluid content than discs removed at surgery (Johnstone et al., 1992). Discs taken at surgery have a lower fluid content in the nucleus and a higher fluid content in the outer annulus than those obtained at autopsy and this will affect mechanical behaviour.

Unlike other studies Wilder and colleagues (Wilder et al., 1988) made a positive effort to avoid the effect of creep on the viscoelastic behaviour of the FSU. The authors make no mention of preconditioning the specimens and, in fact, report their endeavours in reducing the number of unnecessary repetitive loadings to limit such influences. Indeed the consistency of the balance point location prior to loading cycles is cited as evidence to the minimal effect of creep on the tissue mechanics. Load rates and durations were also varied to specifically observe the viscoelastic response. To create a combined flexion and compression load the authors simply applied the axial load 4mm anterior to the original balance point, thus generating an eccentric load. The three dimensional kinematics of the FSUs were studied by means of three points fixed to the upper vertebral body and their subsequent deflections. The experimental results demonstrate that the mechanical characteristics of the motion segment are significantly altered by exposure to 1 hour of simulated static sitting and vibration. The major effect appeared to be a softening or increased compliance in the segments exposed, resulting in a greater tendency towards coupled motion. In some instances, following exposure, the motion segment exhibits a "sudden, unstable, combined (flexion and lateral bend) buckling response to axial loading". Tracking tears through, or avulsion of, the annulus was also demonstrated after prolonged combined loading and vibration thus confirming the possibility of herniation

associated with such conditions. These findings are supported by many other *in vitro* studies (Horst & Brinckmann, 1981; Nachemson, 1963) and by much of the work of Adams, Hutton and co-workers (Adams & Hutton, 1982a; Adams & Hutton, 1982b; Adams et al., 1980). Indeed a simple model to explain the effect of disc wedging was presented by Adams and Hutton (Adams & Hutton, 1985a) in their review of the effect of posture on the lumbar spine (Figure 3.1). In this model the viscoelastic or non-linear behaviour of the anterior annulus, under combined bending moments and compressive forces, was shown to protect the nucleus pulposus from excessive hydrostatic pressures. This mechanism thus prevents failure of the vertebral body end-plates, the site of injury in compression of the erect, neutral or moderately flexed spine. In the flexed, or hyperflexed, posture the site of compression injury is the lamellae of the annulus fibrosus. Once end-plate damage has been sustained, however, the load distribution characteristics of the vertebra become permanently altered and begin a process of mechanical disc disruption (Adams et al., 2000a). This latter *in vitro* study has shown that even moderate trauma may be sufficient to initiate these irreversible changes. Compressive forces resulting in a loss of only 1% of motion segment height were adequate in producing these changes, particularly in discs aged 50-70 years. Recent work has shed light on the physiology of the disc in response to mechanical load (Aigner et al., 1998; Bartels et al., 1998; Crean et al., 1997; Duance et al., 1998; Errington et al., 1998; Ishihara & Urban, 1999; Roberts et al., 1998). As the largest avascular structure in the body, the physiological and biochemical changes taking place within living discs have proved difficult to investigate. Evidence, however, from these studies on human and bovine discs is beginning to suggest that cellular and histochemical mechanisms are present *in vivo* and that they respond, sometimes surprisingly rapidly, to changes in mechanical loading of the disc. Changes in the concentration and expression of enzymes, metabolites and structural proteins may help explain the link between mechanical demands and disc and end-plate degeneration (Aigner et al., 1998; Crean et al., 1997; Duance et al., 1998; Errington et al., 1998; Roberts et al., 1998).

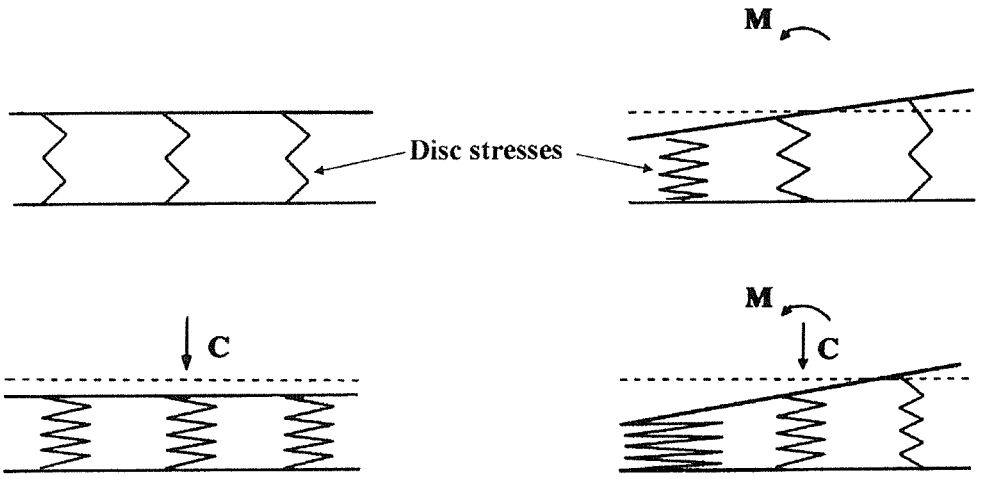
These kinds of experimental studies are important in that they may help explain kinematic behaviour. By improving our understanding of how the disc and other structures behave under differing mechanical conditions *in vitro*, we are better prepared to explain any motion changes witnessed *in vivo*. The sudden change in displacement of spinal specimens noted by Wilder and colleagues (Wilder et al., 1988), for example, may be

analogous to the rapid rotations or irregularities identified during DVF examination in patients suspected of segmental instability (Kondracki & Breen, 1993). A recent update on the work of Wilder and colleagues (Ogon et al., 1997a; Ogon et al., 1997b) has shed more light on this area and will be discussed more fully in Chapter 4.

These studies have also touched upon the issue of viscoelastic behaviour and its role in determining the mechanical responses of the spine to imposed demands. The following section will concern itself with the topic of viscoelasticity and how a better awareness of its effects *in vitro* might shed light on observed *in vivo* kinematics.

FIGURE 3.1

DIAGRAM SHOWING THE RESPONSE OF THE DISC TO WEDGING
(ADAPTED FROM ADAMS AND HUTTON 1985)



C = Compression forces
M = Moments

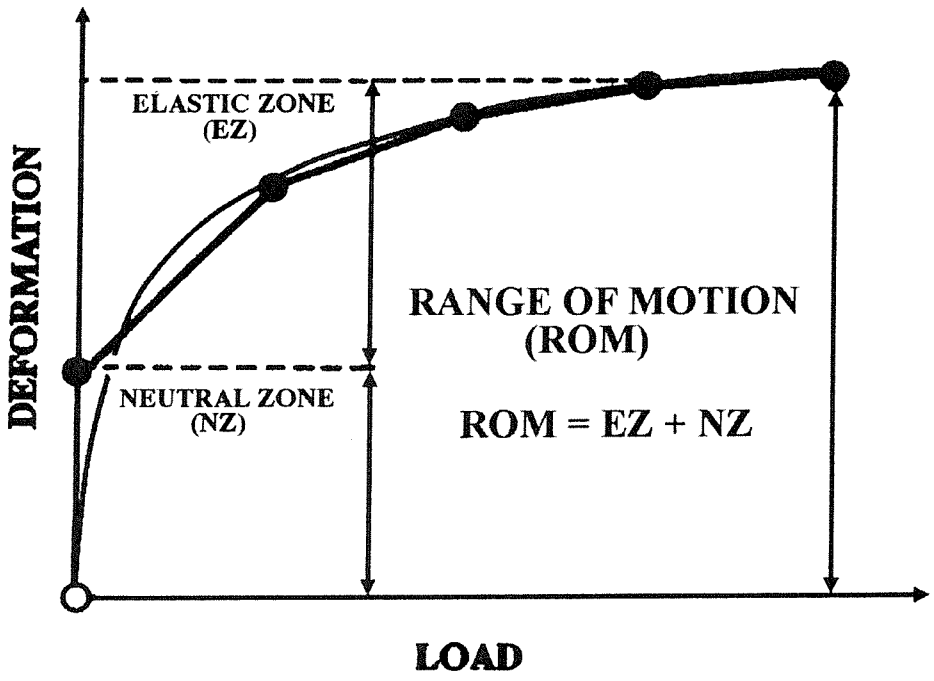
3.5 THE NEUTRAL ZONE

Following on from the work of Wilder et al (Wilder et al., 1988) on human lumbar spine specimens, Panjabi (Panjabi, 1992b) has expressed concern over the methods employed in cadaveric studies. In particular the practice of preconditioning or pre-stressing spinal specimens before load-deformation measurements. This procedure has been used to reduce the viscoelastic effects and produce linear, or near-linear results. In life, however, spinal tissues exhibit highly non-linear behaviour. Indeed this non-linearity in load-deformation may well hold the key to the understanding of spinal dysfunction. Spinal, and many other, ligaments possess the ability to vary their stiffness throughout a range of movement. In other words, stiffness is a strain-dependent phenomenon. This viscoelastic behaviour allows greater movement within and around the neutral position but

progressively limits motion towards the end of the range. The region of relative ligamentous laxity around the neutral position has been termed the "neutral zone" (NZ) and that part of the range of motion associated with increasing ligament stiffness the "elastic zone" (EZ) (Panjabi, 1992b) (Figure 3.2). These zones exist for both rotations and translations in all planes. Furthermore, although no definitive measurement for *in vivo* neutral/elastic zones are presently available, these active counterparts, dependent on resting muscle tone, are thought to have smaller values than their corresponding passive neutral zones.

FIGURE 3.2

DEFINITION OF THE NEUTRAL ZONE
(PANJABI 1992)



A similar finding was noted by Dolan and Adams (Dolan & Adams, 1993) using the 3-SPACE ISOTRAK device (a skin-surface technique) to determine peak flexion angles in human subjects. Using cadaveric data from a previous study (Adams & Dolan, 1991) the authors claimed to be able to convert the *in vivo* measurements of lumbar flexion into bending moments (Figure 3.3). The results of both these studies show the neutral position and much of the flexion range to be associated with low bending moments in the lumbar spine. This suggests that the lumbar spine offers little resistance to bending throughout this range and thus high stresses in the soft tissues are avoided. From this information Dolan and Adams have suggested that subjects with poor mobility in the lumbar spine and hips can generate high, potentially harmful, stresses in the lumbar disc and ligaments on simple forward bending. This is ably illustrated by Figure 3.3, showing that, as the limit of flexion range is approached, relatively small changes in flexion angle result in large bending moments being imposed on the lumbar spine. On the other hand, supple individuals are probably able to touch their toes, for example, without generating these high bending moments.

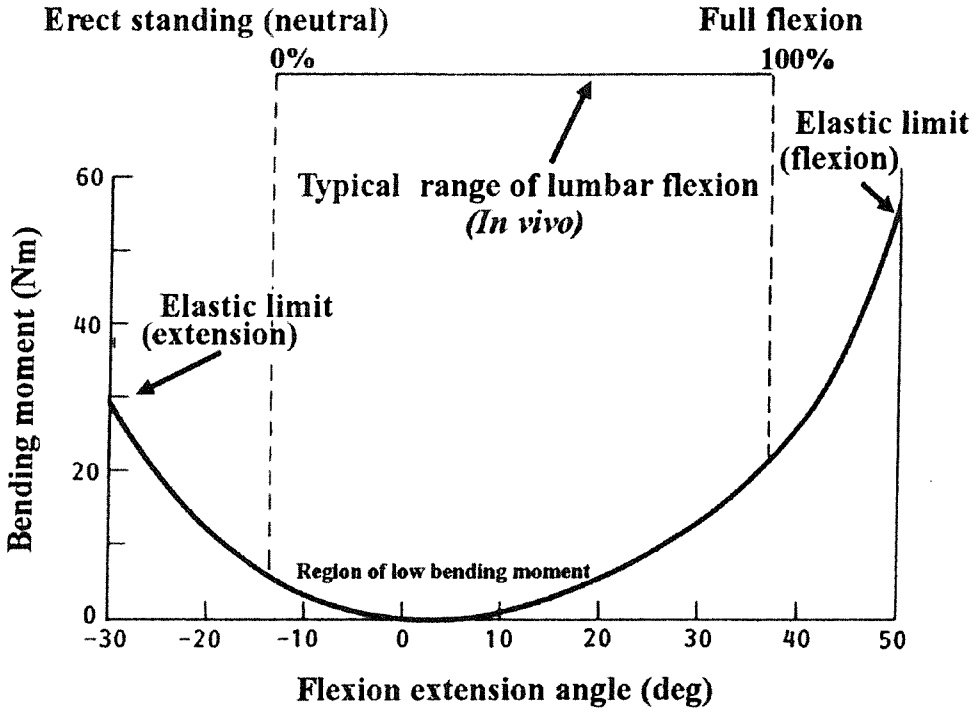
The biphasic nature of spinal motion allows minimum energy expenditure for movements around the neutral position, but provides opposition to potentially damaging movements at the end of range. Of these movements, hyperflexion has probably the greatest deleterious effect on spinal soft tissues. It has been shown that, during full flexion, intradiscal pressure can increase by 100%, under a constant compressive load, as a result of the tension generated in the intervertebral ligaments towards the end of range (Adams & Dolan, 1995).

Utilising data from an earlier study (Yamamoto et al., 1989)), Panjabi (Panjabi, 1992b) has demonstrated a method of measuring the neutral zone *in vitro* and proposed that it represents an index of clinical instability. The procedure for determining the neutral zone involves the repeated loading of a spinal specimen. After removal of the load it was noted that the specimen does not return fully to its initial position but only partially, showing residual displacement. Loading, and hence displacement, can then be undertaken in the opposite direction. When this load/unload cycle is repeated three times, the residual displacement just prior to the third load cycle, for each direction, defines the end of the neutral zone. Further load/displacement from this point defines the elastic zone and the

FIGURE 3.3

DIAGRAMMATIC REPRESENTATION OF THE EFFECT OF THE BENDING MOMENT ACROSS THE LUMBAR SPINE OVER THE FULL RANGE OF FLEXION AND EXTENSION (BASED ON CADAVERIC DATA)

(DOLAN & ADAMS 1993)



point midway between the two neutral zones is taken as the neutral position. Panjabi has shown that the neutral zone is more sensitive to injury and degeneration than the corresponding range of motion a notion that continues to find support in the literature (Panjabi et al., 1998; Tsantrizos et al., 2000; Zhu et al., 1999). As Dolan and Adams (Dolan & Adams, 1993) point out this "region of low bending moment", as they refer to it, is a fairly constant proportion of the range and therefore should not be expected to change under normal circumstances. More recently, a group from Arizona have attempted to

redefine the neutral zone concept (Crawford et al., 1998). These researchers have hypothesized that a different parameter exists, which they have termed the lax zone (LZ), and that it more accurately describes the region of ligamentous laxity than the NZ. Their contention is that the NZ is a smaller subset of the LZ dependent on the frictional characteristics of the joint in question. Crawford and colleagues (Crawford et al., 1998) observed that, using the experimental method for measuring the NZ *in vitro* described by Panjabi (Panjabi, 1992b), the resting position to which the spine returns after loading, was subject to "extreme variation" with small changes in specimen posture. In other words, the upper border of the NZ, and hence the NZ itself, was dependent on alterations in preload and posture and thus susceptible to error if these variables were not controlled. Crawford's group hypothesised that the NZ actually represents a range of spinal orientation where only frictional joint resistance occurs and that a different, less variable, parameter exists that they have termed the lax zone (LZ). This disparity occurs because the neutral position of the spine is influenced by the orientation of, and friction forces within, spinal joints and that a true ligamentous neutral position differs slightly from this. Their contention is that the LZ describes a range of orientations where only minimal ligamentous resistance occurs, irrespective of slight changes in posture and loading. The complex methodology devised by Crawford's team employed six cadaveric cervical spine specimens (C5/6), the experimental results of which determined that the NZ was in all cases smaller than the LZ. This finding supported their hypothesis that the NZ is a subset of the LZ and that both parameters should increase with instability/injury. The authors suggest that the clearest advantage of using the LZ rather than the NZ is that the LZ would be less sensitive to postural shifts. In this regard, any future *in vivo* measurement of LZ, as yet undiscovered, is likely to be more clinically useful since it is impossible to precisely control loading conditions in life.

Although the determination of the neutral zone is an *in vitro* process involving load/deformation data, it may be possible to relate this concept to the time/displacement information generated by DVF studies. Since viscoelasticity is a time-dependent phenomenon one might expect the angular change through the neutral zone, in flexion/extension for example, to be greater per time increment than motion during the elastic zone. Also, since the neutral zone must be found at the commencement of the motion and the elastic zone towards the end of range, by comparing displacement during each half of a motion sequence we have developed a "laxity index" which may be

analogous to the neutral zone concept (Kondracki & Breen, 1993). As yet unproven, this index shows some potential, using DVF, for the *in vivo* quantification of intervertebral laxity and hence acts as a possible parameter of instability.

DVF is, at present, a two-dimensional technique for kinematic analysis. As a uniplanar imaging modality used for dynamic studies of the spine, it is essential to consider the issue of out-of-plane movements. In this regard the most pressing topics are: coupled motions generally and axial rotation specifically.

3.6 COUPLING STUDIES

To fully investigate the kinematics of the lumbar spine and the effects of coupling and other variables, such as posture, necessitates the inclusion of adjacent vertebral levels in the testing procedure. This aspect has not escaped the attention of the *in vitro* investigators and from the earliest days of spinal research whole/intact or long segment lumbar spine specimens have been utilised in biomechanical experiments (Evans & Lissner, 1959; Lovett, 1905). Since the late 1970s there appears to have been a gradual increase in the number of such studies. The application of non-constraining pure moments to whole spine specimens also represents a major shift towards more applicable studies. The work of Goel and colleagues (Goel et al., 1985) has been instrumental in this change of rationale and more recently Lysack and his team have refined and developed these principles (Lysack et al., 2000). Cognisant of the need for continuous loading throughout the neutral zone, Lysack and colleagues have described an apparatus for obtaining data from multi-level specimens across an entire motion sequence. This shift in emphasis and the improved methodologies employed, has been a commendable trend since results obtained from multilevel experiments have much more relevance to normal spinal motion and are more comparable, albeit indirectly, to *in vivo* studies.

The effect of posture on the coupling characteristics of the lumbar spine is at least one area of kinematic concern inaccessible to the methods of single motion segment testing. The coupling patterns of the cervical spine, particularly the lower cervical spine, have been well established (Lysell, 1969; Moroney et al., 1988; Panjabi et al., 1986).

Coupling in the lumbar spine, however, remains controversial especially as regards the association between axial rotation and lateral bending (Pope et al., 1977; Stokes et al., 1981; Tencer et al., 1982). Some researchers report little, or no, such association in the

lumbar spine (Rolander, 1966; Schultz et al., 1979). Whatever patterns of coupling exist in the normal lumbar spine, most authors agree that, for flexion/extension motion, very little accompanying rotations take place in other planes. For axial rotation and lateral bending, however, large accompanying rotations do occur and it is here that most of the controversy exists (Hindle et al., 1990; Pearcy, 1985; Pearcy & Tibrewal, 1984; Plamondon et al., 1988). Coupling patterns may be clinically important and indicate spinal dysfunction (Dupuis et al., 1985; Parnianpour et al., 1988; Pearcy et al., 1985; Pearcy & Tibrewal, 1984; Weitz, 1981). On the other hand coupling characteristics may vary considerably within normal limits and might have a strong dependence on posture and other variables.

Without this fundamental knowledge, observation of coupling patterns *in vivo* has limited clinical significance. In an attempt to address this very question Panjabi and co-workers (Panjabi et al., 1989) applied axial torque and lateral bending moments, separately, to cadaveric whole lumbar spine (L1-S1) specimens. The three-dimensional intervertebral motions of each segment were recorded by stereophotogrammetry and the response to loading studied in five spinal postures (full extension and flexion, half extension and flexion and neutral positions). The authors applied an axial compressive preload of 100 N, to simulate *in vivo* loads, and horizontal forces, either anteriorly or posteriorly, to create the flexed or extended postures. In order to generate lateral bending and axial rotation, only pure moments were applied, through the body of L1, along the relevant axes. This ensured that each intervertebral joint received the same magnitude of moment. The components of the moment vector, however, will vary at each joint as a function of the lumbar lordosis. The moments were applied in three load/unload cycles with a 30-second rest period to allow for creep. Vertebral motion was recorded only after the third load cycle. In other words the specimens were preconditioned in an effort to reduce their viscoelastic properties.

The findings of this study demonstrated that posture and intervertebral level (or intrinsic mechanical properties) are two very important factors in determining the magnitude and characteristics of both the main and coupled motions in the lumbar spine. This study again highlights the functional division between the lumbar and lumbosacral spine. In the neutral position, for example, left axial torque brought about contrasting effects between upper and lower lumbar levels. Upper lumbar segments were driven into right lateral

bending, that is bending to the opposite side of axial rotation. At lower lumbar levels, however, the lateral bending was to the same side, with the L3/4 FSU acting as a transitional segment. The authors noted a distinct lack of mechanical reciprocity in lumbar coupling. In other words, when left axial torque was applied to L4/5, for example, this produced left lateral bending. However, when left lateral bending was applied the coupling was with right, and not left, axial rotation.

Although the distinction between lumbar and lumbosacral levels were not as clear, the findings of this study were in agreement with the *in vivo* findings of Pearcy and Tibrewal (Pearcy & Tibrewal, 1984). In their study the transitional segment for lateral bending direction appeared to be L4/5. The magnitudes of main and coupled motions, however, were remarkably similar. The only other major difference in findings between the two studies was in the associated sagittal plane coupling with axial torque and lateral bending. In addition to lateral bending accompanying the main axial rotation and vice versa, Panjabi and co-workers found a second coupling effect. They noted, in the neutral posture, a sagittal plane rotation, which tended towards flexion at all levels. Pearcy and Tibrewal (Pearcy & Tibrewal, 1984), on the other hand, found the opposite. They noted extension as the predominant sagittal plane coupled motion, with the exception of the lumbosacral segment, which showed an equivocal response. Panjabi and colleagues suggested that this paradox could be explained if Pearcy's subjects were standing in a slightly flexed posture at the time of screening. This, of course, is speculation and the fundamental differences in the two studies make the interpretation of contrasting results difficult. In the Panjabi experiment the active or passive components of the spinal musculature could play no part in coupling effects. With the *in vivo* work of Pearcy and Tibrewal (Pearcy & Tibrewal, 1984), however, muscle influences were present but unquantifiable. In fact these authors suggested that, together with the lordotic shape of the lumbar spine, muscular control is key in determining the nature of combined or accompanying rotations. Nevertheless there was good agreement between findings, despite the obviously dissimilar methodologies, and the complimentary nature of the two papers remains quite unique. It is interesting to note that in a later *in vivo* collaboration (Pearcy & Hindle, 1989) Pearcy (et al's) findings support that of Panjabi and co-workers. Using an electro-magnetic position sensor, the 3Space Isotrak, Pearcy and Hindle showed a strong coupling of flexion with lateral bending.

A recent study combining *in vitro* experimentation and biomechanical/mathematical modelling (Cholewicki et al., 1996) claims results in broad agreement with the *in vivo* work of Pearcy and Tibrewal (Pearcy & Tibrewal, 1984). The authors attempted to distinguish between those coupling effects attributed simply to the degree of lordosis and those arising from the intrinsic mechanical properties of the spine. Their results suggest that lordosis and mechanical properties had an approximately equal effect on predicting coupling between axial rotation and lateral bending. The coupling of flexion associated with lateral bending, however, was thought to be almost wholly a function of lumbar lordosis.

It is interesting to note that, even currently, the effects of the lumbar lordosis, particularly on the biomechanics of lifting, are still not fully understood. Recent conclusions, however, are beginning to agree that full lumbar flexion should be avoided during loading (McGill et al., 2000; Shirazi-Adl & Parnianpour, 1999). These studies disagree with the early work of Adams and colleagues (Adams & Hutton, 1985a), who recommended flattening or flexion of the lumbar spine during heavy lifting. Both of these recent studies suggest that a mildly or slightly flattened spine tends to reduce maximum disc strain and allow optimum function in the back extensor muscles in countering anterior shear forces. Larger flexion angles, however, tend to reverse these changes and place the lumbar segments at risk.

3.7 AXIAL ROTATION STUDIES

Axial rotation of the spine is particularly interesting as it is one of the least studied, but actually one of the most natural *in vivo* spinal movements. In terms of kinematics, axial rotation has been studied mainly in relation to the radiological assessment of scoliosis (Drerup, 1984; Drerup, 1985; Mehta, 1973). The biomechanical investigation of axial rotation or torsion, on the other hand, has focused largely on its role in stability. In comparison to movements in other planes, segmental axial rotation is slight. At individual lumbar levels these movements are not thought to exceed much beyond 1 degree to each side and to this extent it is unlikely that high torsional stresses are generated within the disc (Adams & Hutton, 1981; Cossette et al., 1971; Farfan et al., 1970). Nevertheless, knowledge concerning the *in vivo* stresses acting on the spine with respect to torsion is sparse (Adams & Dolan, 1995). In examining the rotational stability of thoracolumbar specimens (T11-S1) Haher and colleagues (Haher et al., 1989) employed sequential

destruction of the three functional columns of the spine. The anterior, middle and posterior columns, as described by Denis (Denis, 1983), were surgically divided at the L2/3 interspace. Torsional loads of up to 20Nm over 15 degrees were applied to the specimens before and after destruction and load/rotation data plotted. The results showed that the anterior column contributed the greatest effect in resisting torsion. Destruction of the middle and posterior columns alone could only produce a loss of torsional rigidity of less than 35%, the annulus fibrosus again demonstrating its function as the major holding element.

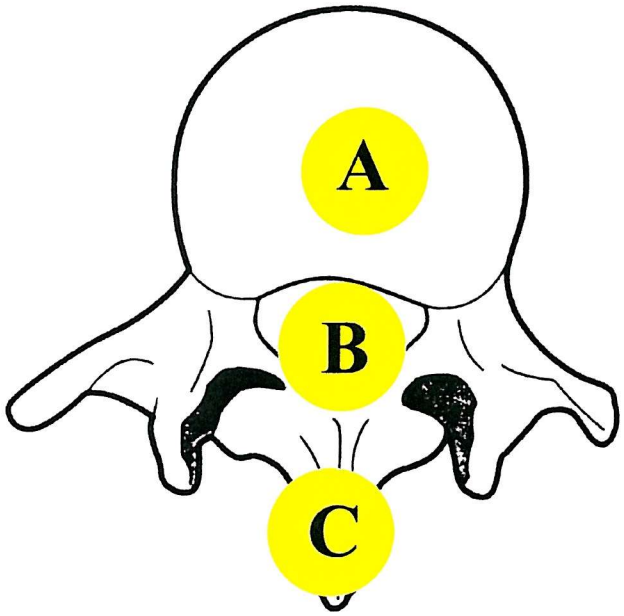
Haher and co-workers undertook a similar study 3 years later (Haher et al., 1992) in which human thoracolumbar specimens (T11-S1) were again sequentially injured and subjected to torsional forces. This study, however, used photography to calculate the IARs for axial rotation. The axis of rotation of the intact lumbar spine was consistently found posteriorly around the facet joints. With destruction of the facet joints alone the IAR is shown to migrate anteriorly and combined annular and facet destruction shifts the IAR posterior to the facet joints (Figure 3.4). Thus sequential destruction seems to cause the IAR to migrate to the remaining intact structures. This study supports their earlier work by demonstrating that the primary rotational stabiliser is the annulus and this is explained because of the distance of its moment arm to the IAR. This study also supports the *in vivo* work of Gregersen and Lucas (Gregersen & Lucas, 1967) in the location of transverse plane IARs for the normal lumbar spine. The authors hypothesised that the transverse plane IARs could be theoretically determined as the intersection of the two perpendicular bisectors of the articular facets. The location of the IARs, therefore, would be a function of the orientation of these surfaces. Using this method they determined that the IARs for thoracic segments would fall within or be anterior to the intervertebral disc and would therefore allow considerable axial rotation. The IARs for lumbar segments, however, would be found posterior to the disc and thus limit axial rotation. By measuring the displacement of Steinmann pins inserted into the thoracolumbar spinous processes of healthy male subjects, Gregersen and Lucas, not surprisingly, observed just such a trend. Before inserting the specially tip-threaded pins into the bony spinous processes, by means of a hand drill, care was taken to make small vertical incisions in the overlying skin to prevent adherence to the pins. The authors also observed a large discrepancy in the magnitude of axial rotation in lumbar

FIGURE 3.4

IARs FOR AXIAL ROTATION

(HAHER ET AL 1992)

Inferior vertebra of an FSU viewed from above



A Location of IAR after destruction of facet joints
B Location of IAR in the intact spine
C Location of IAR after destruction of facet joints and annulus

segments between sitting and standing. This discrepancy was greatest for the lumbosacral joint where the average axial rotation for two subjects was 3 degrees seated compared to 13 degrees standing. In 1991 Pearcy and Hindle (Pearcy & Hindle, 1991), using isolated intervertebral joint specimens, found some intervertebral joints do show an increased ability to axially rotate in sub-maximal flexion, probably as a result of apophysial joint

morphology. In full flexion, however, axial rotation was reduced. The authors suggest this effect is most probably due to tightening of the posterior ligaments and apophysial joint capsules. The study supports the argument that torsion alone is insufficient to damage the intervertebral disc but a combination of flexion and torsion must increase its vulnerability to injury. This association of asymmetrical bending and compression as conditions likely to cause damage to the intervertebral disc, has considerable support (Adams & Dolan, 1995; Adams & Hutton, 1982a; Gordon et al., 1991).

In a more recent *in vitro* study (Gunzburg et al., 1992) a similar decrease in axial rotation was observed during flexion of whole lumbar spine specimens. The aim of this study was to determine the role that each of the capsulo-ligamentous structures play in axial rotation of the lumbar spine. Again the techniques of sequential destruction were employed and angular displacement measured before and after, torsional loads were applied. The specimens were pre-conditioned, i.e. 2 minutes was allotted before measurements were taken in order to allow for creep. After division of the apophysial joint capsules the amount of axial rotation increased significantly for both neutral and flexed positions. The authors, however, point out that although this study demonstrates the importance of apophysial joints in resisting rotation, the resistance is no greater in flexion than in the neutral position. Hence these joints cannot be responsible for the observed decrease in torsion whilst in flexion, as suggested by Pearcy and Hindle (Pearcy & Hindle, 1991). In an earlier study (Gunzburg et al., 1991), Gunzburg combined experimental data obtained from *in vitro* whole lumbar spine specimens and *in vivo* human subjects. Torsion was applied to both groups in a neutral posture and in forward bending and, again, axial rotation was found to be reduced in forward flexion. Interestingly the study also observed the effect of articular tropism (i.e. asymmetrically aligned facet joints) and concluded that it had no influence on the magnitude of rotation. This work lends biomechanical support to the findings of Murtagh and colleagues (Murtagh et al., 1991) who found little, if any, correlation between tropism, facet degeneration and significant disc pathology. In Gunzburg's later study (Gunzburg et al., 1992), it was noted that in some specimens the posterior annulus and posterior longitudinal ligament seemed to limit rotation to a greater degree in flexion than in neutral and suggested that these structures are probably of greatest importance in limiting axial rotation. The supraspinous, interspinous and yellow ligaments were not thought to contribute much in resisting torsion.

CHAPTER 4

SEGMENTAL INSTABILITY

4.1 INTRODUCTION

Fewer concepts in the field of back pain management cause more consternation than that of segmental instability. No acceptable definition appears to exist which successfully combines the clinical, biomechanical and radiological aspects of this perplexing condition (Eisenstein, 1999; Pope & Panjabi, 1985; Sharma et al., 1995).

Knutsson (Knutsson, 1944) originally coined the term "segmental instability", although von Lackum (von Lackum, 1924) had alluded to lumbar spine instability as a possible cause of low back pain in the 1920's. This author considered the lumbosacral joint as inherently unstable by virtue of its transitional nature between mobile and immobile regions of the spine. He was also one of the first advocates of surgical fusion for instability or pain. Surgical fusion remains, at the present time, the most likely procedure for the treatment of segmental instability or intractable back pain (Kanayama et al., 1998; Kotilainen et al., 1997; Papp et al., 1997; Shono et al., 1998).

White and Panjabi (White & Panjabi, 1990) have defined spinal instability as "the loss of the ability of the spine under physiological loads to maintain its pattern of displacement so that there is no initial or additional neurological deficit, no major deformity and no incapacitating pain". The essence of this definition, and most others, is that "normal" loads imposed on the unstable spine lead to "abnormal" deformations or displacement (Frymoyer & Selby, 1985). Some authors suggest that greater acknowledgement should be paid to the magnitude of the destabilising force, or perturbation, required to "upset" the system (Farfan & Gracovetsky, 1984). For others, the emphasis has been on the displacements, believing that instability is always associated with abnormal deformation and loss of tissue stiffness (Scholten et al., 1988).

4.2 RADIOGRAPHIC INSTABILITY

This concept of tissue laxity and excessive movement naturally leads to the conclusion that instability can be defined by vertebral displacements seen on X-ray or other imaging techniques. Indeed, since the work of Knutsson (Knutsson, 1944) instability has,

traditionally, been diagnosed by radiological signs and measuring vertebral displacements from plain lumbar radiographs (Boden & Wiesel, 1990; Dupuis et al., 1985; Frymoyer & Selby, 1985; Morgan & King, 1957; van Akkerveeken et al., 1979). Radiographic evidence considered indicative of segmental instability include traction spurs, narrowing of the disc space (generally or asymmetrically during flexion/extension movements), malalignment of vertebral bodies and abnormal Z-axis (shear) translation either anteriorly (anterolisthesis) or posteriorly (retrolisthesis) (Kotilainen et al., 1997). Other features seen on plain-film X-rays and computerised tomography (CT) scans include the presence of gas in the disc, facet joint degeneration, synovial cysts, capsular swellings or calcification and paraspinal muscle atrophy (Dietemann & Zollner, 1999). Augustus White and colleagues have recently proposed a checklist approach to the radiographic diagnosis of instability (White & Bernhardt, 1999) (Table 4.1).

TABLE 4.1

CHECKLIST FOR DIAGNOSIS OF CLINICAL INSTABILITY IN THE LUMBAR SPINE (WHITE & BERNHARDT, 1999)

Element	**Point value**
Anterior elements destroyed or unable to function	2
Posterior elements destroyed or unable to function	2
Radiographic criteria	4
A. Flexion/extension radiographs:	
1. Sagittal plane translation >4.5mm or 15% (2 points)	
2. Sagittal plane rotation >15^0 at L1/2, L2/3 and L3/4 (2 points)	
>20^0 at L4/5 (2 points)	
>25^0 at L5/S1 (2 points)	
or	
B. Resting radiographs	
1. Sagittal plane displacement >4.5mm or 15% (2 points)	
2. Relative sagittal plane angulation >22^0 (2 points)	
Cauda equina damage	3
Dangerous loading anticipated	1

Total of 5 or >5 points = unstable

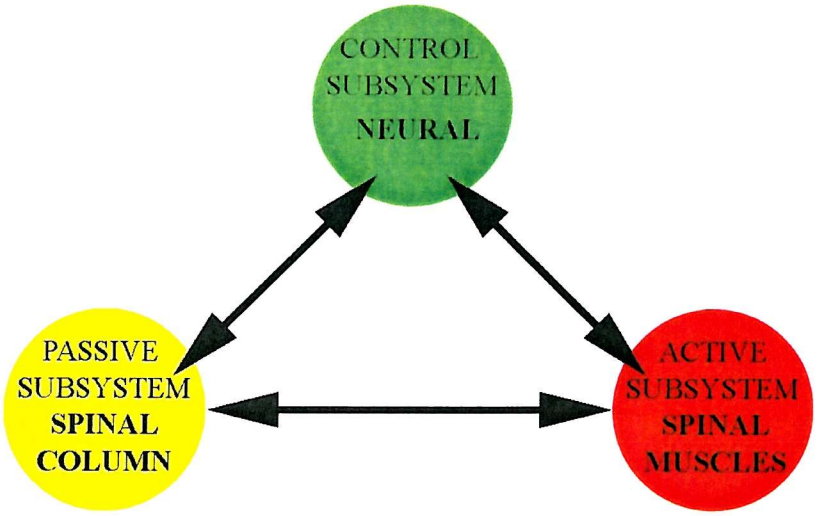
Unfortunately, as with almost all aspects of the human condition, descriptions of this nature raise issues of "normality". Kaigle and colleagues aptly summarise this in their recent paper (Kaigle et al., 1997) remarking; "there is no adequate description of normal motion, and there are large variations in motion, even in asymptomatic people". This is especially true of segmental shear translations, long considered pathognomic of instability, which have steadily defied adequate classification over the years (Hayes et al., 1989; Tallroth et al., 1992). The somewhat arbitrary, but established, watershed value is generally regarded to be 3mm of sagittal plane translation. Thus evidence of translation greater than 3mm or 9% of vertebral body width on flexion/extension radiographs is still, it would seem, considered indicative of instability (Fritz et al., 1998). The problem of translatory measurement, however, is more fully discussed in chapter 2. These challenges have led to novel methods of obtaining images and measuring techniques (Boden & Wiesel, 1990; Friberg, 1987; Putto & Tallroth, 1990). Ora Friberg (Friberg, 1987) attempted to provoke excessive shear translation, during radiographic procedures, in low back pain patients by subjecting them to axial traction and compression. Traction was achieved by allowing them to hang, by the hands, from a horizontal bar and compression was induced by means of a weighted rucksack. Friberg claimed that this technique revealed instability in patients even when conventional flexion/extension films had failed to provoke abnormal movement. However, a more recent comparison study of the two procedures was unable to support Friberg's result (Pitkanen et al., 1997). These authors concluded that the traction-compression method was not useful for the detection of segmental instability either in addition to or instead of the flexion/extension protocol. Intriguingly, as previously mentioned, a study by Wood and co-workers (Wood et al., 1994) has demonstrated promising results in revealing abnormal movements in spondylolisthetic patients suspected of instability. The authors were able to detect, in a majority of these patients, excessive translation only in flexion/extension radiographs taken in a lateral recumbent position. The same patients X-rayed in a conventional, weight-bearing manner showed none of these changes. It should be pointed out, however, that 30% showed abnormal translation in both positions and a minority (13%) displayed more motion whilst standing. Nevertheless, these authors conclude by recommending the recumbent protocol for the evaluation of unstable spondylolisthesis.

4.3 A NEW APPROACH TO INSTABILITY

In 1992 Panjabi published a discussion paper, which attempted to conceptualise a model for spinal stability (Panjabi, 1992a). This model de-emphasised the reductionist approach in favour of a broader view of how stability might be achieved physiologically. The thrust of the argument was that stability is not simply a matter of the passive stiffness of a column resisting buckling. Early attempts to impose the role of stability squarely on the osteo-ligamentous spine showed that the isolated cadaveric spine was remarkably unsuited to supporting loads (Lucas & Bresler, 1961). The critical load for the lumbar spine has been determined, *in vitro*, at around 90 Newtons. Loads greater than this will cause buckling of the isolated lumbar spine. In contrast, physiological loads encountered *in vivo* are thought to be of the order of 1500 N (Panjabi, 1994). The discrepancy between these values underlines the role of spinal musculature, acting as guy wires in stabilisation. The human spine is a dynamic structure and thus stability cannot be reduced to a static resolution of forces. Stability, therefore, must be a function of a rapidly adapting system capable of responding to constantly fluctuating loading conditions. This necessitates the inclusion of neuromuscular elements into any dynamic model of spinal stability. The model Panjabi proposed comprises three interacting subsystems (Figure 4.1) (Panjabi, 1992a).

FIGURE 4.1

PANJABI'S MODEL FOR SPINAL STABILITY (PANJABI, 1992A)



The passive subsystem consists of the solid structures such as the vertebral bodies, facet joints and capsules, discs and ligaments. In addition it also includes the passive mechanical properties of skeletal muscle. It is here that the concept of the neutral zone (NZ) is evident. Around the neutral position the components of the passive subsystem are unable to provide any significant resistance. This subsystem, however, is considered passive only in that these structures, by themselves, do not generate forces or produce movement. Nevertheless, they are dynamic in the sense that transducers, as an integral part of these tissues, are capable of monitoring the mechanical behaviour of the spine

during motion. This information can then be fed-back to the neural subsystem. Since passive elements contribute little resistance throughout the NZ it is likely that, during this phase, they function almost entirely as transducers.

The active subsystem comprises the paraspinal musculature and tendons. These structures generate the forces and moments required in maintaining stability. The force transducers that reside in the muscle tendons, and muscle spindles, are responsible for gathering information on the magnitude of forces being produced by each muscle and as such are part of the neural control subsystem.

The neural subsystem is the "black box" which processes the information received from the various transducers. Acting on this information the active subsystem can then be controlled to achieve the required tension in individual muscles until the conditions for stability are met (Figure 4.1).

The appropriate magnitude of muscle contraction is determined, Panjabi hypothesises, most probably on the basis of information received regarding ligament strain rather than internal stresses. This is particularly likely throughout the NZ where the reactive forces are small compared to the relatively large ligament deformations. This remarkably co-ordinated arrangement is likely to be capable of a great degree of compensation and optimisation and is, furthermore, liable to achieve this in a highly variable fashion. Given that, it is hardly surprising that instability is an elusive beast. With a multitude of compensatory mechanisms in place it is not unexpected that attempts to reveal instability by provocation, a common clinical technique for divulging latent abnormalities, are met with resistance by the patient. Nevertheless, a control system of this nature is, by necessity, complex and must function on an instantaneous basis under almost infinitely variable conditions. It is, therefore, prone to dysfunction. Muscles may be recruited inappropriately, contracting too soon or too late, with insufficient force or too vigorously. Overall the objectives for immediate stability might be accomplished at the expense of long-term component damage. Accumulated injury to various anatomical tissues such as the disc, ligaments and facet joints may result in accelerated degeneration with all its attendant problems of pain and dysfunction. Furthermore, it is not inconceivable that degeneration or damage of this kind can lead to additional stability compromise.

4.4 DEGENERATIVE INSTABILITY

Lumbar instability as a result of severe trauma, neoplastic destruction or infectious disruption is a relatively easy concept to grasp (Galasko, 1999; Nachemson, 1999; Neumann et al., 1995). The controversy arises with instability following degenerative processes (van Akkerveeken, 1999). An example of which is the concept of the degenerative spondylolisthesis. Degenerative spondylolisthesis is considered as a clinical entity although the radiographic findings, in many individuals, are not associated with any history of low back pain (Frymoyer et al., 1990; Kauppila et al., 1998; Mullholland, 1999). The pathomechanics of this disorder are thought to arise from a combination of disc compromise and degenerative remodelling of the facet joints such that they assume a more horizontal position (Eisenstein & Parry, 1987; Giles & Singer, 1997; Jayson, 1992). It occurs almost exclusively at the L4/5 level, where the L5 segment, stiffened by strong lumbosacral ligaments, meets the vulnerable, more mobile L4 vertebra. Progressive disc narrowing combined with osteoarthritic degeneration at the facet joint allows forward drift of L4 on L5. In these cases there is no pars defect and thus the posterior elements are pulled anteriorly with the vertebral body, reducing the cross-sectional area of the spinal canal and predisposing the patient to symptoms of spinal stenosis. Given these facts, it is not surprising that these patients can present with a variety of symptoms from simple backache, facet pain relieved by anaesthetic joint blocking, neurogenic claudication with its picture of bilateral leg pain brought on by walking and eased by crouching, radicular pain and even referral to the testes, groin or perineum and vulva (Jayson, 1992; Mullholland, 1999). As this disorder can be present without pain and with such a wide spectrum of symptom manifestation, it is difficult to see it as a distinct entity within primary segmental instability, although rotational instability has been strongly associated with degenerative instability (Frymoyer et al., 1990). Bogduk, in particular, has voiced concern over this classification (Bogduk, 1997), claiming that; "rotational instability remains only a hypothetical entity". Likewise, since no pars defect is found can degenerative spondylolisthesis be a true spondylolisthesis? It would, as some authors suggest, perhaps be better to refer to this deformity as a pseudospondylolisthesis (Frymoyer et al., 1990).

On the other hand, the isthmic spondylolisthesis at L5/S1 has been conventionally accepted as a stable disorder in the adult spine (Fredrickson et al., 1984; Frymoyer et al., 1990; Pearcy & Shepherd, 1985). Progression of an isthmic, lumbosacral

spondylolisthesis beyond adolescence is traditionally regarded as a very rare event, although the minority of cases that involve the L4/5 level have been associated with a higher probability of progression, pain and instability (Fredrickson et al., 1984; Grobler & Wiltse, 1997). A recent stereophotogrammetric study of motion in the lumbar spine was unable to demonstrate any abnormal segmental movements in a group of spondylolytic patients (Axelsson et al., 2000). These authors used a radiographic technique of motion analysis (discussed more fully in Chapter 1) accepted for its accuracy and concluded that: "The spondylolytic defect in pars interarticularis does not cause permanent instability/hypermobility detectable in the adult patient with low back pain and low-grade olisthesis". It is generally accepted that, in the majority of cases, the anatomical defect in this condition is not associated with pain (Eisenstein et al., 1994; Fredrickson et al., 1984; Kauppila et al., 1998; Libson et al., 1982; Nordstrom et al., 1994). This, in itself, would lend weight to the argument against a classification of true spinal instability. In the minority of patients where pain can be ascribed to a spondylolysis/spondylolisthesis, however, the mechanism is thought to be mechanical stimuli of neural elements within the tissues of the defect itself (Eisenstein et al., 1994; Nordstrom et al., 1994)

It has been a widely received notion that degenerative changes in adulthood will tend to stabilise the spondylolisthesis and inhibit further slip. This long-established view, however, has recently been challenged (Floman, 2000). Yizhar Floman, from Jerusalem, documented the slip progression of 18 patients with previously asymptomatic isthmic lumbosacral slippage over a 6-year period. His findings suggest that disc degeneration at the level of slip can result in further anterolisthesis in adult life and is associated with back pain and even symptoms of spinal stenosis. This study demonstrates that degeneration can compromise the integrity of the intervertebral disc and convert an asymptomatic developmental lesion, present for 20 to 30 years, into a painful, potentially unstable disorder.

Kirkaldy-Willis (Kirkaldy-Willis, 1992) describes a model of spinal degeneration which divides the process into three distinct phases; dysfunction, instability and re-stabilisation. Progression through these stages, according to Kirkaldy-Willis, is not at a constant rate and may differ between individuals. Since the intervertebral disc is considered to be the most important structure in maintaining stability (Dai, 1998), attempts have been made to establish a relationship between disc degeneration and segmental instability (Farfan &

Gracovetsky, 1984; Kirkaldy-Willis & Farfan, 1982; Soini et al., 1991). Using discography and plain-film radiography on a series of 77 patients, this latter group (Soini et al., 1991), concluded that disc degeneration seldom results in abnormal angular movement and instability of the lumbar spine. Furthermore, they suggested that flexion-extension radiography might only have limited diagnostic value. Several years later, a Japanese group using both standard plain-film radiographic methods and MRI imaging of disc changes, were, again, unable to confirm any clear association (Murata et al., 1994). The authors, employing conventional kinematic parameters of vertebral tilting and translation on 109 low back pain patients, could show little correlation with the degree of disc degeneration as evaluated on MRI. Using the methods proposed by Dupuis (Dupuis et al., 1985), measurements of angular and translatory motion were taken from recumbent films. Standing, weight-bearing radiographs were used to measure disc height. With these criteria the authors claimed to identify segmental instability at all lumbar levels, even in patients who appeared to have normal discs or only mildly degenerated ones. More recently a similar study on cervical spine instability and disc degeneration concluded that signs of instability were more likely in the early phases of degeneration (Dai, 1998) thus supporting the work of Kirkaldy-Willis (Kirkaldy-Willis, 1992) and Gertzbein (Gertzbein et al., 1985). Another study employing MRI techniques attempted to use abnormal disc findings to predict lumbar segmental instability (Bram et al., 1998). These authors reviewed case files of 60 patients with both MR images and sagittal flexion/extension radiographs. Instability was, again, defined using measurements of shear translation adapted from Dupuis and colleagues (Dupuis et al., 1985). These measures were taken by radiologists blinded to the MR results of disc abnormalities and instability was assigned where the horizontal translation exceeded 3 mm. They concluded that the presence of annular tears in the disc and traction osteophytes were the findings most related to segmental lumbar instability. These conclusions are interesting but are questionable when the sole basis for the definition of instability rests on a 3mm shear translation. A more recent study has claimed to have established a relationship between disc degeneration, facet arthrosis and segmental instability (Fujiwara et al., 2000a). Again using MRI and the Dupuis method (Dupuis et al., 1985), for determining ranges of rotation and translation, these authors showed a positive association between disc degeneration and anterior translatory instability. Fujiwara's team employed the recumbent radiographic protocol proposed by Wood and colleagues (Wood et al., 1994). This non-weight bearing and unloaded method is thought to reveal abnormal movements concealed

by compression preload. In addition, they noted a negative association with facet joint osteoarthritis and both abnormal tilting movements and anteroposterior translatory instability. In conclusion they suggest that, with increasing degeneration of the disc and facet joints, the disc loses its anterior translational stiffness, but that facet joint osteoarthritis limits abnormal tilting movements and anteroposterior translation. Once again, however, the basis upon which the diagnosis of instability rests is subject to question. In this study, Fujiwara and colleagues subdivided translatory instability into anterior, posterior and anteroposterior on the difference in magnitudes of displacement in flexion and extension. When anterior displacement exceeded posterior displacement, by 1mm or greater, the motion segment was determined to have anterior translatory instability. Their intraobserver error, however, was 1mm for translation and 3.2^0 for rotation. In addition, their sample population comprised 70 patients with low back pain, leg symptoms or both with no matched control group. This approach is likely to lead to false conclusions because of the well-established lack of correlation between degeneration and symptoms. The recent seminal work by Boos and colleagues (Boos et al., 1995), was clearly unable to establish any significant differences between a group of patients with symptomatic disc herniation and asymptomatic volunteers matched for age, sex, and work-related risk factors, in terms of disc degeneration. Studies such as that carried out by Fujiwara and colleagues (Fujiwara et al., 2000a), also tend to draw conclusions about "instability" from samples of patients with nebulous back or leg pain without attempts to ascertain clinical instability. The issue of "clinical instability syndrome", however, will be discussed in the next section.

At this point it is worth, perhaps, a brief mention of the difference between disc degeneration and disc disruption. Much of the "pathology" associated with disc degeneration is viewed as no more than normal age-related change (Bogduk, 1997). Nuclear degradation, initiated by end-plate fracture, is, on the other hand, a process that may result in progressive destruction of the nucleus pulposus and loss of mechanical function (Bogduk, 1991). A recent cadaveric study using stress profilometry on motion segments subjected to minor end-plate trauma, has provided considerable support for this hypothesis (Adams et al., 2000a). This study suggests that minor compressive damage to the vertebral body end-plate can result in decompression of the nucleus and inward collapse of the annulus. The nucleus, now exposed to the blood vascular system for the first time, is likely to initiate an inflammatory or autoimmune response as suggested by

Bogduk (Bogduk, 1991). These changes are, as Bogduk (Bogduk, 1997) puts it, "an active consequence of trauma; not a passive consequence of age". Intuitively, it would seem logical that these profound changes in disc structure and function would be more likely to result in instability than simple age-related change. Nevertheless, confirming the onset of disc disruption *in vivo* is not an easy task and, as yet, no evidence has been generated to suggest a link between this process and true instability.

