THE LUMBAR SPINE MECHANICAL DIAGNOSIS & THERAPY

VOLUME ONE

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Mechanical Diagnosis & Therapy

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Dedication

To dear Joy, whom I love so dearly – who, through thick and thin, has patiently allowed my obsession to freely flow and who has never once complained about the hours, days and months of absence in my search for the final goal.

Foreword

When it first appeared, *The Lumbar Spine* was a slim edition that announced a new concept. It postulated what might be happening in patients with low back pain, and it provided a system of assessment and treatment.

Since its inception, the McKenzie system has grown into a movement. The system captured the imagination of therapists and others, who adopted it. Their numbers grew to form an international organisation that offers training programmes and postgraduate degrees in several countries around the world. The system also attracted the attention of opponents, critics and non-aligned investigators.

Over the years, tensions have developed as the McKenzie system has tried to keep pace with advances in spine science, but also as spine science has tried to keep pace with advances in McKenzie. In basic sciences, our understanding of the structure, function and pathology of the lumbar intervertebral disc has increased enormously. In clinical sciences, the advent of evidence-based medicine has demanded that interventions have evidence of reliability, validity and efficacy. These developments have challenged the McKenzie system, but have not threatened it. Indeed, in many respects, the McKenzie movement has led the way in undertaking research into its precepts, and has implicitly called upon other concepts in physical therapy to catch up. No other system in physical therapy has attracted as much research both from among its proponents and from its detractors.

This new edition of *The Lumbar Spine* has become a tome. It still describes the original concept, albeit updated and revised, but the edition provides students and other readers with a compendium of all the literature pertaining to the lumbar intervertebral disc and the massive literature that now pertains to the McKenzie system.

Readers receive an up-to-date review of information on the structure and function of the disc, its pathology, and new data on its patho-biomechanics. Related entities, such a zygapophysial joint pain and sacro-iliac joint, are comprehensively reviewed.

As befitting a text on this subject, *The Lumbar Spine* contains a complete collection of all studies that have examined the McKenzie

system. These studies have sought the evidence for its reliability, validity and efficacy.

Its reliability is now beyond doubt. Whereas research has shown that other methods of assessment lack reliability, McKenzie assessment has moved from strength to strength. Its reliability, however, is contingent upon training. While anyone can assess according to the system, it cannot be mastered by hearsay or assumption.

Some steps have been taken towards establishing validity. The early studies have been encouragingly positive, but perhaps self-fulfilling. The critical studies have yet to be performed and depend on establishing the efficacy of the treatment.

The Lumbar Spine provides an exhaustive but honest and responsible appraisal of studies of the efficacy of McKenzie treatment. Much of the world finds the evidence insufficiently compelling, but the treatment has not been refuted. Proponents retain the prospect of still vindicating the treatment if and once putatively confounding factors can be eliminated or controlled.

To some observers McKenzie therapy may seem to be a glorified system of special manoeuvres and exercises, but such a view mistakes and understates its virtues. Throughout its history, McKenzie treatment has emphasised educating patients and empowering them to take charge of their own management. Not only did this approach pre-empt contemporary concepts of best practice, it has been vindicated by the evidence. Empowering the patient is seminal to the success of any programme of management.

Although I am not a McKenzie disciple or enthusiast, we have in our own research borrowed from the McKenzie system. In studying the efficacy of evidence-based practice for acute low back pain in primary care,[1] we talked to our patients and we addressed their fears; but to complement that we needed something more for the patients to take with them. For this purpose we drew on some of the simpler exercises described in *The Lumbar Spine*. Not that we believed that these were therapeutic in their own right, but they empowered the patients with sensible things that they could do to cope with their pain and maintain, if not improve, their mobility and function. This approach, a not-too-distant cousin of what McKenzie promotes, was not only successful in a clinical sense, but received great approval from the consumers.

The patho-anatomic concepts and the mechanical aspects of McKenzie therapy may or may not be absolutely material. They may or may not be vindicated in time. But what is already clearly evidence-based is the central theme of McKenzie therapy: to enable patients confidently to care for themselves.

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[1]McGuirk B, King W, Govind J, Lowry J, Bogduk N. The safety, efficacy, and cost-effectiveness of evidence-based guidelines for the management of acute low back pain in primary care. Spine 2001; 26.2615-2622.

Acknowledgments

I would like to give special thanks to my co-author and colleague, Stephen May, MA, MCSP, Dip MDT, MSc, who has provided the necessary expertise to make this second edition an evidence-based text of importance to all health professionals involved in non-operative care of the lower back.

I am also greatly indebted to the many faculty of the McKenzie Institute International, who have either directly or indirectly influenced the refinements that have been made to the descriptions of the procedures of assessment and examination. The value of these contributions is immeasurable.

I would also like to express my gratitude to Kathy Hoyt, a founder of the Institute in the United States, and Helen Clare of Australia, the Institute's Director of Education, who gave so much of their time to read the manuscripts and provide invaluable commentary and criticism.

To Vert Mooney, who opened so many doors, to Ron Donelson for his continued support of the system and the Institute, and to those members of the International Society for the Study of the Lumbar Spine who have encouraged and supported my work, I give my thanks.

Finally, to Jan, my daughter, who reorganised me and coordinated the various specialists required to successfully complete this major task, I give my heartfelt love and thanks.

Robin McKenzie
March 2003

About the Authors

Robin McKenzie was born in Auckland, New Zealand, in 1931 and graduated from the New Zealand School of Physiotherapy in 1952. He commenced private practice in Wellington, New Zealand in 1953, specialising in the diagnosis and treatment of spinal disorders.

During the 1960s, Robin McKenzie developed new concepts of diagnosis and treatment derived from a systematic analysis of patients with both acute and chronic back problems. This system is now practised globally by specialists in physiotherapy, medicine and chiropractic.

The success of the McKenzie concepts of diagnosis and treatment for spinal problems has attracted interest from researchers worldwide. The importance of the diagnostic system is now recognised and the extent of the therapeutic efficacy of the McKenzie Method is subject to ongoing investigation.

Robin McKenzie is an Honorary Life Member of the American Physical Therapy Association "in recognition of distinguished and meritorious service to the art and science of physical therapy and to the welfare of mankind". He is a member of the International Society for the Study of the Lumbar Spine, a Fellow of the American Back Society, an Honorary Fellow of the New Zealand Society of Physiotherapists, an Honorary Life Member of the New Zealand Manipulative Therapists Association, and an Honorary Fellow of the Chartered Society of Physiotherapists in the United Kingdom. In the 1990 Queen's Birthday Honours, he was made an Officer of the Most Excellent Order of the British Empire. In 1993, he received an Honorary Doctorate from the Russian Academy of Medical Sciences. In the 2000 New Year's Honours List, Her Majesty the Queen appointed Robin McKenzie as a Companion of the New Zealand Order of Merit.