Since degeneration is seen as a normal consequence of ageing, this would imply that vertebral instability is an inevitability for us all. The argument put forward by Kirkaldy-Willis, that instability is part of the degenerative process is not without plausibility but is difficult to substantiate. If the process of degeneration is universal then it suggests that at some time or another, given a long enough life span, segmental instability is present in all of the population. Since "clinical instability" is not universal, one can assume that, although the anatomical changes may exist, compensatory mechanisms prevent the expression of symptoms. This, perhaps, highlights the problematic nature of the term "instability". As Eisenstein points out, the term "instability" suggests a disease and diseases are usually associated with symptoms or other manifestations of dysfunction (Eisenstein, 1999). As shown above, the definitions of instability have a common thread and inextricably link the term with joint laxity or loss of stiffness. Problems arise, however, when one attempts to explain symptoms or dysfunction in terms of these mechanical alterations. A recent paper involving knee injuries aptly illustrates this issue (Snyder-Mackler et al., 1997). In this study, twenty patients with proven disruption of the anterior cruciate ligament (an intra-articular structure important for knee stability) were functionally assessed and no correlation could be established between their functional ability and degree of joint laxity. Although, perhaps ultimately, associated with a loss of passive stiffness, clinical instability would appear to be a functional disorder dependent on a great number of variables and compensatory changes. It would seem, then, that the concept of lumbar segmental instability in terms of biomechanical parameters or in terms of degeneration is not as helpful in clinical practice as it might be. In the clinical arena, patients present mainly with pain and it is therefore only with the combination of pain presentation and abnormal findings that one can establish a diagnosis. In a recent study of the role of biomechanics in diagnosing instability, the authors recommend that the decision to perform lumbar fusion be based, primarily, on simply identifying a painful degenerated disc (Krismer et al., 1997).

4.5 CLINICAL INSTABILITY

The clinical entity of "lumbar segmental instability" is vague, to say the least, and does not easily stand out as a distinct diagnosis from the morass of conditions comprising chronic low back pain (Szpalski, 1996). In fact where symptoms do exist, chronic, ill-defined, low back pain seems to be the most prevalent (Borenstein et al., 1995; Porter, 1989). The nature of the pain, however, is what alerts clinicians to the possibility of unstable segments. Professor Porter, in his book on the management of back pain, discusses the concept of tissue deformation and how it relates to symptoms (Porter, 1986). For segmental instability to exist there has to be, by definition, deformation of the restraining elements (disc and ligaments) beyond that considered normal. From this idea one could hypothesise that such patients would have pain when these elements are under load. Either in prolonged weight bearing or during postural change and this, indeed, is what Porter describes. Typically, he suggests, these patients will be symptomatic when resisting shear forces during lengthy weight bearing or momentarily when the displaced segment and deformed tissues return to the pre-deformed position. The former occurs when bony posterior elements such as the facet joints fail, in spondylolisthesis for example, and shear forces are resisted only by the ligaments, disc and muscles, which are then prone to fatigue. Patients, under these conditions, will likely complain of pain whilst walking, especially when carrying, or during prolonged standing. They will subsequently find great relief by lying down. This is in some contrast to the vast majority of simple back pain patients who report alleviation of pain during moderate activities such as walking. In the instability patient, pain of sudden onset is described when changing posture. Typically this is seen when the patient extends from the flexed position or when standing from a seated posture. In such individuals the, normally smooth, extension motion of the trunk is disturbed by the sudden pain and the sufferer will often have to complete the movement by using his hands to support his upper body on his thighs. This gives rise to the so-called "extension catch" sign of clinical instability (Frymoyer et al., 1990; Porter, 1989).

Eisenstein (Eisenstein, 1999), however, disagrees with the notion that clinical instability is a vague ill-defined condition. The lumbar instability syndrome, as he sees it, has an easily recognisable presentation. As mentioned above, the exacerbation by activity and relief obtained by rest, particularly in recumbency, is, Eisenstein feels, central to the syndrome. He further describes the typical patient as one in whom sleep is undisturbed and whose

pain is at a minimum on waking but progressively worsens throughout the day. These patients often report frequent "locked back" attacks, which confine them to bed for several days. These episodes of rapid onset, extremely intense pain is typically associated with apparently innocuous changes in posture or other movements. On examination these individuals will frequently display tense, hypertonic erector spinae muscles combined with an antalgic posture or list to one side. Eisenstein also refers to the jerky "ratchet" movements encountered on flexion or extension, as typical of lumbar instability syndrome.

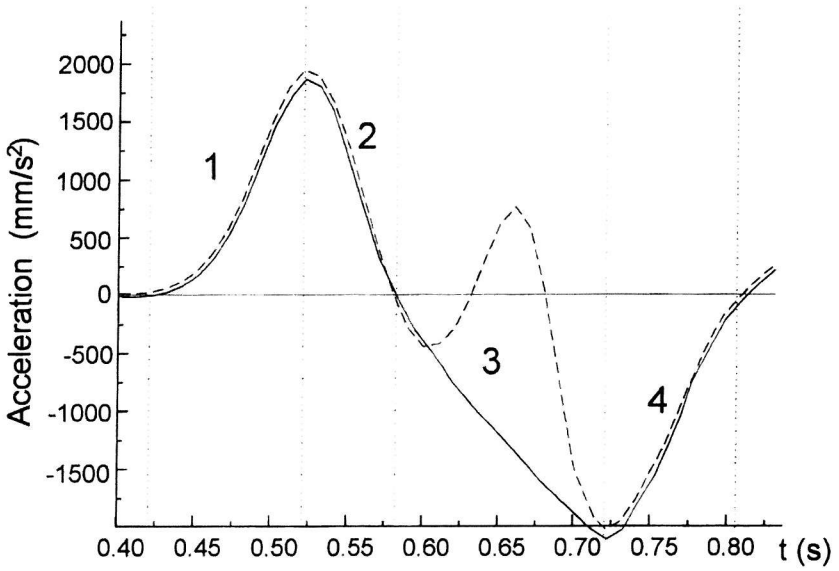
4.6 BIOMECHANICAL INSTABILITY

This sudden "giving way", "catch" or "slipping out" feeling reported by sufferers, has not been ignored by those in the biomechanics field. In their cadaveric study of lumbar disc herniation Wilder, Pope and colleagues noted sudden and excessive translatory or rotary responses during lateral bending or flexion loading, analogous to those described by patients (Wilder et al., 1988). More recently Ogon and co-workers (Ogon et al., 1997b), including Wilder and Pope, updated this study and concentrated their attentions on the motion characteristics associated with these sudden movements or small "jerks". The methodology of this work included moving human lumbar FSUs through the entire range of flexion/extension and right to left sidebending whilst analysing the kinematics by recording the displacement of light-emitting diodes attached to the specimens. Previous *in vitro* studies often separate these movements into single phases, i.e. loading characteristics in flexion or extension only. This practice results in important information regarding stability over the dynamic transition phase between flexion/extension and right/left sidebending, being overlooked or lost (Kaigle et al., 1995; Lysack et al., 2000). Ogon and colleagues (Ogon et al., 1997b), however, specifically targeted this region in order to study the effects of dynamic loading, by a pure moment of 3 Newton-metres, across the whole sequence. They defined these jerks as changes in the acceleration/deceleration patterns of vertebral motion. The normal, overall, pattern of velocity change in an FSU can be subdivided into 4 phases: increasing acceleration (1), decreasing acceleration (2), increasing deceleration (3) and decreasing deceleration (4) (Figure 4.2).

FIGURE 4.2

VELOCITY CHANGE IN THE FSU

(OGON ET AL., 1997B)



Where these small movements, or jerks, caused a slowing down of the motion it was labelled as a *hesitation.* On the other hand where a jerk resulted in a momentary increase in velocity it was defined as *giving way.* These authors also employed a sequential destruction approach to analyse component instability effects. Three conditions were observed, the intact state, post-discectomy and post-facetectomy. Discectomy was performed using a standard surgical approach involving the removal of the ligamentum flavum and as much of the nucleus pulposus as possible. Following the discectomy a facetectomy was performed by excision of the entire right facet joint. The findings of this study suggest that these small *hesitations* or a *giving way*, of around 2 degrees duration, are normal characteristics of fast intersegmental motion in cadaveric specimens. This would support our observations of rapid rotations found during *in vivo* DVF screening of asymptomatic individuals (Breen & Allen, 1996). Furthermore these jerks were found

around the neutral position in the intact FSUs. It is especially surprising that a *hesitation* would occur in the neutral zone since this slowing down would be inconsistent with the region's low resistance. The authors point out, however, that although the neutral zone is associated with reduced resistance to load deformation, the disc, in concert with other viscoelastic tissues, is also responsive to the rate of loading. Thus during the kinds of fast dynamic loading conditions employed in this study, considerable resistance might be met even within the neutral zone. Under the component instability situations, however, it was hypothesised that the frequency of these jerks would increase. This was not the case. The jerks did not increase with component instability but their location within the motion sequence did. It was found that the jerks shifted away from the direction of motion with discectomy and more so with facetectomy. In other words during extension to flexion, (flexion motion) component instability shifted the jerks towards the starting position, i.e. extension. Despite these findings the authors were unable to confirm that these "jerks" and patient reported "catches" were synonymous. Their results actually weigh against this hypothesis since the acceleration rate of the jerks was noted to decrease with increasing component instability. At the same time, however, there was recorded a considerable increase in the maximum acceleration/deceleration values. This, the authors suggest, may indicate a protective role for these jerks, in that a *hesitation* may slow down the maximum acceleration and *giving way* reduce maximum deceleration. In this way rate-sensitive tissues may be protected from rapid loading. Ogon and colleagues also discussed the role of the jerks in the intact specimens. The presence of these movements during the neutral zone was, as mentioned earlier, somewhat of a surprise. Nevertheless, Panjabi has postulated that "micromovements within the physiological NZ may provide the necessary signal to the neuromuscular system for the proper functioning of the spinal stabilising system." (Panjabi, 1992b). Thus the jerks might be the micromovements required to trigger the co-ordinated contraction of deep muscles thought to be essential for spinal stability.

4.6.1 ACTIVE FACTORS

This concept that spinal stability is a function of both passive and active elements connected via a controlling neural subsystem is a compelling one (Panjabi, 1992a). A recent paper by Kaigle and colleagues (Kaigle et al., 1998) provides a great deal of *in vivo* support for this model. This cleverly designed study involved measuring intervertebral

motion, overall trunk motion and myoelectric activity simultaneously in two groups of subjects. Seven patients with chronic low back and six asymptomatic subjects were studied. All seven of the patients were suspected of lumbar segmental instability based on clinical and radiographic findings. These included chronic low back pain of greater than 3 years duration, difficulty in spinal flexion or rising from the flexed position and two or more radiographic changes at the suspected level. The four radiographic criteria considered were: anterior-posterior vertebral translation of greater than 3mm on single static X-ray, angulatory disc space collapse with translation viewed on flexion/extension radiographs, disc space narrowing and traction spurs. Although these criteria fall short of the 5-point Clinical Stability Scale suggested by White and colleagues (White & Bernhardt, 1999), they are, nonetheless, probably more typical of the criteria employed by the majority of clinicians. Nevertheless, it is surprising that a single static radiograph was used to assess translation since it has been known for some time that static AP slip on a single film is not representative of motion or instability (Boden & Wiesel, 1990). Intervertebral motion was measured, non-radiographically, using a new linkage transducer system attached via steel pins into the spinous processes of the motion segments under study. This system was claimed to record displacements with a root mean square (RMS) error of 0.4 degrees and 0.14mm and is similar to the device Kaigle had presented in an earlier work (Kaigle et al., 1992b). Trunk movements were measured using a potentiometric goniometer and myoelectric activity in the lumbar erector spinae muscles by means of surface electromyographic (EMG) electrodes. The main objective of this study was to examine the intersegmental changes occurring during the "flexion relaxation" (FR) response. This phenomenon was described, in 1955, by Floyd and Silver (Floyd & Silver, 1955) and is, in effect, a spontaneous reduction or relaxation in muscle contraction seen at the end of forward trunk flexion in normal subjects. It has been noted by many authors since its discovery and is thought to be due to inhibition of muscle activity initiated by receptors in spinal ligaments, which are activated by the stretch encountered during full flexion. Recent studies have also established that the absence or modification of the FR response in patients with chronic low back pain is a reliable indicator of back muscle dysfunction (Ahern et al., 1988; Ahern et al., 1990; Andersson et al., 1996; McGill & Kippers, 1994; Toussaint et al., 1995; Triano & Schultz, 1987). Absence of the FR response has also been observed in patients with disc herniation (Haig et al., 1993). The FR response, however, is not a simple on/off switching but may, in fact, be a complex and intricate co-ordination of load sharing between the lumbar and thoracic erector spinae and

the passive holding elements (Toussaint et al., 1995). Toussaint and co-workers showed that as the myoelectric activity during flexion diminished, as measured over the lumbar region (L3), the thoracic EMG (T9) became active. The authors point out that this arrangement, due to the differences in force vectors between these two divisions, may have a role in preventing tissue damage at the extremes of flexion. Referring to work by Macintosh and Bogduk (Macintosh & Bogduk, 1991), they note that as the obliquity of insertion changes from the thoracic region to the lumbar region, this may result in the thoracic erector spinae having a protective effect in reducing lumbar compression. Kaigle and colleagues (Kaigle et al., 1998), in agreement with previous authors (Ahern et al., 1988; Sihvonen et al., 1991; Triano & Schultz, 1987), showed a significant difference in FR response between patients and controls. Within the control group they were able to demonstrate a 78% reduction in muscle activity at full flexion. Most of the patient group, on the other hand, showed no reduction in myoelectric activity at all. More importantly than this, perhaps, were the accompanying kinematic changes. In the asymptomatic group, intersegmental flexion was found to have reached its maximum value well before trunk rotation was complete. Thus, it would seem that in the normal spine, segmental rotation could be expected to plateaux before trunk motion reaches its maximum. In these individuals a normal FR response is noted. In the patients where segmental rotation was not complete before full trunk flexion, FR was absent. The authors conclude, therefore, that the FR response is perhaps triggered by full segmental rotation. As with Panjabi's hypothesis discussed earlier (Panjabi, 1992b), these findings suggest that the neuromuscular co-ordination required for spinal stability may depend on triggers related to intersegmental movements. Inferences of this nature tend to highlight the limited usefulness of devices such as Isotrack, which measure gross trunk mobility. However, if the ratio of trunk motion and intersegmental motion can be used as a parameter of function/dysfunction it argues, favourably, for the combined use of trunk and intersegmental measures. Of some significance was the related finding, in this study, that the maximum range of motion did not necessarily occur at the endpoints of flexion or extension. Kaigle had shown this in a previous paper on experimentally induced degeneration on a porcine model. Not only was it noted that the maximum range and end range were often different but that the maximum range of motion was found to be much more sensitive in highlighting differences between intervention and control groups (Kaigle et al., 1997). The authors point out the necessity of using dynamic techniques of analysis, which encompass the whole range of motion, especially including the neutral zone.

4.6.2 AXIAL TRANSLATION

Returning to the most recent work of these researchers (Kaigle et al., 1998), further differences between the patient group and controls have raised some interesting issues. Overall those in the patient group, suspected of segmental instability, demonstrated reduced ranges of motion including anteroposterior or shear translation. Traditionally it has been thought that instability would manifest as an increase in this kinematic parameter (Gertzbein et al., 1984; Nachemson, 1985; Seligman et al., 1984). This, together with work outlining the difficulties in establishing normal values, brings into question the notion of using shear translation as an indicator of instability. Axial, or vertical, translation, on the other hand, showed some curious characteristics. The superior and inferior displacement of vertebral bodies seems to have been largely ignored in most kinematic investigations but in this study appeared to be the most sensitive parameter to change between the groups. Overall axial translation for the patients was significantly reduced when compared to controls. Again the persistent activation of muscles in the patient group thought to be responsible for the reduction in the ROMs may have a compressive effect on the FSU. This effect might well inhibit distraction or axial translation. Not surprisingly the maximum range of motion for axial translation in the control group was associated almost solely with flexion. For the patients, however, the maximum range, although of a lesser magnitude than that of the controls, occurred earlier in the sequence and included the neutral zone. That the lumbar segments should distract during flexion is not unexpected since this is where the tensile forces are generated. Likewise, resistance to these forces are supplied, in the main, by the capsules of the facet joints and the disc (Adams et al., 1980). Other authors (Gracovetsky et al., 1990; Toussaint et al., 1995) have noted axial translation and its effect on the lengthening of the lumbar spine during bending. Alternatively, reductions in spinal length, "spinal shrinkage", have been used to quantify occupational loads imposed on the spine (Leivseth & Drerup, 1997).

In this light axial translation can be seen as an entirely passive phenomenon. The conditions under which it occurs or not, however, may provide more of an insight into the role of active tissues. In an earlier work on an *in vivo* porcine model, Kaigle and colleagues attempted to experimentally induce instability by graded injuries to the disc and facet joints (Kaigle et al., 1995). Using a similar methodology the authors showed significantly greater ranges of axial translation, during flexion/extension, immediately

following injuries to the disc. Injuries to the facet joints, however, were associated with an increase in sagittal rotation and a decrease in shear translation when compared to the sham procedure (intact segment). In a more recent related study, the same porcine model and methodology was employed to study the effects of experimental degeneration on spinal mobility and stability (Kaigle et al., 1997). In this study the graded injuries were specifically intended to produce accelerated degenerative changes in the spine. Accordingly the kinematics was not studied until three months following the injuries. With this approach it was possible to induce chronic degenerative lesions in the pig lumbar spine similar to those seen in human degenerative discs and facet hypertrophy. Again it was found that the most significant kinematic changes caused by the lesions were not those of sagittal rotation or shear translation but of axial translation. For all lesions of the disc and most of the facet injuries, excepting that of bilaterally removing the articular cartilage of the articular processes (Facet Joint Slit), axial translation increased. In an *in vitro* study of human FSU specimens, Ogon and co-workers established a similar increase in axial translation following surgical discectomy and unilateral facetectomy (Ogon et al., 1997a). Recently, a rather crude study of low back pain patients and controls demonstrated a slight difference in lumbar spine elongation during gravitational traction (Tekeoglu et al., 1998). Thirty low back pain patients diagnosed with disc bulging, disc degeneration and segmental instability were compared to 30 age, sex and weight matched controls. No details, however, were supplied on how these diagnoses were reached. By suspending patients and subjects in an upright position during radiographic procedures, these authors showed that the L1-S1 distance increased by 25mm in the patient group compared to 20mm in the controls. Although not an enormous difference between the two groups, it does perhaps lend support to the notion that an increase in axial translation may be a sensitive measure of loss of stiffness. Translation may indeed turn out to be an important factor in assessing instability, it is possible, however, that we have been concentrating on the wrong axis of translation.

4.6.3 HYSTERESIS

In order to fully assess the effects of interventions on the pig lumbar spine *in vivo*, Kaigle and colleagues collected data during both the loading and unloading phases (going into and returning from full flexion) (Kaigle et al., 1995; Kaigle et al., 1997). This was done to record changes in hysteresis behaviour. Both of these studies also included bilateral

stimulation of the paraspinal muscles as an intervention to investigate the influence of active elements on kinematic characteristics. In the earlier paper on experimental instability, Kaigle showed that an intact disc displayed considerable hysteresis in axial translation but only slight hysteresis in shear translation. The overall effect of muscle stimulation, however, was to cause significantly greater ranges of motion in sagittal rotation and shear translation but reduced axial translation range and hysteresis (Kaigle et al., 1995). The more recent study (Kaigle et al., 1997) involved chronic changes and demonstrated some interesting features. As with the previous work there were discrepancies between the maximum range of motion and the end ranges, particularly for axial translation. The maximum range, in the intact FSUs, tended to occur between two-thirds of the way into flexion and halfway into extension, i.e. through the neutral zone, and in magnitude, approached a 40% greater value. This clearly suggests that static flexion/extension radiographs will grossly underestimate true values of axial translation. In terms of muscular stimulation, only the Facet Joint Slit group showed significant changes to ROM. These changes were similar to those seen in the earlier study in which sagittal rotation and shear translation maximum ROMs were increased. For the sham group, with intact FSUs, paraspinal muscle stimulation reduced hysteresis for both rotation and shear translation with significant reduction primarily within the neutral zone. In the groups with more destructive facet lesions, muscular stimulation tended to increase the hysteresis. This at first seems puzzling, the authors, however, hypothesise that the destruction of the facet joint capsule associated with these lesions, disrupt the proprioceptive nerve fibres and hence interfere with the neuromuscular feedback system. Interesting differences were also noted between the disc-injured groups. In those where the lesion disrupted the annulus but did not penetrate the nucleus, an unhealed cavity was produced over time. This was sufficient to affect the segmental stability, manifested by an increase in axial translation range and hysteresis. Lesions that penetrated the nucleus, over time, stimulated more severe degenerative changes including fibrosis and osteophyte formation. This, it was proposed, had a restabilising effect, which reduced the hysteresis in these segments. Muscular stimulation had the effect of increasing hysteresis for the disc annulus lesions but slightly reducing the hysteresis for those segments with penetrating lesions of the nucleus. In these latter lesions there was a marked loss of disc height associated with the degeneration of the nucleus. This, it was suggested, may cause a slackening of the resting paraspinal muscles leading to an increase in laxity. During muscular stimulation this slack would be taken up and might be responsible for the greater

differential in hysteresis behaviour. These findings may help explain, at least partially, why clinical instability is such an elusive diagnosis. It is evident from these data that instability at a given segment is dependent on a number of factors such as type of lesion, resting muscle length and integrity of articular neurology. It is also clear that mechanical conditions involving loading phase and pattern of motion, for example, are important variables in determining which segments display unstable behaviour and which segments do not. Conclusions drawn from the previous work (Kaigle et al., 1995) aptly apply to both of these revealing studies. Overall, paraspinal muscle stimulation would appear to increase the ROM in rotation but have a stabilising effect by reducing the "abrupt patterns of motion in the neutral region" in the injured motion segment. These studies also show that within the neutral region, where muscles are under reduced tension, the FSU is particularly prone to instability.

4.7 MULTIFIDUS

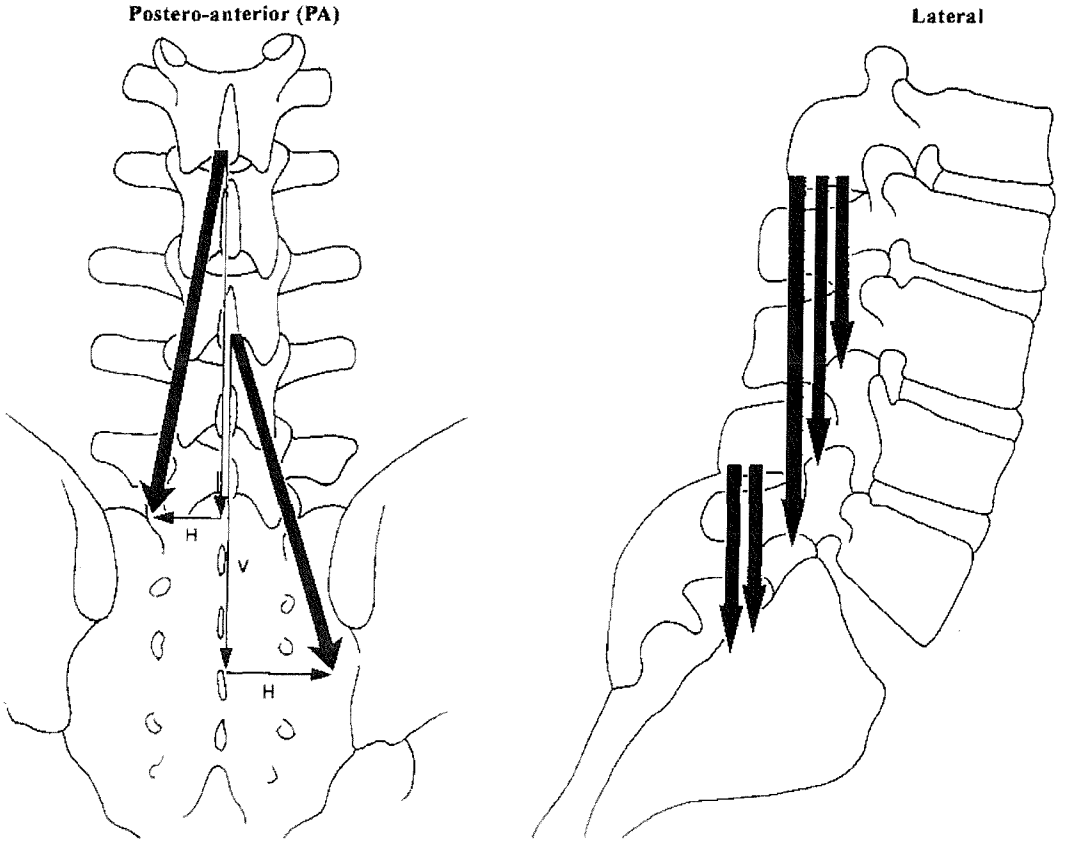
It is clear that the large superficial muscles, which connect the thoracic cage to the pelvis, such as quadratus lumborum and rectus abdominus, exert probably the greatest stiffening effect to the lumbar spine. However, even when forces in these large muscles are high, lumbar segments will buckle if no force is generated by the multifidus and lumbar erector spinae (Cholewicki & McGill, 1996). Bergmark (Bergmark, 1989) has suggested that the number of active elements (muscles) acting on the lumbar spine is far in excess of the minimum required to maintain static equilibrium. Bergmark speculates that this apparent "overkill" provides stability virtually independent of posture but, necessarily, involves complex neural control. In this paper, the author proposes that stability is achieved by two active systems, global and local. The global muscle system include the large superficial muscles that do not arise from or act directly on lumbar segments, but nevertheless have considerable influence on them. The local muscle system, on the other hand, comprises those smaller muscles whose origins and/or insertions are found on lumbar vertebrae. Interestingly, Bergmark also included all of the passive joint properties within the local stability system. The global system, it would appear, is concerned with the distribution of external forces over the trunk, whereas the local system performs actions, which are essentially locally determined (i.e. by the posture of the lumbar spine). Overall stability, therefore, is a function of a complex interplay between these two systems. Furthermore, segmental instability can be reduced by increasing stiffness (activity) in these smaller local

muscles or by increasing stiffness in the passive joint tissues. Since changes in stiffness in the active elements, is much easier to achieve than changes in the properties of the disc and ligaments, it is logical to assume that this would be the preferred method (Cholewicki & McGill, 1996). These authors have attempted to model the various components of stability under a number of *in vivo* loading conditions. Their findings suggest that activity in the small intrinsic muscles of the lumbar spine, by as little as 3% of maximal voluntary contraction, may be sufficient to maintain stability.

The possible role of the multifidus in lumbar stability has been speculated on for some time now (Lewin et al., 1962). Over recent years attention on this muscle, from all fields involved in back pain, has increased (Cholewicki & McGill, 1996; Hides et al., 1994; Indahl et al., 1997; Macintosh & Bogduk, 1986; Macintosh et al., 1986; Solomonow et al., 1998; Stokes et al., 1992). In line with Panjabi's hypothesis regarding the systematic control of spinal stability (Panjabi, 1992a) it is likely that, in life, the neuromuscular component is perhaps the most important aspect. This obviously involves a very complex interplay between the control subsystem (neural) and active subsystem (muscles). Although by no means the only element, the multifidus muscle is increasingly being viewed as the major active component of lumbar segmental stability. This is perhaps not surprising given its unique anatomy. The multifidus is certainly the largest deep muscle to cross the lumbosacral junction and the most medial. Although its gross morphology appears homogenous, closer examination reveals it to be composed of five separate bands each receiving a distinct segmental innervation. This anatomical arrangement fits well with its possible role as a segmental stabiliser (Macintosh et al., 1986). Its principal action is in extending the lumbar segments and countering the large flexion moments acting over the spine and particularly those over the lumbo-sacral joint (Macintosh & Bogduk, 1986). This is achieved without any shear translation due to the near perpendicular attachment to the spinous processes. Their fixture to a structure, which is so posterior to the axis of sagittal rotation, also invests them with considerable mechanical advantage (Figure 4.3).

FIGURE 4.3

FORCE VECTORS OF THE LUMBAR MULTIFIDUS. THE LATERAL VIEW CLEARLY SHOWING THE PERPENDICULAR ATTACHMENTS TO THE SPINOUS PROCESSES (ADAPTED FROM (BOGDUK, 1997)).



Although the muscle has no shear translatory effect, it may have a role in axial translation. A recent EMG study of the multifidus suggests that when bilaterally active, over the same FSU, the muscle has a function in resisting distraction of the two segments (Solomonow et al., 1998). This perhaps helps to explain why axial translation might be greater in back pain patients, since multifidus dysfunction can be demonstrated in these patients (Hides et al., 1994). Solomonow and co-workers (Solomonow et al., 1998) attempted to establish if a reflex arc exists between the supraspinous ligaments and the multifidus. The broad objective of the work was to quantitatively verify the notion that spinal ligaments,

although passive limiters of motion, may have a greater role in the neuromuscular control of stability. They point to the now established view that spinal ligaments are well endowed with sensory mechanoreceptors (Rhalmi et al., 1993; Yahia & Newman, 1991; Yahia et al., 1988). Their own study involved electrically stimulating some of these receptors in the L2-L4 supraspinous ligaments in three human patients undergoing spinal surgery and recording any EMG changes from the multifidi over these levels. Twelve cats also underwent the same procedure over the levels L1-L7. The experiments did, indeed, show that such a reflex exists and that mechanical deformation of the supraspinous ligaments led to activation of the multifidus muscle at the involved level and at least one level above and or below. The net effect of this stimulation was, not surprisingly, to stiffen the motion segment and provide resistance to anterior flexion and distraction moments or forces. Adams et al (Adams et al., 1980) have shown that the supraspinous ligaments contribute little to the mechanical resistance to flexion, which is achieved mainly by the facet capsules and the disc, and in fact are the first structures to be damaged immediately after the limit of flexion is exceeded. This might suggest that their role is primarily that of a transducer for fine control of the multifidus. In this way the multifidus muscle can act to strengthen and stabilise the passive tissues such as the facet joint capsule and the intervertebral disc, when the spinal unit is subjected to destabilising forces. Recent experimental work involving a porcine model supports this view and highlights the role of disc tissues (Indahl et al., 1997). In this simple but elegant study, electrical stimulation of nerve fibres in the disc annulus were shown to elicit reflexes in the multifidus and longissimus muscles. Further, it was shown that injection of saline into the facet joint capsule, simulating stretch of these tissues, reduced this muscular response. The authors suggest that these reflexes form the basis of the flexion-relaxation (FR) response and demonstrate a complex interaction between the neural components of the passive tissues and paraspinal muscles. As Solomonow and colleagues (Solomonow et al., 1998) argue "In essence, if such is the case, the multifidus muscles could be designated as active ligaments, capable of increasing and decreasing their tension on neural control, as opposed to passive ligaments with fixed stress-strain relationships". Of particular interest, in the Solomonow study, was the finding that the load required to elicit a muscular response had a definite and relatively high threshold. A result of this nature tends to support the neutral zone concept, implying that muscle activity would not be triggered until the segment had moved beyond the neutral zone. In an attempt to separate the effects of mechanoreceptors situated in other tissues and stimulated by segmental movement,

Solomonow loaded free segments in the cat lumbar spine before repeating the trial in segments that were rendered immobile by external fixation. In the latter group this ensured that no vertebral movement was possible and that any effect produced would be from isolated supraspinous deformation only. These feline preparations required a load of around 12 to 23% of body weight in the freely mobile segments but required approximately 23 to 42% of body weight to initiate muscle contraction in the immobilised segments. This would suggest that isolated sensory input will require substantial loading to reach threshold, whereas combined mechanoreceptor stimuli from various spinal tissues will converge to regulate joint stability at lower loads. Of further interest was the ascending and descending divergent stimulation from both free and immobile segments. The free segment loading caused activity in multifidus branches, albeit at a lower level, up to three vertebral levels above and below the stimulated segment. In the immobilised segments this activity was seen only at one to two levels above and below. Such a finding would indicate a high degree of neuromuscular co-ordination in the control of segmental stability. As the authors point out, this has important clinical ramifications in that disease or injury at one level may predispose an individual to instability at adjacent levels. This, by chance, was illustrated in one of their patient subjects who had a spondylolisthesis and herniated disc. On EMG testing this individual showed no response to stimulation of his supraspinous ligament, indicating a neurological deficit interrupting the reflex arc. It is therefore conceivable that such lesions could lead to a susceptibility to unstable behaviour. This concept is supported by the earlier work of Stokes and Gardner-Morse (Stokes & Gardnermorse, 1995). Using a three-dimensional lumbar spine model incorporating multijoint muscles, they showed that changes in stiffness at the motion segment had profound effects on the way spinal muscles are recruited and loads transmitted through the lumbar spine. Furthermore, they predicted that an increase in stiffness as a result of age, injury or degeneration would cause a corresponding rise in muscle force and hence predispose the spine to further injury. There does, however, appear to be certain limiting factors in reducing the effect of some neurological damage. Unlike the mechanoreceptor input, motor control of the multifidus is confined to the involved segment. That is, that the efferent innervation of the multifidus muscle is unisegmentary, such that each band of the muscle receives its motor stimulus from one dorsal ramus only. Thus fascicles from the L1 spinouses, for example, are innervated only by the medial branch of the dorsal ramus of the L1 spinal nerve (Kalimo et al., 1989). In the case of a root compression by a herniated disc, therefore, the neurological deficit can be expected to affect only the muscle

fascicles originating from the vertebra with the same segmental number as the compressed root. This might act to minimise loss of neuromuscular control in these lesions and again, perhaps, highlights the importance of the multifidus in segmental stability. The role of multifidus was, once more, brought into relief in an *in vitro* study focusing on the influence of muscles on lumbar stability (Wilke et al., 1995). Using whole lumbar spine specimens (L2-S1) with attached steel cables to simulate muscle forces, Wilke and colleagues have shown that, in general, these forces stiffen the motion segment. This stiffness was manifested by a reduction in both ROM and neutral zone, when measured at the L4/5 FSU. Furthermore, the simulated multifidus muscle forces accounted for more than two-thirds of this effect. The authors do, however, draw attention to the limitations of their *in vitro* work. The simulation included only five muscle pairs and they freely admit that knowledge concerning the combination of muscle forces *in vivo* is unavailable and that these studies are, at best, crude estimations. Nevertheless they conclude that work of this nature is important and recommend that future *in vitro* studies include the effects of at least some of the lumbar musculature. In a recent elaboration of this work, Quint and colleagues (Quint et al., 1998) again used whole lumbar spine specimens to simulate the effects of muscle coactivation on the mechanical behaviour of the L4/5 FSU. Coactivation is, essentially, the combined contraction of agonist/antagonist muscle groups and is thought to be a strategy for maintaining lumbar spine stability in the face of unexpected perturbations (Cholewicki et al., 1997; Gardner-Morse & Stokes, 1998). In the present context this generally refers to anterior flexor muscles such as rectus abdominus, abdominal obliques and psoas, for example, coactivated with the posterior extensor group comprising multifidus and erector spinae. Quint and co-workers (Quint et al., 1998) simulated the coactivation of the psoas major and multifidus muscles using a similar protocol to that used by Wilke (Wilke et al., 1995). Results from this study suggest that coactivation may stiffen the segment in lateral bending and axial rotation but destabilise it in flexion-extension. These findings are somewhat at variance with those of other authors who see coactivation as an unequivocal stabiliser of the lumbar spine (Cholewicki et al., 1997; Gardner-Morse & Stokes, 1998). The *in vitro* model employed by Quint's group, however, had significant limitations. Stability itself was not quantified, only ROM was measured and no attention paid to possible changes in the neutral zone. The application of flexion-extension moments together with simulated coactivation resulted in a 13% increase in sagittal ROM, which was suggested as indicative of instability. In addition no axial preload, simulating body weight, was applied to the whole

lumbar spine specimens during testing. Recent work has implied that antagonistic muscle coactivation is responsive to changes in axial preload (Cholewicki et al., 1997). This notion has been given further support by a recent *in vitro* study using whole lumbar spine specimens (Patwardhan et al., 1999). This cleverly designed study involved the novel application of axial preload by means of cables along a path that approximated the centres of rotation of each segment. In contrast, previous studies (Crisco & Panjabi, 1991; Lucas & Bresler, 1961) had used vertical loads applied to the superior end of the specimens, which had resulted in spinal buckling at loads far less than encountered *in vivo*. Patwardhan and colleagues (Patwardhan et al., 1999) have suggested that their new "follower load" technique can simulate, realistically, *in vivo* compressive preload. Their results have shown a marked increase in the load-carrying capacity of the lumbar spine under these preload conditions and, as they suggest, might explain how the whole lumbar spine can be lordotic and yet resist large compressive loads. They propose that their work may show a mechanism by which muscles can stabilise the lumbar spine under the kinds of compressive loads experienced *in vivo*.

A further limitation of the Wilke study (Wilke et al., 1995) relates to the constant force magnitudes generated throughout the trial. These measures, required to reduce complexity of the model, by necessity, cannot simulate the subtleties of real-life neuromuscular co-ordination. In these respects, *in vitro* models such as this one, are probably too far removed from reality to be of significant value in isolation. The authors themselves comment on these limitations and suggest that future studies should combine *in vivo* and *in vitro* approaches. A recent *in vivo* EMG study of 10 asymptomatic adults attempted to investigate trunk flexion-extension coactivation around the neutral position and whether any changes were noted with increasing loads (Cholewicki et al., 1997). The authors concluded that muscle coactivation is likely to provide mechanical stability around the neutral zone. One of the significant findings of this work was the high variability in the patterns of coactivation for similar loading conditions. This, Cholewicki and colleagues point out, is not surprising considering the vast redundancy in the response of the neuromuscular system. The greatest differences involved the multifidus and abdominal internal oblique muscles. Regardless of other changes, either one of these muscles were often recruited, at a constant level, throughout the motion. This constant level of contraction was maintained despite the change in trunk angle. It seems, from this data, that coactivation is necessary for lumbar stability but can be achieved in a variety of ways.

Likewise, the neuromuscular system can, and does, recruit a variety of different flexion/extension muscle pairs the most important of which seem to be the multifidus and internal oblique. These authors also noted increased coactivation in response to increased axial load on the spine. This data suggests, perhaps, that coactivation in flexion/extension requires axial preload and may help explain the paradoxical findings of Quint and colleagues (Quint et al., 1998) detailed above. In 1993 Lavender and colleagues (Lavender et al., 1993) used EMG on four volunteers subjected to sudden loading through hand held weights. Unfortunately their methodology does not specify if these individuals were asymptomatic in terms of back pain, but one assumes this to be the case. As with Cholewicki (Cholewicki et al., 1997) these authors also noted a high variability in muscle recruitment and coactivation in preparation to loading. Not surprisingly, the neuromuscular response strategies changed with task experience. As weights were dropped into the subject's hands 30 times over a 30-minute period the subjects were able to develop preparatory muscle responses. The average torso flexion, the action against which the responses were directed, across the four subjects was, in the final session, reduced by 78% of its initial value. Concentrating only on prolonged lateral flexion contractions of the trunk, Potvin and O'Brien (Potvin & Obrien, 1998) showed that coactivation increased with fatigue of the agonist muscles. Again it was proposed that this response increased stiffness and hence stability at the cost of some increase in spinal compression. Gardner-Morse and Stokes (Gardner-Morse & Stokes, 1998) also comment on this triad of stability, fatigue and compression in their recent study employing a three-dimensional biomechanical model. These authors also support the notion that coactivation is a strategy for trunk stabilisation and suggest that *in vivo* a compromise will exist between stability and fatigue and spinal compression. They predict that, without active muscle stiffness, the lumbar spine would be unstable in response to small perturbations despite equilibrium conditions and suggest that activated muscles behave as stabilising springs and not simply as force generators.

What is becoming clear from these studies is that a synergistic relationship exists between mechanoreceptors in the viscoelastic structures, including the IVD (Roberts et al., 1995), and the multifidus and possibly longissimus muscles (Gedalia et al., 1999). Deformation of the tissues of the IVD and related ligaments leads to reflexive muscular activity aimed at stiffening spinal joints, reducing strain in ligaments and preventing excessive motion. Where these physiological systems break down, however, the potential for injury, pain and

instability exist. Recent work on cyclic loading of the spine has suggested a possible mechanism for how these conditions might occur (Gedalia et al., 1999; Solomonow et al., 2000; Solomonow et al., 1999). When viscoelastic spinal tissues are subjected to cyclic, prolonged or vibratory loading, creep and laxity result (Adams & Dolan, 1996; Kaigle et al., 1992a; Leivseth & Drerup, 1997). Compression of the disc, and ligamentous strain are thought to lead to an increase in neutral zone and laxity. This laxity appears to desensitise the afferent stimulation from mechanoreceptors and results in a dampening of the reflexive muscle activity (Solomonow et al., 2000; Solomonow et al., 1999). The combination of reduced passive stiffness and absence of protective muscle contraction exposes the spine to an increased risk of injury and pain. Furthermore, the mutual recovery process of both disc and ligament, two very different types of viscoelastic tissue, after cyclic loading is complex and much longer in duration than previously thought. Solomonow and colleagues (Solomonow et al., 2000), however, demonstrated that less than 1 hour of cyclic loading required at least 7 hours of rest before full recovery of all viscoelastic tissues occurred. Reflexive muscular activity measured by EMG, however, was restored to normal levels after only 4 hours. In addition, following recovery of reflex muscle activity, some muscles became hyperexcitable. These muscles then displayed a greater magnitude of EMG response to viscoelastic deformation than witnessed before cyclic loading began. Following on from this study, it has recently been shown that prolonged flexion loading of the cat lumbar spine, also produced waves of unpredictably timed contractions, or spasms, in the multifidus (Williams et al., 2000). The authors speculate that this hyperexcitability is initiated by pain following subacute damage to viscoelastic tissues, but were unable to substantiate this hypothesis.

Further studies are certainly required to fully understand the recruitment patterns and stabilising strategies of trunk musculature in both health and disease. Nevertheless, the literature seems to recommend a detailed knowledge of these factors as a promising tool for objective clinical assessment in low back pain and instability patients. Another promising area for instability research appears to be the use of ultrasound and other imaging techniques in the measurement of spinal musculature (Hides et al., 1992; Hides et al., 1995) and more recently (McGill et al., 2000). Given the importance of muscles such as the lumbar multifidus and erector spinae, any method capable of quantifying the function of these structures must be of considerable value. For some time now it has been known that a relationship between muscle size and strength exists and that atrophy of

muscles around a joint, the knee for example, is a sequela of joint dysfunction (Young et al., 1984). Furthermore, this atrophy or wasting of the muscle, which can begin surprisingly early following injury of the joint, predisposes the associated articulation to instability and further damage (Stokes & Young, 1984). Since it is impossible to isolate the individual paravertebral muscles and perform functional strength testing, an imaging procedure, which could employ muscle size as an indirect measure *in vivo*, would be clinically useful. A recent methodology using ultrasound scanning techniques has established a clear link between acute back pain and wasting of the lumbar multifidus muscle (Hides et al., 1994). In this study, patients with low back and unilateral radicular pain of, on average, 13 days duration, were found to exhibit marked unilateral wasting of the muscle on the symptomatic side. The atrophy was not thought to be due to disuse, which would be expected to take a longer time course, but to reflex inhibition perhaps involving perceived pain pathways. In instability and chronic low back pain, however, one would expect the multifidus, considering its stabilising role, to be tonically stimulated and hence unlikely to atrophy. This notion would appear to be borne-out by the findings of an imaging study from the early 1990's (Stokes et al., 1992). Using computerised tomography (CT), these authors demonstrated that not only was multifidus atrophy not present in chronic back pain patients but that these patients exhibited a relative increase in multifidus dimension on the side of symptoms. This was accompanied by a slight reduction in erector spinae dimension, again on the side of radicular symptoms. The relative increase in multifidus mass was, however, not attributed to overall muscle hypertrophy but to selective changes in fibre type. Skeletal muscles comprise, in general, two distinct fibre types, type I and II. The proportion of type I and II in a given muscle depend on the major function of that muscle, i.e. whether slow, postural contractions are required or rapid, explosive shortening. Type I are the so-called slow twitch fibres that are adapted, because of their high oxidative capacity, for prolonged, tonic contractions at low intensities. Muscles with a high proportion of type I fibres are fatigue-resistant, such as the gastrocnemius/soleus group. Type II are the fast twitch fibres, which are recruited for fast and forceful contractions. Muscles that contain a predominance of type II fibres are explosive muscles and are prone to early fatigue, the biceps brachii, for example. The multifidus has been found to comprise around 67% of type I fibres, perhaps reflecting its role as a postural stabiliser. Considerable variability in fibre proportion, however, has been reported within normal individuals (Jowett et al., 1975; Kalimo et al., 1989). This diversity in fibre composition of the multifidus has been suggested as an explanation for

the wide individual susceptibility to back pain (Kalimo et al., 1989). A recent histochemical study has concentrated on the cellular changes in the multifidus after lumbar disc herniation (Zhao et al., 2000). These authors took bilateral samples of the multifidus from affected intervertebral levels and examined them for changes in fibre type and size. Their results showed that the percentage of type I fibres was slightly greater on the affected side compared to the "normal" side, with mean values of 60.2% and 58.1% respectively. However the range of values was, 39-83% and 37-88% respectively, again showing the variability reported by others. Nevertheless, significant changes in the size of muscle fibres between affected and unaffected sides were reported. On the side of the herniation both type I and II fibres were significantly smaller than those obtained from the opposite side. This was particularly true for those with evidence of spinal nerve compression, as assessed using the straight leg raising (SLR) test. For those patients whose symptoms were more of central low back pain, the reduction in fibre size was predominantly that of type I. The reasons for these changes are unclear and even the question of whether they represent consequence or causation, remains unanswered. The authors lament the lack of control studies and point to the ethical difficulties in obtaining biopsies from healthy subjects. They do, however, suggest that their findings support the use of therapeutic exercises to increase the size of atrophied muscle fibres and improve the strength of back muscles in the management of lumbar disc herniation. Contemporary work on the management of lumbar instability has, also, shown promise in the use of specific exercises aimed at activating the multifidus in these patients (O'Sullivan, 2000; O'Sullivan et al., 1997). These exercises are directed at not simply strengthening the muscle, but in restoring appropriate neural control and perhaps influencing the relative proportions of fibre type.

The lumbar multifidus, as we can see, is without question a vitally important element of segmental stability. It can also be argued that the multifidus is essential to overall stability of the lumbar spine.

In a review paper considering the functions of lumbar spinal ligaments and muscles, (Aspden, 1992) suggests that traditional lever models of the spine are inappropriately simplistic in explaining its dynamic mechanical behaviour. Instead, Aspden proposes an arch-like model that defines spinal stability in terms of its inherent curvature. He argues that the curved nature or posture of the spine is central to this hypothesis of stability. Since

multifidus is, by virtue of its attachment to the spinous processes, the chief active element in controlling local curvature in the lumbar region, it then follows that multifidus determines its stability.

4.8 INTRA-ABDOMINAL PRESSURE CHANGES

A detailed account of the role of intra-abdominal pressure in lumbar spine stability is beyond the scope of the present thesis. Nevertheless, a discussion of instability would be incomplete without at least a passing reference to this proposed mechanism.

In the late 1950's, Bartelink (Bartelink, 1957) alluded to the possibility that intra-abdominal pressure (IAP) could help counteract spinal compression forces during lifting and other activities. Increases in IAP were thought to cause upward forces on the diaphragm, producing a balloon-like resistance to spinal flexion. Further studies, however, failed to unequivocally support this and in 1986 a group led by Nachemson (Nachemson et al., 1986), showed that increasing IAP by a Valsalva manoeuvre increased rather than decreased lumbar spine compression. By this time it became apparent that any mechanism for IAP to result in reduced flexion moment would have to be more complex than a simple "balloon" effect. Attention was then focused on the role of the thoracolumbar fascia (Gracovetsky et al., 1985; Tesh et al., 1987). It was therefore proposed that an increase in IAP resulted in lateral tensile forces being imparted to the thoracolumbar fascia, which, due to the criss-cross fibre orientation of its posterior layer, produces an extension moment on the lumbar spine via the spinous processes. The abdominal muscles would, in effect, brace or even extend the spine during lifting. Nevertheless, experimental work on this mechanism showed the maximum extensor moment possible to be disappointingly low (Macintosh & Bogduk, 1986). A number of hypotheses have since been presented to explain the precise nature of how IAP influences spinal stability but recent accepted thought suggests that the exact principles have yet to be described (Cholewicki et al., 1999). These authors have written a concise and current review of this subject and constructed a simple physical model to illustrate a possible IAP mechanism for spine stabilization. Two separate stabilizing mechanisms were simulated in their study, the first involved antagonistic muscle coactivation, as previously described and the second involved generation of IAP. The flexor/extensor coactivation, however, necessitates that a proportion of the extensor muscle activity be directed at equilibrating the abdominal muscle contraction. Changes in IAP, on the other hand, can be produced

solely by abdominal muscle activity and thus stability is maintained without additional erector spinae coactivation. It is logical, therefore, that this mechanism would be preferred under conditions which require maximum activity in extensor muscles. Cholewicki and colleagues (Cholewicki et al., 1999) concluded that lumbar stability is most likely achieved by a combination of these two mechanisms. Whether these phenomena, *in vivo*, occur in combination or act separately remains unclear. Stabilizing the spine via IAP, they propose, is likely to occur during tasks demanding trunk extensor moments such as lifting or jumping. This would seem to be supported by the earlier work of Hutton and colleagues, who showed that IAP approaches zero in full flexion (Hutton et al., 1979). In this model, stability is brought about, not so much by the magnitude of generated IAP but by the stiffness of the abdominal muscles creating it.

4.9 SUMMARY

Eisenstein (Eisenstein, 1999) regards the challenge of defining instability to be one mainly of semantics and terminology. He contends that no single definition will be acceptable to all those engaged in the study of the human spine. In his thesis he proposes that, since the title "instability" is likely to persist, the term should be more accurately applied. To this purpose Eisenstein offers an ABC classification of instability. The A-instability applies to "apparitional" and indicates instability as defined by imaging techniques. This class includes those radiographic signs previously mentioned. B-instability refers to definitions based on biomechanical data. These are largely experimentally produced instabilities and are usually difficult to relate to the clinical situation. C-instability is that clinical presentation, which, for those involved in diagnosis and management, is suggestive of the clinical instability syndrome. Eisenstein recommends that this appellation be used in scientific communication, to resolve the present confusion that exists between disciplines engaged in instability research. He stresses, however, that in dealing with patients it is not necessary for the three classes to coincide before reaching a diagnosis. Nevertheless, it may prove useful to attempt to gather information from a patient using skills and knowledge from all three disciplines. In this way it might be possible to establish evidence of instability relating to these different classes and thus provide greater certainty in diagnosis. As we have seen, instability is a truly multifactorial phenomenon, with a wide threshold between what is considered stable and what is unstable. There would appear to exist a penumbra encompassing a spectrum of mechanical changes, which may

or may not produce recognisable symptoms. The multitude of factors in determining which segments will display instability include such variables as the type of lesion or damage to a particular restraining component, be it disc or facet joint, for example. Other factors relate to supporting tissues such as muscle, its resting length, state of tension perhaps or degree of compensation. This might be revealed, conceivably, by the presence of hypertrophy. In addition, the balance of muscle fibre type may indicate adaptation to unstable conditions. The neural subsystem is, as previously mentioned, of great importance and the integrity of the articular transducers and their connections may also provide indicative evidence. External factors, as in, loading phases and the spinal responses to them, displayed by the pattern of motion, both segmental and regional, may be useful in the assessment of a suspected instability patient. From a clinical standpoint, knowing when a patient is in most pain and timing its onset with respect to kinematic events may be of particular value. In short, there is a great deal of information, from investigations of a clinical, biomechanical and imaging nature, which can be amassed from patients. In light of the previous discussion, it is unlikely that any one technique or investigation will solve the riddle of segmental instability. What is more plausible is that a battery of tests combined with functional assessment and good history taking, have a greater chance of yielding a credible estimate of the degree of stability. For example, Kaigle and colleagues (Kaigle et al., 1998) have suggested that the cessation of segmental rotation with respect to trunk flexion limits could be an indicator of spinal dysfunction. Using a combined approach they showed that, in patients where segmental rotation was not fully accomplished before the end of trunk flexion, the normal flexion-relaxation (FR) response was absent. Thus the coalition of a dynamic imaging procedure, such as digital fluoroscopy, with an EMG protocol and a surface measure of overall trunk motion, ISOTRAK for example, could reveal data of greater significance than either technique individually. Similarly, by continuously recording myoelectric activity during dynamic imaging, it may be possible to relate kinematic phenomena with muscular responses. As Kaigle and her team suggest, "To properly evaluate the biomechanical stability of the spinal system, the kinematic behaviour of the passive and active components must simultaneously be considered" (Kaigle et al., 1995). Previous attempts to establish instability have, perhaps, concentrated too vigorously on passive elements. Although failure of passive restraining components is probably the most common mechanism in cases of clinical instability, diagnostic approaches have largely ignored active responses or compensatory changes. In this way much valuable information is lost. The preceding

discussion has revealed the multifidus muscle to be a key element in these compensatory mechanisms. The work of Hides, Stokes and others (Hides et al., 1992; Hides et al., 1995; Stokes et al., 1992) have shown that relatively non-invasive, *in vivo* techniques are capable of providing evidence of morphological changes in these tissues in response to pain and disability. This is not to say that passive component integrity should be disregarded. Indeed, a knowledge of which components (disc, facet joint, ligaments) show signs of damage and, perhaps more importantly, what types of lesions exist, can give valuable insight as to the likelihood of instability (Indahl et al., 1997; Kaigle et al., 1995; Kaigle et al., 1997; Sharma et al., 1995). Thus diagnostic imaging techniques such as CT and MRI scanning, together with dynamic imaging and functional assessment can be used to substantiate a hypothesis of instability or other dysfunction (Bram et al., 1998; Burton et al., 1996; Stokes et al., 1992).

Pain is another important factor often overlooked in purely biomechanical investigations of stability. Attempts should be made to correlate painful phases with kinematic events. Is it more painful in flexion, when posterior structures are under greater load? Does the pain coincide with one of the abrupt "hesitations" or "giving way" movements described by Ogon and colleagues (Ogon et al., 1997b)? Perhaps pain associated with axial translation, as reported by Kaigle and co-workers (Kaigle et al., 1998), might be a significant clue as to the abnormality of the deformation? This kind of information could be ascertained by some kind of hand-held, pressure-sensitive device that patients, undergoing dynamic imaging, could squeeze when pain is felt during a motion sequence. Again, in combination with a surface measure of trunk mobility, such investigations could, conceivably, yield a summation of knowledge greater than its component parts.

In terms of dynamic imaging there is a great deal of useful information yet to be gathered. It is clear from the work of Ogon and his team (Ogon et al., 1997a; Ogon et al., 1997b) that particular attention has to be paid to changes in velocity of segments, especially around the neutral zone. Kaigle and colleagues (Kaigle et al., 1998) have established a possible link between instability and changes in axial translation. This is a relatively simple parameter to measure and one that has been largely overlooked in previous research. The same authors have shown, *in vivo*, that differences in hysteresis behaviour may provide data on the stability of a given segment (Kaigle et al., 1995). Unfortunately this requires taking subjects through a range of flexion/extension, for example, more than

once. With fluoroscopic screening this necessitates a slightly increased dose of ionising radiation. However the flexion/extension protocol used by Kaigle (Kaigle et al., 1998) is remarkably similar to the method we have employed in our recent attempts to establish normal values for passive motion. This involves taking subjects from the neutral position to full flexion, returning through the neutral zone to full extension and then back to the neutral position. In contrast to Kaigle's work, however, we have our subjects in the lateral recumbent position during passive screening. Obviously this has implications for loading conditions and changes in hysteresis behaviour, amongst other kinematic parameters, can be expected to differ under non weight-bearing situations (Ogon et al., 1997a; Wood et al., 1994).

4.10 CONCLUSION

No single investigation is likely to be diagnostic for segmental instability and no single definition is likely to be adopted by all disciplines. Only by corroboration of evidence, from a variety of sources, is a *probability* of instability liable to be reached. These sources should include myoelectric data from supporting musculature, imaging of morphological changes in passive and active tissues and kinematic parameters from dynamic imaging. In addition, good clinical history-taking and functional assessment is vital if an informed decision is to be made regarding these patients. When the evidence clearly points towards unstable behaviour and after all conservative methods have failed, only then should a patient be subjected to the trauma of surgical fusion. In this way, hopefully, the patient will be less likely to relapse and will receive the full benefit of this valuable procedure. Dynamic digital fluoroscopy can, potentially, play a key role in the assessment of the spinal instability patient.

PART II

APPLICATIONS OF DVF

INTRODUCTION

DVF, as discussed in chapter 1, was never devised purely for a research role. From its inception, the hope was to develop a robust yet sensitive technique for gathering clinically pertinent data on the mechanical behaviour of the spine. Any technique employing ionising radiation, of course, carries latent risk and this has to be weighed against the benefits provided. The question as to whether other, less hazardous, modalities can supply the required information should always exist. At the present time, however, the use of X-rays for obtaining such information would appear unavoidable. This, hopefully, will not always be so, and the techniques and methodologies developed in DVF should be readily applicable to newer and safer forms of dynamic imaging. The evolution of DVF, nevertheless, has not been as rapid as would be preferred and, in the main, has been due to this reluctance to sacrifice patient/subject safety for improved image quality. Traditionally, the term "fluoroscopy" conjures connotations of high X-ray exposure. Although this is a fallacious view, nonetheless static, plain-film radiography remains the norm for spinal motion studies. The aim, among those working with DVF, has always been to obtain maximum information from minimum exposure. Improvements in low-dose fluoroscopy are ongoing and will ensure that digitised fluoroscopy research can continue to adhere to its objectives.

Currently, DVF is providing useful clinical information in the assessment of lumbar spine function and the following chapters will illustrate its role in two diverse ways. The first reveals how DVF can be used to assess the validity of a surface measurement technique (3SPACE ISOTRAK) in the quantification of back mobility in normal subjects. The second describes a preliminary study involving patients who have undergone instrumented spinal fusion. This chapter demonstrates the part that DVF can play in assessing surgical outcome and determining further intervention.

CHAPTER 5

COMPARISON STUDY BETWEEN DVF AND 3 SPACE ISOTRAK

5.1 INTRODUCTION

3SPACE ISOTRAK is an electromagnetic surface measurement device comprising a source, sensor and data acquisition software. It is capable of continually recording the three-dimensional position and orientation of its sensor with respect to the source. When attached to the surface of the back, therefore, it serves as a relatively inexpensive and safe means for obtaining information on spine and trunk mobility. All authors involved in active research with this device, however, suspect that the ISOTRAK has a tendency to overestimate true angular displacements of the spine. Furthermore, all agree that skin stretch has an effect on the results but make no attempt to quantify such a factor. Accuracy of the 3SPACE ISOTRAK has been studied statically using wedges of known inclination, (Pearcy & Hindle, 1989), but to our knowledge the device has not yet been compared with radiographic measurements. The aim of this present study, therefore, is to directly compare ISOTRAK measurements with those obtained using DVF.

5.2 METHODOLOGY

This pilot study recruited ten adult male subjects between the ages of twenty-two and forty-nine years for screening. Subjects were excluded from the study if they had suffered back pain over the previous six months or were over five-foot ten inches tall (the height requirement ensured that most of the lumbar spine would be visible, on fluoroscopy, throughout the movement). The 3SPACE ISOTRAK was hard-wired to a personal computer to display the angular displacement of the sensor, in degrees, around the three Cartesian axes. The device was surface-mounted to the subjects' backs (Figures 5.1 & 5.2) by means of double-sided tape and elastic strapping. The sensor was positioned over the spinous process of the second lumbar vertebrae using double-sided tape and then secured with an elastic strap around the trunk. The source was first mounted to a plastic pad, contoured to fit the sacrum, and then secured there by an elastic strap around the pelvis. Osseous motion was assessed using DVF.

FIGURE 5.1

ISOTRAK MOUNTED TO SUBJECT'S BACK

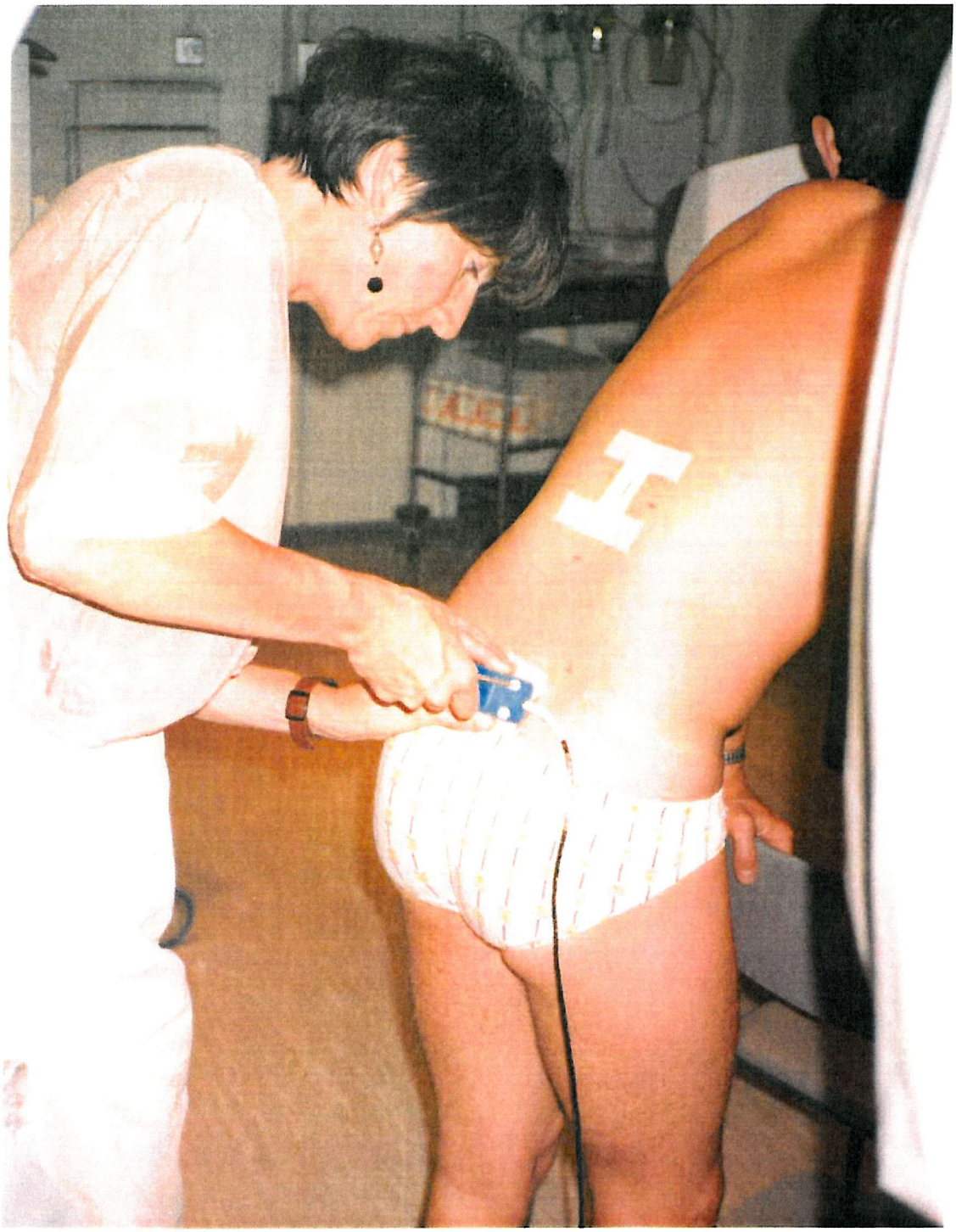
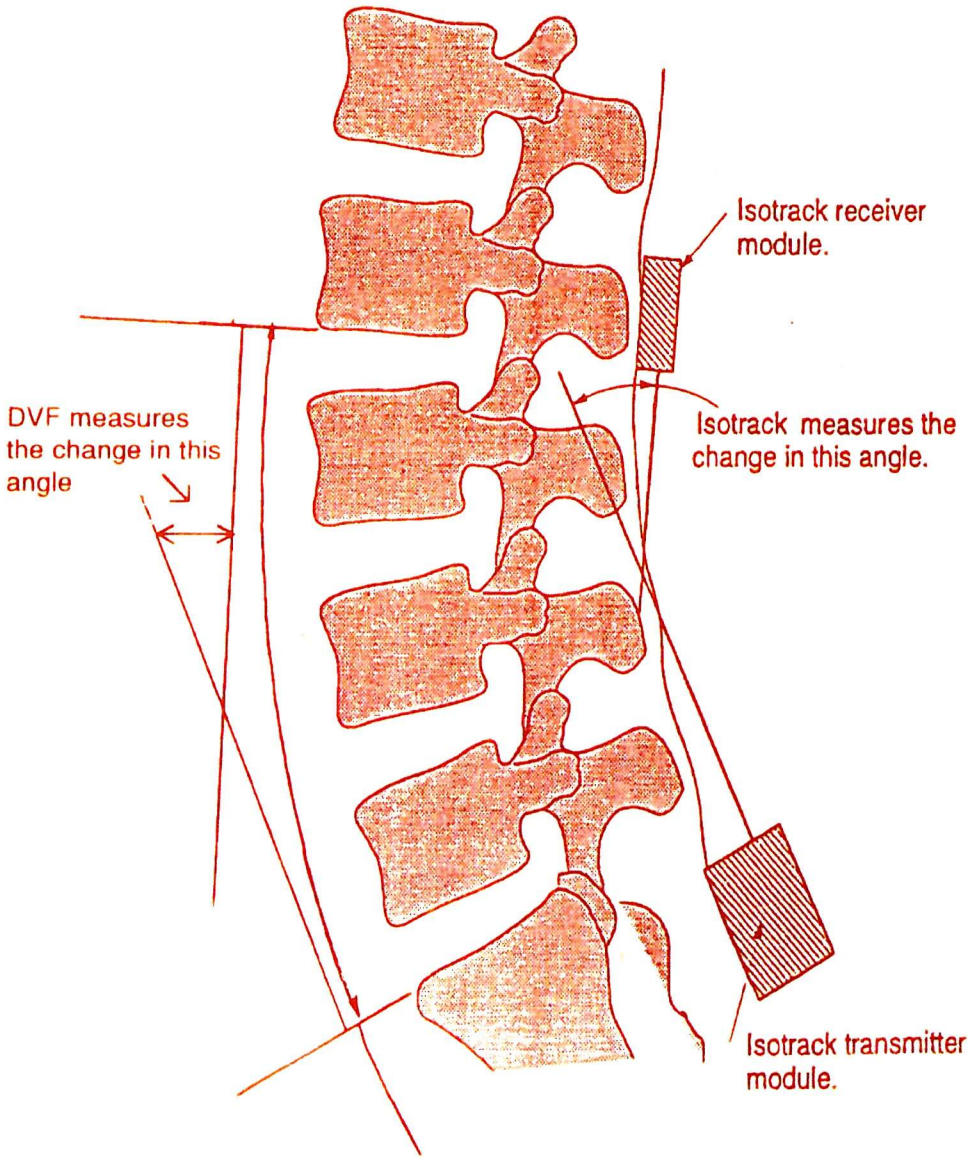


FIGURE 5.2

DIAGRAM SHOWING ISOTRAK AND DVF MEASUREMENTS



The DVF system, in this study, comprised a Thompson CGR X-ray machine with a 12-inch diameter image intensifier, located at the radiology department of Poole General Hospital, Poole, Dorset. With the ISOTRAK device in place, subjects were positioned seated against the vertically aligned X-ray table (Figure 5.3) with arms extended in front of them. An active flexion sequence was rehearsed in this position over a duration of

approximately 3-4 seconds. After final alignment, the flexion sequence was repeated under lumbar spine screening with a focus to intensifier distance of 1 metre. The X-ray motion sequences were recorded onto 3/4-inch low-band U-Matic videotape. Externally generated time-code, by means of a time-code generator, was superimposed on the videotape. This coincided with the 3 SPACE ISOTRAK recordings. The three ISOTRAK values (lateral bending, axial rotation and flexion/extension) were recorded at 0.036-second increments.

The sequences were digitised and stored in sequence on the PC at an approximate sampling rate of 1Hz. A commercially available image processing system (OPTIMAS) was used to manipulate and manually mark the images (Figure 5.4).

Limitations in the radiography hindered marking of the S1 segment and hence DVF quantified the angular displacement between L5 and L2 and not S1to L2 as measured by the ISOTRAK. Using a template system driven by the screen cursor, the spatial co-ordinates and orientations of the lateral images of the L2 and L5 vertebral bodies were extracted for each of the 16 images and exported to an Excel spreadsheet. This, in turn, generated graphs of the motion, expressed as a series of 16 data points representing the angles subtended by the vertebral bodies of L2 and L5. This procedure was repeated, by the same examiner, for reliability. It allowed comparison with the angles generated over the same time period with the ISOTRAK device. Agreement between the measurements generated by the two systems was assessed using the intraclass correlation coefficient (ICC), which is itself based on the one-way Anova test.

5.3 RESULTS

Two of the files were excluded from the analysis (subjects 5 and 10), being below the level of radiographic quality required for reliable measurement. Graphs comparing the two devices for each subject are shown in Figure 5.5. From these it can be seen that the DVF and ISOTRAK measures agree closely only over the first few frames of motion.

FIGURE 5.3

ISOTRAK SUBJECT IN POSITION

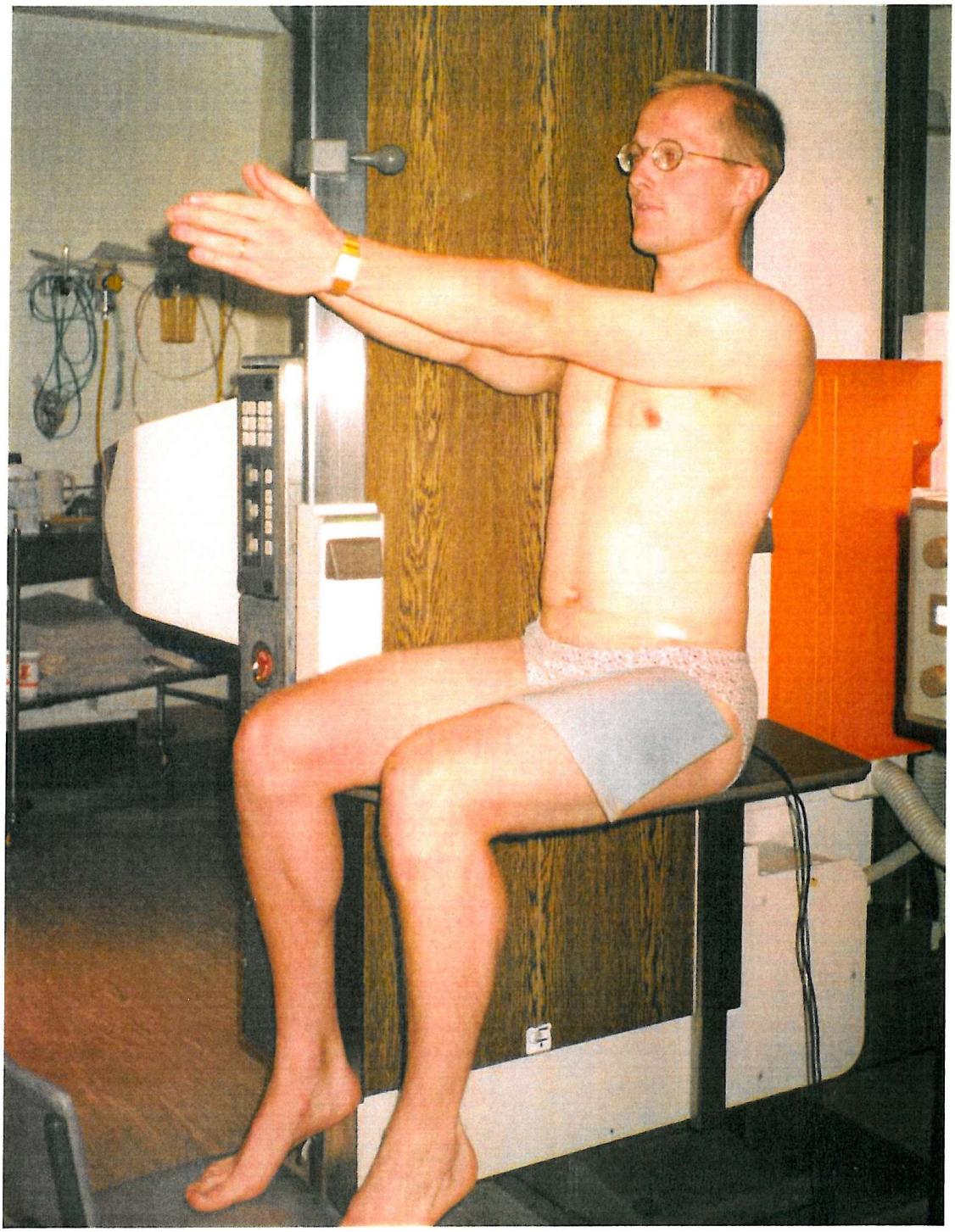


FIGURE 5.4

OPTIMAS IMAGE PROCESSING SYSTEM

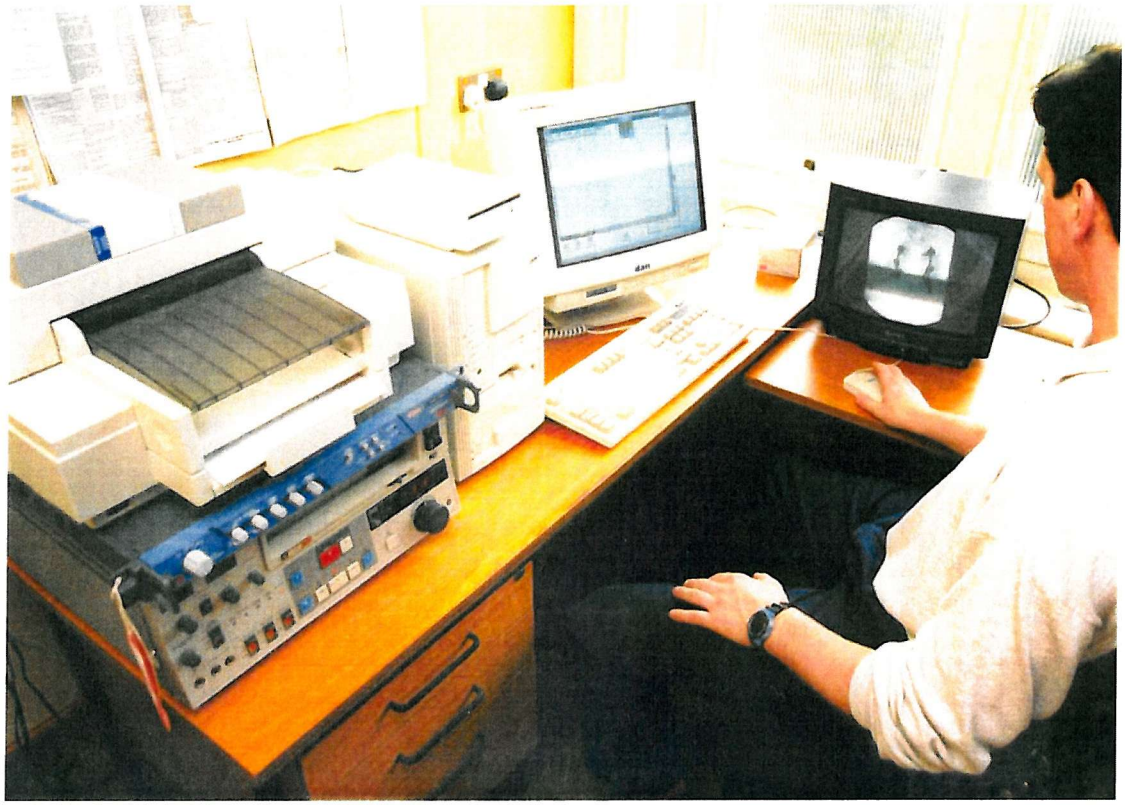
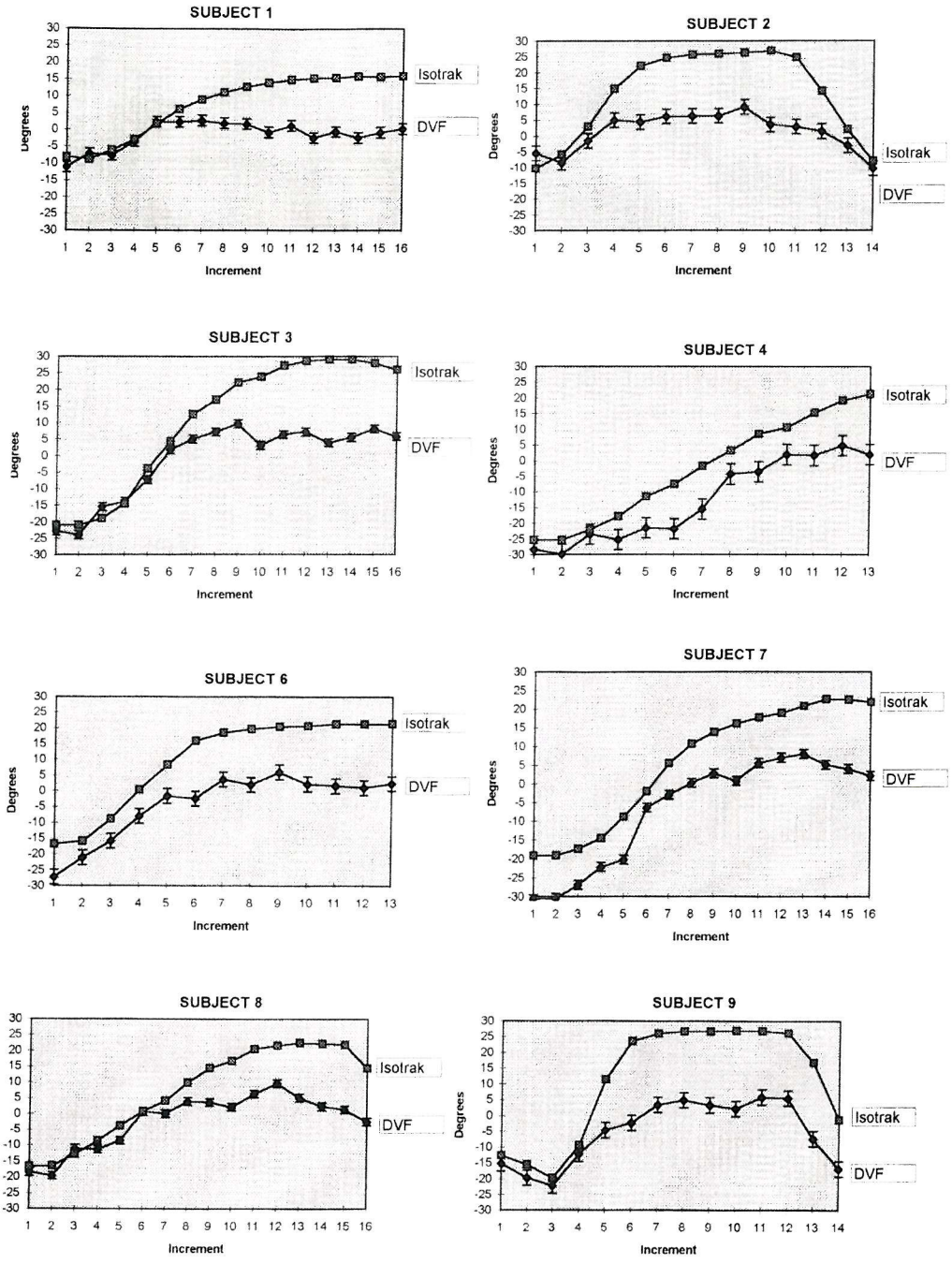


FIGURE 5.5

GRAPHS SHOWING ISOTRAK AND DVF RESULTS



After the fifth or sixth frames, however, these curves separate, with ISOTRAK consistently measuring higher angles of flexion. The agreement between first and second DVF measurements of lumbar flexion angle on a subject by subject basis is shown in Table 5.2. Intraclass correlation coefficients (ICCs) are consistently high for these measurements suggesting good intra-examiner reliability. The reliability data was also pooled, combining the individual subject readings. Overall agreement between DVF measurements 1 and 2 for this pooled data was also high at 0.90 with a mean difference of less than half a degree (Table 5.2).

The statistical comparison, in terms of range, is shown in Table 5.3. There is a strong relationship between lumbar angles as calculated by DVF and ISOTRAK (Pearson's r = 0.91). However, as can be seen from the data, the ISOTRAK records a range for each subject, on average, nearly 12 degrees higher than that of DVF. Actual agreement between the techniques is moderate (ICC = 0.554 p = 0.0001).

5.4 DISCUSSION

Since the ISOTRAK measures angular displacements at around 0.036 second intervals and the approximate grabbing rate for the image processing was 1 second, it was not possible to time match the data exactly. The average time difference between the two sets of data was 0.01 seconds (SD = 0.009). The largest difference was 0.036 seconds, which always occurred at the start of the sequence (Table 5.1). This was due to the fact that the ISOTRAK recordings, unlike DVF, never began exactly at time zero. Since the temporal variance between the two measurements was always less than 0.05 seconds and the back movements in question of low velocity, the measurements were considered to be simultaneous.

TABLE 5.1

EXAMPLE OF TIME DIFFERENCES, IN SECONDS, BETWEEN ISOTRAK AND DVF MEASUREMENTS

ISO TIME	DVF TIME	DIFFERENCE
0.036	0.00	0.036
0.893	0.875	0.018
1.749	1.75	0.001
2.642	2.625	0.017
3.499	3.5	0.001
4.391	4.375	0.016
5.248	5.25	0.002
6.140	6.125	0.015

In this study the differences in flexion angle become apparent after the first six or seven increments (Figure 5.5). This is reflected in the agreement between both techniques, as measured by the ICC, when only the first six increments are compared. Agreement over this early phase of flexion reaches 0.75 compared to a value of 0.55 for the overall motion. The ISOTRAK values of flexion are, on average, 12 degrees larger than those of DVF and the respective graphs can be seen to diverge most when significant lumbar motion, measured using DVF, has ceased. This would suggest that skin distraction of the ISOTRAK sensor over relatively static spinous processes is responsible for the overestimation in flexion values (Figure 5.6). Radiographic constraints, however, prevented marking of the S1 segment and therefore DVF measurements were only taken between L5 and L2 and not S1to L2 as measured by the ISOTRAK. The effect of this apparent mismatch is difficult to quantify but would appear to be minimal considering the close agreement between DVF and ISOTRAK in the early, most mobile, phase of flexion. Flexion ranges were, however, lower than those obtained by Pearcy (Pearcy, 1985), for a similar asymptomatic group using biplanar radiography. This is explicable by the differing methodologies. Pearcy, in his 1985 study, commenced all lumbar motions from the erect standing position with a consequently greater degree of extension than that encountered in the seated subjects from the present study. Also, the entire lumbar spine could not be consistently visualised in this present study. This, coupled with the poorer penetration qualities associated with low-dose fluoroscopy (discussed in chapter 7), limited our lumbar studies to between the L2 and L5 segments.

A number of authors have attempted to relate back surface morphology to underlying skeletal positioning (Bryant et al., 1989; Lee et al., 1995; Sicard & Gagnon, 1993; Walsh & Breen, 1995). These studies, however, have used skin profile changes in static positions and corresponding plain film radiographic techniques. This present study is different in that it has been concerned more with changes during motion rather than quantifying back shape in various positions. Both ISOTRAK and DVF record continuous data, simultaneously, during dynamic bending movements. The incremental changes in angle during these movements, for both techniques, are demonstrated in Figure 5.5. Because vertebrae can be considered as rigid bodies, the change in angle as measured by the spinous processes (ISOTRAK) should be identical to the change in angle as measured by the vertebral bodies (DVF). The differences in measurement between the two techniques must, therefore, be a result of the changes occurring in the overlying skin.

This is not surprising since most authors investigating back mobility via surface measures have found skin folding and skin distraction to be problematic particularly in extension positions (Dolan & Adams, 1993; Pearcy, 1993; Portek et al., 1983; Walsh & Breen, 1995).

5.5 CONCLUSION

This study has demonstrated that there is a strong correlation between lumbar flexion as measured by the 3SPACE ISOTRAK and by radiographic measurement using DVF. Agreement between the two techniques, however, is moderate and the ISOTRAK system appears to overestimate true angular displacement. This phenomenon is most likely to be a result of skin distraction and thus, as with all surface measures, 3SPACE ISOTRAK is limited by its non-invasiveness. Nevertheless the results do show close agreement between the two techniques during early flexion from neutral when the lumbar spine is most mobile. As such, ISOTRAK would appear to be most useful when considering gross changes of within-range lumbar mobility in normal subjects and in some cases, back pain patients. Of particular interest is the ability of the ISOTRAK to provide information on changes in kinematic patterns between and within individuals. These changes may be of considerable value in conditions where the anatomical integrity of spinal structure is intact, but pain or other insult has produced abnormal motor behaviour. Where issues of spinal mechanics, particularly of intervertebral linkages, are fundamental to a clinical problem, however, dynamic radiographic methods must be the investigation of choice. Of these methods, DVF, considering its flexibility and associated low X-ray dosages, would be more appropriate.

FIGURE 5.6

SKIN STRETCH HYPOTHESIS

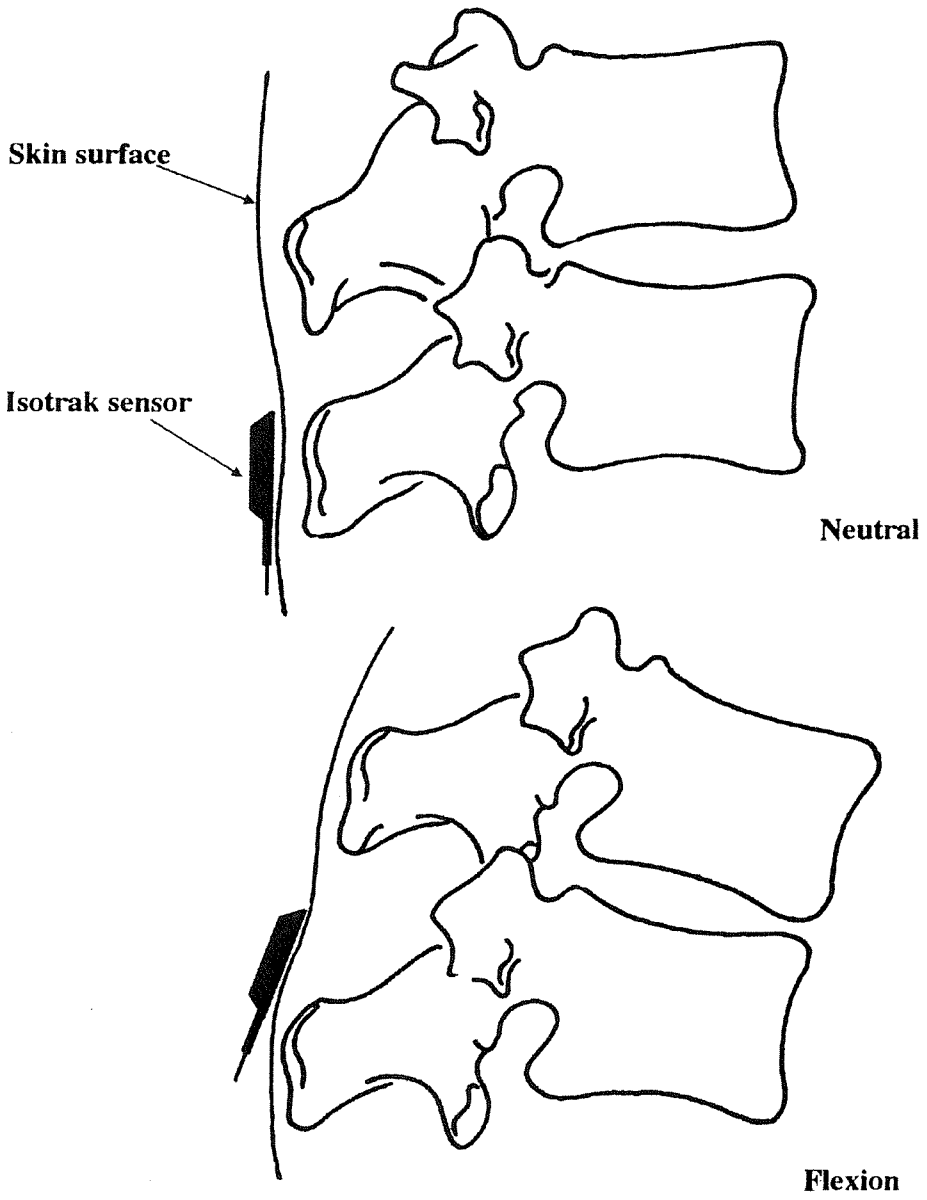


TABLE 5.2

AGREEMENT BETWEEN DVF MEASUREMENTS 1 AND 2 OF LUMBAR FLEXION ANGLE FOR EACH SUBJECT.

Subject	Data Points	Mean Diff	ICC	95% LSD
1	16	-0.27	0.93	3.12
2	14	2.19	0.87	4.93
3	16	0.06	0.99	4.26
4	13	0.11	0.98	5.41
6	13	-0.53	0.99	2.73
7	16	0.21	0.99	2.87
8	16	0.40	0.96	3.72
9	14	0.57	0.98	6.04
Overall	118	0.34	0.90	4.76

TABLE 5.3

COMPARISON OF LUMBAR FLEXION RANGE AS MEASURED BY DVF

Subject	DVF Range	ISOTRAK Range	Difference
1	13.82	24.60	10.78
2	19.35	37.10	17.75
3	33.57	50.00	16.43
4	34.71	46.70	11.99
6	33.13	38.10	4.97
7	38.64	41.70	3.06
8	29.20	39.20	10.00
9	28.01	46.70	18.69
MEAN	28.80	40.51	11.71
SD	8.35	7.93	5.75

CHAPTER 6

DETECTION OF FAILED LUMBAR FUSION BY DIGITAL VIDEOFLUOROSCOPY

6.1 INTRODUCTION

Segmental spinal instability, its diagnosis and management continues to be a contentious topic amongst those involved in spinal studies (Goel & Pope, 1995; Nachemson, 1985). Nevertheless, it is regarded as a major cause of chronic back pain and surgical fusion, or arthrodesis, is becoming an increasingly applied procedure in its management (Katz, 1995). As early as the 1920's its use was being enthusiastically promoted as a treatment for "instability" or chronic pain (von Lackum, 1924). Lumbar surgery with fusion, however, is more invasive, associated with greater complications, is more costly and has no widely accepted efficacy over non-fusion surgery for degenerative spinal disease (Gibson et al., 2000; Malter et al., 1998). As such, its widespread use for chronic degenerative back pain has been questioned and strong recommendations made for good randomised controlled trials to compare its clinical outcomes with placebo and other, less invasive, treatments (Gibson et al., 2000; Malter et al., 1998; Muggleton et al., 2000). Recently a study of patient satisfaction, in terms of pain relief and function, following primary lumbar fusion, produced results in support of this negative view of its efficacy with regard to degenerative disease (Buttermann et al., 1998). Nevertheless, Buttermann and colleagues concluded that patients with developmental conditions, grade III-IV spondylolisthesis for example, expressed a high level of satisfaction from primary surgical fusion.

In 1996, a large multicentre clinical trial was proposed and implemented to investigate the value of spinal fusion in the management of chronic low back pain (Fairbank et al., 1996). As a result of substantial methodological difficulties, however, the trial was prematurely terminated.

Whatever the indications for spinal fusion, the basic objectives remain those of rectifying deformity, relief of pain and improvement of function (Sonntag & Marciano, 1995). Many different types and techniques of fusion exist but almost all involve the use of bone grafting with or without supplementary instrumentation (Chow et al., 1996; Fraser, 1995).

The use of instrumentation itself, however, appears to have no advantage in terms of clinical outcome (France et al., 1999; Gibson et al., 2000) but may improve fusion rate. In any case, the technical success of fusion is difficult to objectively assess in the presence of metal-work (Gibson et al., 2000).

Although there are no clinical trials that compare one method of fusion to another, a consensus exists that fusion between vertebral bodies (interbody) provides a more stable union than posterior element fusion alone (Chow et al., 1996; Fraser, 1995). The mechanical stability of an arthrodesis and long-term clinical outcome, however, are often quite different (Hanley, 1995; Rahm & Hall, 1996). Nevertheless, a relatively recent survey of 150 patients undergoing surgical lumbar arthrodesis supports the view that solid fusion is related to a successful clinical result (Ransom et al., 1994). Furthermore, Ransom and colleagues go on to suggest that failure to achieve solid arthrodesis is the most likely reason for the inability to improve low back pain postoperatively. This inadequate arthrodesis, or pseudarthrosis, is a major complication of lumbar spine fusion (Krodel, 1996). A very recent study has suggested that failure to achieve a solid bony fusion occurs in up to 40% of patients (Boden, 2000). It is not surprising, therefore, that, when patients who undergo lumbar spine fusion suffer a return of their symptoms, this should prompt a second attempt at surgical repair. Buttermann and co-workers (Buttermann et al., 1998) have shown that, after fusion, a lack of improvement in back pain score or disability score was significantly correlated with pseudarthrosis. The main evidence for pseudarthrosis or failure comes, at present, from plain radiographs. Since this investigation does not truly represent motion in the spine, surgical exploration often becomes the only reliable means of determining the integrity of the fusion. This pilot study aimed to investigate the prospect for using digitised videofluoroscopy (DVF) to identify pseudarthroses without the necessity for surgical inspection.

6.2 METHODS

Eight patients at the Centre for Spinal Studies in Oswestry were admitted for inspection of previous lumbar spine fusion as a result of relapse or unresolved pain. All were subjected to videofluoroscopic recording of voluntary lumbar spine sagittal motion in the standing position. Following this, surgical inspection for pseudarthrosis was performed and repaired where necessary. The results of the surgical inspection and any clinical details were withheld from those analysing the motion sequences. The videotaped sequences

were analysed using a computer-based image processing system (DVF). Flexion/extension motion was quantified for all fused segments and displayed in graphical form. The graphs were then visually inspected for obviously excessive movements. A segment was designated as fixed when the motion failed to exceed the associated error.

6.3 RESULTS

Twelve intervertebral linkages from eight lumbar spines were compared. Surgical inspection revealed four pseudarthroses and eight successful grafts. DVF analysis detected all eight successful grafts and three out of the four pseudarthroses (Table 6.1), giving an agreement rate of 92% (Kappa = 0.8, $p<0.01$). The flexion and extension ranges of the segments were compared once the surgical results were known. The mean half-range (i.e. flexion or extension) of the pseudarthroses was 5.7^0 and at the fixed segments 3.7^0 (one-way unpaired t-test $p<0.005$).

TABLE 6.1

COMPARISON BETWEEN DVF & SURGICAL ASSESSMENT OF FUSION

		SURGICAL	
		Pseudo	Fixed
DVF	Pseudo	3	0
	Fixed	1	8

6.4 DISCUSSION

"The anatomic objective of lumbosacral arthrodesis is solid bony union coupled with the clinical objective of decreased postoperative low back pain" (Ransom et al., 1994). When

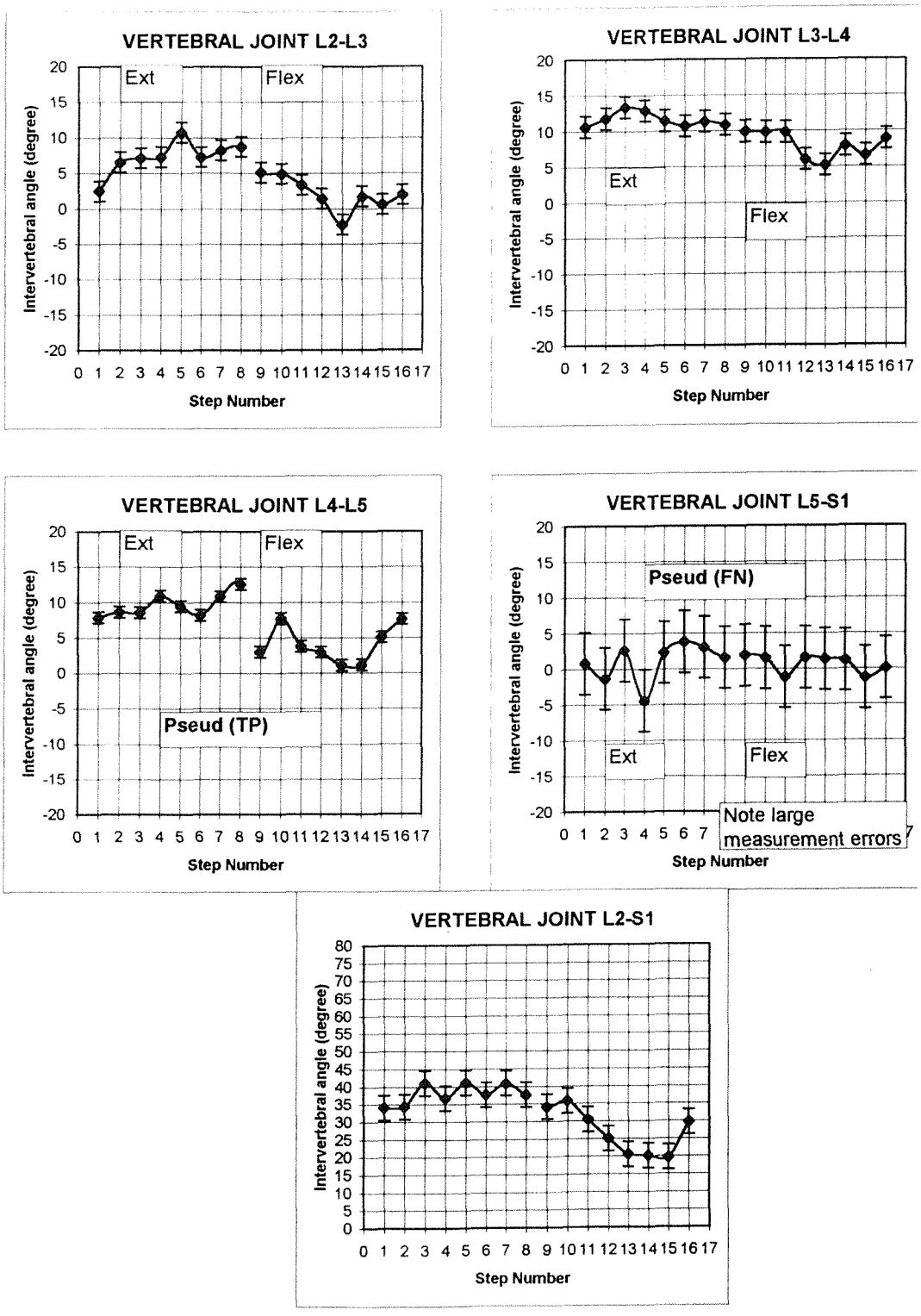
the clinical objective is not met the anatomical objective, naturally, comes under scrutiny and often this can only be achieved by further surgical intervention. In this study an attempt was made to investigate whether or not DVF analysis could help reduce the need for direct inspection via revision surgery. As with all operative procedures there is substantial risk to the patient during surgery. In addition there is often considerable post-operative morbidity, which is especially deleterious for low-back pain sufferers. It was hoped that, using this low-dose fluoroscopic technique, the analysis could assist in identifying those patients who would benefit from surgical repair and those who would not. Although only a preliminary study, the results were encouraging. The most significant of which were the reduced half-ranges (flexion or extension) of the fixed segments. This was further reflected in the ability to detect all eight successful fusions. The failure to detect one of the four pseudarthroses was attributed to observer error. An eagerness to assign all motion sequences as either fixed or mobile resulted in the observer designating equivocal data. The segment in question, due to poor image quality, had large associated measurement errors and no attempt should have been made to assign it. This is illustrated in Figure 6.1, which shows the motion graphs generated from the analysis of that patient, and attention is drawn to the L5-S1 segment and its excessive error-bars. In future studies, greater attention should be paid to the problem of error. The results of this study, although tantalising, are insignificant in terms of numbers and a much larger study should be attempted. Such a study could also include a clinical trial of differing instrumentation and address the kinematic effects of arthrodesis on adjacent segments.

6.5 CONCLUSION

This study suggests that DVF may be an effective, inexpensive and trauma-free means of selecting patients suitable for pseudarthrosis repair. In addition DVF may also provide valuable information on how surgical fusion at one level affects adjacent segments.

FIGURE 6.1

GRAPH DEPICTING PSEUDARTHROSIS IN ONE PATIENT



PART III

NEW DEVELOPMENTS

CHAPTER 7

PASSIVE MOTION ANALYSIS OF LUMBAR SPINE MOTION

7.1 INTRODUCTION

Since the first use of DVF in 1988 (Breen et al., 1988) the radiographic protocol, in regard to human subjects, has remained essentially unchanged. At that time it was thought best to X-ray subjects during natural voluntary movements of the lumbar spine involving flexion/extension and sidebending. Patients, or subjects, were asked to sit upright on a wooden frame attached to the fluoroscopic tilt table and as close to the image intensifier as possible. The frame provided some stabilisation of the sacrum during extension but allowed free unhindered motion throughout flexion. Similarly during sidebending, with the back close to the intensifier, the patients/subjects were allowed voluntary control in bending to either side. In this way, it was hypothesised, the natural weight-bearing movements of the human lumbar spine could be quantified in both health and disease.