In 2003, the University of Otago, in a joint venture with the McKenzie Institute International, instituted a Post Graduate Diploma /Masters programme endorsed in Mechanical Diagnosis and Therapy. Robin McKenzie has been made a Fellow in Physiotherapy at Otago and will be lecturing during the programme.

Robin McKenzie has authored four books: *Treat Your Own Back; Treat Your Own Neck; The Lumbar Spine: Mechanical Diagnosis and Therapy;* and *The Cervical and Thoracic Spine: Mechanical Diagnosis and Therapy.* With the publication of *Mechanical Diagnosis & Therapy of the Human Extremities,* Robin McKenzie, in collaboration with Stephen May, describes the application of his methods for the management of musculoskeletal disorders in general. As with his publications dealing with spine-related problems, the emphasis in this text is directed at providing self-treatment strategies for pain and disability among the general population.

Stephen May was born in Kent, England, in 1958. His first degree was in English Literature from Oxford University. He trained to be a physiotherapist at Leeds and graduated in 1990. Since qualifying, he has worked for the National Health Service in England, principally in Primary Care. In 2002 he became a Senior Lecturer at Sheffield Hallam University.

He developed a special interest in musculoskeletal medicine early in his career and has always maintained a diligent interest in the literature. One of the results of this was a regular supply of articles and reviews to the McKenzie newsletter (UK). In 1995 Stephen completed the McKenzie diploma programme. In 1998 he completed an MSc in Health Services Research and Technology Assessment at Sheffield University.

Stephen is author or co-author of several articles published in international journals. He has previously collaborated with Robin McKenzie on *The Human Extremities: Mechanical Diagnosis & Therapy*.

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Introduction

Many years have passed since the publication of the first edition of my monograph, *The Lumbar Spine: Mechanical Diagnosis and Therapy*. Since 1981, when the book was first released, the conceptual models for the identification of subgroups in the non-specific spectrum of back pain and the methods of treatment I recommended have internationally received wide acceptance.

The extent of the acceptance for what I chose to call Mechanical Diagnosis and Therapy (MDT) was never anticipated. I did not, as a result of dissatisfaction with existing methods, deliberately construct a new system of diagnosis and treatment to manage common mechanical back problems. Rather, from everyday observation and contact with large numbers of patients, I learned from them, unconsciously at first I suspect, that different patients with apparently similar symptoms reacted quite differently when subjected to the same mechanical loadings. On grouping together all those whose symptomatic and mechanical responses to loading were identical, three consistent patterns emerged and became in turn the syndromes whose identification and management are described within these pages.

Because of the stable population in the city of Wellington in New Zealand, many patients with recurrent and chronic problems returned for help over time. Thus I had the opportunity to observe in many individuals the passing spectrum of mechanical and symptomatic changes that progressed during two or even three decades of life. From this experience I learned how to make the changes in management that were dictated by the gradual structural changes resulting from the natural ageing process. The eventual refinement of my observations and techniques of loading were thus merely a function of evolution.

I have recounted the story of "Mr Smith", described later in this volume, on many courses and at many conferences around the world. I do so because it describes an actual event that has had an enormous impact on my life and has, and continues to have, an impact on the way health professionals worldwide think about and manage the spine and musculoskeletal problems in general.

Occasionally I am asked, "Was there really a Mr Smith, or did you invent him to provide an amusing story to go with the effects of extension?" I can only reply that, yes, it is a true story, and no, I did not make it up, but his real name is long forgotten.

Prior to the encounter with Mr Smith, I, along with a few other physiotherapists at that time, was exploring and mastering the multitude of manipulative techniques and the philosophies that lay behind them. Cyriax, Mennell, Stoddard and the chiropractors were the flavour of that period. Maitland and Kaltenborn were yet to appear. In my mind, the only rational explanation to account for the centralisation of Mr Smith's symptoms was to be found in the first volume (1954), written by one James Cyriax, MD.

Cyriax attributed sudden and slow onset back pain respectively to tearing of the annulus and bulging or displacement of the nucleus. If the bulge was large enough, compression of the root would follow. Thus it suggested to me that Mr Smith's centralisation occurred because the pressure on his sciatic nerve was removed. Extension, I thought, was therefore a good thing to apply in these cases. It might even be more effective than the manipulations we practised, which sometimes did – and many times did not – produce a benefit for the patient.

Following the encounter with Mr Smith, the hypothesis to explain the varying responses to loading crystallised and formed the basis of the conceptual models upon which the treatments were developed. Without the conceptual model of displacement and its sequelae, I doubt that I could have developed the explanations and eventually provided the solutions for many of the mechanical disorders presenting in daily practice.

Belief in the conceptual model provided an explanation and better understanding of centralisation and peripheralisation. It explained the changes in pain location and intensity that follow prolonged or repetitive sagittal loading and led to the discovery that offset loading (hips off centre) was required when symptoms were unilateral or asymmetrical. The model suggested that it could be possible, by applying lateral forces, to entice low back and cervical pains to change sides. That phenomenon is now clinically repeatable in certain selected patients.

Identification of the most effective direction for applying therapeutic exercise – the use of prolonged positioning and repeated rather than single movements in assessment; the progressions of force; differentiation between the pain of displacement, from the pain of contracture, and pain arising from normal tissue; the three syndromes; differentiation of limb pain caused by root adherence, entrapment or disc protrusion – all arose directly or indirectly from the conceptual model.

The disc model, the theories and clinical outcomes relative to mechanical diagnosis and therapy are under investigation worldwide. The models are as yet unproven scientifically; even so they provide a sound basis for the management of non-specific disorders of the lower back. Much to my intense satisfaction, the experiments, the conclusions and the results I recorded have successfully been replicated by others.

To this day, belief in the conceptual model, acting on its suggestions and obeying its warnings, guide me in the management of the patient. Many things indirectly arose from the model. Mr Smith was the catalyst. We no longer have to manipulate all patients in order to deliver the procedure to the very few requiring it. We no longer have to apply manipulation to our patients to determine retrospectively if it was indicated. I would never be without the model and Mr Smith is never far from my thoughts.

Mechanical Diagnosis and Therapy is now one of the most commonly used treatment approaches utilised by physiotherapists in the United Kingdom, New Zealand and the United States. It is an approach also utilised and recommended by chiropractors, physicians and surgeons. The increasing interest is reflected in the substantial body of research that has been conducted into aspects of "The McKenzie Method", as it has come to be known. The very nature of MDT lends itself to measurement.

There have been numerous studies into centralisation, symptom response and reliability, as well as studies into the efficacy of MDT. More studies are needed, but much research already strongly endorses aspects of this system of assessment and management. Further recent endorsement of MDT has been given by its inclusion in national back pain guidelines from Denmark and systematic musculoskeletal guidelines from the United States.