From this data it was anticipated that not only ranges of motion but also indices of regularity/irregularity and laxity could be established to help distinguish normal from abnormal motion. The reality of measuring spinal motion in this way, however, gradually became apparent. Given voluntary control in bending the trunk, most individuals, especially those in pain, simply avoided using the lumbar spine to any great extent. The rate of bending also was problematic in that, although the movements were rehearsed, subjects/patients often performed the movements under fluoroscopy at differing rates and at velocities that produced blurring of resultant images. The character of the overall motion was very often jerky and irregular, particularly at the end of range. This made determination of the initial and final positions of vertebral segments difficult to assess. Although these features may be clinically significant, they are difficult to interpret without previous baseline data or repeat screenings. The nature of ionising radiation, however, restricts the routine use of multiple studies on individuals. It is therefore essential to employ a protocol that yields the maximum information on first attempt.

One feature that recurred on analysis of both groups was, what was termed, rapid rotations (Kondracki et al., 1992). These were relatively large rotations occurring over one increment, often paradoxical in direction, with respect to the overall range. With screening during active voluntary movements, however, it was difficult to determine if

these irregular rapid rotations were a momentary loss of tension in the holding elements, reflex changes in spinal musculature or phenomena related to overall voluntary trunk motion. Another feature of fluoroscopic imaging during voluntary movement is that the radiographer must control the X-ray tube and follow the subject's lumbar spine throughout the sequence. This often results in the extreme segments of the lumbar spine (L1/L2 and L5/S1) being lost from the field of view. When calculating indices that depend on comparison between overall lumbar spine and segmental motion, however, this loss of information is problematic. Irregular motion, expressed in terms of the largest incremental change as a proportion of the overall range of motion, (Kondracki & Breen, 1993) appeared to be a consistent feature in DVF studies of both normal subjects and back pain patients. This, again, may have been attributable to the methods of data gathering.

From the preceding discussion it is obvious that, in short, too many variables, introduced by the original radiographic protocol, may hinder full interpretation of the raw data. Experience gained over several years has led to a number of suggested modifications in data collection and analysis. Of most pressing importance, has been identified alterations to the radiographic method such that it is capable of:

- Introducing greater lumbar rotations than previously;
- Producing smooth trunk motion at a consistent rate;
- Providing known start and stop positions for each sequence;
- A reduction in erratic voluntary muscle behaviour;
- Producing good quality images that consistently include the entire lumbar spine;

7.2 DESIGN

To fulfil the above criteria the simplest solution would be a motorised device, on which the patient could lie recumbent, that would take them smoothly and passively through a known rotational range during fluoroscopic screening. The recumbent position was considered essential to obtain passive motion and reduce axial pre-load. Instability of the passive restraining tissues, it was thought, would be much more likely to manifest itself under these conditions. Wood and colleagues (Wood et al., 1994), in a dynamic imaging study of spondylolisthesis, were able to demonstrate significantly greater amounts of

abnormal motion in the lateral recumbent position than in standing weight-bearing films.

This device would then, theoretically, challenge the passive holding elements such as the intervertebral disc and joint capsules without the influence of voluntary muscular contractions. The use of passive motion methods in obtaining functional radiographs of the lumbar spine, especially when assessing possible instability, has gained some support (Dvorak et al., 1991).

With these questions in mind the task of designing a prototype table to meet these requirements commenced. Certain tables used in chiropractic practice allow the lumbar spine to be passively flexed and extended or laterally flexed depending on whether the patient is in the supine or lateral position. These tables, however, are stand-alone devices usually made from materials such as tubular steel and upholstered aluminium. They are, by design, weighty to impart stability and durability and are not readily transportable. They also have the disadvantage of being manufactured from materials that have poor radiolucency. The optimum device would be one capable of the same articulation but of a much lighter design, allow motorization, have no radio-opaque parts, moving or non moving, in the field of view and fit easily onto the fluoroscope plinth (Figure 7.1). With a few preliminary sketches a company, which specialises in the design and manufacture of chiropractic tables (Atlas Clinical Ltd), was approached.

The initial prototype was a simple two-piece structure that was positioned directly on top of the fluoroscope plinth. This device was non-motorised and required an operator to manually rotate the table and subject during screening. It was hoped that motorization would not be necessary for the preliminary work as it is potentially the most costly modification. However, in view of the difficulty in displacing the device in a consistent manner and the hazards of exposure to ionising radiation, it was decided that motorization would take a high priority. Another drawback with this prototype was due to its direct contact with the fluoroscope plinth. The plinth, on which the patient lies, had a concave bevel to provide support and security. This had the effect of causing the prototype table to bow and distort thus impeding its motion. It was therefore decided to secure the table to an aluminium frame, which could then be firmly but temporarily attached to the fluoroscope plinth (Figures 7.2). Following modification the current version comprises three main sections, a two-piece articulating section (upper and lower) on which the patient/subject is placed and a base section that extends the caudal end of the fluoroscope

plinth and provides a surface upon which the table rotates. Fitted to this is a simple step motor that can be controlled remotely from the safety of the X-ray screen. The motor is capable of taking the lower section of the table through a circular arc of around 90 degrees in approximately 12 seconds. For convenience the arc is marked in 5 degree increments. Another attachment allowed us to vary the motor speed by means of a dial transformer. The aluminium frame was additionally fitted with four flanges to prevent lateral movement of the table with respect to the plinth. The entire device could be assembled and attached to the fluoroscope in less than 10 minutes. When not required, the table can be conveniently stored within the radiology department.

FIGURE 7.1

FLUOROSCOPE PLINTH WITH PASSIVE MOTION TABLE ATTACHED

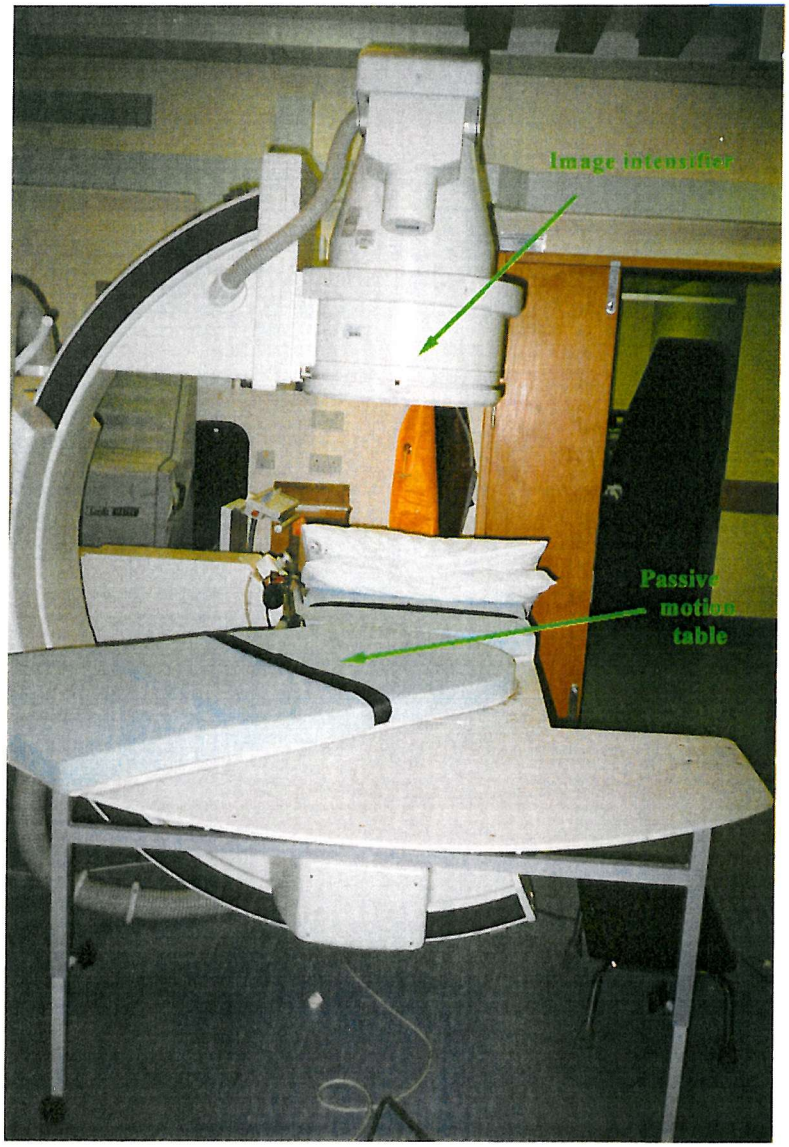
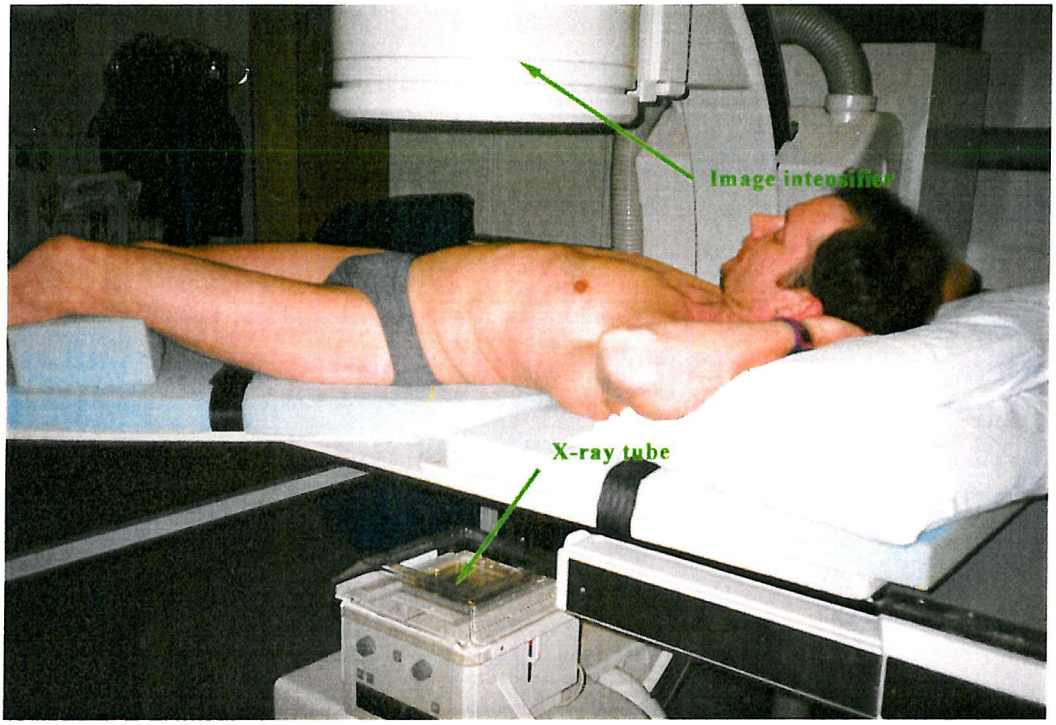


FIGURE 7.2

PASSIVE MOTION TABLE WITH PATIENT



7.3 IMPLEMENTATION

To avoid exposing patients/subjects to unnecessary radiation, a number of non-radiographic trials were undertaken, using volunteers, to determine optimum positioning and stabilisation. Initially the subjects were placed directly onto the surface of the table, since this was thought to provide greater adherence to the table during motion and thus produce maximal movement in the lumbar spine. This, however, proved too uncomfortable for the subjects and the provision of radiolucent foam material between subject and table became necessary. This was found to work well and did not impede movement of the table, it also seemed to somewhat improve adherence between the subjects and the table surface. The greater comfort afforded by this modification had the added benefit of reducing unnecessary changes in subject position. Once correctly placed on the table it is also important that during the arc of motion the lumbar spine does not "drift" out of the field of the X-ray beam. In order to simulate the central ray of the fluoroscope tube, a laser pointer was used. This was suspended directly above the subject with the laser point projected onto the skin overlying the L3 vertebra. The subject could then be positioned such that the point remained relatively static throughout the table's rotation arc. These early trials established that 40^0 of extension was tolerated well but was sufficient to challenge the lumbar spine in all of the asymptomatic volunteers. The same extent of flexion, on the other hand, challenged the volunteer's spine only if a sufficient amount of hip flexion had been introduced first, by bringing the knees towards their chests.

The following two chapters describe a recent study employing the passive motion table in the investigation of i*n vivo* lumbar spine motion in 10 asymptomatic subjects. Chapter 8 outlines the methodology, including assessment of measurement error, and Chapter 9 details the results of the study.

CHAPTER 8

THE PASSIVE INTERVERTEBRAL MOTION OF THE LUMBAR SPINE IN ASYMPTOMATIC SUBJECTS: A PRELIMINARY STUDY (METHODOLOGY)

8.1 PASSIVE MOTION TABLE PROTOCOL

Ten male subjects, from the thirty employed in the passive motion trial, were used in the study. Their ages ranged from 19 to 40 years, with a mean age of 28 years. All subjects were free from back pain at the time of the study and for a period of at least 6 months prior to investigation. Subjects were excluded if they had suffered any significant back pain, i.e. incapacitating pain or moderate pain of greater than 6 weeks duration. Additionally, subjects were excluded if they had undergone any major back surgery such as fusion, laminectomy or discectomy.

Each subject was placed on the passive motion table (PMT), firstly on their right hand side with their back to the radiographer's booth (Figure 8.1). The subject was moved up or down until the interface between the moving lower section and static upper section of the table was at the level of L3. This was thought to maximally concentrate the movement over the whole lumbar spine. Their position was further adjusted by the operator until the subject was comfortable, stable and in attitude as close to the neutral spinal position as possible. A strap running underneath the PMT and over the subject secured the upper body. The strap passed under a restraining clamp attached to the table, the subject's free arm (left arm) and finally a second restraining clamp, which was tightened once the subject was optimally positioned. The strap was tightly applied to the subject's left axilla and chest wall and was cushioned for comfort and to prevent abrasion. The lower body was left unsecured to alleviate excessive discomfort. A pillow was placed between the knees to help keep the pelvis aligned and the knees and hips slightly flexed to avoid over-extension of the lumbar spine. The subject was then taken through the movements several times to establish whether or not they could tolerate the full range (40 degrees of table flexion and extension) and to correct any minor displacements. Most often, full flexion was not perceived as the end-range and greater hip flexion was necessary to fully flex the

lumbar spine. Subjects, on the other hand, always felt full extension, as extreme and mildly uncomfortable. Each subject was then taken through the sequence under fluoroscopic screening. For flexion/extension the sequence began from the initial neutral position and proceeded to full flexion where the table direction was immediately reversed and the subject taken into full extension (Figure 8.2). Again the direction was immediately reversed and the subject taken back to the initial neutral position. Automatic control of X-ray dosage by the image intensifier, however, often resulted in image "flaring" during flexion/extension. This occurred at points in the sequence when the subjects' trunk failed to cover the full extent of the X-ray beam and the image intensifier received unattenuated rays. To reduce this problem flexible lead sheeting was taped to the subjects' back (Figure 8.1). This approach worked well for flexion but occasionally failed during extreme extension as the sheets buckled under compression and lifted away from the tethering. This was unavoidable, as further repeat screening would have resulted in unacceptable exposure. The wider profile offered by the trunk in supine screening obviated the need for such measures in the lateral bending studies.

For lateral flexion screening the subject was placed in the supine position with a small pillow under the knees for comfort and to help reduce excessive extension of the lumbar spine. The same protocol as observed for flexion/extension was then carried out with the subject being taken into right lateral flexion (RLF) first and then into left lateral flexion (LLF) before returning to neutral. Each sequence took around 30 seconds to complete with most subjects screened in both planes for a total of, less than, one minute of exposure. Volunteers were subjected to a second screening of flexion/extension and lateral bending within 20 minutes of the first.

FIGURE 8.1

SUBJECT IN LATERAL POSITION READY FOR FLEXION/EXTENSION SCREENING, SHOWING LEAD SHEET IN PLACE TO REDUCE "FLARING".

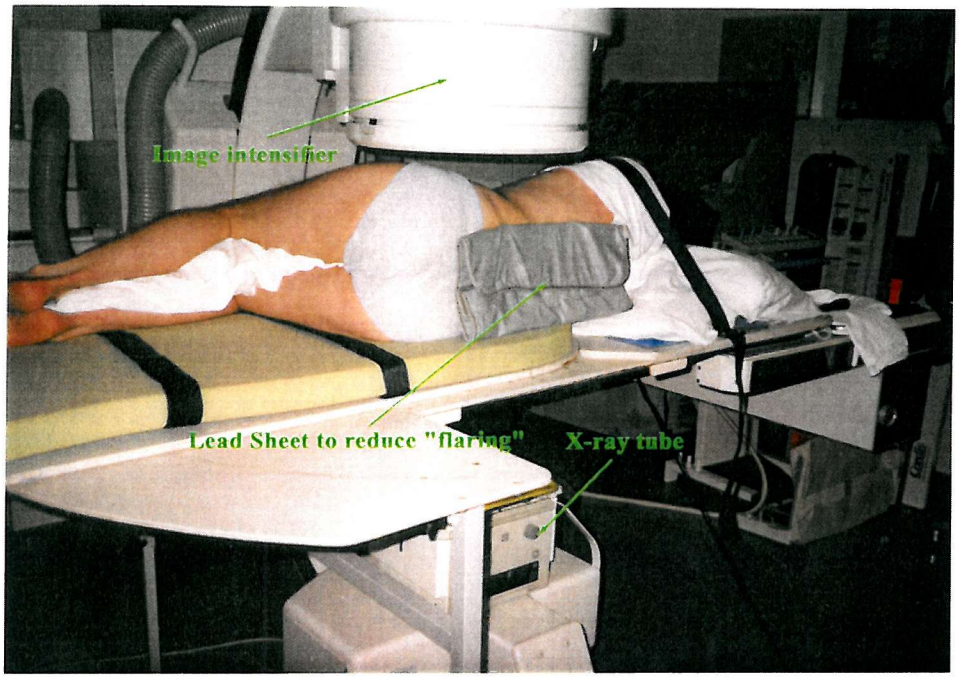
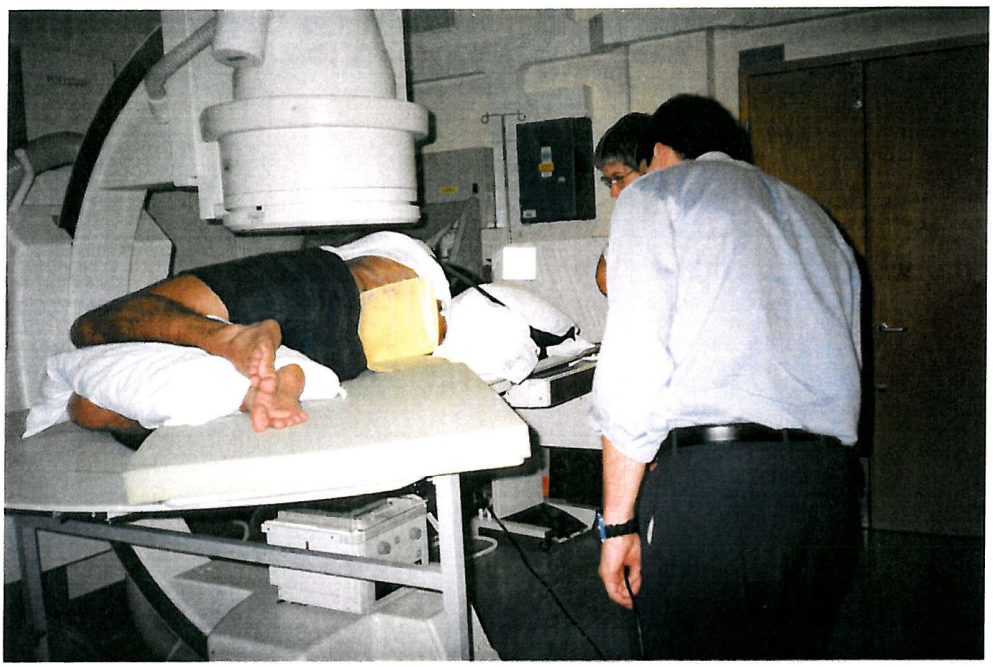


FIGURE 8.2

SUBJECT BEING ASSESSED FOR RANGE OF EXTENSION.



8.2 DATA ACQUISITION

The video images were obtained directly from the video output of the fluoroscope and digitised on-site to the hard disk of a PC. Since this development obviates the need for an intermediate video stage, image degradation is reduced and it becomes appropriate to henceforth use the term digitised fluoroscopy (DF) rather than DVF.

As each of the images required a great deal of memory, it was not possible to predetermine the sampling rate. Instead, the frame grabbing rate was set at "fast as possible" which allowed us to obtain 150 frames for each sequence, i.e. flexion/extension or right/left side bending. This resulted in an image a capture rate of 5 frames per second. Thus 150 frames allowed us a 30 second window for image acquisition of each sequence.

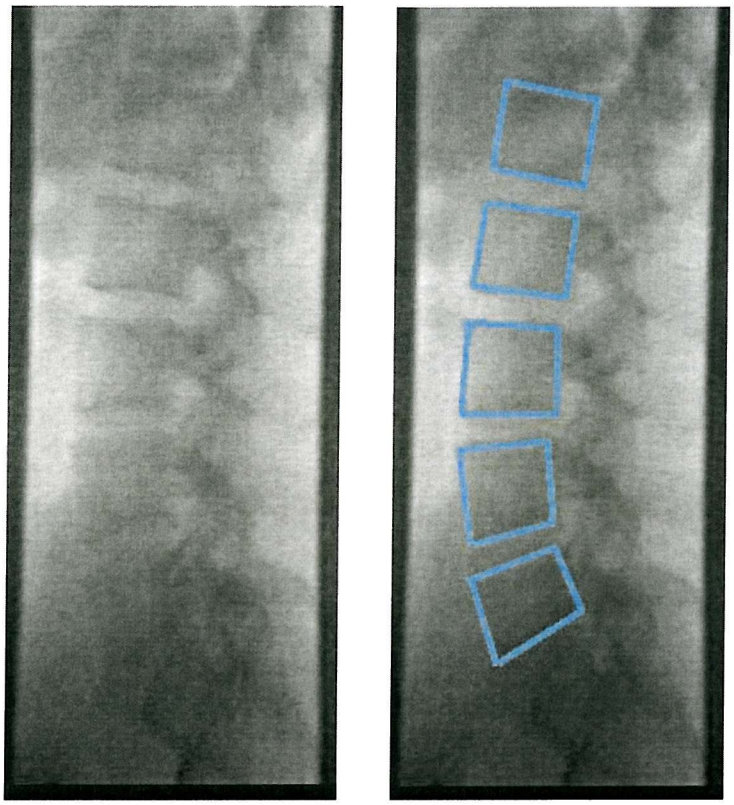
8.3 MANUAL MARKING

Image processing was performed manually using the Optimas system. This requires the identification and selection of each vertebral corner, according to the method described by Frobin and colleagues (Frobin et al., 1996), for all lumbar segments from the initial, neutral, image (Figure 8.3). This process forms a rectangular template around each vertebral body, which can be mapped, using rotation and translation, to its corresponding segment on subsequent images (Figure 8.3). In this way vertebral position, in one plane, can be determined throughout the motion sequence. Manual marking is, by nature, time consuming and wearing and therefore it was not possible to carry out analysis on all frames. In this present study, analysis was performed on every fourth frame, thus encompassing a time span of approximately 0.8 seconds for each increment. With a maximum of 150 frames per sequence, this division required around 37 image analyses and thus increments for each motion cycle. Given that each of the ten volunteers was subjected to both flexion/extension and lateral bending motion studies and screened twice for each, brings the total number of analysed images to 150 per subject.

These objectives were achieved by customising the Optimas package using software developed by Dr Jen Muggleton. Dr Muggleton was also present throughout the data acquisition process for all subjects and whose invaluable assistance is greatly appreciated.

FIGURE 8.3

IMAGES OF LATERAL LUMBAR SPINE WITH AND WITHOUT TEMPLATES



8.4 DATA ANALYSIS

The present study attempted to measure changes in intervertebral angles (IVAs), anteroposterior translation and axial translation in the sagittal plane in ten asymptomatic subjects. Coronal plane studies were restricted to the measurement and analysis of angular change only.

The initial positioning of the vertebral templates was normalised to zero so that all angular measurements were expressed as change relative to the starting neutral position. In this manner, the raw data shows relative movement of each of the vertebral bodies, where possible, from L1 to S1 (Figure 8.4). Since movement of the subject within the image plane was not fixed to a reference point, the relative movement of one segment with its subjacent neighbour was considered more appropriate for study. The intervertebral angles (IVAs) were thus derived by subtraction of the related vertebral angles.

An example of the IVA data over the motion sequence is depicted in Figure 8.5. This typical graph of lateral bending motion shows the expected sinusoidal motion pattern and also the finding that the lumbar spine does not return to its precise starting point. This is probably a result of the combination of subject shift on the table, measurement error and the variability of the neutral posture. The latter point will be discussed more fully in the following section.

FIGURE 8.4

CHANGE IN VERTEBRAL BODY ANGLE L1-L5 FROM NEUTRAL

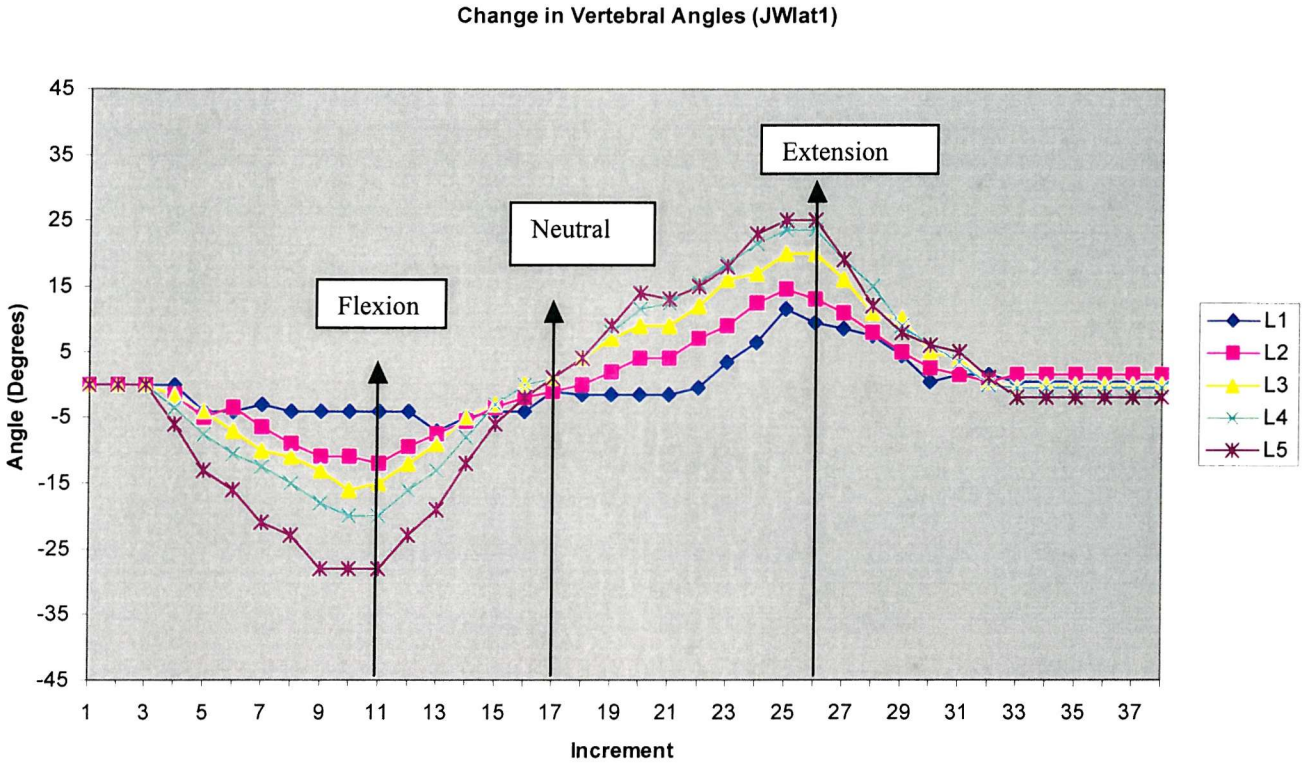
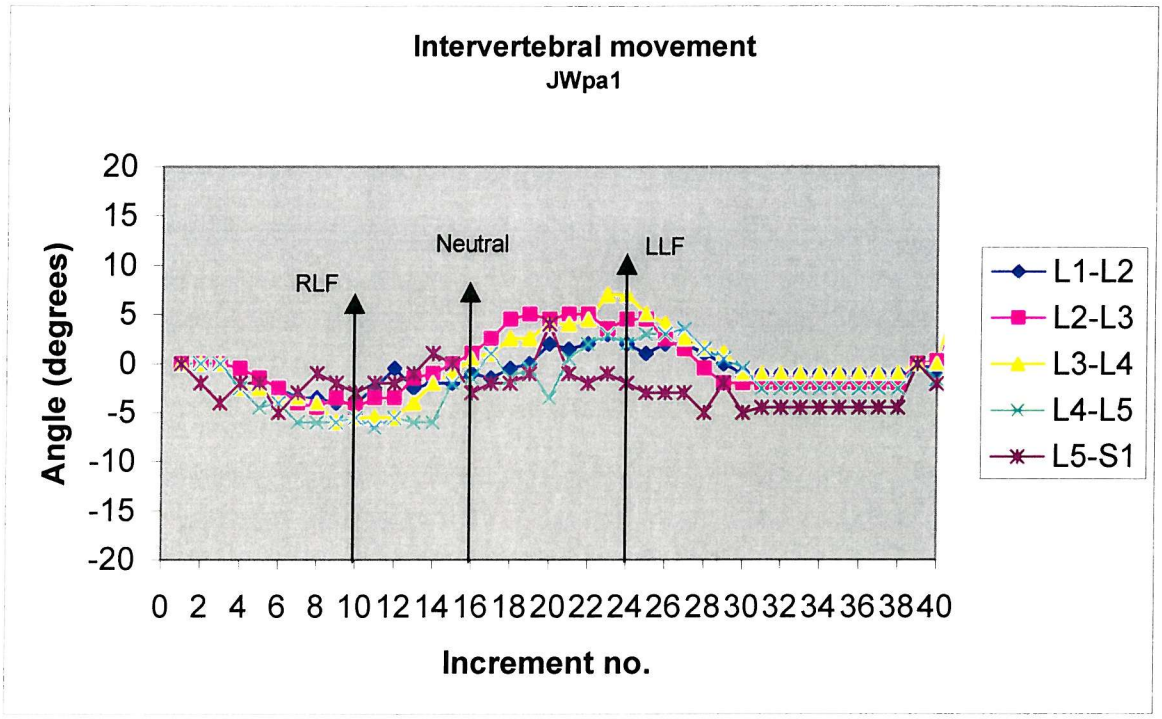


FIGURE 8.5

IVAS FOR FLEXION/EXTENSION AND LATERAL BENDING SHOWING EXPECTED

SINUSOIDAL MOTION



8.5 DETERMINATION OF NEUTRAL POSITION

In the present study all calculated IVAs started from zero and angular change was measured from this point. In this way the neutral posture was defined as the configuration of the lumbar spine at the commencement of the motion sequence. As discussed in the methodology, subjects were positioned in a comfortable manner in a position, as close to, what appeared to be, a neutral trunk posture. With weight bearing studies this is relatively simple, the lumbar spine is considered to be in a neutral position on comfortable erect standing (Lin et al., 1994; Okawa et al., 1998; Pearcy, 1985). Some authors have attempted to be more specific by defining the initial position of the feet and vertex of the head (Yoshioka et al., 1990). Kanayama and colleagues (Kanayama et al., 1996) recognised the need for standardisation in their *in vivo* study and made efforts to define the starting position for all subjects. In that study, neutral erect standing was achieved when

the occipital, midthoracic and sacral regions were aligned perpendicular to the floor. Most recently, another Japanese group, led by Harada (Harada et al., 2000), undertook a cineradiographic study of lumbar flexion and extension in asymptomatics. They too, commented on the difficulty of establishing neutral and employed a continuous motion protocol starting either at full extension and proceeding to full flexion or *vice versa.* Comparison of results with the present study, however, was difficult because of the methodological differences. Of note, however, was the finding by Harada's group that the L5/S1segment exhibited markedly different motion behaviour from other segments. In particular, they report that levels L3/4 and L4/5 showed similar behaviour in both full flexion to full extension and full extension to full flexion. The L5/S1 segment, on the other hand, behaved differently depending on the direction of trunk motion. This, once again, highlights the functional distinctiveness of the lumbosacral joint. Limitations of the present study, not least of which were the radiographic difficulties, unfortunately prevented meaningful investigation of this recurring theme.

In recumbent studies determining neutral is not quite so straightforward as for standing protocols. In a recent MRI investigation (Edmondston et al., 2000) the authors employed a supine protocol, which used rolled towels under the lumbar spine to induce flexion and pillows under the knees to produce extension of the lumbar spine. This type of approach ignores the need for a neutral position but, arguably, loses much information on, what has become, a very revealing position of the human spine in terms of its mechanical integrity. Another recent dynamic MRI study (Vitzthum et al., 2000) using a seated protocol, again, avoided neutral and opted for a flexion/extension only method. In the present study, the neutral posture was approximated by positioning the subject in a comfortable mid-position. In doing so, this starting intervertebral configuration defined neutral for the entire sequence.

8.6 ESTIMATION OF MEASUREMENT ERROR (ACCURACY)

Previous incarnations of this work have relied on the use of a bony calibration model to assess accuracy of the system in measuring angles (Breen, 1991). This model comprised two human lumbar vertebrae linked by a universal joint and connected to a protractor for accurate angular displacement. The results of this earlier work reported a 2SD error of around 2^0 for images of the model degraded by soft-tissue scatter. For the present study it was decided to use a simpler, more direct, method for assessing systematic accuracy. The

reasons for this were twofold, firstly the original calibration model proved difficult to use with the present fluoroscope. Previous work had involved using an upright protocol with a free standing X-ray tube and image intensifier, this enabled the model to be placed directly in front of the image intensifier where subjects would sit. With the present fluoroscope and recumbent protocol this was not possible without considerable modifications. Secondly it was felt that any calibration should be applied throughout the viewable area and not confined to the region of the central beam. Furthermore, in order for any such model to be truly realistic it should include all lumbar segments and pelvic bones to simulate the radiographic difficulties encountered at L5/S1. In addition, the spinal model should be fixed within a simulated "trunk" to provide a realistic displacement from the tube and intensifier and also replicate soft-tissue scatter. To achieve this would involve the development of an articulated "phantom" beyond the budgetary scope of the present study. Instead, a compromise was reached by using an aluminium grid (Figure 8.6). The grid was formed by precision-drilled holes of 1mm diameter at 1cm spacing over an area of 14cm by 14cm. By placing the grid on the fluoroscope table, it was possible to move the table towards or away from the image intensifier and thus simulate the estimated positions of the lumbar spine in subjects of varying trunk diameters (Figure 8.7). The images of the grid were then stored for each of the estimated lumbar spine positions relative to the trunk (Figure 8.7). For calibration purposes a selection of three of these distances were used: 5, 10 and 13cm.

FIGURE 8.6

ALUMINIUM CALIBRATION GRID

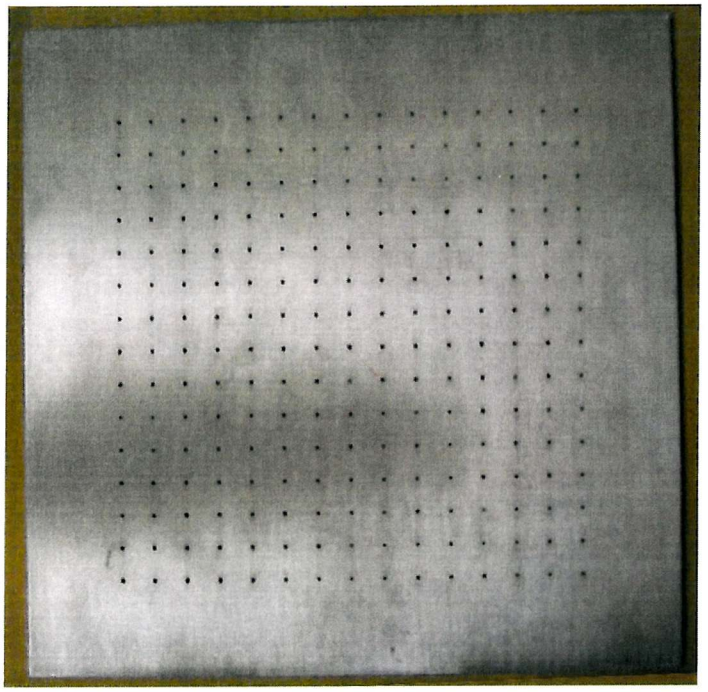
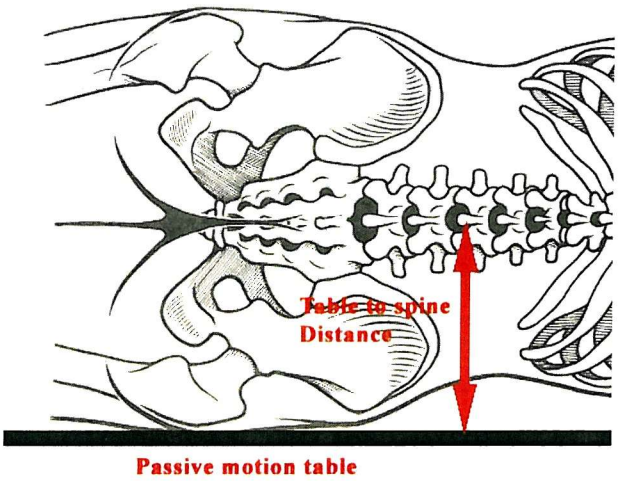


FIGURE 8.7

MEASUREMENT OF SPINE-TO-TABLE DISTANCES USED TO ASSESS THE POSITION OF THE LUMBAR SPINE RELATIVE TO THE IMAGE INTENSIFIER



For each of these images five areas of the grid were selected for measurement, central, lower right, lower left, upper right and upper left (Figure 8.8). At the selected regions the "Line" function of the image processing software was used to measure the angles between lines drawn on the images. Firstly a horizontal was drawn across three of the 1cm squares approximating the average AP diameter of a lumbar vertebra. Secondly, another line was drawn vertical to the first one and lastly, a third, diagonal line was drawn. These measurements were repeated five times for each of the five selected regions of the grid. To account for slight malpositioning of the grid on the fluoroscope table, all angles were normalised to the horizontal. The mean discrepancy and SD for all five selected grid regions across the three spine "distances" were, respectively, $1.2^0$and 2.04^0. These figures, together with values for each grid region, are shown in Table 8.1

The aspect ratio, across the three lumbar spine-to-table distances, was 0.93.

FIGURE 8.8

IMAGE OF CALIBRATION GRID WITH MARKED ANGLES USED TO ASSESS ACCURACY

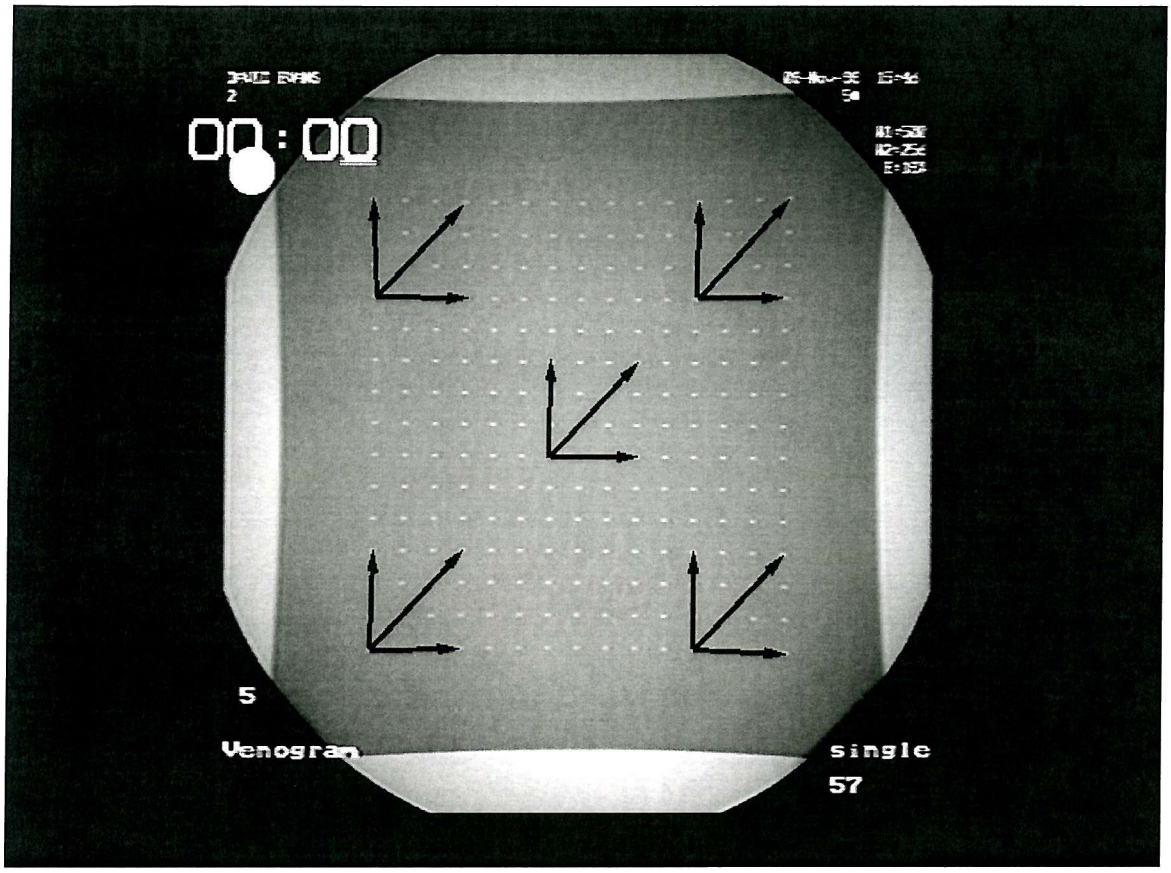


TABLE 8.1

RESULTS OF CALIBRATION STUDY SHOWING MEAN DISCREPANCY AND STANDARD DEVIATION OF ANGLE MEASUREMENTS FOR EACH OF THE FIVE SELECTED REGIONS OF THE GRID AND ALL REGIONS COMBINED (OVERALL)

Calibration	Overall	Centre	Upper Right	Upper Left	Lower Right	Lower left
Mean Discrepancy (Degrees)	1.20	1.88	0.52	1.52	3.25	0.06
(+/- 1SD)	(+/- 2.04)	(+/- 1.18)	(+/- 1.98)	(+/- 1.53)	(+/- 0.93)	(+/- 2.38)

8.7 ESTIMATION OF MEASUREMENT ERROR (REPEATABILITY)

Much of the variation in establishing vertebral kinematics results from the selection of anatomical landmarks (Harvey & Hukins, 1998; Panjabi et al., 1992; Panjabi & White, 1971; Pearcy & Bogduk, 1988). The irregular nature of vertebral morphology, the influence of positional distortion and the use of low dose X-ray techniques, renders the reliable marking of spinal landmarks difficult (Frobin et al., 1996). Using a template method reduces the need for continuous re-selection of these landmarks but introduces other potential sources of error. Analysis by mapping sequential images in one plane with a template assumes a rigid body constraint. However, it does not account for slight movements in other planes or minor shifts in the central beam of the X-ray producing changes in projection characteristics for vertebral segments. In this way, the contours of a segment may appear to change in subsequent images and therefore hinder the accurate placement of the template.

All data for the present study was obtained by one observer (observer 1), and thus the major effort involved establishing intra-observer measurement variation. At the same

time, however, inter-observer data was gathered using 3 additional researchers of varying experience in the manual marking of spinal images.

For intra-observer analysis, a total of five repeated measurements were used. This value was thought to represent a reasonable amount of manual processing to tolerate at any one sitting. This way, it was thought, error as a result of excessive fatigue would not unduly influence the result but at the same time would characterize a realistic simulation of normal conditions. For the inter-observer study, a single image for all three positions was marked by each observer; neutral, full flexion or right lateral flexion and full extension or left lateral flexion. Again, it was felt, this would be sufficient for individuals of varying experience with this type of data acquisition.

In all cases three frames from a randomly selected file were used. The first, starting neutral, frame established the templates which were then applied to two further images representing each extreme of the motion sequence. The raw data, co-ordinates, obtained by these repeated measurements were then used in the calculation of the various parameters, IVA, ICA, translation and so on, in order to establish limits of variation.

8.8 IVA MEASUREMENT VARIATION

Variation was estimated from repeated measurements of the absolute vertebral movement from which the IVA's are derived. Using absolute angles also has the advantage of helping us to determine exactly which vertebral segments are associated with the greatest error rather than the less specific FSU. Tables 8.2 & 8.3 show the intra-observer variation from five repeated measures of subject SA, flexion/extension and right/left lateral flexion. The standard deviation (SD) of the measured angles shows clearly that measurements of the lower segments (L5/S1) are less repeatable than middle or upper segments. This is, of course, a recurring problem and is most likely related to image quality associated with anatomical factors peculiar to this region of the lumbar spine. The S1 vertebral segment and much of the L5 vertebra are shielded by the pelvic bones and hence require greater radiographic penetration in order to visualise them. Images of these segments are therefore less distinct than upper segments covered only by soft tissue. Also, the S1 segment is often more irregular in shape than other vertebrae and difficulties in identifying the contours, especially the inferior corners, are well recognized (Frobin et al., 1996).

In addition, the SD values suggest that, generally, extension measurements are less repeatable than those of flexion. This is possibly a result of the radiographic "flaring" problem associated with the extremes of extension.

Lateral flexion measurements also demonstrate a similar disparity between the L5/S1 segments and the rest of the lumbar spine but not to quite the same extent. In general, anteroposterior (AP) images are "cleaner" and provide clearer, more easily identifiable vertebral corners.

TABLE 8.2

INTRA-OBSERVER VARIATION FOR 5 REPEATED MEASUREMENTS OF VERTEBRAL MOVEMENT RELATIVE TO NEUTRAL (OBSERVER 1)

(SAGITTAL PLANE)

Full Flexion Angle (Degrees)	**L1**	**L2**	**L3**	**L4**	**L5**	**S1**
	-5	-8	-12	-18	-23	-4
	-5	-10	-14	-18	-19	-5
	-5	-9	-14	-17	-22	-4
	-5	-9	-14	-19	-23	-7
	-5	-10	-13	-18	-21	-4
Mean	***-5.00***	***-9.20***	***-13.40***	***-18.00***	***-21.60***	***-4.80***
+/- (1SD)	***(0.00)***	***(0.84)***	***(0.89)***	***(0.71)***	***(1.67)***	***(1.30)***

Full Extension Angle (Degrees)	**L1**	**L2**	**L3**	**L4**	**L5**	**S1**
	9	15	20	26	26	17
	10	13	18	26	28	16
	7	14	21	25	28	15
	7	15	20	26	29	10
	7	15	19	24	29	9
Mean	***8.00***	***14.40***	***19.60***	***25.40***	***28.00***	***13.40***
+/- (1SD)	***(1.41)***	***(0.89)***	***(1.14)***	***(0.89)***	***(1.22)***	***(3.65)***

TABLE 8.3

INTRA-OBSERVER VARIATION FOR 5 REPEATED MEASUREMENTS OF VERTEBRAL MOVEMENT RELATIVE TO NEUTRAL (OBSERVER 1)

(CORONAL PLANE)

Right Lateral Flexion Angle (Degrees)	**L1**	**L2**	**L3**	**L4**	**L5**	**S1**
	-13	-15	-20	-25	-26	-32
	-13	-15	-18	-23	-26	-33
	-13	-16	-19	-23	-28	-32
	-13	-15	-18	-25	-28	-34
	-13	-16	-18	-24	-27	-32
Mean	***-13.00***	***-15.40***	***-18.60***	***-24.00***	***-27.00***	***-32.60***
+/- (1SD)	***(0.00)***	***(0.55)***	***(0.89)***	***(1.00)***	***(1.00)***	***0.89***
Left Lateral Flexion Angle (Degrees)	**L1**	**L2**	**L3**	**L4**	**L5**	**S1**
	6	9	13	16	17	23
	7	8	14	16	20	22
	7	8	13	16	20	22
	7	8	14	16	19	20
	6	8	13	18	20	23
Mean	***6.60***	***8.20***	***13.40***	***16.40***	***19.20***	***22.00***
+/- (1SD)	***(0.55)***	***(0.45)***	***(0.55)***	***(0.89)***	***(1.30)***	***(1.22)***

The inter-observer variation of IVA measurements for three and four observers is shown in tables 8.4 to 8.7. In these studies observer 1 and three additional observers manually marked three images from the same file representing neutral, full flexion and full extension. This was repeated for the lateral bending sequence. In each case the neutral image allowed the observer to create the templates of all segments before applying them to the images of the extremes of motion. Observer 1 had the greatest current experience of manual marking amongst the group whilst observer 2 had past previous experience of marking using a prototype system. Observer 3 had only recently begun using image-processing techniques on lumbar spine images, whereas observer 4 was without any previous familiarity with this type of work and this was his first attempt at a template fitting procedure.

Subsequent to the data collection it became apparent that observer 4 had made an error in fitting the S1 template to the flexion image (Table 8.4). Measurements of IVAs made by observer 4 were then removed from the analysis to study the overall effect on variation (Table 8.5). As can be seen from the SD of these measurements, apart from nullifying the effect of the erroneous flexion value for S1, removal of observer 4 data has only a minimally beneficial effect on repeatability. Given the current level of image quality then, the technique would appear reasonably robust given the limitations of manual marking.