Centralisation has been shown to have clear prognostic as well as diagnostic significance. It is one of the few clinical factors that have been found to have more prognostic implications than psychosocial factors. Study after study has asserted the poor reliability of assessment that is based on palpation or observation, while symptom response consistently shows good reliability.

Education in MDT has now been structured to enable the formalised teaching of clinicians and provide a base upon which rigorous scientific inquiry may proceed. Educational programmes are provided under the auspices of the McKenzie Institute International and its branches and are conducted in all continents and attended annually by thousands of clinicians. Some appreciation of the extent of the adoption of MDT can be seen from the request by the Director of the Chinese Ministry of Health, Department of Rehabilitation, to provide the Institute's education programme for Chinese physicians and surgeons involved in the management of back disorders throughout the world's most populous country.

It is now common knowledge that management of musculoskeletal problems must involve patient understanding, including a knowledge of the problem and proffered solution. Patients must be actively involved in treatment. This was a message first stated over twenty years ago in the first edition of this title. Sadly, it seems, with the continued usage of ultrasound and other passive treatment modalities by clinicians, despite clear evidence for lack of efficacy; this is a message that health professionals have still not clearly heard. "How many randomised controlled trials does it take to convince clinicians about the lack of efficacy for ultrasound and other passive treatments?" (Nachemson, 2001).

The clinical utility and worth of the system is attested to by the thousands of 'studies of one' conducted by clinicians on their patients throughout the world every year. It is used and continues to be used because it is effective.

Ultimately, do we wish to make the patient feel 'better', albeit briefly, or do we wish to offer the patient a means of self-treatment and understanding so that there is a strong possibility they will benefit from our services in the long-term? Are we creating patient dependence on therapy, or providing a chance of independence

through self-management? A key role for clinicians must be as educators, rather than 'healers'.

The second edition of this title is presented to the reader with the knowledge and hindsight of experience gained since the production of the first. In the first edition of 1981, there were few, if any, references to quote in support of the methods and theories I propounded. Prior descriptions of the use of repetitive end-range motion and its effects on pain location and intensity; the phenomenon of clinically induced centralisation and peripheralisation; the prognostic value of centralisation and non-centralisation; the theoretical models; identification of subgroup syndromes; the progressions of therapeutic forces; and most importantly self-treatment and management strategies did not exist in the literature of the day. Fortunately that is not the case today.

I believe that with the involvement of Stephen May in the writing of this edition, the imperfections that abound in my first excursion into the literary world have been eliminated. Stephen's understanding of "McKenzie", combined with his literary talents and global familiarity with the scientific literature, have brought to this edition a quality that far exceeds my own capabilities. This will become apparent to the reader on advancing through the chapters within.

We have provided for you in this second edition, a monograph that describes in explicit detail what the "McKenzie Method" is, how to apply it and the evidence that substantiates and justifies its use for the management of non-specific low back pain.

I believe these chapters will allow better understanding and more appropriate investigation of MDT. Above all, I trust it will serve its prime purpose in helping our patients.

Robin McKenzie
Raumati Beach
New Zealand

1: The Problem of Back Pain

Introduction

It is important to understand the extent to which any health problem impacts upon the population. This provides an understanding of that problem, as well as suggestions as to how it should be addressed by health care providers. Clearly it is inappropriate for health professionals to deal with a benign, self-limiting and endemic problem such as the common cold in the same way that they address possibly life-threatening disorders such as heart attacks. The study and description of the spread of a disease in a population is known as *epidemiology*.

Modern clinical epidemiology is concerned with the distribution, natural history and clinical course of a disease, risk factors associated with it, the health needs it produces and the determination of the most effective methods of treatment and management (Streiner and Norman 1996). Epidemiology thus offers various insights that are critical to an understanding of any health problem (Andersson 1991; Nachemson *et al.* 2000). It provides information about the extent of a problem and the resultant demand on services. An understanding of the natural history informs patient counselling about prognosis and helps determine the effects of treatment. Associations between symptoms and individual and external factors allow the identification and modification of risk factors. Outcomes from studies about interventions should provide the evidence for the most effective management strategies.

The sections in this chapter are as follows:

- prevalence
- natural history
- disability
- cost
- health care
- treatment
- effectiveness.

Risk and prognostic factors are discussed in the next chapter. This information provides a background understanding that should influence the management that health professionals provide.

Prevalence

Trying to measure the frequency of back pain, its clinical course or the rate of care-seeking related to back pain is not straightforward. There is considerable variability in the way data has been gathered – in different countries, at different times, employing different definitions, asking slightly different questions and using different methods to gather this information. There is often a lack of objective measurement, the problem is frequently intermittent and recall can be plagued by bias. Thus there is a problem with the validity and reliability of the data, and the figures offered should be seen as estimations rather than exact facts (Andersson 1991; Nachemson *et al.* 2000). Nonetheless, certain figures appear consistently enough to give a reasonably reliable overall picture of the extent of the problem and its natural history.

Despite all methodological difficulties, it can be stated that back pain is about the most prevalent pain complaint, possibly along with headaches (Raspe 1993). In adults, between one-half and three-quarters of the population will experience back pain at some point in their life. About 40% will experience an episode of back pain in any one year, and about 15 – 20% are experiencing back pain at any given time. Similar figures are given in reviews and primary research from different countries around the world (Croft *et al.* 1997; Klaber Moffett *et al.* 1995; Evans and Richards 1996; Waddell 1994; Shekelle 1997; Papageorgiou and Rigby 1991; Papageorgiou *et al.* 1995; Linton *et al.* 1998; Brown *et al.* 1998; Leboeuf-Yde *et al.* 1996; McKinnon *et al.* 1997; Szpalski *et al.* 1995; Heliovaara *et al.* 1989; Toroptsova *et al.* 1995; Cassidy *et al.* 1998). Apparently only 10 – 20% of the adult population seems to have never had back problems (Raspe 1993).

Table 1.1 contains a sample of international studies that have been conducted in great numbers of the general population. Large representative surveys are the best evidence for a problem in the greater population (Nachemson *et al.* 2000). Commonly these surveys describe the proportion of people who report back pain at the time or that month (point prevalence), in that year (year prevalence) or back pain ever (lifetime prevalence).

Table 1.1 Prevalence of back pain in selected large population-based studies

Reference	*Country*	*Point prevalence*	*Year prevalence*	*Lifetime prevalence*
Hillman *et al.* 1996	UK	19%	39%	59%
Papageorgiou *et al.* 1995	UK	39%		
Brown *et al.* 1984	Canada (police force)		42%	
Heliovaara *et al.* 1989	Finland	20%		75%
Toroptsova *et al.* 1995	Russia	11%	31%	48%
Leboeuf-Yde *et al.* 1996	Nordic countries (review)		50%	66%
Linton *et al.* 1998	Sweden		66%	
McKinnon *et al.* 1997	UK	16%	48%	62%
Skovron *et al.* 1994	Belgium	33%		59%
Walsh *et al.* 1992	UK		36%	58%
Dodd 1997	UK	15%	40%	
Waxman *et al.* 2000	UK		41%	59%
Average rates of selected studies		**22%**	**44%**	**61%**

These gross figures disguise differences in the characteristics of different episodes of back pain relative to duration, severity and effect on a person's lifestyle.