TABLE 8.4

INTER-OBSERVER VARIATION BETWEEN 4 OBSERVERS FOR 2 IMAGES (FULL FLEXION, FULL EXTENSION)

(SAGITTAL PLANE)

Full Flexion Angle (Degrees) Observers 1,2,3,4	**L1**	**L2**	**L3**	**L4**	**L5**	**S1**
Mean	*-5.25*	*-8.50*	*-13.75*	*-17.25*	*-23.25*	*-11.00*
+/- (1SD)	*(0.96)*	*(0.58)*	*(1.71)*	*(2.22)*	*(2.87)*	*(10.74)*
Full Extension Angle (Degrees) Observers 1,2,3,4	**L1**	**L2**	**L3**	**L4**	**L5**	**S1**
Mean	*8.25*	*14.00*	*19.25*	*23.50*	*24.75*	*16.00*
+/- (1SD)	*(0.96)*	*(1.41)*	*(0.96)*	*(2.38)*	*(1.89)*	*(2.71)*

TABLE 8.5

INTER-OBSERVER VARIATION BETWEEN 3 OBSERVERS FOR 2 IMAGES (FULL FLEXION, FULL EXTENSION)

(SAGITTAL PLANE)

Full Flexion Angle (Degrees) Observers 1,2,3,	**L1**	**L2**	**L3**	**L4**	**L5**	**S1**
Mean	*-5.67*	*-8.67*	*-13.00*	*-16.33*	*-22.00*	*-5.67*
+/- (1SD)	*(0.58)*	*(0.58)*	*(1.00)*	*(1.53)*	*(1.73)*	*(1.53)*
Full Extension Angle (Degrees) Observers 1,2,3,	**L1**	**L2**	**L3**	**L4**	**L5**	**S1**
Mean	*8.00*	*14.67*	*19.67*	*24.00*	*24.33*	*15.33*
+/- (1SD)	*(1.00)*	*(0.58)*	*(0.58)*	*(2.65)*	*(2.08)*	*(2.89)*

TABLE 8.6

INTER-OBSERVER VARIATION BETWEEN 4 OBSERVERS FOR 2 IMAGES (RLF, LLF)

(CORONAL PLANE)

Right Lateral Flexion Angle (Degrees) Observers 1,2,3,4	**L1**	**L2**	**L3**	**L4**	**L5**	**S1**
Mean	*-13.00*	*-16.25*	*-19.00*	*-24.25*	*-27.00*	*-30.00*
+/- (1SD)	*(0.82)*	*(2.22)*	*(1.83)*	*(0.96)*	*(1.15)*	*(2.16)*

Left Lateral Flexion Angle (Degrees) Observers 1,2,3,4	**L1**	**L2**	**L3**	**L4**	**L5**	**S1**
Mean	*7.00*	*7.75*	*12.75*	*15.75*	*18.50*	*21.50*
+/- (1SD)	*(1.41)*	*(0.96)*	*(0.96)*	*(0.50)*	*(1.91)*	*(1.91)*

TABLE 8.7

INTER-OBSERVER VARIATION BETWEEN 3 OBSERVERS FOR 2 IMAGES (FULL FLEXION, FULL EXTENSION)

(CORONAL PLANE)

Right Lateral Flexion Angle (Degrees) Observers 1,2,3,	**L1**	**L2**	**L3**	**L4**	**L5**	**S1**
Mean	*-12.67*	*-16.00*	*-18.33*	*-24.33*	*-27.33*	*-29.67*
+/- (1SD)	*(0.58)*	*(2.65)*	*(1.53)*	*(1.15)*	*(1.15)*	*(2.52)*

Left Lateral Flexion Angle (Degrees) Observers 1,2,3,	**L1**	**L2**	**L3**	**L4**	**L5**	**S1**
Mean	*7.33*	*7.67*	*13.00*	*16.00*	*19.00*	*21.00*
+/- (1SD)	*(1.53)*	*(1.15)*	*(1.00)*	*(0.00)*	*(2.00)*	*(2.00*

8.9 AP TRANSLATION MEASUREMENT VARIATION

These measurements, as with all other measurements in the present study, employed the co-ordinates of the anterior and posterior corners as obtained from the vertebral templates. Once acquired, these points formed the input data for the calculation of relative translation using the method described by Frobin and colleagues (Frobin et al., 1996) (Figure 8.9). This method uses the bisectrix of the intervertebral angle and a scalar sliding parameter to define the anteroposterior displacement of the vertebral body centre points (Appendix I). These values were derived using the Microsoft Excel spreadsheet software. The calculation coding was achieved with the invaluable assistance of Dr Chris Howls of the Department of Mathematics, University of Southampton.

Once the magnitude and direction of the translation vector has been established it can then be expressed as a percentage of the superior vertebral body depth. The average depth of the segment was determined by calculation of the distances between the superior, middle and inferior points anteriorly and posteriorly. This was carried out to account for any irregularities in vertebral body shape.

The intra-observer variation for five repeated measurements of all levels in neutral, flexion and extension is shown in Figures 8.10-8.12. Inter-observer variation for single measurements of neutral, full flexion and full extension by three observers is shown in Figures 8.13-8.15

FIGURE 8.9

METHOD OF MEASURING AP TRANSLATION ATTRIBUTED TO FROBIN AND COLLEAGUES (1996). ALL VALUES FOR AP TRANSLATION IN THE PRESENT STUDY REPRESENT THE DISTANCE DC AS A PERCENTAGE OF SUPERIOR BODY DEPTH.

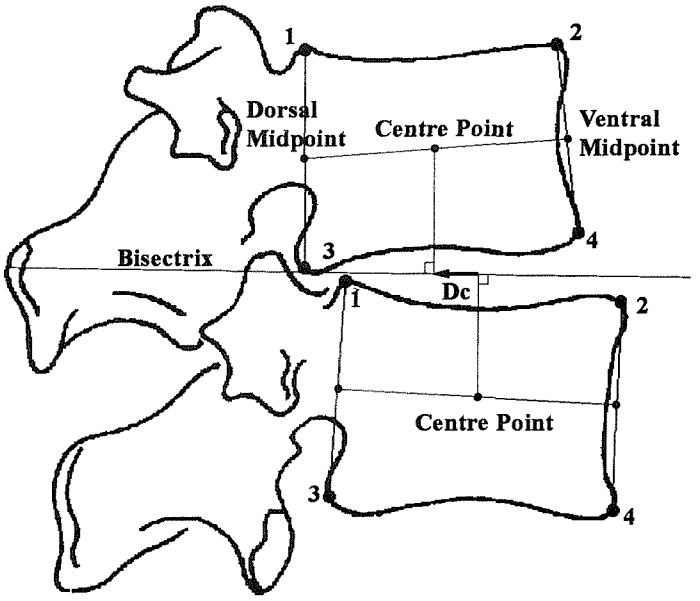


FIGURE 8.10

INTRA-OBSERVER VARIATION (OBSERVER 1) FOR AP TRANSLATION BASED ON 5 REPEATED MEASUREMENTS OF ONE LATERAL NEUTRAL IMAGE

(ERROR BARS = +/-1SD)

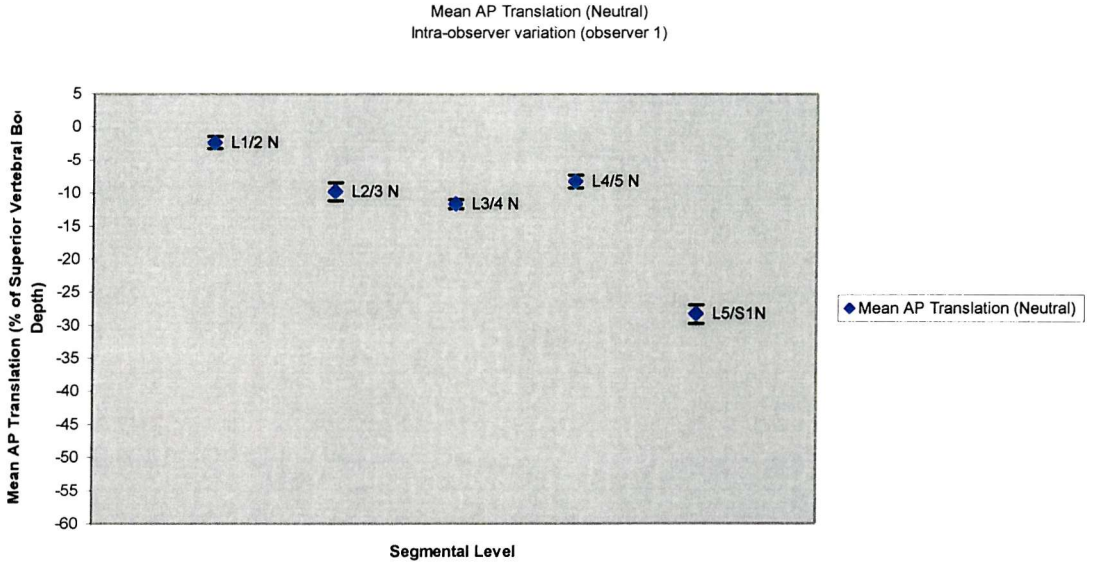


FIGURE 8.11

INTRA-OBSERVER VARIATION (OBSERVER 1) FOR AP TRANSLATION BASED ON 5 REPEATED MEASUREMENTS OF ONE LATERAL FULL FLEXION IMAGE

(ERROR BARS = +/-1SD)

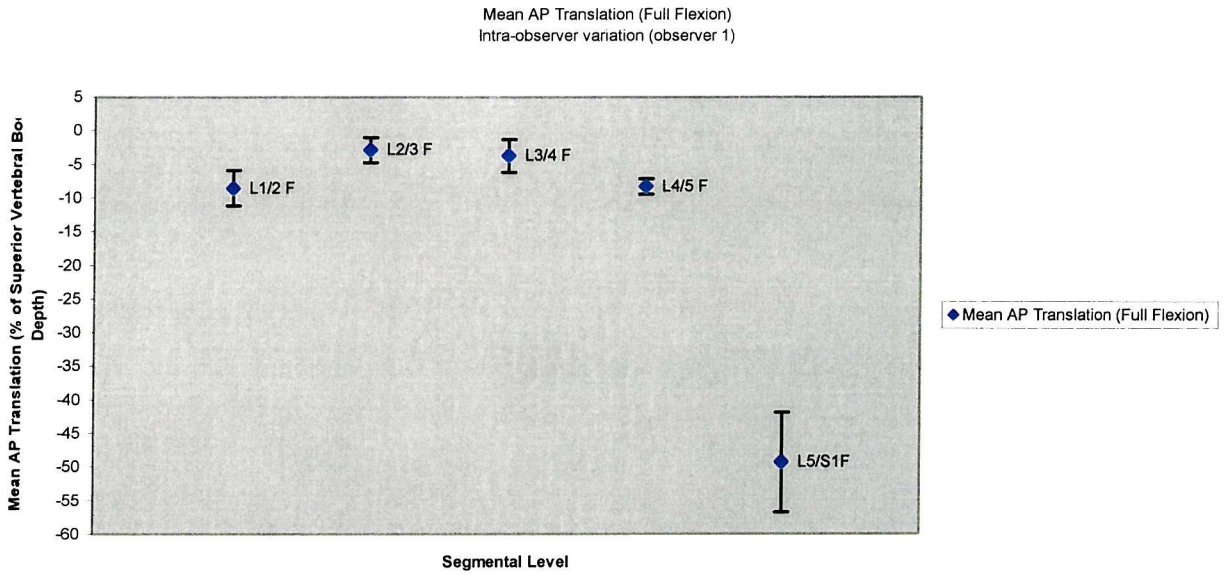


FIGURE 8.12

INTRA-OBSERVER VARIATION (OBSERVER 1) FOR AP TRANSLATION BASED ON 5 REPEATED MEASUREMENTS OF ONE LATERAL FULL EXTENSION IMAGE

(ERROR BARS = +/-1SD)

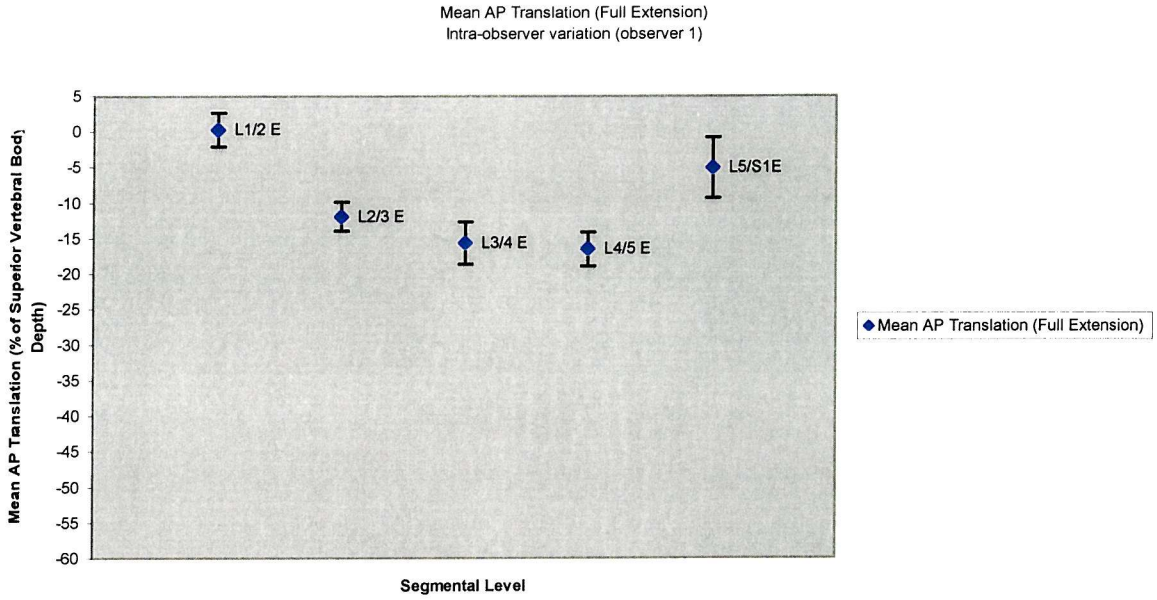


FIGURE 8.13

INTER-OBSERVER VARIATION (OBSERVERS 1,2,3) FOR AP TRANSLATION BASED ON A SINGLE MEASUREMENT BY THREE OBSERVERS OF ONE LATERAL NEUTRAL IMAGE

(ERROR BARS = +/-1SD)

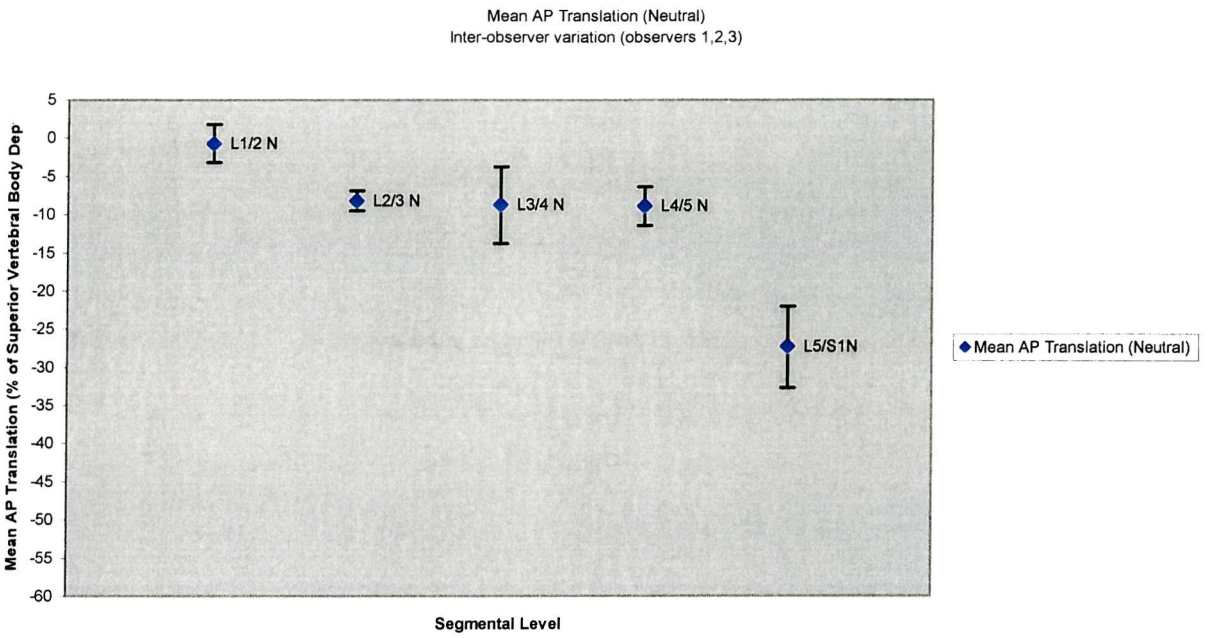


FIGURE 8.14

INTER-OBSERVER VARIATION (OBSERVERS 1,2,3) FOR AP TRANSLATION BASED ON A SINGLE MEASUREMENT BY THREE OBSERVERS OF ONE LATERAL FULL FLEXION IMAGE (ERROR BARS = +/-1SD)

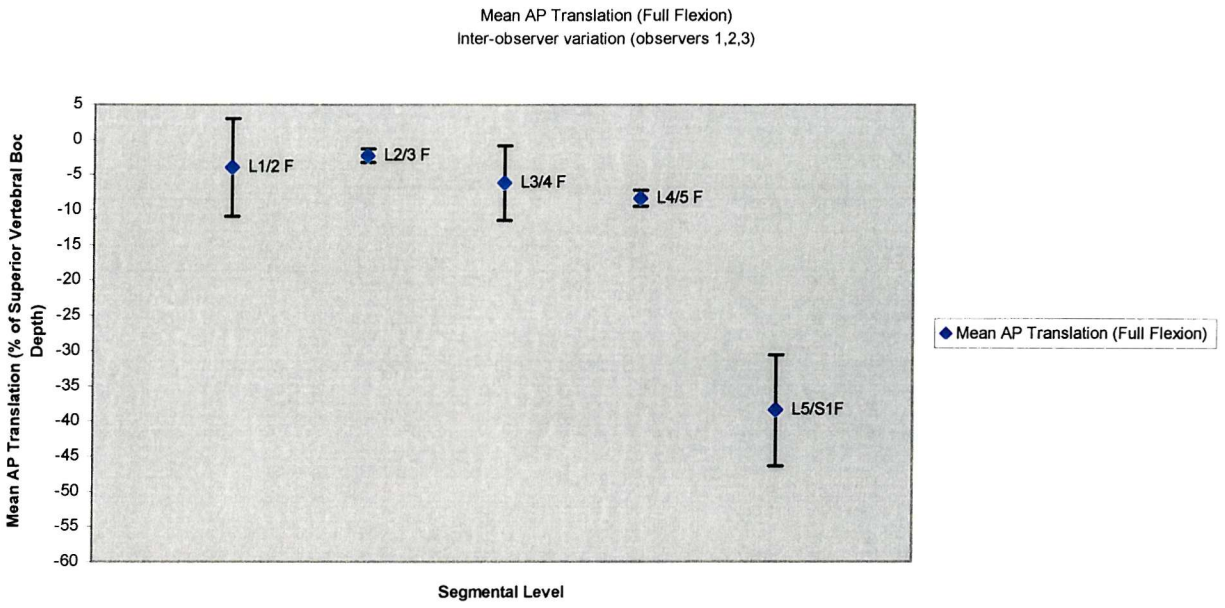
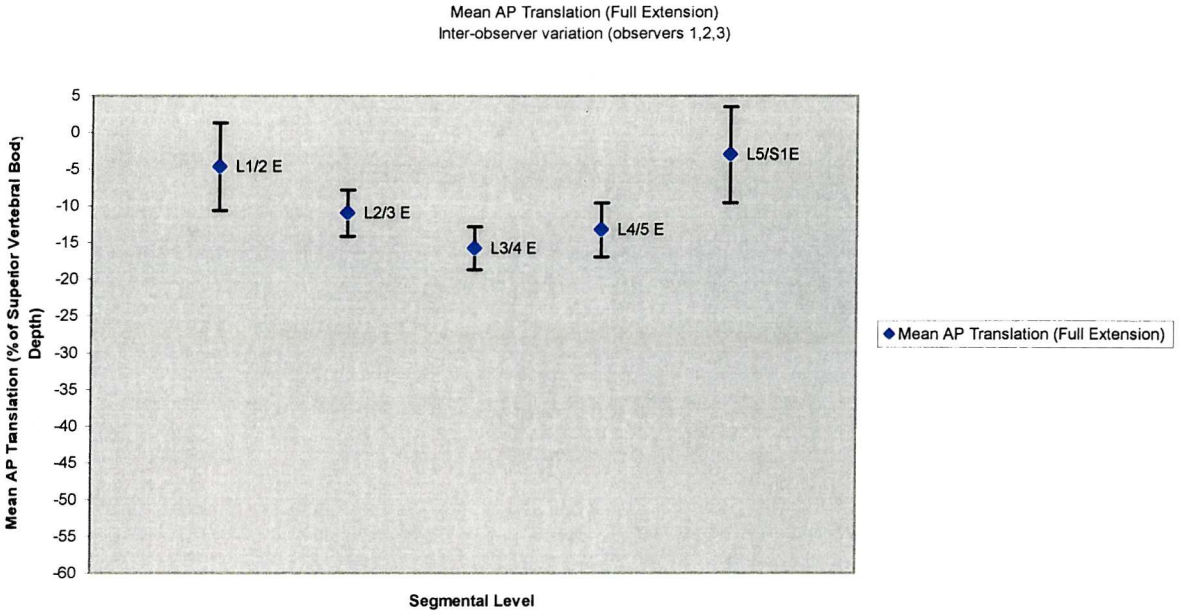


FIGURE 8.15

INTER-OBSERVER VARIATION (OBSERVERS 1,2,3) FOR AP TRANSLATION BASED ON A SINGLE MEASUREMENT BY THREE OBSERVERS OF ONE LATERAL FULL EXTENSION IMAGE (ERROR BARS = +/-1SD)



The variation in measurements of AP translation was, as demonstrated in Figures 8.10-8.15, greater between observers than the intra-observer values determined for observer 1(Figures 8.10-8.12). Standard deviations of between 2 and nearly 8 percent of the upper vertebral AP diameter between examiners do not represent good measurement reliability. Given a distribution of 2SD around the mean values equates to, at worst, a variation of the order of +/- 6mm of a 35mm vertebral body. Nevertheless, the values associated with the greatest variation are almost always those of the L5/S1 segment and when these are excluded from analysis, the single examiner variation is found to be around 2 percent and no greater than 3 percent. Measurement variations of this order result in a 2SD of approximately 2mm around mean values. If AP translation is, indeed, normally only 2-3mm in magnitude then, plainly, measurement variations as described are unacceptable for meaningful interpretation. With a greater range of normal AP translation as suggested by some authors (Dvorak et al., 1991; Hayes et al., 1989; Tallroth et al., 1992) then, perhaps, trend inferences from these data are still valid. It is also worth emphasising that the inter-observer variation in this present study was calculated from single measurements of multiple images and repeated measurements by each examiner may have resulted in more acceptable values.

8.10 AXIAL TRANSLATION MEASUREMENT VARIATION

As suggested by Kaigle and colleagues (Kaigle et al., 1995), shear or AP translation may not yield as much clinically valuable information as previously thought. These researchers and others (Ogon et al., 1997a) have raised the possibility that axial translation may be a more sensitive indicator of loss of intervertebral stiffness. Accordingly an attempt was made, in the present study, to obtain baseline information regarding these types of movements amongst an asymptomatic group. Axial translation, or axial shift, was not measured directly in the present study but inferred from the changes in mean intervertebral disc height and additionally by measuring the distance between centre points. Mean intervertebral disc height was estimated by averaging the distances between 6 points (Figure 8.16). The change in this average value was expressed as a percentage of the mean height of the upper segment, which was also estimated by the distances between 6 points (Figure 8.17). The intra-observer variation for five repeated measurements of all levels in neutral, flexion and extension is shown in Figures 8.18-8.20. As with AP

translation, inter-observer variation for single measurements of neutral, full flexion and full extension by three observers is shown in Figures 8.21-8.23.

FIGURE 8.16

POINTS USED IN THE CALCULATION OF MEAN IVD HEIGHT EMPLOYED AS A MEASURE OF AXIAL TRANSLATION

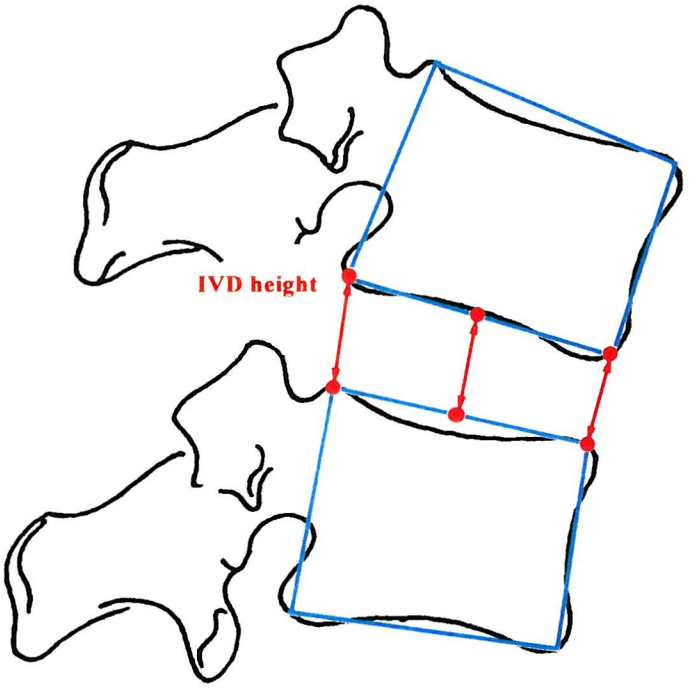


FIGURE 8.17

POINTS USED TO CALCULATE MEAN VERTEBRAL BODY HEIGHT

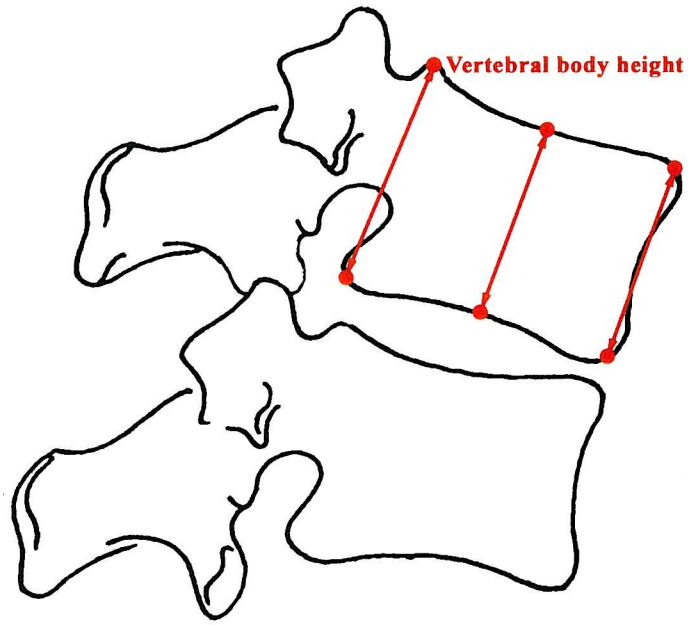


FIGURE 8.18

INTRA-OBSERVER VARIATION (OBSERVER 1) FOR AXIAL TRANSLATION/IVD HEIGHT BASED ON 5 REPEATED MEASUREMENTS OF ONE LATERAL NEUTRAL IMAGE

(ERROR BARS = +/-1SD)

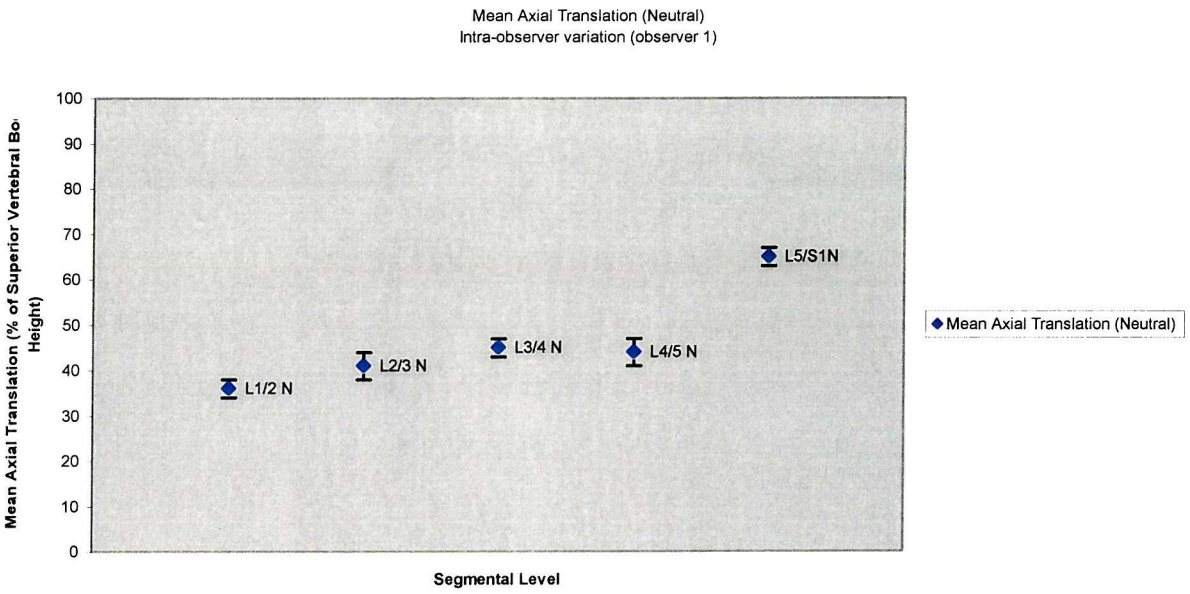


FIGURE 8.19

INTRA-OBSERVER VARIATION (OBSERVER 1) FOR AXIAL TRANSLATION/IVD HEIGHT BASED ON 5 REPEATED MEASUREMENTS OF ONE LATERAL FULL FLEXION IMAGE (ERROR BARS = +/-1SD)

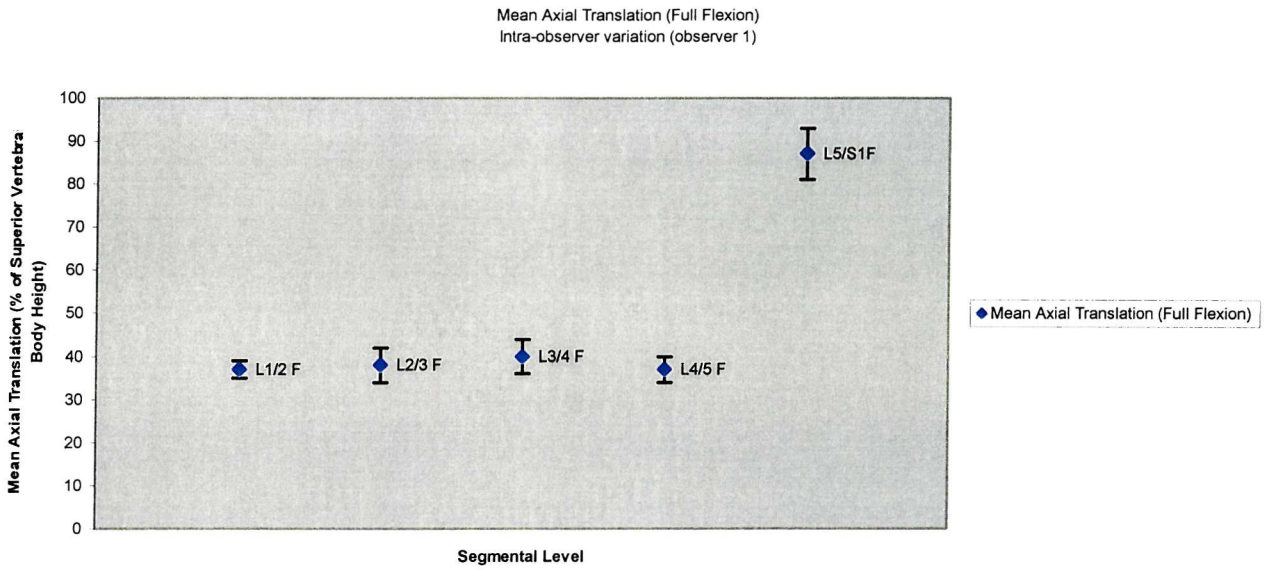


FIGURE 8.20

INTRA-OBSERVER VARIATION (OBSERVER 1) FOR AXIAL TRANSLATION/IVD HEIGHT BASED ON 5 REPEATED MEASUREMENTS OF ONE LATERAL FULL EXTENSION IMAGE (ERROR BARS = +/-1SD)

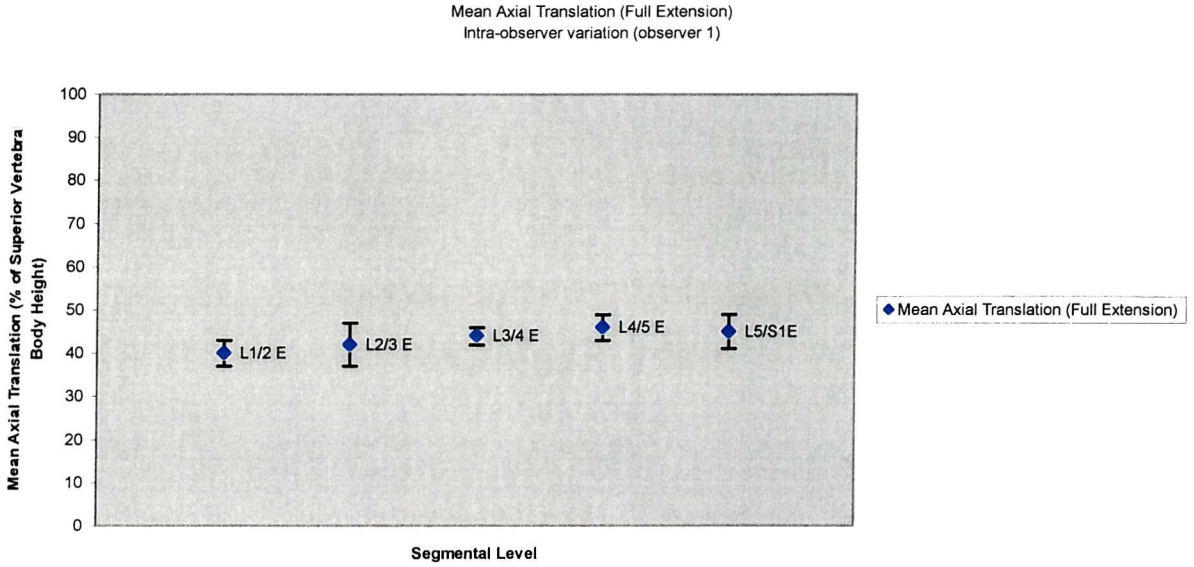


FIGURE 8.21

INTER-OBSERVER VARIATION (OBSERVERS 1,2,3) FOR AXIAL TRANSLATION/IVD HEIGHT BASED ON A SINGLE MEASUREMENT BY THREE OBSERVERS OF ONE LATERAL NEUTRAL IMAGE (ERROR BARS = +/-1SD)

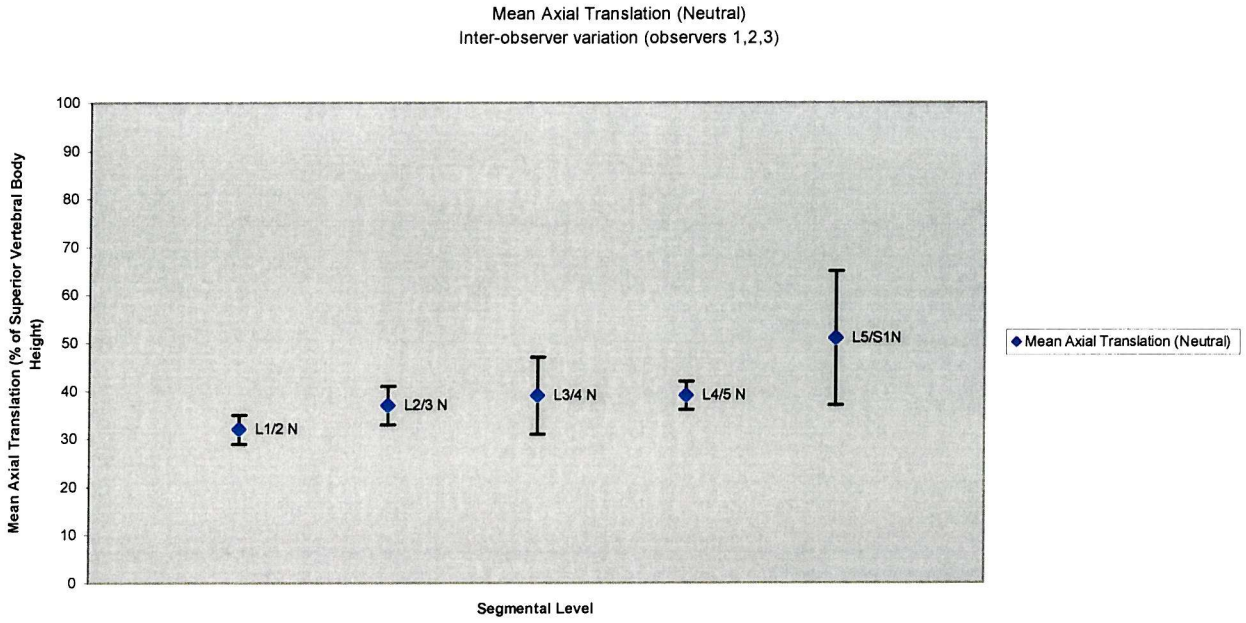


FIGURE 8.22

INTER-OBSERVER VARIATION (OBSERVERS 1,2,3) FOR AXIAL TRANSLATION/IVD HEIGHT BASED ON A SINGLE MEASUREMENT BY THREE OBSERVERS OF ONE LATERAL FULL FLEXION IMAGE (ERROR BARS = +/-1SD)

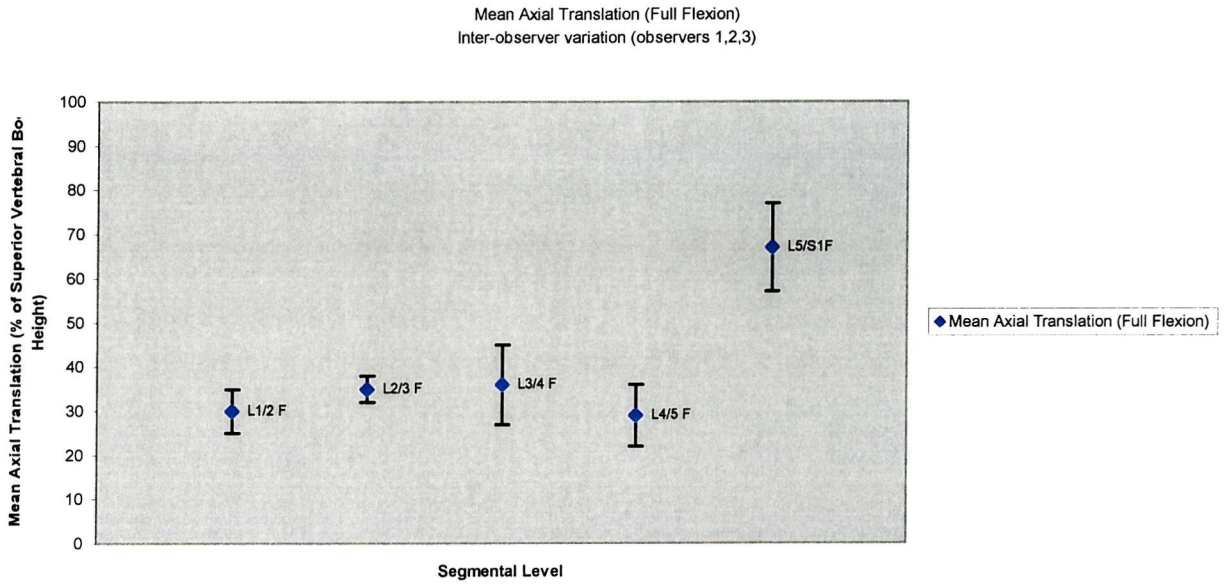
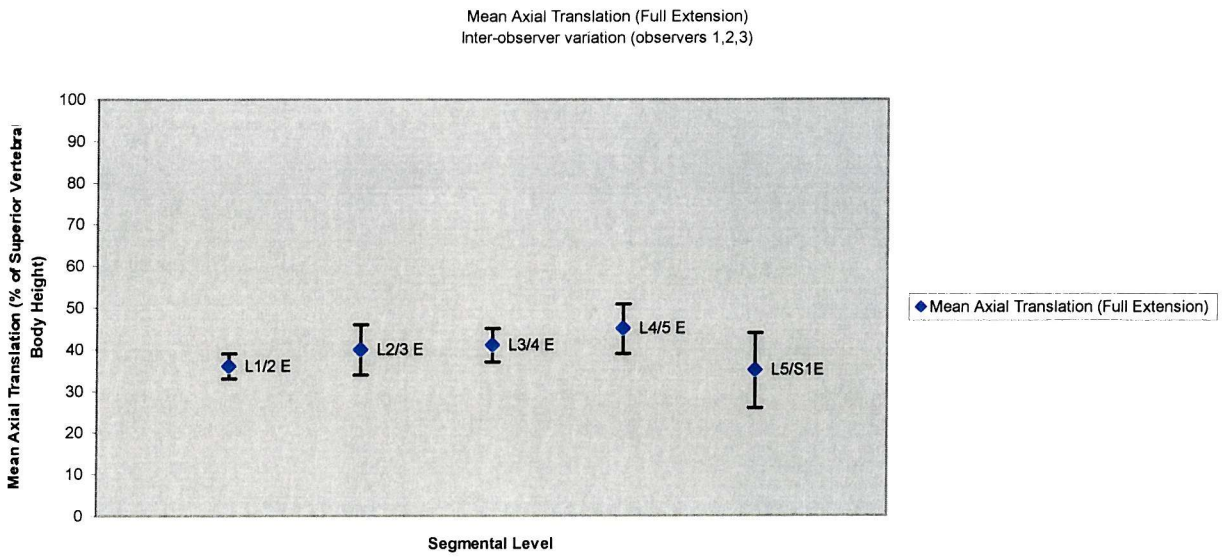


FIGURE 8.23

INTER-OBSERVER VARIATION (OBSERVERS 1,2,3) FOR AXIAL TRANSLATION/IVD HEIGHT BASED ON A SINGLE MEASUREMENT BY THREE OBSERVERS OF ONE LATERAL FULL EXTENSION IMAGE (ERROR BARS = +/-1SD)



Nikolai Bogduk, in his textbook on the clinical anatomy of the lumbar spine (Bogduk, 1997), quotes figures on disc and vertebral body dimensions derived from the, as yet, unpublished data of fellow researcher Lance Twomey. This work involved measurements from over 200 cadavers across a wide age range. Using the appropriate measurements for the age range involved in the present study, it was possible to calculate the approximate disc height for each level as a percentage of the upper segment (Table 8.8). Given these data it seems unlikely that the measured disc heights of the present study, with respect to the L5/S1 level, are truly representative. Values of 65 and 87% of upper vertebral height are not plausible at a region where lumbar disc height is normally less than most of its neighbouring segments. These figures are, therefore, most likely a result of measurement error given the imaging difficulties associated with its anatomy. In consequence, disc height/axial translation data relating to the L5/S1 segment was excluded from the present study.

As a separate exercise, the distances between the centre points (CPs) of each FSU were calculated from the data derived for AP translation and compared with the mean IVD height (Figure 8.24). The change in distances between these points mirror very closely the changes in average disc height and comparison of the data demonstrates a high correlation (Table 8.9).

Since the likelihood of distortion and coupled movements associated with the lateral flexion are high, measurements of translation were not attempted for coronal plane motion.

TABLE 8.8

MEAN DISC AND VERTEBRAL BODY DIMENSIONS MEASURED FROM 20-35 YEAR OLD MALES CADAVERS

(BOGDUK 1997)

	L1	L2	L3	L4	L5
Body Height (mm)	*25.3*	*25.8*	*25.6*	*25.5*	*24.1*
Disc Height (mm)	*6*	*10.4*	*11*	*11.5*	*10.7*
Disc Height (% of superior body height)	*23.7*	*40.3*	*43*	*45.1*	*44.4*

FIGURE 8.24

COMPARISON OF ASSESSMENT OF AXIAL TRANSLATION BY MEASUREMENT OF THE CHANGE IN MEAN IVD HEIGHT AND CHANGE IN DISTANCE BETWEEN CENTRE POINTS (CP)

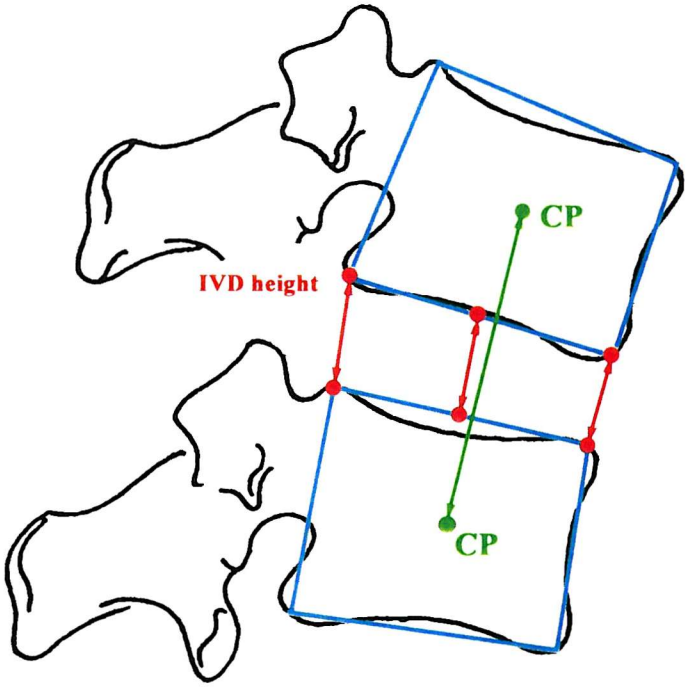


TABLE 8.9

CORRELATION BETWEEN MEASUREMENT OF CHANGE IN MEAN IVD HEIGHT AND CHANGE IN CP DISTANCES FOR LEVELS L1-L5 FOR ALL SUBJECTS (SAGITTAL PLANE ONLY)

Correlation Change in mean IVD height/change in CP distance	**L1/2**	**L2/3**	**L3/4**	**L4/5**
Pearson's r	***0.95***	***0.88***	***0.86***	***0.94***

8.11 RADIATION DOSAGE

The use of human subjects in this present study necessitated the approval of the local medical ethics committee at Salisbury District Hospital. Approval was granted for the screening of thirty male volunteers on the basis of a total of two minutes fluoroscopic screening for each subject with an associated expected dose of approximately 2.14 Gy cm^2. The total dosage for each of the ten subjects reported in the present study, are given in Table 8.10.

The dose-area product (DAP) is a way of estimating the amount of radiation received over the total body area exposed by the patient. This value represents the radiation exposure for the entire area and the overall patient exposure under all conditions (Schubert, 1995). Also depicted in Table 8.10 are the Effective Dose Equivalents for the subjects screened in this present study. This measure, expressed in milliSieverts, incorporates an estimate of cancer risk based on exposure. In effect, these values indicate the level of whole-body irradiation that would generate the equivalent radiation injury as the examination under consideration (McCollough & Schueler, 2000). Effective dose is considered to be the most relevant quantity for expressing and comparing "the dose given to a patient" (Wall & Hart, 1997). A recent European directive, based on recommendations from the International Commission on Radiological Protection (ICRP), has established maximum permissible limits for all practices involving risk from ionising radiation (Dendy & Heaton, 1999). This directive stipulates, for "members of the public", a maximum effective dose of 1 mSv/year or no greater than 1 mSv over any 5 year period. The new dose limits for exposed workers and students over 18 years of age are 50 mSv in any one-year or a total of 100 mSv over a consecutive 5 year period. In this way the previous ICRP recommendations of 20 mSv in consecutive years, for industrial workers, is preserved (ICRP, 1991a). It must be understood, however, that all medical exposures are, and always have been, excluded from these dose limitations.

For comparison, Table 8.11 shows the dose-area products for a number of common hospital procedures. These tables demonstrate that the digitised fluoroscopy work carried out in this present study compares favourably with many of these common radiological investigations. In particular the median dose for a plain film examination of the lumbar spine, probably the most common initial investigatory procedure for low back pain, is over four times that of a one-minute fluoroscopic screening under the current protocol. In the present study, the average DAP across all ten subjects for screening one (PA and lateral) was 2.6 Gy cm^2. Nevertheless, it is true to say that dose levels for common medical

procedures have reduced over recent years (Shrimpton et al., 1999; Warren-Forward et al., 1998). A recent investigation of radiation dose associated with fluoroscopic and plain film examinations (Warren-Forward et al., 1998), has suggested a median dose for lumbar spine plain film of 7.65 Gy cm^2, considerably lower than the NRPB reference dose of 15 Gy cm^2 .

TABLE 8.10

RADIATION DOSAGE AND SCREENING TIMES FOR ALL TEN SUBJECTS

Subjects	Total Time (min)	Total Dose-area product (Gy cm^2)	Effective dose equivalent (mSV)
BM	1.7	6.82	0.99
CR	1.9	3.43	0.46
DE	1.6	5.87	0.68
DO	1.7	4.88	0.61
GD	1.7	3.85	0.44
GP	1.8	5.34	0.65
JM	1.8	4.05	0.50
JW	1.9	5.98	0.70
NW	1.9	4.77	0.57
RM	2.3	6.01	0.62
Mean values for all ten subjects	1.83	5.1	0.62

TABLE 8.11

DISTRIBUTION OF INDIVIDUAL DOSE-AREA PRODUCTS FOR ADULT PATIENTS AT A RANDOM SAMPLE OF 20 ENGLISH HOSPITALS WITH NRPB REFERENCE VALUES OF DOSE-AREA PRODUCT (BASED ON SHRIMPTON ET AL 1986 AND SHRIMPTON ET AL 1999)

Examination	Dose-area product (Gy cm^2) Minimum	Dose-area product (Gy cm^2) Median	Dose-area product (Gy cm^2) Maximum	*NRPB Reference dose (Gy cm^2) (rounded value of 3rd quartile)*
Lumbar spine (3.4 films)	2.0	12	93	***15***
Barium enema (8.5 films, 3.73 mins)	6.2	41	272	***60***
Barium meal (7.8 films, 3.22 mins)	0.49	17	163	***25***
Intravenous urography (8.2 films)	3.3	29	251	***40***
Abdomen (1.4 films)	0.70	4.9	30	***8***
Pelvis (1.1 films)	0.49	3.8	19	***5***

CHAPTER 9

THE PASSIVE INTERVERTEBRAL MOTION OF THE LUMBAR SPINE IN ASYMPTOMATIC SUBJECTS: A PRELIMINARY STUDY (RESULTS)

9.1 INTRODUCTION

All of the ten subject files were included in the study, although complete data for L5/S1 was missing from two of the files. In one of the subjects it was the consequence of a transitional segment rendering the L5/S1 joint immobile. In the other volunteer, it was due to poor image quality as a result of the combination of inadequate radiographic penetration at this level and the inferior migration of the S1 segment out of the viewing area, in lateral views. In two subjects inferior drift of the lumbosacral region caused a transient loss of data, 3 and 5 increments respectively. Similarly, L1 was noted to drift superiorly from the viewing area in 6 subjects. In all cases this superior migration was around full extension with the segment lost from view between 2 and 10 increments.

For analysis of sagittal plane data, changes in angles, anteroposterior and axial translation were considered. For the coronal plane studies only angular changes were considered.

9.2 IVA RANGES (FLEXION/EXTENSION)

As previously described in Chapter 8, all subjects began the motion sequence in neutral, were then taken into full flexion where the motion momentarily stopped as the movement direction was reversed. From full flexion, the subjects were then taken into full extension in one continuous sweep where the movement, again, ceased briefly before reversing the direction, bringing the subjects back, finally, to neutral again.

For sagittal plane rotations at each level the IVA results are depicted in Table 9.1.

The greatest mean range across the entire motion sequence was found at the L5/S1 level (14.22^0). The smallest mean range was found at the L3/4 level (10.95^0). Separate ranges for flexion were ascertained by examination of the motion between the initial (neutral) position and full flexion. Likewise, the extension range was determined between full

extension and the end of the motion sequence (neutral). The greatest mean range for flexion was found at the L5/S1 level (8.34^0) and the least at L2/3 (4.65^0). In extension most sagittal rotation occurred at the L1/L2 joint (7.60^0) and least at L4/L5 (6.33^0). Summation of these ranges indicates that most motion throughout the whole lumbar spine (i.e. L1-S1) occurred in extension (35.16^0) compared to flexion (29.47^0).

TABLE 9.1

SUMMARISED IVA DATA FOR FLEXION/EXTENSION. MEAN RANGES FOR ALL TEN SUBJECTS ACROSS BOTH SCREENINGS.

IVA Data (Degrees)	L1/2	L2/3	L3/4	L4/5	L5/S1
Mean Range	12.38	11.03	10.95	11.68	14.22
(+/- 1SD)	(+/- 3.31)	(+/- 2.71)	(+/- 2.91)	(+/- 3.46)	(+/- 3.77)
Mean Flexion Range	5.13	4.65	4.75	6.60	8.34
(+/- 1SD)	(+/- 1.88)	(+/- 1.61)	(+/- 1.97)	(+/- 2.12)	(+/- 3.21)
Mean Extension Range	7.60	6.70	7.48	6.33	7.06
(+/- 1SD)	(+/- 7)	(+/- 4.5)	(+/- 8)	(+/- 9)	(+/- 11)

9.3 IVA RANGES (LATERAL FLEXION)

For lateral flexion, the motion sequence protocol was similar to that described for flexion/extension. All subjects were taken from neutral into full right lateral flexion, where the movement momentarily ceased, before being rotated in a continuous arc through neutral into full left lateral flexion. Bringing the subjects back to the neutral position completed the sequence.

For coronal plane rotations at each level the results are depicted in Table 9.2.

The greatest mean range across the entire motion sequence was found at the L4/L5 level (11.90^0). The smallest mean range was found at the L5/S1 level (8.94^0). Separate ranges for right lateral flexion were ascertained by examination of the motion between the initial (neutral) position and extreme right lateral flexion (RLF). Likewise, the left lateral flexion (LLF) range was determined between extreme LLF and the end of the motion sequence (neutral). The greatest mean range for RLF was found at the L4/5 level (6.88^0) and the least at L1/2 (4.40^0). In LLF most side bending occurred at L2/3 (7.03^0) and least at L5/S1 (4.38^0). Again, the sum of these ranges suggests that most side bending over the whole lumbar spine occurred in LLF (28.83^0) compared to RLF (25.91^0), although the difference was not as marked as in flexion/extension.

TABLE 9.2

SUMMARISED IVA DATA FOR LATERAL FLEXION. MEAN RANGES FOR ALL TEN SUBJECTS ACROSS BOTH SCREENINGS.

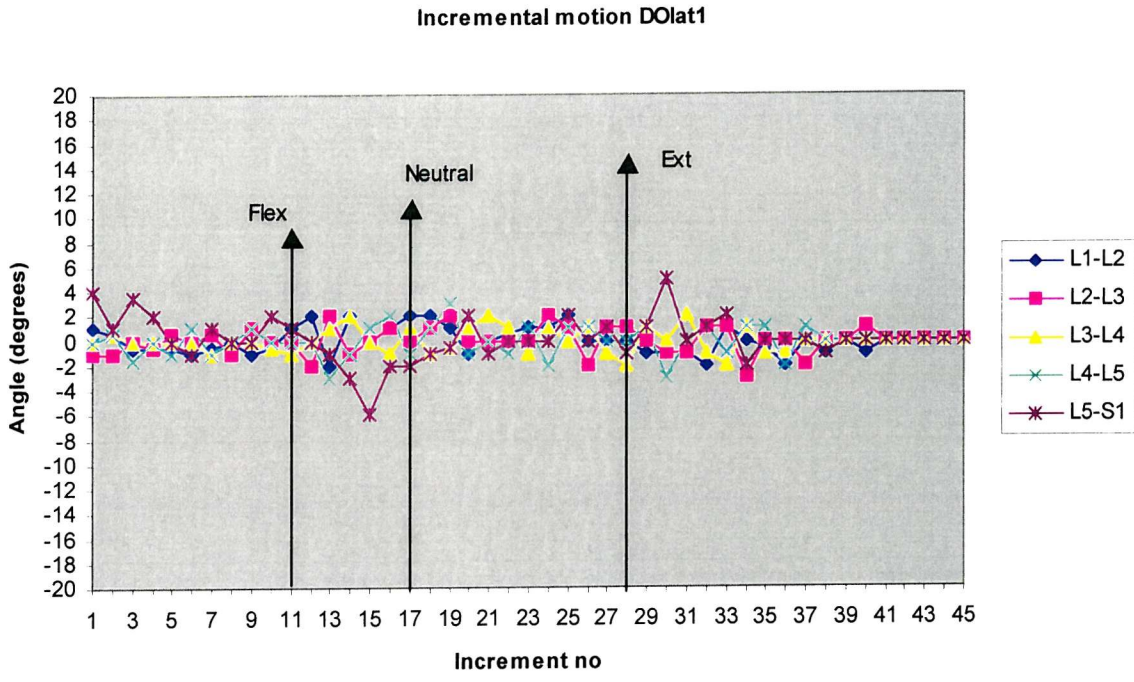
IVA Data (Degrees)	L1/2	L2/3	L3/4	L4/5	L5/S1
Mean Range	9.13	11.65	11.10	11.90	8.94
(+/- 1SD)	(+/- 1.73)	(+/- 2.85)	(+/- 1.44)	(+/- 3.23)	(+/- 2.72)
Mean RLF Range	4.40	5.08	4.68	6.88	4.88
(+/- 1SD)	(+/- 1.07)	(+/- 1.54)	(+/- 1.36)	(+/- 2.10)	(+/- 1.51)
Mean LLF Range	5.38	7.03	6.38	5.68	4.38
(+/- 1SD)	(+/- 1.66)	(+/- 1.68)	(+/- 2.17)	(+/- 2.33)	(+/- 1.96)

9.4 ICA RANGES (FLEXION/EXTENSION)

In order to study segmental behaviour across the motion sequence it is helpful to look not only at the ranges of intervertebral angles but also at the change in angle between increments. To see these changes clearly one has to determine the incremental angle or ICA. This is achieved by subtraction of the IVA at a given level and increment by its preceding angle. If intervertebral movement were smooth and even across the sequence, one would expect little change in the ICA. If, however, segments displayed erratic behaviour, then, clearly, the ICA should reflect these changes. With these values it becomes a simple task to see the direction and magnitude of change in angle across the range (Figures 9.1& 9.2). Generally, for flexion/extension, the mean ICA, irrespective of direction, was less than 2^0. As illustrated in Figure 9.1, however, L5/S1 did display incremental rotations suggestive of erratic motion. Unfortunately, due to the measurement variation associated with this region, it was impossible to determine if these changes reflected true erratic behaviour or simply artefact.

FIGURE 9.1

EXAMPLE OF A TYPICAL FLEXION/EXTENSION ICA GRAPH

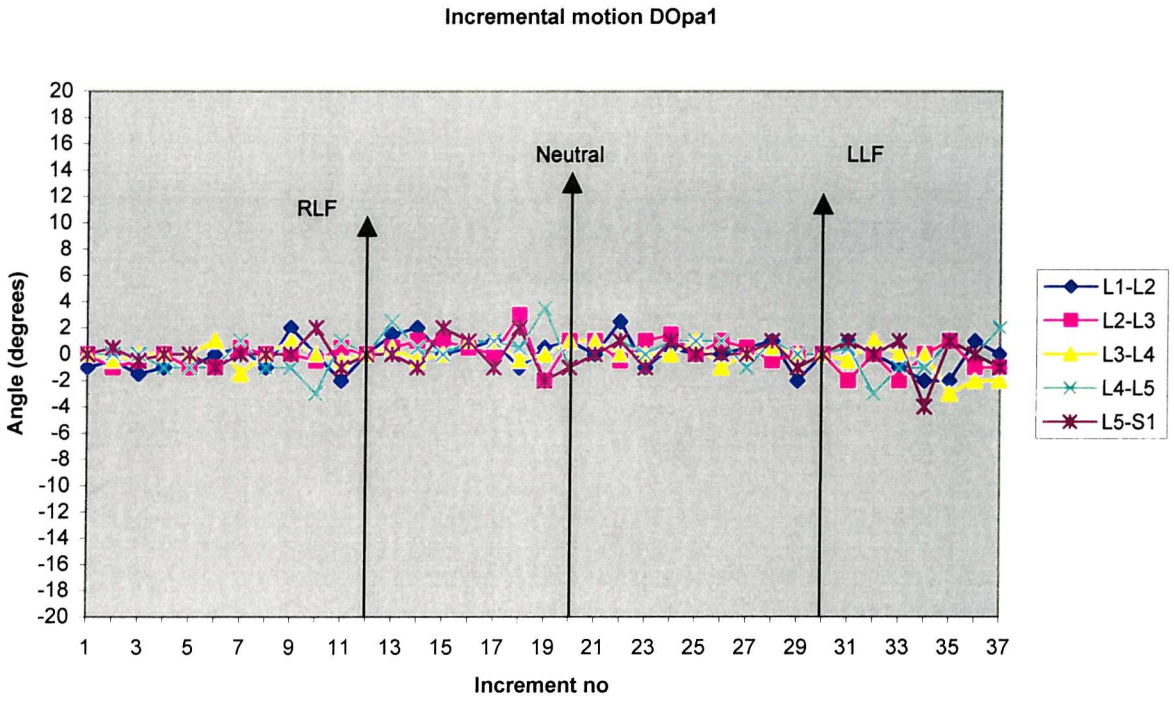


9.5 ICA RANGES (LATERAL FLEXION)

As discussed previously for flexion/extension, absolute ICA values were determined for lateral bending motion. Overall ICA values for coronal plane rotations were notably less, as would be expected, than those for the sagittal plane. In general, ICAs for lateral bending were around 1^0.

FIGURE 9.2

EXAMPLE OF A TYPICAL LATERAL FLEXION ICA GRAPH



9.6 SAGITTAL TRANSLATION

Anteroposterior (AP) Translation was measured by the method attributed to Frobin et al (Frobin et al., 1996). In all subjects over both screenings and at all levels, with the exception of L1 in subject CR, the cranial or superior segment was found, initially, posterior to its caudal or inferior partner. The average initial displacement at the starting neutral position is given in Table 9.3 as a percentage of the average depth of the superior vertebral body. As shown in Table 9.3, this initial retrolisthetic position seems to increase from L1 to L5. During the motion sequence the tendency is for the cranial or superior vertebrae to translate anteriorly somewhat in flexion and posteriorly in extension. In almost all instances the anterior translation was insufficient to bring the superior segment anterior to its subjacent neighbour. In other words, the majority of vertebral segments remained in a slightly retrolisthetic position throughout the motion sequence and in anterior translation merely approached the neutral or zero displacement position. In a very small number of instances the superior segment was found, momentarily, in anterolisthesis. The mean range of translation displacement, combined anterior and posterior, for each level are, again, expressed as a percentage of the average depth of the superior segment and are shown in Table 9.3

Axial translation data will be presented in the discussion section.

TABLE 9.3

SUMMARISED TRANSLATION DATA FOR FLEXION/EXTENSION. MEAN VALUES FOR ALL TEN SUBJECTS ACROSS BOTH SCREENINGS

Sagittal Translation (% of superior body depth)	**L1/2**	**L2/3**	**L3/4**	**L4/5**	**L5/S1**
Mean Initial Displacement	-5.83	-8.42	-9.87	-21.07	-21.06
(+/- 1SD)	(+/- 3.47)	(+/- 3.90)	(+/- 3.28)	(+/- 6.02)	(+/- 10.12)
Mean Flexion/Extension Range (combined)	16.60	16.78	16.34	17.22	27.05
(+/- 1SD)	(+/- 4.81)	(+/- 4.75)	(+/- 3.83)	(+/- 3.92)	(+/- 6.45)
Mean Flexion Range	9.49	7.99	8.53	11.39	17.81
(+/- 1SD)	(+/- 4.15)	(+/- 3.06)	(+/- 2.66)	(+/- 3.87)	(+/- 5.78)
Mean Extension Range	9.72	11.97	10.14	11.10	17.21
(+/- 1SD)	(+/-2.43)	(+/-5.96)	(+/-2.94)	(+/-4.29)	(+/-6.93)

9.7 DISCUSSION

9.7.1 DEFINING THE NEUTRAL POSITION

As discussed in the previous chapter, the determination of the initial neutral position was achieved by positioning the subjects in a comfortable mid-position, the resulting intervertebral configuration was then defined as neutral for the lumbar spine. All vertebral angles, at this attitude, were normalised to zero and movement expressed as a displacement relative to this starting point.

During the analysis, however, it became obvious that this neutral position varied considerably between subjects and between screenings (Figure 9.3). It also raised some interesting questions; how do you determine the neutral position again, once the sequence has begun? Is neutral mid-way between flexion/extension, right and left side-bending? Is neutral, under this protocol, defined when all intervertebral angles approach zero? In some of the subjects it seemed that the initial position, neutral, was closer to flexion, for example, than extension or closer to right side-bending than to left. In others, neutral appeared to occur between increments or at an increment not used for analysis. The question of determining, precisely, the true neutral position has obvious implications for those concerned with the application of the neutral zone (NZ) concept (Panjabi, 1992b). Indeed, a spinal biomechanics group from Arizona have recently revisited the *in vitro* NZ work of Panjabi, demonstrating some interesting observations (Crawford et al., 1998). These researchers have suggested that the NZ, as measured by Panjabi (Panjabi, 1992b), does not represent a zone of true ligamentous laxity but is influenced more by the orientation and friction forces of spinal joints. In effect the optimum neutral position, representing a position of minimal ligamentous stress, differs from that of the actual resting position of the spine, which is dependent on structural joint factors. This issue aptly illustrates the dichotomy between the biomechanics of materials versus the biomechanics of structures.

For the present study, the mid-sequence neutral was visually estimated by careful analysis of each frame between the extreme positions. These frames were then compared to the initial image to establish similarities in vertebral arrangement. This was necessary since it was obvious that subjects often shifted in position slightly on the table. Under these circumstances the attitude of the whole lumbar spine and pelvis might have altered whilst largely maintaining the intervertebral configuration. The fact that the table allowed

volunteers to shift, even slightly, must have reduced the concentration of movement on the lumbar spine and, also, precluded any meaningful investigation of hysteresis. Given the possible permutations of intervertebral arrangements it may be that the exact initial configuration between all lumbar segments is never precisely replicated once movement has begun. Indeed, it was often very difficult to match motion sequence images with the static initial image and so a "best fit" approach was adopted (Figure 9.4). In addition, since only every fourth image was analysed, an estimated neutral image had to be approximated to the nearest image analysed. In practice this did not present great difficulties since most estimated images did, indeed, correspond to analysed images. Obviously, therefore, a greater number of images acquired and analysed per second of motion would be likely to increase the probability of identifying the mid-sequence neutral position.

Since, in the present study, all intervertebral angles began at zero, it seemed logical that, should the spine return to this initial position, the sum of the IVAs would approach zero. In this way it was hoped that a numerical method could be found to confirm the neutral position. However, probably as a result of minor changes in segmental rotation in both direction and magnitude, this method did not produce meaningful results. Nevertheless, the square root of the sum of the squares of these angles did provide an apparently reliable means of predicting neutral. As these values approached zero a strong agreement was noted between the minimum value and the previously estimated neutral image. For all subjects, in both flexion/extension and lateral bending studies including both screenings, 78% of these minima were found within +/- 1 increment of the estimated neutral and 95% within +/- 2 increments. Again, it would be expected that a greater number of increments acquired and analysed would improve the resolution of this method further.

FIGURE 9.3

EXAMPLES OF VARIATION IN THE LOCATION OF THE MID-SEQUENCE NEUTRAL POSITION

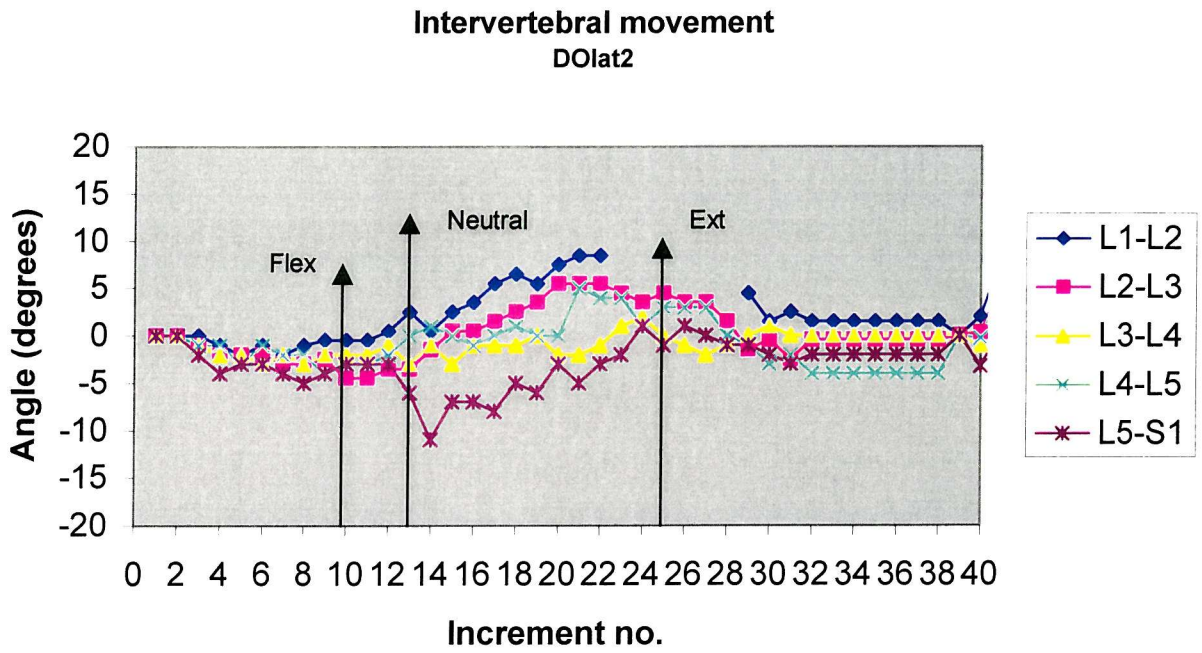


FIGURE 9.4

EXAMPLES OF THE INITIAL "TRUE" NEUTRAL AND ESTIMATED MID-SEQUENCE NEUTRAL.

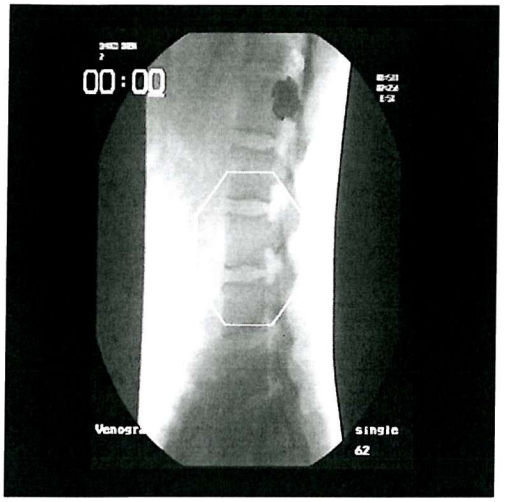


Image one, "true" neutral

neutral

(subject DOlat2)

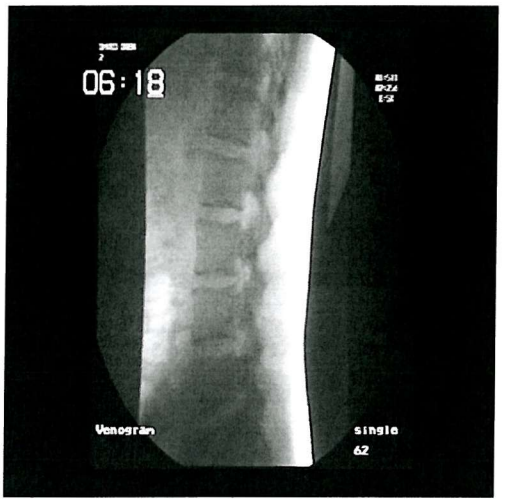


Image 57, estimated mid-sequence

(subject DOlat2)

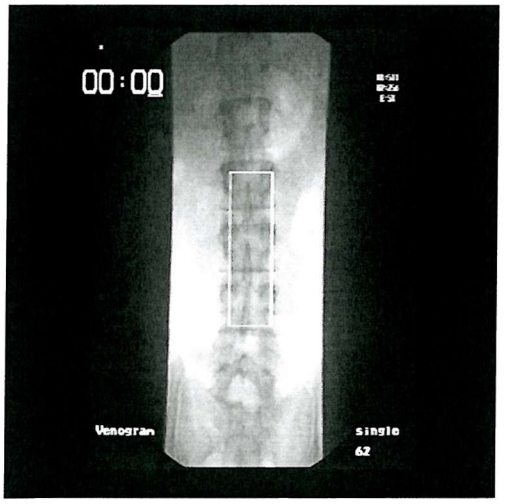


Image one, "true" neutral

neutral

(subject JMpa1)

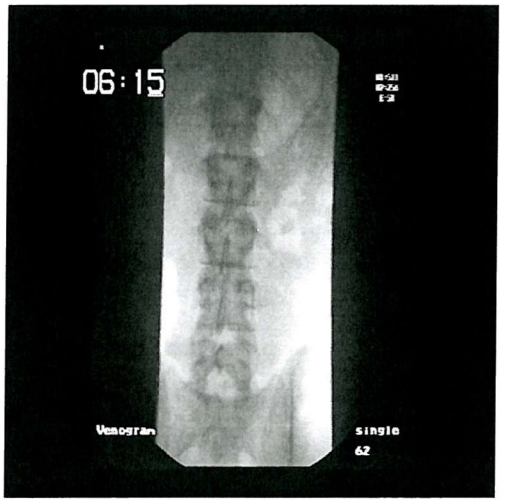


Image 61, estimated mid-sequence

(subject JMpa1)

9.7.2 IVA RANGES

The ranges of rotational motion for these subjects are broadly in agreement with those found in the established literature for *in vivo* human subjects (Tables 9.4 to 9.8). Interestingly, our representative values for combined flexion and extension, are closer to those achieved by active methods (Pearcy, 1985) than the published values obtained by a passive protocol (Dvorak 1989 as cited by (White & Panjabi, 1990)). This is all the more intriguing since our study employed a protocol where the volunteers were recumbent throughout the motion sequence. In 1994, a radiographic study on spondylolisthesis, demonstrated that of the 31 patients displaying signs of abnormal motion, 18 (58%) did so only under recumbent conditions and not when standing (Wood et al., 1994). Ogon and colleagues (Ogon et al., 1997a), using an *in vitro* methodology, detail results that suggest that an absence of axial preload should generate ranges of motion significantly higher than those with preload (standing weight bearing). Although, one year earlier, the group lead by Frobin (Frobin et al., 1996) had shown no difference in ROM between side-lying and standing protocols. Frobin and colleagues (Frobin et al., 1996) based their measurements on a set of radiographs from 61 asymptomatic individuals. It may simply be, perhaps, that non-weight bearing studies are likely to reveal differences only when anatomical integrity is lost. Thirty-three of these asymptomatic volunteers performed the flexion/extension movements passively, whilst 28 were subjected to an active protocol. Since these authors do not specify which protocol generated which results, we must assume that the values published are based on a combination of these methodologies.

Although the average values of rotation for combined flexion/extension obtained in the present study generally agree with the majority of other *in vivo* studies, disparities become apparent when examining studies that separate the motion phases. A marked difference, for instance, was noted between the values for separate flexion and extension established in the present study and those reported by Pearcy (Pearcy, 1985). In Pearcy's study the values for flexion are considerably higher, and those for extension correspondingly lower, than the values reported here. These differences are, perhaps, explained by the distinction in methodologies. Pearcy's subjects were radiographed upright and weight bearing with a frame opposing forward movement of the pelvis and a strap limiting backward movement. By comparison, our subjects were non-weight bearing and relatively unrestrained. A greater degree of flexion may therefore be expected in a spine subjected to the forward moment of the trunk with a rigidly fixated pelvis. This is particularly likely if the subject

is allowed to remain in a fully flexed position inducing the creep phenomenon. The lumbar spine is significantly less stiff in flexion than extension and creep may have had an influence on these values, although Pearcy does not discuss the amount of time required to take the radiographs (Oliver & Twomey, 1995). During our study the subjects were in full flexion only momentarily as the passive motion table motor was immediately switched to extension. Additionally, our protocol moved the lower body, including the pelvis, with respect to the trunk. When the lower section of the table had traversed its full flexion range (40^0), any further flexion of the lumbar spine would have had to be achieved by subjects actively flexing or "curling" the pelvis. Under our passive motion protocol, movements of this kind were eliminated and therefore it is likely that we did not subject our volunteers to their upper limit of full flexion. In contrast, however, it seems probable that we were able to induce a greater degree of extension in our subjects.

Apart from the obvious influence of the normal lumbar lordosis, other factors may also play a part in producing these differences. As the lumbar spine is limited in extension largely by bony impaction (Adams et al., 1988), it is conceivable that extension is sequentially more rapid in onset. That is, as one segment reaches a definite bony cessation of movement, so the next segment is forced more rapidly towards its limit. In this way it is likely that we were able to passively generate more extension than flexion in the same time interval. Pearcy, on the other hand, used an active protocol that depended on the subjective appreciation of a volunteer reaching their limit of extension. As Adams and colleagues have described (Adams et al., 1988), erect standing induces around 15^0 of extension and further extension from this posture, in healthy adults, is limited to between $6\text{-}15^0$. Depending on the degree of lordosis adopted in the starting position, the subject may already have, subjectively at least, been relatively close to their end-range. With our protocol, subjects were non-weight bearing and began the sequence in a position of relative flexion with the hips and knees slightly flexed. Volunteers were asked if they could *tolerate* the full extension range of the table (40^0) and not terminate the movement based on their own perception. Not surprisingly, all subjects in our study reported feeling uncomfortable at the end-point of extension but not so in flexion. Ranges for extension also showed more variability, as evidenced by the greater SDs (Table 9.1). This, again, is most probably a result of the larger movement produced in extension. As subjects were taken closer to their limit in extension than in flexion, individual differences in passive range would be more likely to become apparent and thus result in greater variability.

Lateral bending values in the present study generally approximated those reported by Pearcy (Pearcy, 1985) and Pearcy and Tibrewal (Pearcy & Tibrewal, 1984) (Tables 9.7 & 9.8). In particular, our results show a similar pattern of motion, with the exception of L5/S1, between LLF and RLF as noted by Pearcy (Pearcy, 1985). Pearcy revealed a slight increase in motion of segments L1/2 and L2/3 between RLF and LLF and a slight reduction in the mean values of L4/5. These findings were mirrored in our study and to a slightly greater extent. This would be consistent with the passive nature of the methodology. Pearcy's study (Pearcy, 1985), however, was unable to establish any lateral bending at L5/S1 on bending to the right and only 2^0 during bending to the left. Our finding of 4^0 and 5^0 respectively exceeds Pearcy's and is greater, even, than the proposed representative value of 3^0 published by White and Panjabi (White & Panjabi, 1990), these values are for one-side only and are based on "careful review of the literature". A passive method, particularly one such as ours that initiates the movement distally, is likely to provoke more of a lateral bending motion at the L5/S1 segment than one relying on voluntary movement. Nevertheless, the greater error associated with measurements at this level would suggest caution when interpreting data derived from these segments. Indeed Pearcy and Tibrewal (Pearcy & Tibrewal, 1984) have shown that extension movements, in addition to the established coupling with axial rotation, accompany lateral bending. This, they suggest, is likely to provide an additional source of error associated with two-dimensional studies.

TABLE 9.4

COMPARISON OF REPRESENTATIVE *IN VIVO* ANGLES FOR COMBINED INTERVERTEBRAL FLEXION/EXTENSION (DEGREES).

Level	Present study *n=10*	Albrook (1957) *n=20*	Pearcy (1985) *n=11*	Dvorak (1989) *n=41*	Yamamoto et al (1989) *n=10*	Hayes et al (1989) *n=59*	White &Panjabi (1990) *Composite values*	Lin et al (1994) *n=100*	Frobin et al (1996) *n=43**
L1/2	12	6	13	12	10	7	12	7	12
L2/3	11	8	14	14	11	9	14	9	14
L3/4	11	13	13	15	11	10	15	13	14
L4/5	12	19	16	18	14	13	16	14	16
L5/S1	14	18	14	17	18	14	17	12	13

** The number of subjects for this study is an average since the number of subjects varied for each level as a result of radiographic factors.*

TABLE 9.5

COMPARISON OF REPRESENTATIVE *IN VIVO* ANGLES FOR INTERVERTEBRAL FLEXION (DEGREES).

Level	Present study *n=10*	Pearcy (1985) *n=11*	Lin et al (1994) *n=100*
L1/2	5	8	6
L2/3	5	10	8
L3/4	5	12	10
L4/5	7	13	13
L5/S1	8	9	8

TABLE 9.6

COMPARISON OF REPRESENTATIVE *IN VIVO* ANGLES FOR INTERVERTEBRAL EXTENSION (DEGREES).

Level	Present study *n=10*	Pearcy (1985) *n=11*	Lin et al (1994) *n=100*
L1/2	8	5	1
L2/3	7	3	1
L3/4	7	1	2
L4/5	6	2	2
L5/S1	7	5	3

TABLE 9.7

COMPARISON OF REPRESENTATIVE *IN VIVO* ANGLES FOR INTERVERTEBRAL RLF (DEGREES).

Level	Present study	Pearcy (1985)	White &Panjabi (1990) *one-side only*
L1/2	4	5	6
L2/3	5	5	6
L3/4	5	5	8
L4/5	7	3	6
L5/S1	5	0	3

TABLE 9.8

COMPARISON OF REPRESENTATIVE *IN VIVO* ANGLES FOR INTERVERTEBRAL LLF (DEGREES).

Level	Present study	Pearcy (1985)	White &Panjabi (1990) *one-side only*
L1/2	5	6	6
L2/3	7	6	6
L3/4	6	5	8
L4/5	6	2	6
L5/S1	4	2	3

Of significance, in the present study, is the location of the minima and maxima, hence the range, within the motion sequence. Examples of diagrammatic representations of the location of these maximum and minimum values are depicted in Figures 9.5 & 9.6. These figures show where the ranges occur, for each intervertebral level, as a percentage of neutral to full flexion (or RLF), full flexion to full extension (or RLF to LLF) and full extension (or LLF) to the end of the motion sequence. It has been reported that these do not always coincide with the extremes of movement (Kaigle et al., 1997), and the results of the present study would appear to confirm this. Indeed, for flexion/extension across both screenings, only 5% of the intervertebral ranges coincided with both extremes. In other words, had we only taken radiographs at full flexion and full extension, we would have underestimated the ranges in 95% of these subjects. Furthermore, the maximum and minimum IVA's may lie at some distance from the extremes of movement hence making an estimation of the true range even more difficult without dynamic imaging. In the present study only 17% of the intervertebral ranges fell on or within one increment of flexion or extension. It is clear from these data that the maximum values, in general, lie closer to full extension than the corresponding minima do to full flexion. This is probably a function of the passive motion table producing a more extreme movement in extension than in flexion.

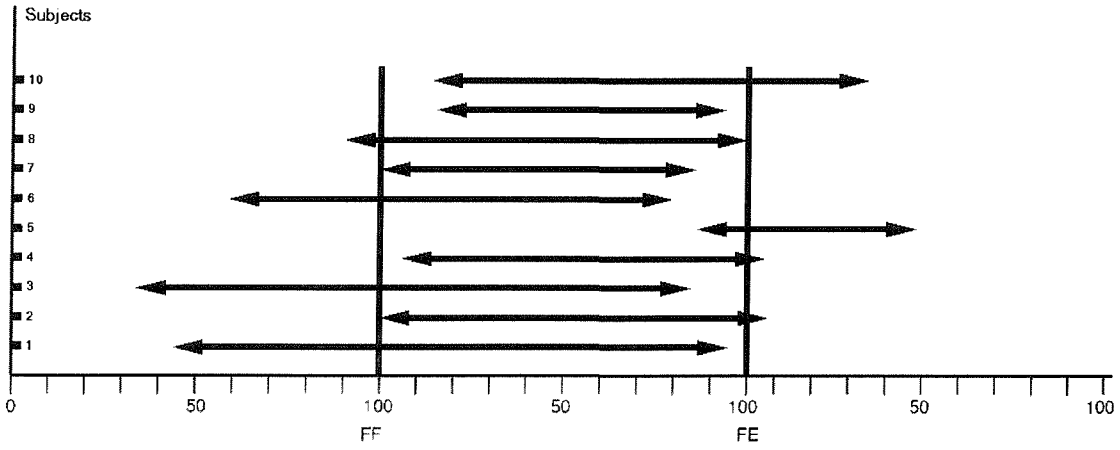
For side-bending the picture is slightly different with 17% of the ranges coinciding with both RLF and LLF. For all lateral bending, 26% of the intervertebral ranges fell on or within one increment of RLF or LLF. For lateral bending a similar finding as with flexion/extension was noted with respect to the extremes. Cursory examination of the data relating to the location of the ROM within the sequence show that, like flexion/extension, the maximum values are closer and more tightly grouped towards LLF than the minima are in relation to RLF. This might be explained, perhaps, by the relatively greater range of motion produced in both LLF and extension compared to RLF and flexion, encountered in this study. This is easier to rationalize in terms of subject positioning for flexion/extension than for lateral bending, where symmetrical placement and consistency are easier to achieve. In lateral bending the difference may be related to handedness and the tendency for greater movement towards the left noted by other authors of *in vivo*

studies (Pearcy, 1985; Pearcy & Tibrewal, 1984). It is interesting to note that ranges of lateral bending are often quoted for one side only and hence forestalls any discussion of differences (Steffen et al., 1997; White & Panjabi, 1990). Nikolai Bogduk, in the latest edition of his textbook on the lumbar spine (Bogduk, 1997), displays a table (page 95) showing mean angles of rotation for both right and left lateral bending. These figures suggest that right side bending has a slightly greater value than left side bending. However, on examination of the papers these figures are based upon, it would appear that the mean angles given by Bogduk are in error.

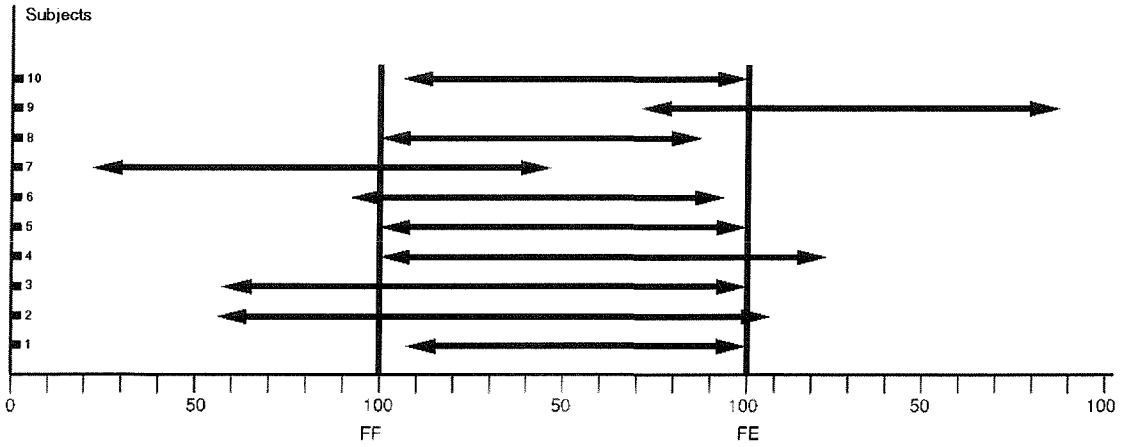
FIGURE 9.5

EXAMPLES OF THE LOCATION OF MAXIMUM AND MINIMUM VALUES FOR IVAS THROUGHOUT THE MOTION SEQUENCE

(SAGITTAL PLANE)



Location of flexion/extension ROM within the motion sequence (L2/3 screening 1)

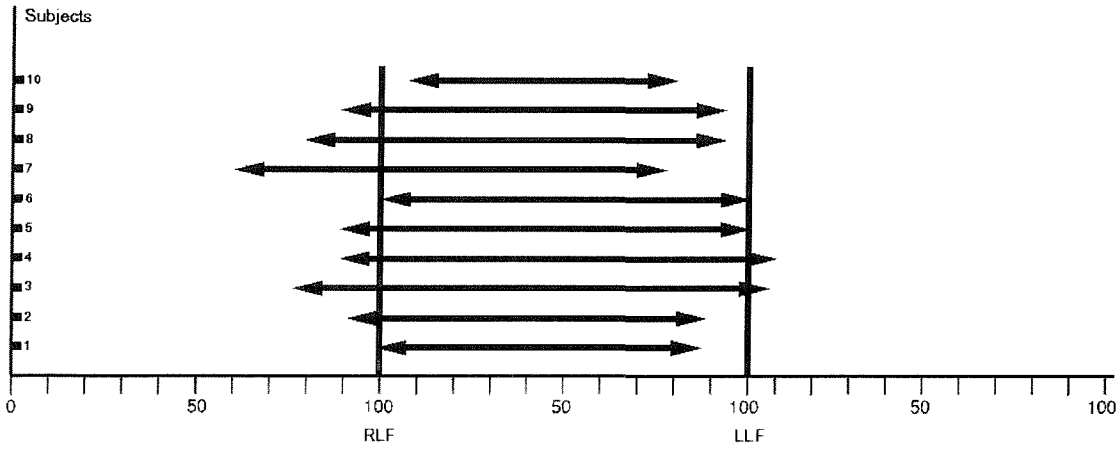


Location of flexion/extension ROM within the motion sequence (L3/4 screening 1)

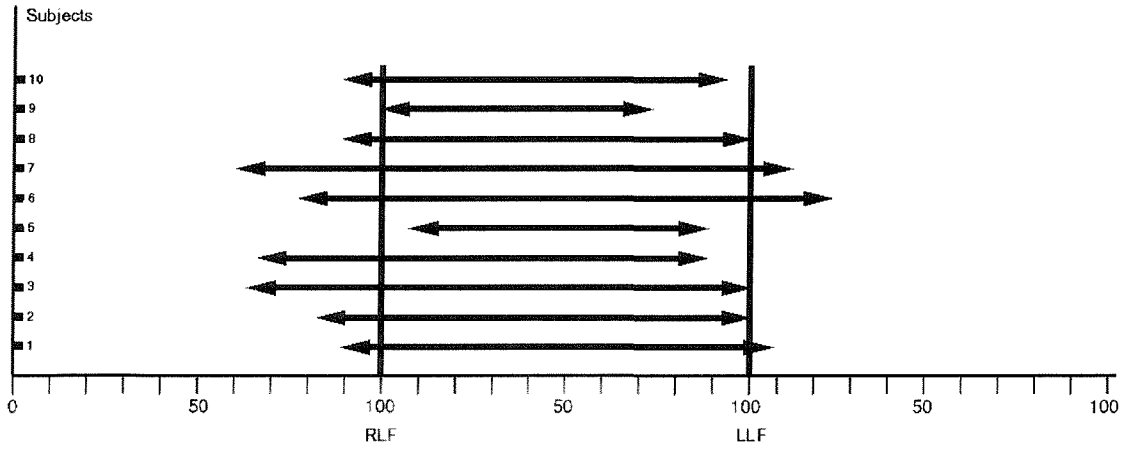
FIGURE 9.6

EXAMPLES OF THE LOCATION OF MAXIMUM AND MINIMUM VALUES FOR IVAS THROUGHOUT THE MOTION SEQUENCE

(CORONAL PLANE)



Location of lateral flexion ROM within the motion sequence (L2/3 screening 2)



Location of lateral flexion ROM within the motion sequence (L3/4 screening 2)

The original angles given by Pearcy and Tibrewal (Pearcy & Tibrewal, 1984) would seem to have been reversed by Bogduk, with right replacing left. In addition the mean angles themselves are partially misquoted. The mean angle for L3/4 bending to the left, as reported by Pearcy and Tibrewal, is 5 degrees not 6 and for L4/5 is 2 degrees in magnitude and not 5 as quoted by Bogduk. In any case the differences in mean angle between right and left lateral bending would appear to be slight and may or may not explain the differences noted in this present study.

9.7.3 ICA RANGES

These values are useful in identifying large intervertebral rotations occurring over short incremental periods. Following on from the work of the research group headed by Wilder (Wilder et al., 1988) and more recently Ogon and colleagues (Ogon et al., 1997b), it was thought that examination of the ICAs *in vivo* might help explain the "jerks" noted in the *in vitro* work of these authors. As Ogon and co-workers (Ogon et al., 1997b) had used a constant moment technique on human lumbar FSUs, analogous to our passive motion table, and had still discovered these intervertebral *hesitations* and *giving way* responses, it was conceivable that we might uncover their *in vivo* counterparts. The main outcome of the Ogon study was that, although these jerks did not increase in magnitude with increasing component instability, they did, however, shift in their location within the motion sequence. For that reason, in the present study, the locations of significant ICAs were noted with respect to neutral and extreme positions. ICAs of up to 3^0 were extremely common in the data derived from these ten volunteers, especially in the sagittal plane. In other words, a change in angle of around 3^0 over one increment is likely to be a normal and commonplace finding in asymptomatics. As one increment represents, on average, approximately one second of elapsed time, this value would not be unexpected. Given this, it was decided to concentrate on ICAs of greater than 3^0 irrespective of direction, i.e. absolute angles, for the purposes of this study.

As previously noted, the mean ICAs for flexion/extension are of the order of 2^0 and for lateral flexion 1^0. For all ten subjects and across all levels, over both screenings, a total of 210 ICAs greater than 3^0 were found, representing around 6.5% of all ICAs for flexion/extension.

The concept of the neutral zone predicts that relatively large ICAs, if analogous to the jerks described by Ogon and colleagues (Ogon et al., 1997b), are likely to occur around the neutral position. In the present study no consistent trend was noted in the location of these larger ICAs although limitations of the present study may have prevented the detection of such rapid changes. Given a greater number of increments and reduced measurement error, however, it is possible that these "jerks" might be demonstrable *in vivo*.

For lateral flexion, as previously mentioned, the absolute values for ICAs were considerably less than those for flexion/extension. Likewise, the occurrence of ICAs greater than 3^0 is of a lesser frequency than those for flexion/extension motion. ICAs of more than 3^0 in magnitude for combined right and left lateral flexion totalled 62 across both screenings, representing some 2% of all incremental angles. Again, no consistent trend in the location of these larger ICAs could be determined from the data.

9.7.4 PARADOXICAL ROTATIONS

Since so-called "paradoxical" motion has been considered as a possible sign of instability (White & Panjabi, 1990), it was actively sought in this present study. As commonly described, paradoxical motion is when, for example, a segment undergoes a typical flexion pattern of movement whilst the overall motion for a given region of the spine is in extension. In the present study, examination of the ICA data for flexion/extension for all volunteers over both screenings revealed a total of 64 paradoxical rotations of more than 3^0 in magnitude, representing some 30% of the 210 ICAs greater than 3^0. The majority of these rotations (30) occurred at the L5/S1 level, which is, perhaps, not surprising. Given the measurement variation associated with this region, however, the total and percentage values were also calculated with L5/S1 excluded from the data. Thus, ignoring the contribution of L5/S1, the total number of paradoxical rotations of 3^0 or more for flexion/extension was 34 (24%). The numbers of these paradoxical rotations for each segmental level are shown in Table 9.9.

In an *in vivo* study of eight asymptomatic subjects, Kanayama and co-workers (Kanayama et al., 1996) used cineradiography to examine lumbar spine motion in flexion/extension. They defined paradoxical motion as "reverse" rotation of more than 3^0 but could not find evidence of any in their study. As mentioned previously, however, these authors used an

active, standing protocol, which allowed the spine to be stiffened by compression pre-load. It is possible, therefore, that the passive nature of the present study, together with the absence of any stiffening effect from weight bearing compression forces, has allowed the segments the freedom to undergo this type of motion. In addition, our present study concentrated on changes in intervertebral angles and thus considered the relative motion between two vertebral bodies. Under these circumstances slight differences in velocity between segments could produce relative paradoxical rotations where the overall movement is in concert. Likewise, the present study did not reveal the phase lag motion pattern described by Kanayama and colleagues (Kanayama et al., 1996). Again this is most likely explained by the methodological differences.

For lateral flexion the total number of paradoxical rotations greater than 3^0 was determined as 14, accounting for around 23% of the 62 ICAs greater than 3^0 found for frontal plane rotations. As with flexion/extension, L5/S1 provided the majority of these rotations. Indeed for lateral bending L5/S1 accounts for almost all of the significant paradoxical rotations (12) which, considering the normal biomechanics of this region, is somewhat unexpected. Again, for the reasons discussed above, the L5/S1 data were excluded which resulted in a total of only 2 paradoxical rotations of greater than 3^0. This suggests that, in lateral bending, only 7% of all ICAs greater than 3^0 are paradoxical in nature (Table 9.9). In this respect the proportions of significant paradoxical and non-paradoxical rotations are dissimilar between movements in both planes. The much greater influence of coupling in frontal plane movements is, perhaps, a possible explanation of the differences found between planes.

From this present study of asymptomatic subjects it appears likely that paradoxical rotations, particularly in the sagittal plane, are not uncommon and therefore not, in themselves, *a priori* evidence of instability.

TABLE 9.9

TABLE SHOWING THE TOTAL NUMBERS OF PARADOXICAL ICAS GREATER THAN 3^0. DATA ARE FOR FLEXION/EXTENSION, RLF AND LLF COMBINED AND INCLUDES BOTH SCREENINGS.

Number of paradoxical ICAs $>3^0$	L1/2	L2/3	L3/4	L4/5	L5/S1	Total for all levels (% of all ICAs $>3^0$)	Total for levels L1-L5 (% of L1-L5 ICAs $>3^0$)
Flex/ext	11	4	4	15	30	64 (30%)	34 (24%)
Lateral Flex	1	0	0	1	12	14 (23%)	2 (7%)

9.7.5 SAGITTAL TRANSLATION

9.7.5.1 AP TRANSLATION

Anteroposterior (AP), or z-axis translation, in lumbar segments of asymptomatic individuals has been traditionally regarded as minimal in magnitude (Boden & Wiesel, 1990; Frobin et al., 1996; Lin et al., 1994; Pearcy, 1985; Rolander, 1966). The consensus figures suggest that AP translation should be around 1-2mm and no more than 3mm in normal subjects. These correspond to around 3-6% and no greater than 8.5% of vertebral body depth with a 35mm deep vertebral body or 3-7% and less than 10% given a 30mm deep vertebral body. For the purposes of comparison with other studies we will assume a vertebral body depth of 35mm (Frobin et al., 1996). As discussed in Chapter 2, however, some authors have suggested that these figures underestimate lumbar translation and propose that as much as 11-14% (4-5mm) may be within normal limits (Hayes et al., 1989; Tallroth et al., 1992).
Many of the studies that consider *in vivo* ranges of translation, though, are difficult to compare, as there is little standardisation in methods of calculating translation (Boden & Wiesel, 1990). Dvorak and colleagues (Dvorak et al., 1991), for example, found that using points on the superior end-plate of the segment generated translations two to three times greater than those employing inferior end-plate landmarks.

In the present study the range of translation appears to be well in excess of the established values for asymptomatics (Table 9.3) being around 17% for levels L1 to L4 and 27% for L5/S1. This may be explained, simply, as a measurement aberration resulting from poor reliability. Alternatively, values of this order may be a consequence of the recumbent protocol employed. With little axial preload acting on the lumbar segments an increase in gapping between the zygapophysial joints might be allowed to occur which could permit greater AP slippage. However, the study carried out by Frobin and co-workers (Frobin et al., 1996) included a comparison of standing and recumbent postures and concluded that these factors had no influence on the "pattern of segmental motion". Nevertheless, Frobin and colleagues make no mention of whether this statement includes the magnitude of translation or if it applies only to the pattern of rotational movement.

In the present study, predictably, measurements from the L5/S1 region are error-prone and for the above value of 27% AP translation carries an SD of 6.45. For calculation of the

mean initial displacement (i.e. the position of L5 at neutral) the SD is greater than 10. Given this, AP translation measurements for this level in the present study convey little credibility. For other levels, although the absolute values are not likely to be truly representative, they can serve as a guide and perhaps provide useful information in terms of general trends throughout the motion sequence. This is particularly so since few, if any, *in vivo* studies have examined translation outside of the extreme positions. In this regard, as with intervertebral angles, the present study has revealed that maximum and minimum values for AP translation are not always represented at the extreme positions (Tables 9.10 & 9.11). From Table 9.11 it can be seen that only 10% of the anterior translation maxima occur exactly at full flexion, correspondingly only 11.25% of the posterior maxima occur exactly at full extension. Not surprisingly the majority of the anterior translation maxima lie between neutral and full flexion, whilst the greatest number of posterior maxima are found between flexion and extension.

TABLE 9.10

PERCENTAGE OF MAXIMUM AND MINIMUM VALUES FOR TRANSLATION WITH RESPECT TO THEIR LOCATION WITHIN THE MOTION SEQUENCE

	Translation	Neutral to flexion	Full flexion	Flexion to extension	Full extension	Extension to neutral
L1	Posterior	15%	0%	55%	20%	10%
L1	Anterior	55%	5%	30%	0%	10%
L2	Posterior	0%	0%	50%	5%	45%
L2	Anterior	45%	5%	30%	15%	5%
L3	Posterior	0%	0%	75%	10%	15%
L3	Anterior	50%	10%	30%	0%	10%
L4	Posterior	10%	0%	55%	10%	25%
L4	Anterior	35%	20%	35%	0%	10%

(L1 TO L4).

TABLE 9.11

PERCENTAGE OF MAXIMUM AND MINIMUM VALUES FOR TRANSLATION WITH RESPECT TO THEIR LOCATION WITHIN THE MOTION SEQUENCE (MEAN VALUES ACROSS ALL LEVELS).

Translation (Mean values across all levels	Neutral to flexion	Full flexion	Flexion to extension	Full extension	Extension to neutral
Posterior	6.25%	0%	58.75%	11.25%	23.75%
Anterior	46.25%	10%	31.25%	3.75%	8.75%

The mean ranges of AP translation between extension and flexion only are given in Table 9.12. As can be seen these values are considerably less than those obtained by subtraction of maxima and minima, wheresoever found in the motion sequence (Table 9.3). Indeed, examination of the values reported in Table 9.12 compare well to the established values of 1-3mm given by other authors.