Clearly back pain is an endemic problem, widespread throughout the community. It is a problem that will affect the majority of adults at some point in their lives. Back pain is normal.

Natural history

The traditional concept of back pain was the acute/chronic dichotomy, in which it was thought that most patients have brief finite episodes and only a few progress to a chronic problem. It is frequently stated that for most people the prognosis is good (Klaber Moffett *et al.* 1995; Evans and Richards 1996; Waddell 1994): *"80 – 90% of attacks of low back pain recover in about 6 weeks"* regardless of the treatment applied, or lack of it (Waddell 1987). However, a picture of the natural history of back pain that suggests the majority will have a brief self-limiting episode denies recent epidemiological evidence and paints an over-optimistic summary of many individuals' experience of this problem.

It is certainly true that a great number of acute episodes of back pain resolve quickly and spontaneously (Coste *et al.* 1994; Carey *et al.* 1995a). Coste *et al.* (1994) followed 103 acute patients in primary care for three months and found that 90% had recovered in two weeks and that only two developed chronic back pain. However, this study sample contained patients with a very brief history of back pain (less than 72 hours), no referral of pain below the gluteal fold and excluded those who had experienced a previous episode in the last three months – all characteristics with a good prognosis.

Dillane *et al.* (1966) reported that the duration of the episode in over 90% of those who visited their GP with acute back pain was less than four weeks. However, the duration was defined as the time between the first and last consultation with the doctor. An episode of back pain cannot be defined in this way. Although patients may stop attending their medical practitioner, this does not necessarily mean that their back pain has resolved. More recently it was found that while most patients only visited their GP once or twice because of the problem, one year later 75% of them were still not symptom-free (Croft *et al.* 1998).

Other studies that have looked at the natural history of new episodes of back pain in primary care settings also paint a more pessimistic picture, although outcome depends partly on what is being measured (Carey *et al.* 1995a; Cherkin *et al.* 1996a). Studies have found that only 30 – 40% of their sample are completely resolved at about two to three months, with little further improvement at six or twelve months (Cherkin *et al.* 1996a; Philips and Grant 1991; Klenerman *et al.* 1995). Thomas *et al.* (1999) interviewed patients who had presented to primary care with new episodes of back pain – 48% still reported disabling symptoms at three months, 42% at one year and 34% were classified as having persistent disabling back pain at both reporting times.

Recurrences in the following year after onset are extremely common, reported in about three-quarters of samples (Klenerman *et al.* 1995; van den Hoogen *et al.* 1998). In a large group of patients in primary care studied (von Korff *et al.* 1993) one year after seeking medical treatment for back pain, the majority with both recent and non-recent onset of back problems reported pain in the previous month (69% and 82% respectively). In those whose problem had started recently, only 21% were pain-free in the previous month; in those

whose problem was of a longer duration, only 12% were pain-free in the previous month.

Table 1.2 gives a selection of studies that have described relapse rates and persistent symptoms. Relapse rates refer to those in the back pain population who report more than one episode in a year, and persistence refers to back pain that has lasted for several months or more. Exact definitions vary between different studies, but a history of recurrences and non-resolving symptoms is clearly a very common experience.

Table 1.2 Relapse rate and persistent symptoms in selected studies

Reference	*Relapse rate*	*Persistent symptoms*
Linton *et al.* 1998	57%	43%
Brown *et al.* 1998	55%	
Szpalski *et al.* 1995		36%
Heliovaara *et al.* 1989	45%	
Toroptsova *et al.* 1995	65%	23%
Hillman *et al.* 1996		47%
Philips and Grant 1991		40%
Klenerman *et al.* 1995	71%	
Thomas *et al.* 1999		48%
Van den Hoogen *et al.* 1998	76%	35%
Miedema *et al.* 1998		28%
Croft *et al.* 1998		79%
Carey *et al.* 1999	39%	
Waxman *et al.* 2000		42%
Average rates from selected studies	**58%**	**42%**

"The message from the figures is that, in any one year, recurrences, exacerbations and persistence dominate the experience of low back pain in the community" (Croft *et al.* 1997, p. 14).

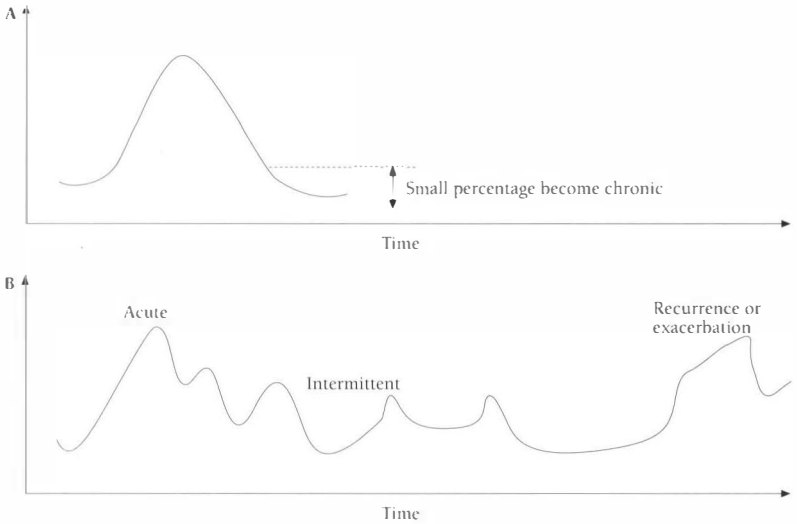
It is clear that for many individuals, recovery from an acute episode of backache is not the end of their back pain experience. The strongest known risk factor for developing back pain is a history of a previous episode (Croft *et al.* 1997; Shekelle 1997; Smedley *et al.* 1997). The chance of having a recurrence of back pain after a first episode is greater than 50%. Many recurrences are common and more than one-third of the back pain population have a long-term problem (Croft *et al.* 1997; Evans and Richards 1996; Waddell 1994;

Papageorgiou and Rigby 1991; Linton *et al.* 1998; Brown *et al.* 1998; Szpalski *et al.* 1995; Heliovaara *et al.* 1989; Toroptsova *et al.* 1995). There is also the suggestion from one population study that those with persistent or episodic pain may gradually deteriorate, being significantly more likely to report chronic low back pain and associated disability at a later date (Waxman *et al.* 2000). However, the risk of recurrence or persistence of back pain appears to lessen with the passage of time since the last episode (Biering-Sorensen 1983a).

The inference from these figures is clear – an individual's experience of back pain may well encompass their life history. The high rate of recurrences, episodes and persistence of symptoms seriously challenges the myth of an acute/chronic dichotomy. Back pain is "*a recurrent condition for which definitions of acute and chronic pain based on a single episode are inadequate, characterised by variation and change, rather than an acute, self-limiting episode. Chronic back pain, defined as back pain present on at least half the days during an extended period is far from rare...*" (Von Korff and Saunders 1996).

It would appear from the evidence that the much-quoted speedy recovery of back pain does not conform to many people's experience and that the division of the back pain population into chronic and acute categories presents a false dichotomy (Figure 1.1). This is not to deny that many people have brief acute episodes that resolve in days, nor that there is a small group of seriously disabled chronic sufferers, but that for large numbers, "*low back pain should be viewed as a chronic problem with an untidy pattern of grumbling symptoms and periods of relative freedom from pain and disability interspersed with acute episodes, exacerbations, and recurrences*" (Croft *et al.* 1998). Back pain should be viewed from the perspective of the sufferer's lifetime – and given such a perspective, the logic of self-management is overwhelming.

Figure 1.1 The assumed and real natural history of back pain



A: Assumed course of acute low back pain
B: Real course of back pain

Reproduced with permission from Croft P, Papageorgiou A and McNally R (1997) *Low Back Pain*. In: A Stevens and J Raftery (eds) Health Care Needs Assessment. Second Series: Radcliffe Medical Press, Oxford.

In summary, many episodes of back pain are brief and self-limiting; however, a significant proportion of individuals will experience persistent symptoms, while a minority develop chronic pain. The natural improvement rate stabilises after the first few months, and after this time resolution is much less likely. Up to one-third of new episodes result in prolonged periods of symptoms. Half of those having an initial episode of back pain will experience relapses. Lack of clinical follow-up creates the mistaken impression that there is common resolution of problems, which is not confirmed by more stringent research methods.

Disability

Not all back pain is the same. There is variability between individuals in the persistence of symptoms, in severity and in functional disability (von Korff *et al.* 1990). One review of the literature found that between 7% and 18% of population samples that have been studied are affected frequently, daily or constantly by back pain (Raspe 1993). Persistent symptoms have been reported by about 40% and longstanding, disabling backache by about 10% of all those who suffer from the problem (Croft *et al.* 1997; Evans and Richards 1996; Fordyce 1995;

Waddell 1994; Linton *et al.* 1998; Szpalski *et al.* 1995; Heliovaara *et al.* 1989; Toroptsova *et al.* 1995; Carey *et al.* 2000). Levels of disability, even among those with persistent symptoms, vary widely.

Musculoskeletal disorders are the most common cause of chronic incapacity, with back pain accounting for a significant proportion of this total (Bennett *et al.* 1995; Badley *et al.* 1994). Back pain is thus one of the most common causes of disability, especially during the productive middle years of life. It has been estimated (Waddell 1994) that 10% of the adult population, or 30% of those with back pain, report some limitation of their normal activity in the past month because of it. Work loss due to backache occurs for 2% of the adult population each month, just less than 10% each year and in 25 – 30% of the working population across their lifetimes (Waddell 1994).

Heliovaara *et al.* (1989) reported from a population survey that 40% of those with back pain had been forced to reduce leisure activities permanently, 20% had marked limitation of daily activities and 5% had severe limitations. In a one-year period, 22% of those with back pain who were employed went on sick leave because of it, representing a prevalence rate in the adult population of 6% (Hillman *et al.* 1996). According to one study, serious disability and work loss affects 5 – 10% of the population in any year, and in a lifetime over one-quarter of the population take time off work due to back pain (Walsh *et al.* 1992).

Table 1.3 Disability and work loss due to back pain in general population

	Men One year	*Men Lifetime*	*Women One year*	*Women Lifetime*
Disability	5%	16%	4.5%	13%
Work loss	11%	34%	7%	23%

Source: Walsh *et al.* 1992

Disability due to back pain has varied over time. In the UK during the 1980s, the payment of sickness and invalidity benefit rose by 208%, compared to an average rise of 54% for all incapacities (Waddell 1994). There is no evidence of an increased prevalence of back pain over recent decades (Nachemson *et al.* 2000; Leboeuf-Yde *et al.* 1996); the increased incapacity is thought to relate to changed attitudes and expectations, changed medical ideas and management, and changed social provision (Waddell 1994). It might also be seen to reflect a time of high unemployment and social change within the UK. Indeed,

more recent evidence from the US reports that rather than being on the increase, the estimate of annual occupational back pain for which workers claimed compensation actually declined by 34% between 1987 and 1995 (Murphy and Volinn 1999).

It is important to be aware that patients with chronic back pain represent a diverse group, not all of whom are fated to a poor prognosis. When ninety-four individuals with chronic back pain were questioned about work and social disability, less than 8% indicated an interruption of normal activities over a six-month period (McGorry *et al.* 2000). Attempts have been made to classify chronic pain states relative to severity and associated disability, which indicated that over half of those with chronic pain report a low level of restriction on their lifestyle and low levels of depression.

Several large population-based studies of chronic pain and back pain (von Korff *et al.* 1990, 1992; Cassidy *et al.* 1998) and a study of chronic back pain patients (Klapow *et al.* 1993) reveal reasonably consistent levels of limitation of activity due to persistent pain problems. About half of those with chronic pain report a low level of disability and a good level of coping. About a quarter report moderate levels of disability, and another quarter report severe incapacity due to the problem (see Table 1.4). In those attending primary care for back pain, about 60% had low disability and about 40% had high disability at presentation (von Korff *et al.* 1993). After one year, less than 20% were pain-free, 65% had minimal disability and between 14% and 20% had high disability, so even in those with persistent symptoms the severity and disability is variable, with the majority reporting minimal reduction of function.

Table 1.4 Grading of chronic back pain

Grade	*von Korff et al. 1992 (N = 1213)*	*Klapow et al. 1993 (N = 96)*	*Cassidy et al. 1998 (N = 1110)*
Low disability and low intensity	35%	49%	48%
Low disability and high intensity	28%	25%	12%
High disability	37% (Moderate 20%; Severe 17%)	26%	11%

"There was considerable heterogeneity in manifestations of pain dysfunction among persons with seemingly comparable pain experience. A considerable proportion of persons with severe and

persistent pain did not evidence significant pain-related disability. Some persons with severe and persistent pain did not evidence psychological impairment, although many did" (Von Korff *et al.* 1990).

The pain status of individuals is not static, but dynamic (Table 1.5). Symptoms and associated disability fluctuate over time, and many patients leave the pool of persistent pain sufferers if followed over a few years. The overall pool of those with chronic pain appears to stay about the same, but a proportion leave that group and either become pain-free or are less severely affected, while a similar number join it over a period of a year or more (Cedraschi *et al.* 1999; Croft *et al.* 1997; Troup *et al.* 1987; von Korff *et al.* 1990; McGorry *et al.* 2000).