Table 9.12 also demonstrates that, in general, translation displacement from extension to flexion tends to be anteriorward. A positive value signifying anterior slip and negative signifying posterior, along the z-axis. The general pattern of AP translation found in the present study agrees largely with that found in recent *in vivo* studies of normal subjects (Frobin et al., 1996; Lin et al., 1994). Namely that initial vertebral position, in neutral, tends to be slightly retrolisthetic and increasingly so towards extension. Furthermore rotational movements from extension to flexion are accompanied by anterior translation towards neutral displacement and almost never extend beyond this into anterolisthesis.

TABLE 9.12

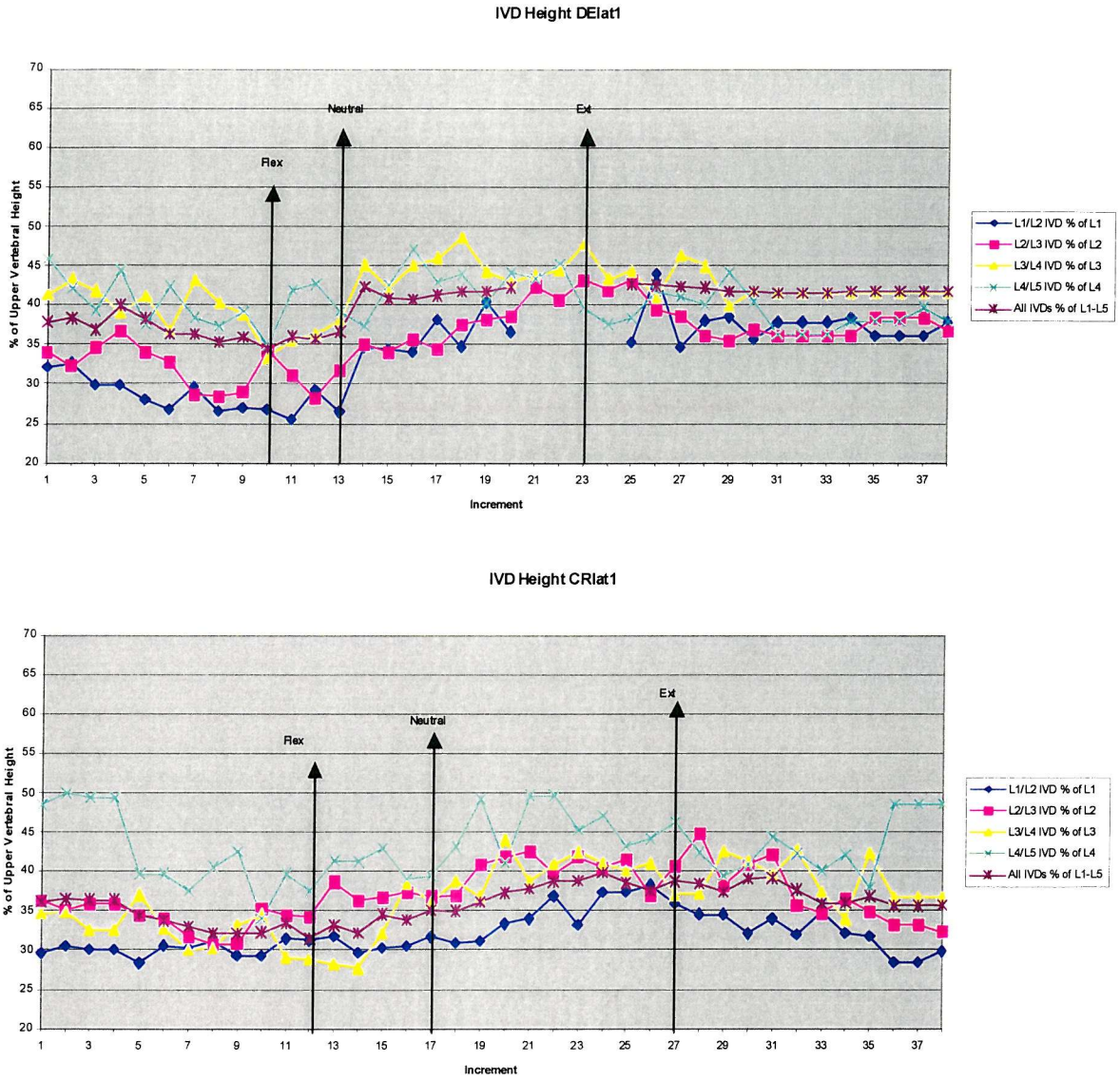
MEAN TRANSLATION DISPLACEMENT VALUES FOR EACH SEGMENT AS A PERCENTAGE OF THE AVERAGE SUPERIOR VERTEBRAL BODY DEPTH BETWEEN FULL EXTENSION AND FULL FLEXION (THE NEGATIVE SIGN DENOTES THE CRANIAL SEGMENT AS TRANSLATING POSTERIOR TO ITS SUBJACENT NEIGHBOUR)

AP translation (% of mean superior vertebral body depth)	**L1/2 (Flex/ext range)**	**L2/3 (Flex/ext range)**	**L3/4 (Flex/ext range)**	**L4/5 (Flex/ext range)**	**L5/S1 (Flex/ext range)**
Mean	*4.52*	*6.14*	*9.41*	*5.64*	*-6.42*
StDev	*6.20*	*7.34*	*5.79*	*8.08*	*10.68*

9.7.5.2 AXIAL TRANSLATION

Axial translation, axial shift or distraction of vertebral segments has been recently proposed as a sensitive measure of laxity (Kaigle et al., 1997; Ogon et al., 1997a). Consequently, the present study attempted to derive information regarding these types of movement throughout the motion sequence. Distraction or longitudinal shift was inferred by measuring changes in mean intervertebral disc (IVD) height, using the method described in Chapter 8 (Figures 8.16 & 9.8), and expressing these changes as a percentage of the mean superior vertebral body height. In general terms IVD height tended to reduce towards flexion and increase towards extension (Figure 9.7).

FIGURE 9.7
EXAMPLES OF CHANGE IN IVD HEIGHT ACROSS THE MOTION SEQUENCE



The mean IVD heights for all subjects and levels across the motion sequence are depicted in Table 9.13. These values are comparable, with the exception of L1/2, with the dimensions provided by Twomey (unpublished data) and quoted by Bogduk (Bogduk, 1997). The results given by Twomey are similar to those described by Panjabi and co-workers (Panjabi et al., 1992) but included data on disc dimensions, which the Panjabi study did not.

TABLE 9.13

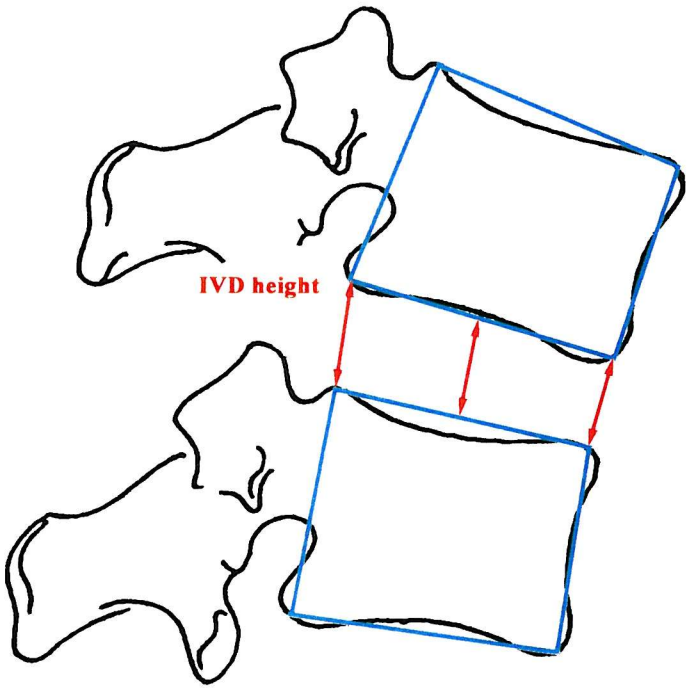
MEAN IVD HEIGHTS FOR ALL SUBJECTS AND LUMBAR LEVELS AND L1-L5 IVDS COMBINED, ACROSS THE ENTIRE MOTION SEQUENCE AS A PERCENTAGE OF THE SUPERIOR VERTEBRAL BODY HEIGHT (OR THE SUM OF L1-L5 BODY HEIGHTS FOR THE COMBINED DATA)

IVD Height Across motion sequence	L1/2 (% of superior body height)	L2/3 (% of superior body height)	L3/4 (% of superior body height)	L4/5 (% of superior body height)	L1-L5 (% of total body height)
Mean (+/- 1SD)	35 (+/- 3)	39 (+/- 4)	38 (+/- 5)	43 (+/- 7)	39 (+/- 4)

The dimensions in the present study are, however, somewhat less than those determined by Twomey from cadaveric specimens. This discrepancy might have been explained by the compressive effect of the trunk musculature acting on the lumbar spine *in vivo*. The compressive effect would, of course, be expected to have had a greater influence had our methodology been one of an upright weight bearing nature. A recent paper, using digitised CT images of the lumbar spine in patients with low back pain, has revealed similar results to those of the *in vitro* measurements but of slightly lesser magnitude (Zhou et al., 2000). In this study only the lumbar segments and discs of L3, L4 and L5 were measured and the methodology of recumbent CT imaging, like that of DVF, reduces the axial preload of standing weight bearing studies. It seems more likely, however, that, in the present study, the method of measurement may be more responsible for the apparent reduction in disc height when compared to other studies. The current methodology employed for kinematic analysis depends on mapping a template to each successive image of the respective vertebral bodies. In turn the average axial distance between these templates is used to indicate the change in disc height and give an estimate of vertical translation or separation (Figure 9.8).

FIGURE 9.8

MEASUREMENT OF AXIAL TRANSLATION/IVD HEIGHT, SHOWING DISPARITY BETWEEN "TRUE" DISC HEIGHT AND DISTANCE BETWEEN TEMPLATES.



In other words, true disc height is not measured and no account is taken of the concavity of the superior or inferior end-plates, which would result in an underestimation of the actual cross-sectional height of these structures. For the purposes of estimating axial translation, however, the mean distance between templates was considered adequate and, furthermore, avoided the influence of disc wedging.

In addition, no account was taken of diurnal changes in disc height, which are well recognised (Adams et al., 1987; Adams et al., 1990; Botsford et al., 1994). Changes in IVD height with loading have been thought to arise from a combination of fluid or volume loss and radial bulging of the disc (Adams et al., 1987; Adams et al., 1990). Radial bulging will be invisible on plain film and fluoroscopic imaging and, therefore, cannot be considered in the present study. An *in vivo* study using MRI findings, however, has suggested that most of the diurnal loss in disc height is due to volume changes and that

radial bulging effects are minimal (Botsford et al., 1994). Interestingly, recent work has suggested that these diurnal changes are greater than previously thought and that up to 60% of the total height loss occurs immediately on axial loading of the spine (Keller & Nathan, 1999).

In common with the findings of Kaigle et al (Kaigle et al., 1997), maximum and minimum values for axial translation or IVD height did not occur at the extremes of full flexion or full flexion. Table 9.14 compares the maximum range of axial translation with the range found between flexion and extension.

TABLE 9.14

COMPARISON OF MAXIMUM RANGE OF IVD HEIGHT WITH FLEXION/EXTENSION RANGE (ALL EXPRESSED AS PERCENTAGES OF THE RESPECTIVE SUPERIOR VERTEBRAL HEIGHT EXCEPT L1-L5 WHICH IS EXPRESSED AS A PERCENTAGE OF THE TOTAL COMBINED VERTICAL HEIGHT OF L1-L5)

Range of IVD Height (% of superior vertebral body height)	**L1/2**	**L2/3**	**L3/4**	**L4/5**	**L1-L5**
Mean Maximum Range (+/- 1SD)	*17 (+/- 8)*	*14 (+/- 6)*	*12 (+/- 3)*	*18 (+/- 7)*	*6 (+/- 2)*
Mean Flex/Ext Range (+/- 1SD)	*9 (+/- 4)*	*6 (+/- 4)*	*5 (+/- 4)*	*8 (+/- 6)*	*3 (+/- 3)*

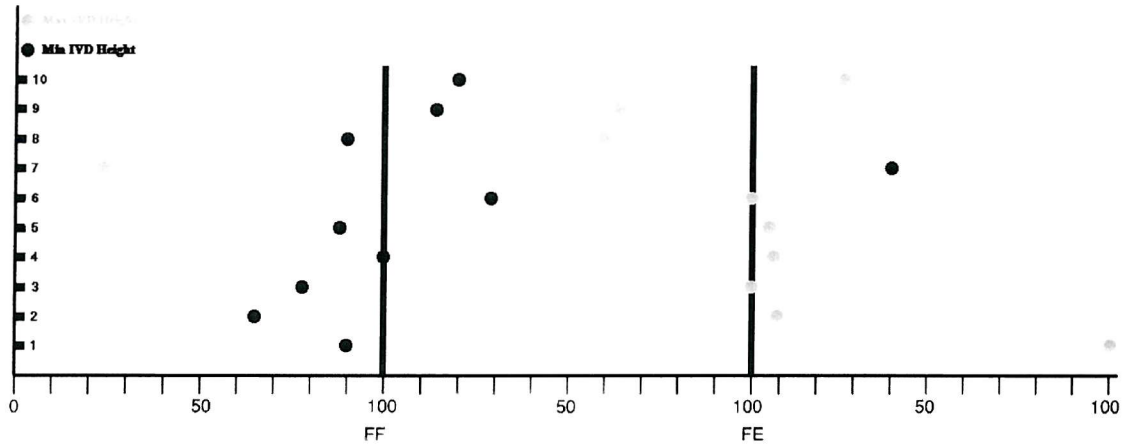
In all cases the range found only between flexion/extension underestimates the true maximum range of axial translation. The flexion/extension range was, on average, 8.25% (of superior vertebral height) smaller than the maximum range for each segment between L1 and L5. This discrepancy could perhaps, as noted by Kaigle and colleagues (Kaigle et al., 1997), explain why many studies of flexion/extension range have failed to establish clear differences between asymptomatics and low back pain patients (Dvorak et al., 1991; Penning et al., 1984; Stokes & Frymoyer, 1987).

Figure 9.9 shows an example of a diagrammatic representation of how these maxima and minima are distributed throughout the movement. These figures display the locations of the maximum and minimum values of IVD height, for each level, as a percentage of the motion sequence. Examination of these data, demonstrate how rare it is for the maximum and minimum values to coincide with the extreme positions. In only one case, subject 4 (DO level L4/5) screening 1, do both the maximum and minimum IVD heights agree with full flexion and extension positions. There appears, however, to exist a curious pattern in the distribution of these maxima and minima across the motion sequence.

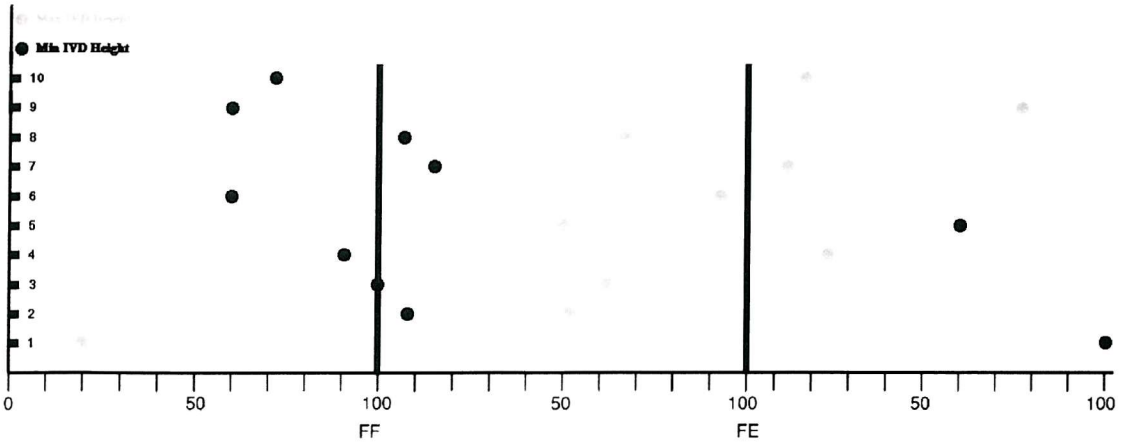
FIGURE 9.9

EXAMPLES OF THE LOCATION OF MAXIMUM AND MINIMUM VALUES FOR IVD HEIGHT, FOR ALL SUBJECTS, THROUGHOUT THE MOTION SEQUENCE

(SAGITTAL PLANE ONLY)



L2/3 maximum and minimum IVD heights (screening 1)



L3/4 maximum and minimum IVD heights (screening 1)

For L1/2 the IVD height minima tend to cluster between 20% into flexion and 30% between flexion/extension (90% for screenings 1and 2 combined). This would be consistent with a compressive moment at this level occurring early in the movement, as might be expected with a methodology that fixes the upper trunk and rotates the pelvis into flexion. Likewise, the maximum IVD heights for the L1/2 segment tend to group around extension with the majority (95% for both screenings) being found between 50% amid flexion/extension to 50% after extension. Again this could be explained by tensile or distraction moments occurring towards extension at this level. This pattern appears to progress in a caudal fashion throughout the motion sequence with the L4/5 segment showing a pattern almost the reverse of that seen at L1/2. Here at L4/5 the majority of the IVD height maxima are found gathering around flexion, with 75% (screenings 1 and 2 combined) located within 50% either side of full flexion. The minimum values show a similar, but not so marked, distribution with 55% occurring between 60% towards and 20% after full extension. Kaigle and colleagues (Kaigle et al., 1995) have reported that the greatest change in axial translation noted in their passive motion study, was found on returning the subject from full flexion or extension with relatively little occurring towards full flexion or extension. This study, however, was carried out on a porcine model and was confined to the pig L3/4 segment only. Nevertheless, similarities were noted with the present study, with most of the change in disc height appearing to take place on returning from full flexion with minimal change taking place approaching full flexion.

9.8 CONCLUSION

Determining the neutral position once the motion sequence has begun is, potentially, of considerable value in the characterisation of spinal motion. In the present study it was difficult to achieve this with any accuracy since the intervertebral configuration of the lumbar spine is subject to much variability within its global range of motion. It may not be possible to determine exactly the point of neutral positioning for the whole lumbar spine since it is variable for each FSU but it is important to attempt to estimate the "range" of neutral in order to assess sudden changes in movements suggestive of instability. In this respect, future methodologies of passive motion should attempt to standardise the initial position and more accurately determine when the lumbar spine has returned to this phase of the sequence. A more adjustable and rigid method of restraint may be all that is required to ensure that subjects are taken through a more reproducible movement.

Ranges of rotational motion, in both sagittal and coronal planes, for the 10 volunteers included in the present study, are largely in agreement with established values for *in vivo* human subjects.

The most striking result in the present study, however, was that only 17% of the intervertebral ranges fell on or within one increment of flexion or extension. This demonstrates that the majority of intervertebral ranges fall outside of the extremes of motion and suggests that traditional radiographic methods of assessing range may underestimate the true extent of movement. Similarly, as with intervertebral angles, the present study has revealed that maximum and minimum values for AP and axial translation are not fully represented by measurements obtained at the extremes of planar motion. Errors in these measurements, especially those of AP translation, prevented a reliable quantitative assessment of the extent of this disparity between ranges.

The present study demonstrated that paradoxical rotations, in asymptomatic subjects, are a relatively common finding. This was especially true of movements in the sagittal plane and would suggest that, in isolation, paradoxical motion does not provide *a priori* evidence of instability.

9.9 FUTURE WORK

Measurement error associated with manual marking is, without doubt, the major stumbling block to the development of this technique. Improvements in image quality are likely to continue but are not expected to substantially reduce measurement error. The enhancement of measurement validity and reliability are most likely to occur as a result of the development of edge detection and automated marking methods. The facility for greatly augmenting the numbers of repeated measures offers the ability to average measurements, as well as increase and overlap increments to produce data of a much more representative nature. Automated marking will allow the development of reliable indices of motion based on iterative calculations over greater numbers of smaller increments.

The prototype passive motion table worked well but was somewhat limited in design. Future versions should allow much greater adjustability for subject dimensions. A more comfortable table surface, such as provided by viscoelastic foams, would enhance patient/subject stability and reduce unwanted movements. From the present study it seems

likely that the volunteers were not subjected to as great a degree of lumbar flexion than extension. Modifications such as improved subject stability and restraint should help increase the ability of the passive motion table in challenging the lumbar spine in flexion. Nevertheless, the natural posture of the lumbar region means that symmetrical changes in angular displacement are unlikely to generate equal deformation between flexion and extension. Future modifications should, therefore, address this lack of angular symmetry and, perhaps, allow more complex movements. If, for instance, during the flexion phase, the lower section of the table were to provide a tensile, inferior distraction force on the lumbar spine, this might produce sufficient pre-stress to increase the bending moment over the lumbar segments. As mentioned earlier in this chapter, axial distraction or translation of spinal segments may reveal more information on the integrity of the disc and other tissues than previously thought.

Alternatively, a padded cylinder, perpendicular to the table and in apposition to the subject's abdomen, may also increase the bending moment. If this device were to be reciprocally linked to the flexion movement such that increasing flexion yielded greater apposition to the abdomen, the lumbar spine could be, in effect, curled around the device in the fashion of a pipe-bending machine.

Whatever form future versions of this prototype take, the main objective of such an apparatus should remain, namely to challenge, as much as possible, the passive elements of the lumbar intervertebral joints *in vivo*. In patients in pain, however, this may prove difficult and the administration of analgesics and even anaesthetics may be necessary to reduce or abolish the influence of the neuromuscular system. However it is achieved, it is clear that only by truly challenging these tissues under conditions which also reduce the effects of any compensatory mechanisms, can we hope to identify abnormal mechanical behaviour *in vivo*.

CONCLUSIONS AND SUMMARY

Spinal pain is a significant problem both for the individual and society. Investigating the detailed movements of the spine, which is necessary to evaluate the integrity of intervertebral linkages, requires penetrative imaging combined with reliable kinematic measurement. Recent attempts have been made to use MR imaging on lumbar spine motion (Edmondston et al., 2000; Vitzthum et al., 2000). Although these studies represent a significant step forward in the *in vivo* investigation of spinal movement, they do not yet allow the true dynamic analysis of motion. Digitised videofluoroscopy (DVF) and now digitised fluoroscopy (DF), on the other hand, can provide genuine dynamic assessment of spinal motion across the whole motion sequence. DVF has shown some promise in the assessment of post-fusion patients and as an aid in selecting those suitable for repeat surgery.

The major limitation of DF, however, lies with measurement error, which can be attributed, largely, to manual marking techniques. Automated analysis, with its potential for vastly increased iterative measurement, will almost certainly offer more meaningful data and transform DF from a research tool into a useable clinical investigation. In terms of minimal risk to patients and computational power, DF represents the most likely and cost-effective approach, currently available, to assess spinal mechanical integrity *in vivo*.

Kinematic analysis of intervertebral segments, for all its apparent complexity, relies on the basic measurement of rotation and translation. There is no doubt that, under ideal conditions, such analysis should be carried out in three dimensions. In its present form, digitised fluoroscopy (DF) is unable to provide three-dimensional information. There is little reason, however, why DF cannot be modified to employ bi-planar image intensifiers and hence offer the opportunity for three-dimensional analysis. As far as we are aware, no bi-planar fluoroscopy system is commercially available at present and adaptation would require a cooperative and supportive manufacturer. Given this, X-ray exposure need not be significantly increased beyond that of present levels if the orthogonal X-ray tubes were synchronously pulsed.

This thesis has reviewed relevant biomechanical literature concerning the lumbar spine, both *in vivo* and *in vitro*, and attempted to explain how findings in the laboratory might

relate to data obtained from our DVF/DF research. In respect of coupled motions we can say that they are of little significance for sagittal plane studies, but are important when considering lateral bending movements. For the lumbar spine, accompanying rotations appear to have no strict pattern and depend, to some extent on posture. Furthermore, there is a clear distinction between coupling at the lumbar and lumbosacral regions.

Axial rotation, it is thought, may have an important role in the causation of lumbar segmental instability but is difficult to assess *in vivo* with present two-dimensional techniques.

We can conclude that *in vitro* biomechanical studies are still an important means of enhancing our understanding of the component tissues of the spine and how surgery, pathology and other damage might alter responses to loading and hence spinal kinematics. This is particularly true for the growing trend in methodologies that employ whole lumbar spine specimens (Adams & Dolan, 1995; Lu et al., 1999; Lysack et al., 2000; Patwardhan et al., 1999; Yamamoto et al., 1989). Nevertheless, it is with *in vivo* studies that we have the greatest chance of obtaining the most clinically valuable data. In this respect, however, it is important that we establish strong baseline data on normal spinal motion under controlled conditions and this thesis has made a preliminary attempt at that objective. Research questions based on abnormal or symptomatic populations should be cautiously designed and carefully interpreted.

It is becoming clear, also, that any kinematic analysis should be carried out throughout the motion sequence and not just at the extremes of trunk motion (Kaigle et al., 1997). The work presented in this thesis unequivocally supports this notion. In terms of vertebral translation in the sagittal plane, information concerning z-axis motion may prove less clinically relevant than previously thought. Nevertheless, translation along the y-axis, axial translation, shows promise in helping to determine segmental stability/instability (Kaigle et al., 1997; Ogon et al., 1997a). In any event, these parameters may have to be redefined in terms of the unloaded, recumbent and passively challenged spine.

In respect of the instantaneous centre of rotation (ICR), we can conclude that it is an effective kinematic index for spinal motion studies but is, at present, too error sensitive for common clinical usage (Harvey & Hukins, 1998). Direct measurement of translations and rotations may, at this time, hold the best return for clinical assessment (Frobin et al., 1997;

Frobin et al., 1996).

Segmental instability is a multifactorial disorder and, as yet, has no single definition acceptable to all scientists and clinicians involved in its investigation. The identification of the neutral or lax zones and their role in segmental stability shows great promise as an area of clinically relevant biomechanical research (Crawford et al., 1998; Kaigle et al., 1995; Kaigle et al., 1997; Kumar & Panjabi, 1995; Ogon et al., 1997b; Panjabi, 1992b). The experimental work presented in this thesis was, nevertheless, unable to establish any clear method of inferring load-deformation characteristics from *in vivo* kinematics. Changes in the neutral or lax zones, if detectable *in vivo*, potentially offers the greatest chance of success in distinguishing normal from abnormal intervertebral holding elements in back pain patients. Given this premise, the study of intervertebral motion, under passive conditions and throughout the full range of movement seems the most likely way forward to increasing our understanding of spinal instability. Kinematic data gathering techniques that give us information on relative motion between spinal segments can help determine probable instability, particularly in the quantification of small movements such as axial translation and movements around the neutral zone. Confirmation of segmental instability, however, is likely to be made only on the basis of combined information from the clinical, radiographic and biomechanical disciplines. These conclusions argue strongly for a multi-disciplinary approach to the assessment of instability. A method such as this would possibly involve obtaining data on muscle contraction from functional EMG, ultrasound studies of muscular dimensions together with kinematic data on spinal motion linked to levels of perceived pain.

APPENDIX I

CALCULATION OF VERTEBRAL DISPLACEMENT

(FROBIN et al 1996)

Calculation of Displacement of Vertebrae relative to one another

Notation

$\mathbf{r}_1 = x_1\mathbf{i} + y_1\mathbf{j}$: Position vector of dorsal midpoint in vertebra 1
$\mathbf{a}_1 = a_{1,x}\mathbf{i} + a_{1,y}\mathbf{j}$: Direction vector of line through dorsal and ventral midpoints in vertebra 1
λ_1: Scalar sliding parameter for vector equation of line through dm and vm of vertebra 1

$\mathbf{r}_2 = x_2\mathbf{i} + y_2\mathbf{j}$: Position vector of dorsal midpoint in vertebra 2
$\mathbf{a}_2 = a_{2,x}\mathbf{i} + a_{2,y}\mathbf{j}$: Direction vector of line through dorsal and ventral midpoints in vertebra 2
λ_2: Scalar sliding parameter for vector equation of line through dm and vm of vertebra 2

$\mathbf{r}_3 = x_3\mathbf{i} + y_3\mathbf{j}$: Position vector of intersection of lines 1 and 2, a fixed point on the bisectrix
$\mathbf{a}_3 = a_{3,x}\mathbf{i} + a_{3,y}\mathbf{j}$: Direction vector of bisectrix
λ_3: Scalar sliding parameter for vector equation of bisectrix

$\mathbf{r} = \mathbf{r}_1 + \lambda_1\mathbf{a}_1$: vector equation of line through dm and vm of verteba 1
$\mathbf{r} = \mathbf{r}_2 + \lambda_2\mathbf{a}_2$: vector equation of line through dm and vm of verteba 1
$\mathbf{r} = \mathbf{r}_3 + \lambda_3\mathbf{a}_3$: vector equation of bisectrix

$\mathbf{c}_{p,1} = c_{p,1,x}\mathbf{i} + c_{p,1,y}\mathbf{j}$ position vector of centre point of vertebra 1, calculated as the average of the dorsal and ventral midpoints.
$\mathbf{c}_{p,2} = c_{p,2,x}\mathbf{i} + c_{p,2,y}\mathbf{j}$ position vector of centre point of vertebra 2, calculated as the average of the dorsal and ventral midpoints

$\lambda_{3,1}$: Value of the bisectrix sliding parameter λ_3 at the projection point of $\mathbf{c}_{p,1}$ on the bisectrix
$\lambda_{3,2}$: Value of the bisectrix sliding parameter λ_3 at the projection point of $\mathbf{c}_{p,2}$ on the bisectrix

$\mathbf{Dc} = Dc_x\mathbf{i} + Dc_x\mathbf{j}$: final displacement vector between vertebrae 1 and 2.

- Condition for $\mathbf{a}_3$ to be the direction vector bisecting the angle between lines 1 and 2:

$$\mathbf{a}_3 = \frac{|\mathbf{a}_1|\mathbf{a}_2 + |\mathbf{a}_2|\mathbf{a}_1}{\|\mathbf{a}_1|\mathbf{a}_2 + \mathbf{a}_1|\mathbf{a}_2\|} \quad \text{(A)}$$

Given the vectors $\mathbf{a}_1$ and $\mathbf{a}_2$ this determines a unit vector $\mathbf{a}_3$.

- Condition for intersection point of lines 1 and 2, $\mathbf{r}_3$

$$\mathbf{r}_1 + \lambda_1 \mathbf{a}_1 = \mathbf{r}_2 + \lambda_2 \mathbf{a}_2$$

This gives two simultaneous equations in λ_1, λ_2 which can be solved to give

$$\lambda_1 = \frac{a_{2,y}(x_1 - x_2) - a_{2,x}(y_1 - y_2)}{a_{1,y}a_{2,x} - a_{1,x}a_{2,y}}$$

$$\lambda_2 = \frac{a_{1,y}(x_1 - x_2) - a_{1,x}(y_1 - y_2)}{a_{1,y}a_{2,x} - a_{1,x}a_{2,y}}$$

We only require one of these (λ_1 or λ_2) to substitute into the vector equations (1 or 2 respectively) to give the intersection point. Which you choose is irrelevant as both answers should be the same (it's a good check of the algebra to try both anyway), i.e.,

$$\mathbf{r}_3 = \mathbf{r}_1 + \lambda_1 \mathbf{a}_1 \quad \text{or} \quad \mathbf{r}_3 = \mathbf{r}_2 + \lambda_1 \mathbf{a}_2 \quad \text{(B)}$$

- Having got the intersection point $\mathbf{r}_3$ from (B) and the direction vector $\mathbf{a}_3$ from (A) we can now write down the equation of the bisectrix:

$$\mathbf{r} = \mathbf{r}_3 + \lambda_3 \mathbf{a}_3$$

- Condition for the value of λ_3 at the perpendicular point of projection from $\mathbf{c}_{p,1}$ onto the bisectrix :

$$\left(\mathbf{c}_{p,1} - [\mathbf{r}_3 + \mathbf{a}_3 \lambda_3]\right)\mathbf{a}_3 = 0$$

i.e., the vector $\left(\mathbf{c}_{p,1} - [\mathbf{r}_3 + \mathbf{a}_3 \lambda_3]\right)$ is perpendicular to the direction vector of the bisectrix $\mathbf{a}_3$.

This can be solved to give

$$\lambda_{3,1} = \frac{a_{3,x}\left(r_{3,x} - c_{p,1,x}\right) + a_{3,y}\left(r_{3,y} - c_{p,1,y}\right)}{a_{3,x}^2 + a_{3,y}^2}$$

Thus the position vector of the projection point from $\mathbf{c}_{p,1}$ on the bisectrix is

$$\mathbf{r}_{3,1}^{*} = \mathbf{r}_3 + \lambda_{3,1}\mathbf{a}_3$$

- An analogous calculation for the projection point from $\mathbf{c}_{p,2}$ onto the bisectrix gives

$$\lambda_{3,2} = \frac{a_{3,x}\left(r_{3,x} - c_{p,2,x}\right) + a_{3,y}\left(r_{3,y} - c_{p,2,y}\right)}{a_{3,x}^2 + a_{3,y}^2}$$

$$\mathbf{r}_{3,2}^{*} = \mathbf{r}_3 + \lambda_{3,2}\mathbf{a}_3$$

The vector **Dc** is then just

$$\mathbf{Dc} = \mathbf{r}_{3,1}^{*} - \mathbf{r}_{3,2}^{*}$$

The absolute length of Dc is then

$$|\mathbf{Dc}| = \sqrt{Dc_x^2 + Dc_y^2}$$

To get the relative (dimensionless) length, you divide this number by whatever length scale you are using.

REFERENCES

Adams, M.A. (1999). Biomechanics of the Intervertebral Disc, Vertebra and Ligaments. In *Lumbar Segmental Instability*, Szpalski, M., Gunzburg, R. & Pope, M.H. (eds) pp. 3-13. Lippincott Williams and Wilkins: Philadelphia.

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

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

Adams, M.A. & Dolan, P. (1996). Time-dependent changes in the lumbar spine: resistance to bending. *Clin Biomech (Bristol, Avon)*, **11,** 194-200.

Adams, M.A., Dolan, P. & Hutton, W.C. (1987). Diurnal variations in the stresses on the lumbar spine. *Spine*, **12,** 130-7.

Adams, M.A., Dolan, P. & Hutton, W.C. (1988). The lumbar spine in backward bending. *Spine*, **13,** 1019-26.

Adams, M.A., Dolan, P., Hutton, W.C. & Porter, R.W. (1990). Diurnal changes in spinal mechanics and their clinical significance. *Journal of Bone and Joint Surgery. British Volume*, **72,** 266-70.

Adams, M.A., Freeman, B.J., Morrison, H.P., Nelson, I.W. & Dolan, P. (2000a). Mechanical initiation of intervertebral disc degeneration. *Spine*, **25,** 1625-36.

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

Adams, M.A. & Hutton, W.C. (1982a). The mechanics of prolapsed intervertebral disc. *International Orthopaedics*, **6,** 249-253.

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

Adams, M.A. & Hutton, W.C. (1985a). The effect of posture on the lumbar spine. *Journal of Bone and Joint Surgery - Series B*, **67,** 625-629.

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

Adams, M.A., Hutton, W.C. & Stott, J.R.R. (1980). The Resistance to Flexion of the Lumbar Intervertebral Joint. *Spine*, **5,** 245-253.

Adams, M.A., May, S., Freeman, B.J., Morrison, H.P. & Dolan, P. (2000b). Effects of backward bending on lumbar intervertebral discs. Relevance to physical therapy treatments for low back pain. *Spine*, **25,** 431-7; discussion 438.

Ahern, D.K., Follick, M.J., Council, J.R., Laserwolston, N. & Litchman, H. (1988). Comparison of Lumbar Paravertebral Emg Patterns in Chronic Low-Back Pain Patients and Non-Patient Controls. *Pain*, **34,** 153-160.

Ahern, D.K., Hannon, D.J., Goreczny, A.J., Follick, M.J. & Parziale, J.R. (1990). Correlation of Chronic Low-Back-Pain Behavior and Muscle Function Examination of the Flexion-Relaxation Response. *Spine*, **15,** 92-95.

Aigner, T., Gresk-otter, K.R., Fairbank, J.C., von der Mark, K. & Urban, J.P. (1998). Variation with age in the pattern of type X collagen expression in normal and scoliotic human intervertebral discs. *Calcified Tissue International*, **63,** 263-8.

Amevo, B., Macintosh, J.E., Worth, D. & Bogduk, N. (1991a). Instantaneous axes of rotation of the typical cervical motion segments: I. An empirical study of technical errors. *Clinical Biomechanics*, **6,** 31-37.

Amevo, B., Worth, D. & Bogduk, N. (1991b). Instantaneous axes of rotation of the typical cervical motion segments: A study in normal volunteers. *Clinical Biomechanics*, **6,** 111-117.

Amevo, B., Worth, D. & Bogduk, N. (1991c). Instantaneous axes of rotation of the typical cervical motion segments: II. Optimization of technical errors. *Clinical Biomechanics*, **6,** 38-46.

Anderson, J.A. & Sweetman, B.J. (1975). A combined flexi-rule/hydrogoniometer for measurement of lumbar spine and its sagittal movement. *Rheumatol Rehabil*, **14,** 173-9.

Andersson, E.A., Oddsson, L.I.E., Grundstrom, H., Nilsson, J. & Thorstensson, A. (1996). EMG activities of the quadratus lumborum and erector spinae muscles during flexion - Relaxation and other motor tasks. *Clinical Biomechanics*, **11,** 392-400.

Aspden, R.M. (1992). Review of the functional anatomy of the spinal ligaments and the lumbar erector spinae muscles. *Clinical Anatomy*, **5,** 372-387.

Axelsson, P., Johnsson, R. & Stromqvist, B. (1992). Effect of lumbar orthosis on intervertebral mobility. A roentgen stereophotogrammetric analysis. *Spine*, **17,** 678-81.

Axelsson, P., Johnsson, R. & Stromqvist, B. (2000). Is there increased intervertebral mobility in isthmic adult spondylolisthesis? A matched comparative study using roentgen stereophotogrammetry. *Spine*, **25,** 1701-3.

Baltzopoulos, V. (1995). A videofluoroscopy method for optical distortion correction and measurement of knee-joint kinematics. *Clinical Biomechanics*, **10,** 85-92.

Bartelink, D.L. (1957). The Role of Abdominal Pressure in Relieving the Pressure on the Lumbar Intervertebral Discs. *J Bone Joint Surg*, **39B,** 718-725.

Bartels, E.M., Fairbank, J.C., Winlove, C.P. & Urban, J.P. (1998). Oxygen and lactate concentrations measured in vivo in the intervertebral discs of patients with scoliosis and back pain. *Spine*, **23,** 1-7; discussion 8.

Bennett, P.H. & Burch, T.A. (1967). New York Symposium on Population Studies in the Rheumatic Diseases: New diagnostic criteria. *Bull Rheum Dis*, **17,** 453.

Bergmark, A. (1989). Stability of the lumbar spine. A study in mechanical engineering. *Acta Orthopaedica Scandinavica. Supplementum*, **230,** 1-54.

Boden, S.D. (2000). Biology of lumbar spine fusion and use of bone graft substitutes: present, future, and next generation. *Tissue Eng*, **6,** 383-99.

Boden, S.D. & Wiesel, S.W. (1990). Lumbosacral Segmental Motion in Normal Individuals - Have We Been Measuring Instability Properly. *Spine*, **15,** 571-576.

Bogduk, N. (1991). The lumbar disc and low back pain. *Neurosurgery Clinics of North America*, **2,** 791-806.

Bogduk, N. (1997). *Clinical Anatomy of the Lumbar spine and Sacrum*. CHURCHILL LIVINGSTONE: New York.

Boos, N., Rieder, R., Schade, V., Spratt, K.F., Semmer, N. & Aebi, M. (1995). 1995 Volvo Award in clinical sciences. The diagnostic accuracy of magnetic resonance imaging, work perception, and psychosocial factors in identifying symptomatic disc herniations. *Spine*, **20,** 2613-25.

Borenstein, D.G., Wiesel, S.W. & Boden, S.D. (1995). Therapy. In *Low back pain: medical diagnosis and comprehensive management* pp. 658-659. W. B. Saunders Company: Philadelphia.

Botsford, D.J., Esses, S.I. & Ogilvie-Harris, D.J. (1994). In vivo diurnal variation in intervertebral disc volume and morphology. *Spine*, **19,** 935-40.

Bram, J., Zanetti, M., Min, K. & Hodler, J. (1998). MR abnormalities of the intervertebral disks and adjacent bone marrow as predictors of segmental instability of the lumbar spine. *Acta Radiologica*, **39,** 18-23.

Breen, A., Allen, R. & Morris, A. (1988). An image processing method for spine kinematics: preliminary studies. *Clinical Biomechanics*, **3,** 5-10.

Breen, A.C. (1991). The measurement of the kinematics of the human spine using videofluoroscopy and image processing. In *Department of Mechanical Engineering* pp. 216. University of Southampton: Southampton.

Breen, A.C. & Allen, R. (1996). Digital videofluoroscopy and abnormal spinal movement. In *Lumbar Spine Disorders: Current Concepts*, Aspden, R.M. & Porter, R.W. (eds), Vol. 2. pp. 79-88. World Scientific: Singapore.

Breen, A.C., Allen, R. & Morris, A. (1989). Spine kinematics: a digital videofluoroscopic technique. *J Biomed Eng*, **11,** 224-228.

Bryant, J.T., Reid, J.G., Smith, B.L. & Stevenson, J.M. (1989). Method for determining vertebral body positions in the sagittal plane using skin markers. *Spine*, **14,** 258-265.

Bryant, J.T., Wevers, H.W. & Lowe, P.J. (1984). One parameter model for error in instantaneous centre of rotation measurements. *Journal of Biomechanics*, **17,** 317-323.

Burton, A.K., Battie, M.C., Gibbons, L., Videman, T. & Tillotson, K.M. (1996). Lumbar disc degeneration and sagittal flexibility. *Journal of Spinal Disorders*, **9,** 418-424.

Burton, A.K. & Tillotson, K.M. (1988). Reference values for 'normal' regional lumbar sagittal mobility. *Clinical Biomechanics*, **3,** 106-113.

Buttermann, G.R., Garvey, T.A., Hunt, A.F., Transfeldt, E.E., Bradford, D.S., Boachie-Adjei, O. & Ogilvie, J.W. (1998). Lumbar fusion results related to diagnosis. *Spine*, **23,** 116-27.

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

Cholewicki, J., Juluru, K. & McGill, S.M. (1999). Intra-abdominal pressure mechanism for stabilizing the lumbar spine. *Journal of Biomechanics*, **32,** 13-7.

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

Cholewicki, J., McGill, S.M., Wells, R.P. & Vernon, H. (1991). Method for measuring vertebral kinematics from videofluoroscopy. *Clinical Biomechanics*, **6,** 73-78.

Cholewicki, J., Panjabi, M.M. & Khachatryan, A. (1997). Stabilizing function of trunk flexor-extensor muscles around a neutral spine posture. *Spine*, **22,** 2207-2212.

Chow, D.H.K., Luk, K.D.K., Evans, J.H. & Leong, J.C.Y. (1996). Effects of short anterior lumbar interbody fusion on biomechanics of neighboring unfused segments. *Spine*, **21,** 549-555.

Cossette, J.W., Farfan, H.F., Robertson, G.H. & Wells, R.V. (1971). The instantaneous center of rotation of the third lumbar intervertebral joint. *J Biomech*, **4,** 149-53.

Crawford, N.R., Peles, J.D. & Dickman, C.A. (1998). The spinal lax zone and neutral zone: measurement techniques and parameter comparisons. *Journal of Spinal Disorders*, **11,** 416-29.

Crean, J.K., Roberts, S., Jaffray, D.C., Eisenstein, S.M. & Duance, V.C. (1997). Matrix metalloproteinases in the human intervertebral disc: role in disc degeneration and scoliosis. *Spine*, **22,** 2877-84.

Crisco, J.J.d. & Panjabi, M.M. (1991). The intersegmental and multisegmental muscles of the lumbar spine. A biomechanical model comparing lateral stabilizing potential. *Spine*, **16,** 793-9.

CSAG. (1994). Epidemiology review: The epidemiology and cost of back pain. The Annex to the Clinical Standards Advisory Group's report on back pain 1994.: London.

Dai, L.Y. (1998). Disc degeneration and cervical instability - Correlation of magnetic resonance imaging with radiography. *Spine*, **23,** 1734-1738.

Davies, A.M., Fowler, J., Tyrrell, P.N., Millar, J.S., Leahy, J.F., Patel, K. & Hill, J.S. (1993). Detection of significant abnormalities on lumbar spine radiographs. *Br J Radiol*, **66,** 37-43.

Dendy, P.P. & Heaton, B. (1999). *Physics for Diagnostic Radiology*. Institute of Physics Publishing: Bristol.

Denis, F. (1983). The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. *Spine*, **8,** 817-831.

Dietemann, J. & Zollner, G. (1999). Radiologic Investigations. In *Lumbar Segmental Instability*, Szpalski, M., Gunzburg, R. & Pope, M.H. (eds) pp. 115-121. Lippincott Williams and Wilkins: Philadelphia.

Dietrich, M., Kedzior, K. & Zagrajek, T. (1991). A biomechanical model of the human spinal system. *Proc Inst Mech Eng H*, **205,** 19-26.

Dimnet, J. (1980). The improvement in the results of kinematics of in vivo joints. *Journal of Biomechanics*, **13,** 653-661.

Dimnet, J., Carret, J.P., Gonon, G. & Fischer, L.P. (1976). A technique for joint center analysis using a stored program calculator. *J Biomech*, **9,** 771-8.

Dimnet, J. & Guinguand, M. (1984). The finite displacements vector's method: An application to the scoliotic spine. *Journal of Biomechanics*, **17,** 397-408.

Dolan, P. & Adams, M.A. (1993). Influence of lumbar and hip mobility on the bending stresses acting on the lumbar spine. *Clin Biomech*, **8,** 185-192.

Drerup, B. (1984). Principles of measurement of vertebral rotation from frontal projections of the pedicles. *Journal of Biomechanics*, **17,** 923-935.

Drerup, B. (1985). Improvements in measuring vertebral rotation from the projections of the pedicles. *Journal of Biomechanics*, **18,** 369-378.

Duance, V.C., Crean, J.K., Sims, T.J., Avery, N., Smith, S., Menage, J., Eisenstein, S.M. & Roberts, S. (1998). Changes in collagen cross-linking in degenerative disc disease and scoliosis. *Spine*, **23,** 2545-51.

Dunham, W.F. (1949). Ankylosing spondylitis; measurement of hip and spine movements. *Brit J Phys Med*, **12,** 126.

Dupuis, P.R., YongHing, K., Cassidy, J.D. & KirkaldyWillis, W.H. (1985). Radiologic diagnosis of degenerative lumbar spinal instability. *Spine*, **10,** 262-276.

Dvorak, J., Panjabi, M.M., Chang, D.G., Theiler, R. & Grob, D. (1991). Functional radiographic diagnosis of the lumbar spine. Flexion-extension and lateral bending. *Spine*, **16,** 562-71.

Ebara, S., Harada, T., Hosono, N., Inoue, M., Tanaka, M., Morimoto, Y. & Ono, K. (1992). Intraoperative measurement of lumbar spinal instability. *Spine*, **17,** S44-50.

Edmondston, S.J., Song, S., Bricknell, R.V., Davies, P.A., Fersum, K., Humphries, P., Wickenden, D. & Singer, K.P. (2000). MRI evaluation of lumbar spine flexion and extension in asymptomatic individuals. *Man Ther*, **5.**

Eisenstein, S.M. (1999). "Instability" and Low Back Pain: A Way Out of the Semantic Maze. In *Lumbar Segmental Instability*, Szpalski, M., Gunzburg, R. & Pope, M.H. (eds) pp. 39-51. Lippincott Williams and Wilkins: Philadelphia.

Eisenstein, S.M., Ashton, I.K., Roberts, S., Darby, A.J., Kanse, P., Menage, J. & Evans, H. (1994). Innervation of the spondylolysis ligament . *Spine*, **19,** 912-6.

Eisenstein, S.M. & Parry, C.R. (1987). The lumbar facet arthrosis syndrome. Clinical presentation and articular surface changes. *Journal of Bone and Joint Surgery. British Volume*, **69,** 3-7.

Errington, R.J., Puustjarvi, K., White, I.R., Roberts, S. & Urban, J.P. (1998). Characterisation of cytoplasm-filled processes in cells of the intervertebral disc. *Journal of Anatomy*, **192 (Pt 3),** 369-78.

Evans, F.G. & Lissner, H.R. (1959). Biomechanical studies on the lumbar spine and pelvis. *J Bone and Joint Surg*, **41A,** 278-290.

Fairbank, J.C.T., Frost, H. & Wilson-Macdonald, J. (1996). Clinical trial to investigate stabilisation of the spine. In *Lumbar Spine Disorders: Current Concepts*, Aspden, R.M. & Porter, R.W. (eds), Vol. 2. pp. 143-160. World Scientific: Singapore.

Farfan, H.F., Cossette, J.W., Robertson, G.H., Wells, R.V. & Kraus, H. (1970). The effects of torsion on the lumbar intervertebral joints: the role of torsion in the production of disc degeneration. *J Bone Joint Surg Am*, **52,** 468-97.

Farfan, H.F. & Gracovetsky, S. (1984). The Nature of Instability. *Spine*, **9,** 714-719.

Fick, R. (1904). *Handbuch der anatomie und mechanik der gelenke unter Berucksichtigung der beweglichen muskein.* S. Fischer Verlag: Jena.

Fielding, J.W. (1957). Cineroentgenography of the normal cervical spine. *J Bone Joint Surg*, **39A,** 1280-1288.

Floman, Y. (2000). Progression of lumbosacral isthmic spondylolisthesis in adults. *Spine*, **25,** 342-7.

Floyd, W.F. & Silver, P.H.S. (1955). The function of the erectores spinae muscles in certain movements and postures in man. *J. Physiol*, **129,** 184-203.

France, J.C., Yaszemski, M.J., Lauerman, W.C., Cain, J.E., Glover, J.M., Lawson, K.J., Coe, J.D. & Topper, S.M. (1999). A randomized prospective study of posterolateral lumbar fusion. Outcomes with and without pedicle screw instrumentation. *Spine*, **24,** 553-60.

Frankel, V.H., Burstein, A.H. & Brooks, D.B. (1971). Biomechanics of internal derangement of the knee. Pathomechanics as determined by analysis of the instant centers of motion. *J Bone Joint Surg Am*, **53,** 945-62.

Fraser, R.D. (1995). Interbody, posterior, and combined lumbar fusions. *Spine*, **20,** 167s-177s.

Fredrickson, B.E., Baker, D., McHolick, W.J., Yuan, H.A. & Lubicky, J.P. (1984). The natural history of spondylolysis and spondylolisthesis. *Journal of Bone and Joint Surgery. American Volume*, **66,** 699-707.

Friberg, O. (1987). Lumbar Instability - a Dynamic Approach By Traction-Compression Radiography. *Spine*, **12,** 119-129.

Fritz, J.M., Erhard, R.E. & Hagen, B.F. (1998). Segmental instability of the lumbar spine. *Physical Therapy*, **78,** 889-896.