Table 1.5 The dynamic state of chronic back pain

	CLBP or chronic pain who become pain-free	*CLBP who improve*	*CLBP who remain ISQ*
Croft *et al.* 1997		33%	67%
Cedraschi *et al.* 1999		53%	47%
Troup *et al.* 1987	8%	9%	83%
Crook *et al.* 1989	13% (pain clinic) 36% (primary care)		

CLBP = chronic low back pain

ISQ = in status quo

Back pain is a symptom that describes a heterogeneous and dynamic state. Individuals vary in their experience of backache relative to time, severity and disability. Many individuals have persistent problems. Most chronic back pain is of low intensity and low disability; high levels of severity and disability affect only the minority. Some of those with chronic backache do become pain-free; however, because of high prevalence rates, back pain produces extensive disability and work loss and thus impacts considerably on individuals and on society.

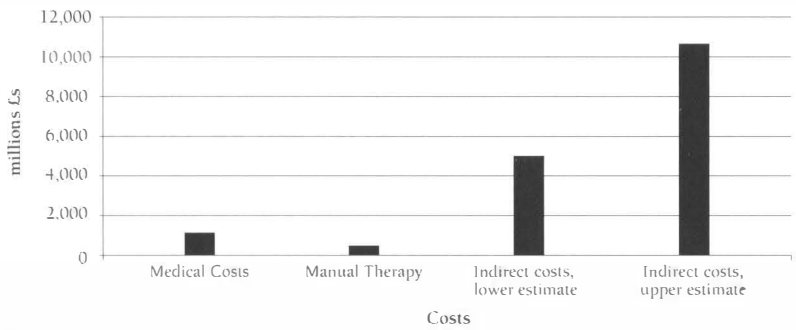
Cost

Even though not everyone with back pain seeks health care, the prevalence of the problem is so great that high numbers of patients are entering the health services. A major concern is the cost associated with back pain, although this is difficult to calculate. It is made up of the direct cost of health care borne by society or by the patient and

the indirect costs associated with absence from work. In the UK costs to the NHS alone in 1992/3 have been estimated at between £265 and £383 million, which constitutes 0.65 – 0.93% of total NHS spending (Klaber Moffett *et al.* 1995). A more recent estimate of the direct health care costs of back pain in the UK for 1998 put the cost at £1,632 million (Maniadakis and Gray 2000). In the US, medical care costs have been estimated at between $8 and $18 billion (Shekelle *et al.* 1995).

The medical costs of back pain, however, are only a part of the whole cost of the problem that society pays. Indirect costs, such as disability or compensation payments, production losses at workplaces and informal care, dwarf the amount that is spent directly on patient care. The total societal cost of back pain in the US has been estimated at $75 – $100 billion in 1990 (Frymoyer and Cats-Baril 1991). Cost data from insurance companies from two separate studies shows that medical care represents about 34% of the total costs, while ind costs make up about 66% (Webster and Snook 1990; Williams *et al.* 1998a). Total employment-related costs in the UK have been estimated at between £5 and £10 billion (Maniadakis and Gray 2000), which means that direct costs only account for between 13% and 24% of the total costs (Figure 1.2). In the Netherlands, the direct health care costs have been estimated as only 7% of the total cost, with the total cost representing 1.7% of the gross national product (van Tulder *et al.* 1995).

Figure 1.2 The direct and indirect costs of back pain



Source: Maniadakis and Gray 2000

Medical costs include medicines and x-rays; manual therapy includes physiotherapy, osteopathy and chiropractic; indirect costs include production losses and informal care. Some of these costs can only be estimated. The direct and indirect costs of back pain are so great that

the economic burden is larger than for any other disease for which economic analysis was available in the UK in 1998 (Maniadakis and Gray 2000). It is more costly than coronary heart disease and the combined costs of rheumatoid arthritis, respiratory infections, Alzheimer's disease, stroke, diabetes, arthritis, multiple sclerosis, thrombosis and embolism, depression, diabetes, ischaemia and epilepsy.

A minority of patients consume the majority of health care and indirect costs for low back pain. Combining data from multiple studies suggests that about 15% of the back pain population account for about 70% of costs (Spitzer *et al.* 1987; Webster and Snook 1990; Williams *et al.* 1998a; Linton *et al.* 1998).

Thus, not only is the cost of back pain huge, but the majority of this money is not spent directly on patient care, but on indirect societal 'costs'. Furthermore, it is the chronic few who consume the largest proportion of this expense.

Health care

Not everybody with back pain seeks professional help. Most surveys reveal that about a quarter to a half of all people with back pain will consult their medical practitioner (Croft *et al.* 1997; Papageorgiou and Rigby 1991; McKinnon *et al.* 1997; Carey *et al.* 1996). A survey in Belgium found that 63% of those with back pain had seen a health professional for the most recent episode (Szpalski *et al.* 1995). Where chiropractic care is available, 13% of back pain sufferers seek their help (Linton *et al.* 1998; Carey *et al.* 1996). Seeking care appears to vary widely; one survey in the UK found those seeking consultation with local physicians to range from 24 – 59% of those with back pain in different areas (Walsh *et al.* 1992). Care-seeking among those with chronic back pain may be slightly higher (Carey *et al.* 1995b, 2000). Many people with low back pain cope independently in the community and do not seek help, whether medical or alternative.

Table 1.6 Proportion of back pain population who seek health care

Reference	*Country*	*% who consult GP*	*% who consult osteopath*	*% who consult chiropractor*	*% who consult physio-therapist*
Dodd 1997	UK	38%	6%	3%	9%
Walsh *et al.* 1992	UK	40%			
Hillman *et al.* 1996	UK	37%	4.5%	1%	10%
Linton *et al.* 1998	Sweden	8%		13%	5%
Carey *et al.* 1996	US	24%		13%	
McKinnon *et al.* 1997	UK	24%			
Santos-Eggimann *et al.* 2000	Switzer-land	25%			

In the UK, Waddell (1994) estimated a population prevalence of 16.5 million people with back pain in 1993. Of these he estimated that 18 – 42% consult their GP, 10% attend a hospital outpatient department, 6% are seen by NHS physiotherapists, 4% by osteopaths, less than 2% each by private physiotherapists and chiropractors, 0.2% become inpatients and 0.14% go to surgery.

Even though many people with back pain do not attend a health professional, because of the large prevalence rate in the community the numbers actually seeking health care are considerable and constitute a significant burden in primary care. For instance, in the US it is estimated that it is the reason for 15 million visits to physicians annually, the fifth-largest reason for attendance, representing nearly 3% of all visits (Hart *et al.* 1995). In a rural primary care setting in Finland and practices in the UK, low back pain patients make up about 5% of all GP consultations (Rekola *et al.* 1993; Hackett *et al.* 1993; Waddell 1994). In the UK it has been estimated that one-third of those attending primary care with back pain will present with a new episode, one-third will present with a recurrence and one-third will present with a persistent disabling problem (Croft *et al.* 1997).

There are no clear clinical features that distinguish those patients who seek health care from those who do not. Hillman *et al.* (1996) found that those who consult tended to report higher levels of pain, greater disability and longer episodes, but also that some individuals with the same characteristics did not seek health care. Carey *et al.* (1999) found that recurrences of back pain, the presence of sciatica

and greater disability were associated with care-seeking. Longer duration of an episode of back pain is more likely to cause people to consult (Santos-Eggimann *et al.* 2000), and failure to improve is associated with seeking care from multiple providers of health care (Sundararajan *et al.* 1998).

Those who attend tertiary care tend to be at the more severe end of the spectrum of symptoms. However, one-fifth of non-consulters had constant pain and needed bed-rest, one-third had had pain for over three months in the previous year and nearly half had leg pain and restricted activity (Croft *et al.* 1997).

In the US, Carey *et al.* (1996) found that those who sought care were more likely to have pain for longer than two weeks that radiated into the leg and had come on at work. However, considerable numbers of those not seeking care also had these characteristics. Szpalski *et al.* (1995) found that back pain frequency, health beliefs and sociocultural factors influenced health care-seeking. Other studies have also found that psychosocial factors have some impact on care-seeking for back pain (Wright *et al.* 1995; Vingard *et al.* 2000). The type of health provider that patients first see may have an effect on subsequent consultation rates, with those who see a chiropractor being twice as likely to seek further help compared to those who saw a medical doctor (Carey *et al.* 1999).

The message in the epidemiological literature – that many people with back pain cope independently from professional help – is reinforced by evidence from qualitative research using interviews of people with back pain. Skelton *et al.* (1996) in the UK found a large number of his sample to be actively working on their problem by adopting various preventive strategies. These included use of certain body postures when bending, sitting and lifting; taking light exercise; resting; doing back and abdominal exercises; and, for some, constant awareness of a back problem in day-to-day activities. In contrast, a smaller group of patients reported taking a minimalist approach to self-management, despite having some knowledge about preventive measures. In between these two extremes were a few who reported that they were in the process of recognising a need to do something about their problem and were beginning to perceive the need to adopt self-management strategies.

Borkan *et al.* (1995) also found patients adopting a range of intellectual and behavioural strategies that were designed to minimise pain or maximise function. Information about back care is a common expectation of those who do seek professional help (Fitzpatrick *et al.* 1987).

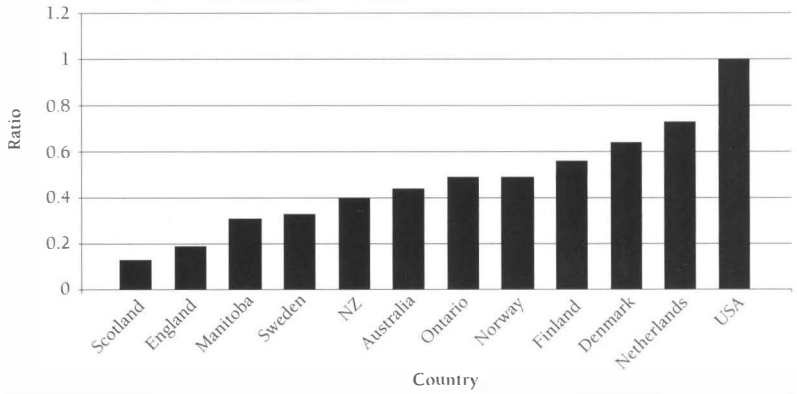
Less than half of those in the community with back pain actually seek health care. It is thus clear that self-management of back problems is both attainable and practised by many. Some of those who do not seek health care have constant, persistent and referred pain with reduced function. The majority of people with back pain manage independently of health professionals. Of those who do seek help, many are looking for things that they can do to help themselves to manage their problem better. There are others who are neglectful of adopting the necessary strategies, but who may be convinced of the necessity of doing so if they are sufficiently informed. Nonetheless, because of the high prevalence rate, back pain constitutes a considerable burden to primary care.

Treatment

The range of treatments offered to patients with back pain varies considerably. There is no consensus on the best type of treatment for back pain, and so the treatment given is chosen on the inclination of the practitioner. It depends more on whom the patient sees than their clinical presentation (Deyo 1993).

A back pain patient in the United States is five times more likely to be a surgical candidate than if they were a patient in England or Scotland (Figure 1.3 from Cherkin *et al.* 1994a). Back surgery rates increased almost linearly with the local supply of orthopaedic and neurosurgeons.

Figure 1.3 Ratios of back surgery rates to back surgery rate in the US (1988 – 1989)



Source: Cherkin *et al.* 1994.

In the US, non-surgical hospitalisation and surgery rates vary considerably, both over time and place. For instance, patients are twice as likely to be hospitalised in the south than in the west, and between 1979 and 1990 there was a 100% increase in the rate of fusion operations (Taylor *et al.* 1994).

In the Netherlands, a descriptive study of general practitioners' approaches to chronic back pain patients has shown that there is little consistency between clinicians (van Tulder *et al.* 1997a). Cherkin *et al.* (1994b) found there was little consensus among physicians about which diagnostic tests should be used for back pain patients with certain clinical presentations and concluded that, for the patient, 'who you see is what you get'.

Equally, in physical therapy there is no standardised management of back pain. Surveys of reported management styles have been conducted in the US (Battie *et al.* 1994; Jette *et al.* 1994; Jette and Delitto 1997; Mielenz *et al.* 1997), in the Netherlands (van Baar *et al.* 1998) and in the UK (Foster *et al.* 1999). These surveys show that a wide range of thermal and electrotherapy modalities, massage, mobilisation and manipulation, exercises and mixed treatment regimes are used. Exercises are commonly used, but these are frequently combined with the use of passive treatment modalities, such as ultrasound, heat or electrical stimulation and, less frequently, with the use of manual therapy. Passive treatment modalities tend to be used by some clinicians, whatever the duration of symptoms.

In a survey in the US, The McKenzie Method was deemed the most useful approach for managing back pain, although in practice clinicians were likely to use a variety of treatment approaches (Battie *et al.* 1994). In the UK and Ireland, the Maitland and McKenzie approaches were reportedly used most often to manage back problems, although electrotherapy modalities (interferential, ultrasound, TENS and short-wave diathermy) and passive stretching and abdominal exercises are also commonly used (Foster *et al.* 1999). Internationally, physiotherapy practice is eclectic and apparently little influenced by the movement towards evidence-based practice.

Back care regimes are clearly eclectic and non-standardised. When so much variety of treatment is on offer, what patients get is more likely to reflect the clinicians' biases rather than to be based on their clinical presentation or the best evidence. Under these circumstances there must be occasions when the management offered is sub-optimal and is not in the best long-term interest of the patient.