Frobin, W., Brinckmann, P., Biggemann, M., Tillotson, M. & Burton, K. (1997). Precision measurement of disc height, vertebral height and sagittal plane displacement from lateral radiographic views of the lumbar spine. *Clinical Biomechanics*, **12,** S1-S64.

Frobin, W., Brinckmann, P., Leivseth, G., Biggermann, M. & Reikeras, O. (1996). Precision measurement of segmental motion from flexion-extension radiographs of the lumbar spine. *Clinical Biomechanics*, **11,** 457-465.

Frymoyer, J.W., Phillips, R.B., Newberg, A.H. & MacPherson, B.V. (1986). A comparative analysis of the interpretations of lumbar spinal radiographs by chiropractors and medical doctors. *Spine*, **11,** 1020-3.

Frymoyer, J.W., Pope, M.H. & Wilder, D.G. (1990). Segmental Instability. In *The Lumbar Spine*, Wickland, E.H. (ed) pp. 617-618. W. B. Saunders Company: Philidelphia.

Frymoyer, J.W. & Selby, D.K. (1985). Segmental Instability - Rationale For Treatment. *Spine*, **10,** 280-286.

Fujiwara, A., Tamai, K., An, H.S., Kurihashi, A., Lim, T., Yoshida, H. & Saotome, K. (2000a). The relationship between disc degeneration, facet joint osteoarthritis, and stability of the degenerative lumbar spine. *Journal of Spinal Disorders*, **13,** 444-450.

Fujiwara, A., Tamai, K., An, H.S., Shimizu, K., Yoshida, H. & Saotome, K. (2000b). The interspinous ligament of the lumbar spine. Magnetic resonance images and their clinical significance. *Spine*, **25,** 358-63.

Galasko, G.S.B. (1999). Spinal Instability Secondary to Metastatic Cancer. In *Lumbar Segmental Instability*, Szpalski, M., Gunzburg, R. & Pope, M.H. (eds) pp. 85-90. Lippincott Williams and Wilkins: Philadelphia.

Gardner-Morse, M.G. & Stokes, I.A.F. (1998). The effects of abdominal muscle coactivation on lumbar spine stability. *Spine*, **23,** 86-91.

Gedalia, U., Solomonow, M., Zhou, B.H., Baratta, R.V., Lu, Y. & Harris, M. (1999). Biomechanics of increased exposure to lumbar injury caused by cyclic loading. Part 2. Recovery of reflexive muscular stability with rest. *Spine*, **24,** 2461-7.

Gertzbein, S.D., Holtby, R., Tile, M. & et, a. (1984). Determination of a locus of instantaneous centers of rotation of the lumbar disc by Moire fringes. A new technique. *Spine*, **9,** 409-413.

Gertzbein, S.D., Seligman, J., Holtby, R. & et, a. (1985). Centrode patterns and segmental instability in degenerative disc disease. *Spine*, **10,** 257-261.

Gianturco, C. (1944). A roentgen analysis of the motion of the lower lumbar vertebrae in normal individuals and in patients with low back pain. *Amer J Roentgenol*, **52,** 261.

Gibson, J.N., Waddell, G. & Grant, I.C. (2000). Surgery for degenerative lumbar spondylosis. *Cochrane Database Syst Rev*, CD001352.

Giles, L.G.F. & Singer, K.P. (1997). *Clinical Anatomy and Management of Low Back Pain*. Butterworth-Heinemann: Oxford.

Goel, V.K., Goyal, S., Clark, C. & et, a. (1985). Kinematics of the whole lumbar spine: Effect of discectomy. *Spine*, **10,** 543-564.

Goel, V.K. & Pope, M.H. (1995). Biomechanics of fusion and stabilization. *Spine*, **20,** 85s-99s.

Gordon, S.J., Yang, K.H., Mayer, P.J., Mace, A.H., Kish, V.L. & Radin, E.L. (1991). Mechanism of Disk Rupture - a Preliminary-Report. *Spine*, **16,** 450-456.

Gracovetsky, S. (1988). *The Spinal Engine*. Vol. 16. Orthopaedic Review. Springer-Verlag: Wien.

Gracovetsky, S., Farfan, H. & Helleur, C. (1985). The abdominal mechanism. *Spine*, **10,** 317-24.

Gracovetsky, S., Kary, M., Levy, S., Said, R.B., Pitchen, I. & Helie, J. (1990). Analysis of Spinal and Muscular-Activity During Flexion Extension and Free Lifts. *Spine*, **15,** 1333-1339.

Grant, P.G. (1973). Biomechanical significance of the instantaneous center of rotation: the human temporomandibular joint. *J Biomech*, **6,** 109-13 *LHM: This title held at the Health Services Library - Southampton General Hospital.

Gregersen, G.G. & Lucas, D.B. (1967). An in vivo study of the axial rotation of the human thoracolumbar spine. *J Bone Joint Surg*, **49A,** 247-262.

Grobler, L.J. & Wiltse, L.L. (1997). Classification, and Nonoperative and Operative Treatment of Spondylolisthesis. In *The Adult Spine: Principles and Practice*, Frymoyer, J.W. (ed), Vol. 2. pp. 1865-1883. Lippincott-Raven: Philadelphia.

Gunzburg, R., Hutton, W. & Fraser, R. (1991). Axial rotation of the lumbar spine and the effect of flexion: An in vitro and in vivo biomechanical study. *Spine*, **16,** 22-28.

Gunzburg, R., Hutton, W.C., Crane, G. & Fraser, R.D. (1992). Role of the capsulo-ligamentous structures in rotation and combined flexion-rotation of the lumbar spine. *J Spinal Disord*, **5,** 1-7.

Haher, T.R., Felmy, W., Baruch, H., Devlin, V., Welin, D., Obrien, M., Ahmad, J., Valenza, J. & Parish, S. (1989). The contribution of the three columns of the spine to rotational stability. A biomechanial model. *Spine*, **14,** 663-669.

Haher, T.R., M, O.B., Felmly, W.T., Welin, D., Perrier, G., Choueka, J., Devlin, V., Vassiliou, A. & Chow, G. (1992). Instantaneous axis of rotation as a function of the three columns of the spine. *Spine*, **17,** S149-54.

Haher, T.R., O. Brien, M., Dryer, J.W., Nucci, R., Zipnick, R. & Leone, D.J. (1994). The role of the lumbar facet joints in spinal stability. Identification of alternative paths of loading. *Spine*, **19,** 2667-70 discussion 2671.

Haig, A.J., Weismann, G., Haugh, L.D., Pope, M. & Grobler, L.J. (1993). Prospective Evidence For Change in Paraspinal Muscle-Activity After Herniated Nucleus Pulposus. *Spine*, **18,** 926-930.

Hall, R.E. (1929). An analysis of the work and ideas of investigators and authors of relations and movements of the mandible. *J Amer Dental Association*, **16**.

Hanley, E.N., Jr. (1995). The indications for lumbar spinal fusion with and without instrumentation. *Spine*, **20,** 143s-153s.

Harada, M., Abumi, K., Ito, M. & Kaneda, K. (2000). Cineradiographic motion analysis of normal lumbar spine during forward and backward flexion. *Spine*, **25,** 1932-7.

Harvey, S.B. & Hukins, D.W. (1998). Measurement of lumbar spinal flexion-extension kinematics from lateral radiographs: simulation of the effects of out-of-plane movement and errors in reference point placement. *Medical Engineering and Physics*, **20,** 403-9.

Hayes, M.A., Howard, T.C., Gruel, C.R. & Kopta, J.A. (1989). Roentgenographic Evaluation of Lumbar Spine Flexion-Extension in Asymptomatic Individuals. *Spine*, **14,** 327-331.

Hides, J.A., Cooper, D.H. & Stokes, M.J. (1992). Diagnostic ultrasound imaging for measurement of the lumbar multifidus muscle in normal young adults. *Physiotherapy Theory and Practice*, **8,** 19-26.

Hides, J.A., Richardson, C.A. & Jull, G.A. (1995). Magnetic resonance imaging and ultrasonography of the lumbar multifidus muscle. Comparison of two different modalities. *Spine*, **20,** 54-8.

Hides, J.A., Stokes, M.J., Saide, M., Jull, G.A. & Cooper, D.H. (1994). Evidence of Lumbar Multifidus Muscle Wasting Ipsilateral to Symptoms in Patients With Acute Subacute Low-Back-Pain. *Spine*, **19,** 165-172.

Hindle, R.J., Pearcy, M.J., Cross, A.T. & Miller, D.H.T. (1990). Three-dimensional kinematics of the human back. *Clinical Biomechanics*, **5,** 218-228.

Hirsch, C. & Galante, J. (1967). Laboratory conditions for tensile tests in annulus fibrosus from human intervertebral discs. *Acta Orthop Scand*, **38,** 148-62.

Horst, M. & Brinckmann, P. (1981). Measurement of the distribution of axial stress on the end-plate of the vertebral body. *Spine*, **6,** 217-232.

Hutton, W.C., Cyron, B.M. & Stott, J.R. (1979). The compressive strength of lumbar vertebrae. *Journal of Anatomy*, **129,** 753-8.

ICRP. (1991a). *1990 Recommendations of the International Commission on Radiological Protection.* Annals of the ICRP 21(1-3). Pergamon Press: Oxford.

Indahl, A., Kaigle, A.M., Reikeras, O. & Holm, S.H. (1997). Interaction between the porcine lumbar intervertebral disc, zygapophysial joints, and paraspinal muscles. *Spine*, **22,** 2834-2840.

Ishihara, H. & Urban, J.P. (1999). Effects of low oxygen concentrations and metabolic inhibitors on proteoglycan and protein synthesis rates in the intervertebral disc. *Journal of Orthopaedic Research*, **17,** 829-35.

Jayson, M.I.V. (1992). *The Lumbar Spine and Back Pain.* Churchill Livingstone: Edinburgh.

Johnsson, R., Selvik, G., Stromqvist, B. & Sunden, G. (1990). Mobility of the lower lumbar spine after posterolateral fusion determined by roentgen stereophotogrammetric analysis. *Spine*, **15,** 347-50.

Johnsson, R., Stromqvist, B., Axelsson, P. & Selvik, G. (1992). Influence of spinal immobilization on consolidation of posterolateral lumbosacral fusion. A roentgen stereophotogrammetric and radiographic analysis. *Spine*, **17,** 16-21.

Johnstone, B., Urban, J.P., Roberts, S. & Menage, J. (1992). The fluid content of the human intervertebral disc. Comparison between fluid content and swelling pressure profiles of discs removed at surgery and those taken postmortem. *Spine*, **17,** 412-6.

Jones, M.D. (1960). Cineradiographic studies of the normal cervical spine. *California Medicine*, **93,** 293-296.

Jowett, R.L., Fidler, M.W. & Troop, J.O.G. (1975). Histochemical changes in the multifidus in mechanical derangements of the spine. *Orthop Clin North Amer*, **6,** 145-161.

Junghanns, H. (1931). Die Zwischenwirbelscheiben im Rontegenbild. *Fortschr Roentgenstr*, **43,** 275-305.

Kaigle, A.M., Holm, S.H. & Hansson, T.H. (1995). Experimental Instability in the Lumbar Spine. *Spine*, **20,** 421-430.

Kaigle, A.M., Holm, S.H. & Hansson, T.H. (1997). 1997 Volvo Award winner in biomechanical studies - Kinematic behavior of the porcine lumbar spine: A chronic lesion model. *Spine*, **22,** 2796-2806.

Kaigle, A.M., Magnusson, M., Broman, H. & Hansson, T. (1992a). In vivo measurement of intervertebral creep: a preliminary report. *Clin Biomech (Bristol, Avon)*, **7,** 59-62.

Kaigle, A.M., Pope, M.H., Fleming, B.C. & Hansson, T. (1992b). A Method For the Intravital Measurement of Interspinal Kinematics. *Journal of Biomechanics*, **25,** 451 et seq.

Kaigle, A.M., Wessberg, P. & Hansson, T.H. (1998). Muscular and kinematic behavior of the lumbar spine during flexion- extension. *Journal of Spinal Disorders*, **11,** 163-174.

Kalimo, H., Rantanen, J., Viljanen, T. & Einola, S. (1989). Lumbar Muscles - Structure and Function. *Annals of Medicine*, **21,** 353-359.

Kanayama, M., Abumi, K., Kaneda, K., Tadano, S. & Ukai, T. (1996). Phase lag of the intersegmental motion in flexion-extension of the lumbar and lumbosacral spine. An in vivo study. *Spine*, **21,** 1416-22.

Kanayama, M., Cunningham, B.W., Weis, J.C., Parker, L.M., Kaneda, K. & McAfee, P.C. (1998). The effects of rigid spinal instrumentation and solid bony fusion on spinal kinematics - A posterolateral spinal arthrodesis model. *Spine*, **23,** 767-773.

Katz, J.N. (1995). Lumbar spinal fusion. Surgical rates, costs, and complications. *Spine*, **20,** 78s-83s.

Kauppila, L.I., Eustace, S., Kiel, D.P., Felson, D.T. & Wright, A.M. (1998). Degenerative displacement of lumbar vertebrae. A 25-year follow-up study in Framingham. *Spine*, **23,** 1868-73; discussion 1873-4.

Keller, T.S. & Nathan, M. (1999). Height change caused by creep in intervertebral discs: a sagittal plane model. *Journal of Spinal Disorders*, **12,** 313-24.

Kirkaldy-Willis, W.H. (1992). Pathology and pathogenesis of low back pain. In *Managing Low Back Pain*, Kirkaldy-Willis, W.H. & Burton, C.V. (eds) pp. 49-79. Churchill Livingstone: New York.

Kirkaldy-Willis, W.H. & Farfan, H.F. (1982). Instability of the Lumbar Spine. *Clinical Orthopaedics and Related Research*, 110-123.

Knutsson, F. (1944). The instability associated with disc degeneration in the lumbar spine. *Acta Radiol*, **24,** 593-609.

Kondracki, M., Breen, A. & Allen, R. (1992). The use of digitised videofluoroscopic images in the analysis of spinal mechanics in-vivo. In *VI Mediterranean Conference on Medical and Biological Engineering*, Bracale, M. & Denoth, F. (eds), Vol. 1. pp. 703-706. Area Di Ricerca - CNR - Pisa: Capri.

Kondracki, M. & Breen, A.C. (1993). Indices of regularity and laxity in the motion between lumbar vertebrae in the sagittal plane. In *Society for Back Pain Research*: London.

Kondracki, M.P. (1991). Sources of error in the estimation of instantaneous axes of rotation in spinal joints. In *Department of Bioengineering* pp. 60. University of Strathclyde: Glasgow.

Kotilainen, E., Heinanen, J., Gullichsen, E., Koivunen, T. & Aro, H.T. (1997). Spondylodesis in the treatment of segmental instability of the lumbar spine with special reference to clinically verified instability. *Acta Neurochirurgica*, **139,** 629-635.

Krismer, M., Haid, C., Ogon, M., Behensky, H. & Wimmer, C. (1997). Biomechanics of lumbar instability. *Orthopade*, **26,** 516-520.

Krodel, A. (1996). Mechanical principles of compressive interbody fusion. *Spine*, **21,** 821-6.

Kumar, S. & Panjabi, M.M. (1995). In vivo axial rotations and neutral zones of the thoracolumbar spine. *Journal of Spinal Disorders*, **8,** 253-63.

Lavender, S.A., Marras, W.S. & Miller, R.A. (1993). The Development of Response Strategies in Preparation For Sudden Loading to the Torso. *Spine*, **18,** 2097-2105.

Lee, Y.-H., Chiou, W.-K., Chen, W.-J., Lee, M.-Y. & Lin, Y.-H. (1995). Predictive model of intersegmental mobility of lumbar spine in the sagittal plane from skin markers. *Clinical Biomechanics*, **10,** 413-420.

Leivseth, G., Brinckmann, P., Frobin, W., Johnsson, R. & Stromqvist, B. (1998). Assessment of sagittal plane segmental motion in the lumbar spine. A comparison between distortion-compensated and stereophotogrammetric roentgen analysis. *Spine*, **23,** 2648-55.

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

Leong, J.C., Luk, K.D., Chow, D.H. & Woo, C.W. (1987). The biomechanical functions of the iliolumbar ligament in maintaining stability of the lumbosacral junction. *Spine*, **12,** 669-74.

Lewin, T., Moffett, B. & Viidik, A. (1962). The Morphology of the Lumbar Synovial Intervertebral Joints. *Acta Morph Neer Scand*, **4,** 299-419.

Lewis, T.T. (1991). Radiological assessment of prolapsed disc [see comments]. *Br J Hosp Med*, **46,** 48-51.

Libson, E., Bloom, R.A. & Dinari, G. (1982). Symptomatic and asymptomatic spondylolysis and spondylolisthesis in young adults. *International Orthopaedics*, **6,** 259-61.

Lin, R.M., Yu, C.Y., Chang, Z.J., Lee, C.C. & Su, F.C. (1994). Flexion-extension rhythm in the lumbosacral spine. *Spine*, **19,** 2204-9.

Liu, X.C., Fabry, G., Labey, L., Van den Berghe, L., Van Audekercke, R., Molenaers, G. & Moens, P. (1997). A new technique for the three-dimensional study of the spine in vitro and in vivo by using a motion-analysis system. *Journal of Spinal Disorders*, **10,** 329-38.

Lovett, R.W. (1905). The mechanism of the normal spine and its relation to scoliosis. *Boston Medical and Surgical Journal*, **153,** 349-358.

Lu, W.W., Luk, K.D., Ruan, D.K., Fei, Z.Q. & Leong, J.C. (1999). Stability of the whole lumbar spine after multilevel fenestration and discectomy. *Spine*, **24,** 1277-82.

Lucas, D.B. & Bresler, B. (1961). Stability of the ligamentous spine. Biomechanics Lab University of California: San Francisco.

Lumsden, R.M. & Morris, J.M. (1968). An in vivo study of axial rotation and immobilization at the lumbosacral joint. *J Bone Joint Surg*, **50A,** 1591-1602.

Lysack, J.T., Dickey, J.P., Dumas, G.A. & Yen, D. (2000). A continuous pure moment loading apparatus for biomechanical testing of multi-segment spine specimens. *Journal of Biomechanics*, **33,** 765-70.

Lysell, E. (1969). Motion in the cervical spine. An experimental study on autopsy specimens. *Acta Orthop Scand*, **123**.

Macintosh, J.E. & Bogduk, N. (1986). The Biomechanics of the Lumbar Multifidus. *Clinical Biomechanics*, **1,** 205-213.

Macintosh, J.E. & Bogduk, N. (1991). The Attachments of the Lumbar Erector Spinae. *Spine*, **16,** 783-792.

Macintosh, J.E., Valencia, F., Bogduk, N. & Munro, R.R. (1986). The Morphology of the Human Lumbar Multifidus. *Clinical Biomechanics*, **1,** 196-204.

Macrae, I.F. & Wright, V. (1969). Measurement of back movement. *Ann Rheum Dis*, **28,** 584-9 *LHM: This title held at the Health Services Library - Southampton General Hospital.

Magora, A. & Schwartz, A. (1976). Relation between the low back pain syndrome and X-ray findings-1. degenerative osteoarthritis. *Scand J Rehabil Med*, **8,** 115-125.

Malter, A.D., McNeney, B., Loeser, J.D. & Deyo, R.A. (1998). 5-year reoperation rates after different types of lumbar spine surgery. *Spine*, **23,** 814-20.

Markolf, K.L. & Morris, J.M. (1974). The structural components of the intervertebral disc. A study of their contributions to the ability of the disc to withstand compressive forces. *J Bone Joint Surg Am*, **56,** 675-87.

Maxwell, R.L. (1960). *Kinematics and dynamics of machinery.* Vol. 1. Prentice-Hall: London.

McCollough, C.H. & Schueler, B.A. (2000). Calculation of effective dose. *Medical Physics*, **27,** 828-37.

McGill, S.M. & Brown, S. (1992). Creep response of the lumbar spine to prolonged full flexion. *Clinical Biomechanics*, **7,** 43-46.

McGill, S.M., Hughson, R.L. & Parks, K. (2000). Changes in lumbar lordosis modify the role of the extensor muscles. *Clinical Biomechanics*, **15,** 777-780.

McGill, S.M. & Kippers, V. (1994). Transfer of Loads Between Lumbar Tissues During the Flexion- Relaxation Phenomenon. *Spine*, **19,** 2190-2196.

Meek, R.N., Martens, M. & Demets, D. (1975). Correlation of instant center displacement with internal derangements of the knee. In *Annual Meeting of the Canadian Orthopaedic Association.*

Mehta, M.H. (1973). Radiographic estimation of vertebral rotation in scoliosis. *J Bone Joint Surg Br*, **55,** 513-20.

Moffett, J.A.K. & Richardson, G. (1995). Costs and effectiveness: Approaches to the management of back pain. In *Lumbar spine disorders: Current concepts*, Aspden, R.M. & Porter, R.W. (eds), Vol. 1. pp. 225-235. World Scientific: Singapore.

Moll, J.M.H. & Wright, V. (1971). Normal range of spinal mobility. *Ann Rheum Dis*, **30,** 381-386.

Moores, B.M. (1987). Digital X-ray imaging. *Proceedings of the Institution of Electrical Engineers*, **134A,** 115-125.

Morgan, F.P. & King, T. (1957). Primary instability of lumbar vertebrae as a common cause of low back pain. *J Bone Joint Surg*, **39B,** 6-22.

Moroney, S.P., Schultz, A.B., Miller, J.A.A. & Andersson, G.B.J. (1988). Load-displacement properties of lower cervical spine motion segments. *Journal of Biomechanics*, **21,** 769-779.

Morrey, B.F. & Chao, E.Y. (1976). Passive motion of the elbow joint. *J Bone Joint Surg Am*, **58,** 501-8.

Muggleton, J.M., Kondracki, M. & Allen, R. (2000). Spinal fusion for lumbar instability: does it have a scientific basis? *J Spinal Disord*, **13,** 200-4.

Mullholland, R.C. (1999). Clinical definition of instability. In *Lumbar Segmental Instability*, Szpalski, M., Gunzburg, R. & Pope, M.H. (eds) pp. 55-61. Lippincott Williams and Wilkins: Philadelphia.

Murata, M., Morio, Y. & Kuranobu, K. (1994). Lumbar Disc Degeneration and Segmental Instability - a Comparison of Magnetic-Resonance Images and Plain Radiographs of Patients With Low- Back-Pain. *Archives of Orthopaedic and Trauma Surgery*, **113,** 297-301.

Murtagh, F.R., Paulsen, R.D. & Rechtine, G.R. (1991). The role and incidence of facet tropism in lumbar spine degenerative disc disease. *J Spinal Disord*, **4,** 86-9.

Nachemson, A. (1981a). The role of spine fusion: Question 8. *Spine*, **6,** 306-307.

Nachemson, A. (1985). Lumbar spine instability: A critical update and symposium summary. *Spine*, **10,** 290-291.

Nachemson, A.L. (1963). The influence of spinal movement on the lumbar intradiscal pressure and on the tensile stresses in the annulus fibrosus. *Acta Orthop Scand*, **33,** 183-207.

Nachemson, A.L. (1981b). Disc pressure measurements. *Spine*, **6,** 93-97.

Nachemson, A.L. (1999). Scientific Diagnosis or Unproved Label for Back Pain Patients? In *Lumbar Segmental Instability*, Szpalski, M., Gunzburg, R. & Pope, M.H. (eds) pp. 297-301. Lippincott Williams and Wilkins: Philadelphia.

Nachemson, A.L., Andersson, B.J. & Schultz, A.B. (1986). Valsalva maneuver biomechanics. Effects on lumbar trunk loads of elevated intraabdominal pressures. *Spine*, **11,** 476-9.

Nachemson, A.L., Schultz, A.B. & Berkson, M.H. (1979). Mechanical properties of human lumbar spine motion segments: influences of age, sex, disc level and degeneration. *Spine*, **4,** 1-8.

Neumann, P., Nordwall, A. & Osvalder, A.L. (1995). Traumatic Instability of the Lumbar Spine - a Dynamic in-Vitro Study of Flexion - Distraction Injury. *Spine*, **20,** 1111-1121.

Nordstrom, D., Santavirta, S., Seitsalo, S., Hukkanen, M., Polak, J.M., Nordsletten, L. & Konttinen, Y.T. (1994). Symptomatic lumbar spondylolysis. Neuroimmunologic studies. *Spine*, **19,** 2752-8.

Ogon, M., Bender, B.R., Hooper, D.M., Spratt, K.F., Goel, V.K., Wilder, D.G. & Pope, M.H. (1997a). A dynamic approach to spinal instability - Part I: Sensitization of intersegmental motion profiles to motion direction and load condition by instability. *Spine*, **22,** 2841-2858.

Ogon, M., Bender, B.R., Hooper, D.M., Spratt, K.F., Goel, V.K., Wilder, D.G. & Pope, M.H. (1997b). A dynamic approach to spinal instability - Part II: Hesitation and giving-way during interspinal motion. *Spine*, **22,** 2859-2866.

Ogston, N.G., King, G.J., Gertzbein, S.D. & et, a. (1986). Centrode patterns in the lumbar spine: Baseline studies in normal subjects. *Spine*, **11,** 591-595.

Okawa, A., Shinomiya, K., Komori, H., Muneta, T., Arai, Y. & Nakai, O. (1998). Dynamic motion study of the whole lumbar spine by videofluoroscopy. *Spine*, **23,** 1743-9.

Oliver, M.J. & Twomey, L.T. (1995). Extension creep in the lumbar spine. *Clin Biomech (Bristol, Avon)*, **10,** 363-368.

Olmarker, K. & Myers, R.R. (1998). Pathogenesis of sciatic pain: role of herniated nucleus pulposus and deformation of spinal nerve root and dorsal root ganglion. *Pain*, **78,** 99-105.

O'Sullivan, P.B. (2000). Lumbar segmental 'instability': clinical presentation and specific stabilizing exercise management. *Man Ther*, **5,** 2-12.

O'Sullivan, P.B., Phyty, G.D., Twomey, L.T. & Allison, G.T. (1997). Evaluation of specific stabilizing exercise in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. *Spine*, **22,** 2959-67.

Oxland, T.R., Crisco, J.J., Panjabi, M.M. & Yamamoto, I. (1992). The effect of injury on rotational coupling at the lumbosacral joint: a biomechanical investigation. *Spine*, **17,** 74-80.

Panjabi, M., Chang, D. & Dvorak, J. (1992). An analysis of errors in kinematic parameters associated with in vivo functional radiographs. *Spine*, **17,** 200-5.

Panjabi, M. & White, A.A. (1971). A mathematical approach for three dimensional analysis of mechanics of the spine. *J Biomech*, **4,** 203-211.

Panjabi, M., Yamamoto, I., Oxland, T. & Crisco, J. (1989). How does posture affect coupling in the lumbar spine? *Spine*, **14,** 1002-1011.

Panjabi, M.M. (1973). Three-dimensional mathematical model of the human spine structure. *J Biomech*, **6,** 671-80.

Panjabi, M.M. (1979). Centers and angles of rotation of body joints: a study of errors and optimization. *J Biomech*, **12,** 911-20.

Panjabi, M.M. (1992a). The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. *J Spinal Disord*, **5,** 383-9.

Panjabi, M.M. (1992b). The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis. *J Spinal Disord*, **5,** 390-6.

Panjabi, M.M. (1994). Lumbar Spine Instability: A Biochemical Challenge. *Current Orthopaedics*, **8,** 100-105.

Panjabi, M.M., Brand, R.A., Jr. & White, A.A.d. (1976). Mechanical properties of the human thoracic spine as shown by three-dimensional load-displacement curves. *J Bone Joint Surg Am*, **58,** 642-52.

Panjabi, M.M., Krag, M.H. & Goel, V.K. (1981). A technique for measurement and description of three-dimensional six degree-of-freedom motion of a body joint with an application to the human spine. *Journal of Biomechanics*, **14,** 447-460.

Panjabi, M.M., Nibu, K. & Cholewicki, J. (1998). Whiplash injuries and the potential for mechanical instability. *European Spine Journal*, **7,** 484-92.

Panjabi, M.M., Summers, D.J., Pelker, R.R. & et, a. (1986). Three-dimensional load-displacement curves due to forces on the cervical spine. *Journal of Orthopaedic Research*, **4,** 152-161.

Panjabi, M.M., White, A.A.d. & Johnson, R.M. (1975). Cervical spine mechanics as a function of transection of components. *J Biomech*, **8,** 327-36.

Papp, T., Porter, R.W., Aspden, R.M. & Shepperd, J.A.N. (1997). An in vitro study of the biomechanical effects of flexible stabilization on the lumbar spine. *Spine*, **22,** 151-155.

Parnianpour, M., Nordin, M., Kahanovitz, N. & Frankel, V. (1988). 1988 Volvo award in biomechanics. The triaxial coupling of torque generation of trunk muscles during isometric exertions and the effect of fatiguing isoinertial movements on the motor output and movement patterns. *Spine*, **13,** 982-92.

Patwardhan, A.G., Havey, R.M., Meade, K.P., Lee, B. & Dunlap, B. (1999). A follower load increases the load-carrying capacity of the lumbar spine in compression. *Spine*, **24,** 1003-9.

Pearcy, M. (1986). Measurement of back and spinal mobility. *Clinical Biomechanics*, **1,** 44-51.

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

Pearcy, M., Portek, I. & Shepherd, J. (1985). The effect of low-back pain on lumbar spinal movements measured by three-dimensional X-ray analysis. *Spine*, **10,** 150-153.

Pearcy, M. & Shepherd, J. (1985). Is there instability in spondylolisthesis? *Spine*, **10,** 175-7.

Pearcy, M.J. (1985). Stereo radiography of lumbar spine motion. *Acta Orthopaedica Scandinavica*, **56,** 45 p.

Pearcy, M.J. (1993). Twisting mobility of the human back in flexed postures. *Spine*, **18,** 114-9.

Pearcy, M.J. & Bogduk, N. (1988). Instantaneous axes of rotation of the lumbar intervertebral joints. *Spine*, **13,** 1033-1041.

Pearcy, M.J. & Hindle, R.J. (1989). New method for the non-invasive three-dimensional measurement of human back movement. *Clinical Biomechanics*, **4,** 73-79.

Pearcy, M.J. & Hindle, R.J. (1991). Axial rotation of lumbar intervertebral joints in forward flexion. *Proc Inst Mech Eng H*, **205,** 205-9.

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

Pennal, G.F., Conn, G.S., McDonald, G., Dale, G. & Garside, H. (1972). Motion studies of the lumbar spine: a preliminary report. *J Bone Joint Surg Br*, **54,** 442-52.

Penning, L. (1978). Normal movements of the cervical spine. *AJR Am J Roentgenol*, **130,** 317-26.

Penning, L. & Blickman, J.R. (1980). Instability in lumbar spondylolisthesis: a radiologic study of several concepts. *Ajr. American Journal of Roentgenology*, **134,** 293-301.

Penning, L., Wilmink, J.T. & Van Woerden, H.H. (1984). Inability to prove instability. A critical appraisal of clinical- radiological flexion-extension studies in lumbar disc degeneration. *Diagnostic Imaging in Clinical Medicine*, **53,** 186-192.

Pitkanen, M., Manninen, H.I., Lindgren, K.A., Turunen, M. & Airaksinen, O. (1997). Limited usefulness of traction-compression films in the radiographic diagnosis of lumbar spinal instability - Comparison with flexion- extension films. *Spine*, **22,** 193-197.

Pittman, P., Colbaugh, R., Glass, K. & Rowen, B. (1992). An approach to determining the kinematic parameters of biomechanical systems with applications to the wrist. *Proc Inst Mech Eng H*, **206,** 213-23.

Plamondon, A., Gagnon, M. & Maurais, G. (1988). Application of a stereoradiographic method for the study of intervertebral motion. *Spine*, **13,** 1027-1032.

Pope, M.H. & Panjabi, M. (1985). Biomechanical Definitions of Spinal Instability. *Spine*, **10,** 255-256.

Pope, M.H., Svensson, M., Broman, H. & Andersson, G.B. (1986). Mounting of the transducers in measurement of segmental motion of the spine. *Journal of Biomechanics*, **19,** 675-7.

Pope, M.H., Wilder, D.G., Matteri, R.E. & Frymoyer, J.W. (1977). Experimental measurements of vertebral motion under load. *Orthop Clin North Am*, **8,** 155-67.

Portek, I., Pearcy, M.J., Reader, G.P. & Mowat, A.G. (1983). Correlation between radiographic and clinical measurement of lumbar spine movement. *British Journal of Rheumatology*, **22,** 197-205.

Porter, R.W. (1986). *Management of Back Pain.* Churchill Livingstone: Edinburgh.

Porter, R.W. (1989). Mechanical Disorders of the Lumbar Spine. *Ann Med*, **21,** 361-366.

Posner, I., White Aa, III, Edwards, W.T. & Hayes, W.C. (1982). A biomechanical analysis of the clinical stability of the lumbar and lumbosacral spine. *Spine*, **7,** 374-389.

Potvin, J.R. & O'Brien, P.R. (1998). Trunk muscle co-contraction increases during fatiguing, isometric, lateral bend exertions - Possible implications for spine stability. *Spine*, **23,** 774-780.

Putto, E. & Tallroth, K. (1990). Extension-Flexion Radiographs For Motion Studies of the Lumbar Spine a Comparison of 2 Methods. *Spine*, **15,** 107-110.

Quinnell, R.C. & Stockdale, H.R. (1982). The significance of osteophytes on lumbar vertebral bodies in relation to discographic findings. *Clin Radiol*, **33,** 197-203.

Quinnell, R.C. & Stockdale, H.R. (1983). Flexion and extension radiography of the lumbar spine: A comparison with lumbar discography. *Clinical Radiology*, **34,** 405-411.

Quint, U., Wilke, H.J., ShiraziAdl, A., Parnianpour, M., Loer, F. & Claes, L.E. (1998). Importance of the intersegmental trunk muscles for the stability of the lumbar spine - A biomechanical study in vitro. *Spine*, **23,** 1937-1945.

Rahm, M.D. & Hall, B.B. (1996). Adjacent-segment degeneration after lumbar fusion with instrumentation: A retrospective study. *Journal of Spinal Disorders*, **9,** 392-400.

Ransom, N., La Rocca, S.H. & Thalgott, J. (1994). The case for pedicle fixation of the lumbar spine. *Spine*, **19,** 2702-6.

Reuleaux, F. (1876). *Kinematics of machinery: outline of a theory of machines.* MacMillan: London.

Rhalmi, S., Yahia, L., Newman, N. & Isler, M. (1993). Immunohistochemical Study of Nerves in Lumbar Spine Ligaments. *Spine*, **18,** 264-267.

Roberts, S., Bains, M.A., Kwan, A., Menage, J. & Eisenstein, S.M. (1998). Type X collagen in the human invertebral disc: an indication of repair or remodelling? *Histochemical Journal*, **30,** 89-95.

Roberts, S., Eisenstein, S.M., Menage, J., Evans, E.H. & Ashton, I.K. (1995). Mechanoreceptors in intervertebral discs. Morphology, distribution, and neuropeptides [see comments]. *Spine*, **20,** 2645-51.

Rolander, S.D. (1966). Motion of the lumbar spine with special reference to the stabilizing effect of posterior fusion. An experimental study on autopsy specimens. *Acta Orthop Scand*, **90,** 1-144.

Rosenberg, P. (1955). The R-Center method: a new method for analyzing vertebral motion by X-rays. *J American Osteopathic Association*, **55,** 103-111.

Schober, P. (1937). Lendenwirbelsaule und Kreuzschmerzen. *Munch Med Wschr*, **84,** 336.

Scholten, P.J.M., Veldhuizen, A.G. & Grootenboer, H.J. (1988). Stability of the Human Spine - a Biomechanical Study. *Clinical Biomechanics*, **3,** 27-33.

Schubert, R. (1995). What is dose area product? *Radiologic Technology*, **66,** 329-30.

Schultz, A.B., Warwick, D.N., Berkson, M.H. & Nachemson, A.L. (1979). Mechanical properties of human lumbar spine motion segments: Part 1. Responses in flexion, extension, lateral bending and torsion. *J Biomech Eng*, **101,** 46-52.

Sedlin, E.D. & Hirsch, C. (1966). Factors affecting the determination of the physical properties of femoral cortical bone. *Acta Orthop Scand*, **37,** 29-48.

Seligman, J.V., Gertzbein, S.D., Tile, M. & Kapasouri, A. (1984). Computer analysis of spinal segment motion in degenerative disc disease with and without axial loading. *Spine*, **9,** 566-573.

Selvik, G. (1989). Roentgen stereophotogrammetry. A method for the study of the kinematics of the skeletal system. *Acta Orthopaedica Scandinavica. Supplementum*, **232,** 1-51.

Selvik, G. (1990). Roentgen stereophotogrammetric analysis. *Acta Radiologica*, **31,** 113-26.

Shaffer, W.O., Spratt, K.F., Weinstein, J., Lehmann, T.R. & Goel, V. (1990). 1990 Volvo Award in clinical sciences. The consistency and accuracy of roentgenograms for measuring sagittal translation in the lumbar vertebral motion segment. An experimental model. *Spine*, **15,** 741-50.

Sharma, M., Langrana, N.A. & Rodriguez, J. (1995). Role of Ligaments and Facets in Lumbar Spinal Stability. *Spine*, **20,** 887-900.

Shirazi-Adl, A. & Parnianpour, M. (1999). Effect of changes in lordosis on mechanics of the lumbar spine-lumbar curvature in lifting. *Journal of Spinal Disorders*, **12,** 436-47.

Shono, Y., Kaneda, K., Abumi, K., McAfee, P.C. & Cunningham, B.W. (1998). Stability of posterior spinal instrumentation and its effects on adjacent motion segments in the lumbosacral spine. *Spine*, **23,** 1550-1558.

Shrimpton, P.C., Wall, B.F. & Hart, D. (1999). Diagnostic medical exposures in the U.K. *Applied Radiation and Isotopes*, **50,** 261-9.

Sicard, C. & Gagnon, M. (1993). A geometric model of the lumbar spine in the sagittal plane. *Spine*, **18,** 646-58.

Sihvonen, T., Partanen, J., Hanninen, O. & Soimakallio, S. (1991). Electric Behavior of Low-Back Muscles During Lumbar Pelvic Rhythm in Low-Back-Pain Patients and Healthy Controls. *Archives of Physical Medicine and Rehabilitation*, **72,** 1080-1087.

Skouen, J.S., Larsen, J.L. & Vollset, S.E. (1993). Cerebrospinal fluid proteins as indicators of nerve root compression in patients with sciatica caused by disc herniation. *Spine*, **18,** 72-9.

Snyder-Mackler, L., Fitzgerald, G.K., Bartolozzi, A.R. & Ciccotti, M.G. (1997). The relationship between passive joint laxity and functional outcome after anterior cruciate ligament injury. *American Journal of Sports Medicine*, **25,** 191-195.

Soini, J., Antti-Poika, I., Tallroth, K., Konttinen, Y.T., Honkanen, V. & Santavirta, S. (1991). Disc degeneration and angular movement of the lumbar spine: comparative study using plain and flexion-extension radiography and discography. *Journal of Spinal Disorders*, **4,** 183-7.

Solomonow, M., He Zhou, B., Baratta, R.V., Lu, Y., Zhu, M. & Harris, M. (2000). Biexponential recovery model of lumbar viscoelastic laxity and reflexive muscular activity after prolonged cyclic loading. *Clin Biomech (Bristol, Avon)*, **15,** 167-75.

Solomonow, M., Zhou, B.H., Baratta, R.V., Lu, Y. & Harris, M. (1999). Biomechanics of increased exposure to lumbar injury caused by cyclic loading: Part 1. Loss of reflexive muscular stabilization. *Spine*, **24,** 2426-34.

Solomonow, M., Zhou, B.H., Harris, M., Lu, Y. & Baratta, R.V. (1998). The ligamento-muscular stabilizing system of the spine. *Spine*, **23,** 2552-2562.

Sonntag, V.K. & Marciano, F.F. (1995). Is fusion indicated for lumbar spinal disorders? *Spine*, **20,** 138s-142s.

Soudan, K., Van Audekercke, R. & Martens, M. (1979). Methods, difficulties and inaccuracies in the study of human joint kinematics and pathokinematics by the instant axis concept. Example: the knee joint. *J Biomech*, **12,** 27-33.

Spiegelman, J.J. & Woo, S.L. (1987). A rigid-body method for finding centers of rotation and angular displacements of planar joint motion. *J Biomech*, **20,** 715-21.

Steffen, T., Rubin, R.K., Baramki, H.G., Antoniou, J., Marchesi, D. & Aebi, M. (1997). A new technique for measuring lumbar segmental motion in vivo. Method, accuracy, and preliminary results. *Spine*, **22,** 156-66.

Stokes, I.A., Wilder, D.G., Frymoyer, J.W. & Pope, M.H. (1981). 1980 Volvo award in clinical sciences. Assessment of patients with low-back pain by biplanar radiographic measurement of intervertebral motion. *Spine*, **6,** 233-40.

Stokes, I.A.F. & Frymoyer, J.W. (1987). Segmental motion and instability. *Spine*, **12,** 688-691.

Stokes, I.A.F. & Gardnermorse, M. (1995). Lumbar Spine Maximum Efforts and Muscle Recruitment Patterns Predicted By a Model With Multijoint Muscles and Joints With Stiffness. *Journal of Biomechanics*, **28,** 173-186.

Stokes, M., Cooper, R. & Jayson, M. (1992). Selective changes in multifidus dimensions in patients with chronic low back pain. *Eur Spine J*, **1,** 38-42.

Stokes, M. & Young, A. (1984). The Contribution of Reflex Inhibition to Arthrogenous Muscle Weakness. *Clinical Science*, **67,** 7-14.

Szpalski, M. (1996). The Mysteries of Segmental Instability. *Bull Hosp Joint Diseases*, **55,** 147-148.

Tallroth, K., Alaranta, H. & Soukka, A. (1992). Lumbar mobility in asymptomatic individuals. *J Spinal Disord*, **5,** 481-4.

Tanz, S.S. (1953). Motion of the lumbar spine. A roentgenologic study. *Amer J Roentgenol*, **69,** 399-412.

Tao, D.C. (1967). *Fundamentals of applied kinematics*. Vol. 1. Addison-Wesley: Reading, Massachusetts.

Tekeoglu, I., Adak, B., Bozkurt, M. & Gurbuzoglu, N. (1998). Distraction of lumbar vertebrae in gravitational traction. *Spine*, **23,** 1061-1063.

Tencer, A.F., Ahmed, A.M. & Burke, D.L. (1982). Some static mechanical properties of the lumbar intervertebral joint, intact and injured. *J Biomech Eng*, **104,** 193-201.

Tesh, K.M., Dunn, J.S. & Evans, J.H. (1987). The abdominal muscles and vertebral stability. *Spine*, **12,** 501-8.

Teves, M.C. (1955). The application of the X-ray image intensifiers I-IV. *Philips Tech 1955/6 Rev*, **17,** 69-97.

Tibrewal, S.B., Pearcy, M.J., Portek, I. & et, a. (1985). A prospective study of lumbar spinal movements before and after discectomy using biplanar radiography. Correlation of clinical and radiographic findings. *Spine*, **10,** 455-460.

Tkaczuk, H. (1968). Tensile properties of human lumbar longitudinal ligaments. *Acta Orthop Scand*, **115,** 1-68.

Todd, T.W. & Pyle, I.S. (1928). A quantitative study of the vertebral column by direct roentgenologic methods. *Amer J Phys Anthr*, **12,** 321.

Toussaint, H.M., Dewinter, A.F., Delooze, M.P., Vandieen, J.H., Kingma, I. & Dehaas, Y. (1995). Flexion Relaxation During Lifting - Implications For Torque Production By Muscle-Activity and Tissue Strain At the Lumbosacral Joint. *Journal of Biomechanics*, **28,** 199-210.

Triano, J.J. & Schultz, A.B. (1987). Correlation of Objective-Measure of Trunk Motion and Muscle Function With Low-Back Disability Ratings. *Spine*, **12,** 561-565.

Tsantrizos, A., Baramki, H.G., Zeidman, S. & Steffen, T. (2000). Segmental stability and compressive strength of posterior lumbar interbody fusion implants. *Spine*, **25,** 1899-907.

van Akkerveeken, P.F. (1999). Is Lateral Stenosis a Feature of Lumbar Segmental Instability? In *Lumbar Segmental Instability*, Szpalski, M., Gunzburg, R. & Pope, M.H. (eds) pp. 95-101. Lippincott Williams and Wilkins: Philadelphia.

van Akkerveeken, P.F., O. Brien, J.P. & Park, W.M. (1979). Experimentally induced hypermobility in the lumbar spine. A pathologic and radiologic study of the posterior ligament and annulus fibrosus. *Spine*, **4,** 236-41.

van Mameren, H., Sanches, H., Beursgens, J. & Drukker, J. (1992). Cervical spine motion in the sagittal plane. II. Position of segmental averaged instantaneous centers of rotation--a cineradiographic study. *Spine*, **17,** 467-74.

Vitzthum, H.E., Konig, A. & Seifert, V. (2000). Dynamic examination of the lumbar spine by using vertical, open magnetic resonance imaging. *Journal of Neurosurgery*, **93,** 58-64.

von Lackum, H.L. (1924). The Lumbosacral Region: An Anatomic Study and Some Clinical Observations. *J Amer Med Assoc*, **82,** 1109-1114.

Waddell, G. (1998). *The Back Pain Revolution.* Churchill Livingstone: Edinburgh.

Wall, B.F. & Hart, D. (1997). Revised radiation doses for typical X-ray examinations. Report on a recent review of doses to patients from medical X-ray examinations in the UK by NRPB. National Radiological Protection Board. *British Journal of Radiology*, **70,** 437-9.

Walsh, M. & Breen, A.C. (1995). Reliability and validity of the Metrecom Skeletal Analysis System in the assessment of sagittal plane lumbar angles. *Clinical Biomechanics*, **10,** 222-223.

Warren-Forward, H.M., Haddaway, M.J., Temperton, D.H. & McCall, I.W. (1998). Dose-area product readings for fluoroscopic and plain film examinations, including an analysis of the source of variation for barium enema examinations. *British Journal of Radiology*, **71,** 961-7.

Weber, W. & Weber, E.H. (1836). *Mechanik der menschlichen gewerkzeuge*. Dieterich: Gottingen.

Weiler, P.J., King, G.J. & Gertzbein, S.D. (1990). Analysis of sagittal plane instability of the lumbar spine in vivo. *Spine*, **15,** 1300-6.

Weitz, E.M. (1981). The lateral bending sign. *Spine*, **6,** 388-397.

White, A.A. & Bernhardt, M. (1999). Clinical biomechanics and lumbar spine instability. In *Lumbar Segmental Instability*, Szpalski, M., Gunzburg, R. & Pope, M.H. (eds) pp. 15-25. Lippincott Williams and Wilkins: Philadelphia.

White, A.A., Johnson, R.M., Panjabi, M.M. & Southwick, W.O. (1975). Biomechanical analysis of clinical stability in the cervical spine. *Clin Orthop*, **109,** 85-96.

White, A.A. & Panjabi, M.M. (1978). The basic kinematics of the human spine. A review of past and current knowledge. *Spine*, **3,** 12-20.

White, A.A. & Panjabi, M.M. (1990). *Clinical Biomechanics of the Spine*. J.B. Lippincott: Philidelphia.

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

Wilke, H.J., Wolf, S., Claes, L.E., Arand, M. & Wiesend, A. (1995). Stability Increase of the Lumbar Spine With Different Muscle Groups - a Biomechanical in-Vitro Study. *Spine*, **20,** 192-198.

Williams, M., Solomonow, M., Zhou, B.H., Baratta, R.V. & Harris, M. (2000). Multifidus spasms elicited by prolonged lumbar flexion. *Spine*, **25,** 2916-2924.

Wiltse, L.L., Newman, P.H. & Macnab, I. (1976). Classification of spondylolisis and spondylolisthesis. *Clinical Orthopaedics and Related Research*, 23-9.

Woltring, H., De Lange, A., Kauer, J. & Huiskes, R. (1986). Instantaneous helical axis estimation via natural, cross-validated splines. In *Biomechanics: Basic and Applied Research* pp. 121-128. Martinus Nijhoff.

Woltring, H.J., Huiskes, R., de Lange, A. & Veldpaus, F.E. (1985). Finite centroid and helical axis estimation from noisy landmark measurements in the study of human joint kinematics. *J Biomech*, **18,** 379-89.

Woo, S.-Y., Camp, J., Gomez, M.A. & Akeson, W.H. (1983). Comparison of the biomechanical properties of fresh and frozen ligaments. In *Advances in Bioengineering*. American Society of Mechanical Engineers: Boston.

Woo, S.-Y., Orlando, C.A., Suto, S., Camp, J.F., Gomez, M.S. & Akeson, W.H. (1984). The effects of aging, temperature, and post mortem storage on ligament tensile behaviour. In *1984 Advances in Bioengineering*, Spilker, R.L. (ed). American Society of Mechanical Engineers: New York.

Wood, K.B., Popp, C.A., Transfeldt, E.E. & Geissele, A.E. (1994). Radiographic evaluation of instability in spondylolisthesis. *Spine*, **19,** 1697-703.

Yahia, L.H. & Newman, N. (1991). Innervation of Spinal Ligaments of Patients With Disk Herniation - an Immunohistochemical Study. *Pathology Research and Practice*, **187,** 936-938.

Yahia, L.H., Newman, N. & Rivard, C.H. (1988). Neurohistology of Lumbar Spine Ligaments. *Acta Orthopaedica Scandinavica*, **59,** 508-512.

Yamamoto, I., Panjabi, M.M., Crisco, T. & Oxland, T. (1989). Three-dimensional movements of the whole lumbar spine and lumbosacral joint. *Spine*, **14,** 1256-60.

Yochum, T.R. & Rowe, L.J. (1996). *Essentials of Skeletal Radiology*. Vol. 1. Williams & Wilkins: Baltimore.

Yoshioka, T., Tsuji, H., Hirano, N. & Sainoh, S. (1990). Motion characteristic of the normal lumbar spine in young adults: instantaneous axis of rotation and vertebral center motion analyses. *Journal of Spinal Disorders*, **3,** 103-13.

Young, A., Stokes, M. & Crowe, M. (1984). Size and Strength of the Quadriceps Muscles of Old and Young-Women. *European Journal of Clinical Investigation*, **14,** 282-287.

Zhao, W.P., Kawaguchi, Y., Matsui, H., Kanamori, M. & Kimura, T. (2000). Histochemistry and morphology of the multifidus muscle in lumbar disc herniation: comparative study between diseased and normal sides. *Spine*, **25,** 2191-9.

Zhou, S.H., McCarthy, I.D., McGregor, A.H., Coombs, R.R. & Hughes, S.P. (2000). Geometrical dimensions of the lower lumbar vertebrae: analysis of data from digitised CT images. *Eur Spine J*, **9,** 242-248.

Zhu, Q., Ouyang, J., Lu, W., Lu, H., Li, Z., Guo, X. & Zhong, S. (1999). Traumatic instabilities of the cervical spine caused by high-speed axial compression in a human model. An in vitro biomechanical study. *Spine*, **24,** 440-4.