Effectiveness

Unfortunately, seeking health care does not, for many, solve their back problem (Von Korff *et al.* 1993; Linton *et al.* 1998; van den Hoogen *et al.* 1997; Croft *et al.* 1998). Despite the vast numbers who are treated for this condition by different health professionals, the underlying epidemiology of back pain, with its high prevalence and recurrence rates, remains unchanged (Waddell 1994). Indeed, there is even the accusation that traditional methods of care, involving rest and passive treatment modalities rather than activity, have been partly implicated in the alarming rise of those disabled by back pain (Waddell 1987).

Some studies have challenged the notion that outcomes are necessarily better in those who are treated with physiotherapy or chiropractic (Indahl *et al.* 1995; van den Hoogen *et al.* 1997). For instance, Indahl *et al.*'s study (1995,) followed nearly 1,000 patients who were randomised either to normal care or to a group who were given a thorough explanation of the importance of activity and the negative effects of being 'too careful'. At 200 days, 60% of the normal care group were still on sick leave, compared to 30% of those instructed to keep active. Of those in the normal care group, 62% received physical therapy and 42% chiropractic, of which 79% and 70%

respectively reported that treatment made the situation worse or had little or no effect.

Various reviews and systematic reviews have been undertaken into interventions used in the treatment of back pain. These universally only include prospective randomised controlled trials, which, with their supposed adherence to strict methodological criteria, are seen as the 'gold standard' by which to judge interventions. This adherence to specific study designs is rarely achieved, but the focus on study design tends to distract from the intervention itself. Restricted recruitment and follow-up may limit generalisability; interventions may not reflect clinical practice, because mostly they are given in a standardised way with no attempt at assessment of individual suitability for that treatment regime; the outcome measures may not be appropriate for the condition.

Nonetheless, the underlying message is impossible to evade – no intervention to date offers a straightforward, curative resolution of back problems (Spitzer *et al.* 1987; AHCPR 1994; Evans and Richards 1996; Croft *et al.* 1997; van Tulder *et al.* 1997b). These are all major reviews conducted in the last decade or so that question the efficacy of a wide range of commonly used interventions.

"Research to date has been insufficiently rigorous to give clear indications of the value of treatment for non-specific LBP patients. No treatment has been shown beyond doubt to be effective. .... There is ... no clear indication of the value of treatments compared to no treatment, or of the relative benefit of different treatments" (Evans and Richards 1996, pp. 2–3).

Specific systematic reviews have been conducted on individual interventions. The use of ultrasound in the treatment of musculoskeletal problems in general has been seriously challenged by all comprehensive systematic reviews to date, which report that active ultrasound is no more effective than placebo (van der Windt *et al.* 1999; Gam and Johannsen 1995; Robertson and Baker 2001). There is no clear evidence for the effectiveness of laser therapy (de Bie *et al.* 1998).

A systematic review found the evidence concerning traction to be inconclusive (van der Heijden *et al.* 1995a), so a randomised sham-controlled trial was constructed avoiding earlier study flaws. Despite

favourable results in a pilot study (van der Heijden *et al.* 1995b), larger numbers and short and long-term follow-up revealed lack of efficacy for lumbar traction (Beurskens *et al.* 1995, 1997).

Results of another systematic review show there was no evidence that acupuncture is more effective than no treatment and some evidence to show it is no more effective than placebo or sham acupuncture for chronic back pain (van Tulder *et al.* 1999).

A recent systematic review of the use of TENS for chronic back pain found no difference in outcomes between active and sham treatments (Milne *et al.* 2001). There would appear to be little role in the management of back pain for such passive therapies.

"No controlled studies have proved the efficacy of physical agents in the treatment of patients who have acute, subacute, or chronic low back pain. The effect of using a passive modality is equal to or worse than a placebo effect" (Nordin and Campello 1999, p. 80).

The lack of efficacy of passive therapies is reinforced by systematic reviews of bed-rest compared to keeping active. There is a consistent finding that bed-rest has no value, but may actually delay recovery in acute back pain. Advice to stay active and resume normal activities as soon as possible results in faster return to work, less chronic disability and fewer recurrent problems. If patients are forced to rest in the acute stage, this should be limited to two or three days (Koes and van den Hoogen 1994; Waddell *et al.* 1997; Hagen *et al.* 2000). Even for sciatica the same rules apply (Vroomen *et al.* 1999).

There is some evidence that non-steroidal anti-inflammatory drugs (NSAIDs) might provide short-term symptomatic relief in cases of acute back pain, but these are not clearly better than ordinary analgesics, and no NSAID is better than another. There is no evidence to suggest that NSAIDs are helpful in chronic back pain or in sciatica (Koes *et al.* 1997; van Tulder *et al.* 2000b).

Several systematic reviews found little evidence for the efficacy of group education or 'back schools' (Di Fabio 1995; Cohen *et al.* 1994; Linton and Kamwendo 1987), but there was some evidence for benefit to chronic back pain patients, especially in an occupational setting (van Tulder *et al.* 1999b).

Several more recent randomised controlled trials would suggest that there is a role for education in the management of back pain (Indahl *et al.* 1998; Burton *et al.* 1999; von Korff *et al.* 1998; Moore *et al.* 2000; Roland and Dixon 1989). These studies used a variety of methods to provide appropriate information about normal activity, self-management and removal of fear of movement, and affected the attitudes and beliefs of several patients, as well as function and behaviour.

In line with the emergence of the concept of patients' attitudes and beliefs influencing illness behaviour, there have been attempts to reduce chronic disability through the modification of environmental contingencies and patients' cognitive processes using behavioural therapy. Systematic reviews suggest that behavioural therapy can be effective when compared to no treatment, but is less clearly so when compared to other active interventions (Morley *et al.* 1999; van Tulder *et al.* 2000c). Compared to a 'treatment as usual' group, one cognitive-behavioural intervention produced a range of improved outcomes of clinical importance, including reducing the risk of long-term sick leave by threefold (Linton and Ryberg 2001).

There have been multiple reviews of manipulation for back pain; there are more reviews than trials (Assendelft *et al.* 1995). Some reviews suggest that manipulation is effective (Anderson *et al.* 1992; Shekelle *et al.* 1992; Bronfort 1999), but others suggest that its efficacy is unproven because of contradictory results (Koes *et al.* 1991, 1996). Even when the conclusion favours manipulation, there are limitations to its value. Most reviews note that the benefit of manipulation is short-term only, and also largely confined to a sub-acute group with back pain only. The value of manipulation in other sub-groups of the back pain population is unclear. If the individual trials are examined in detail, it is also apparent that the treatment effect, when present, is mostly rather trivial, with clinically unimportant differences between the treatment groups. Furthermore, many of the trials reviewed as being about manipulation in fact include non-thrust mobilisation as part of the treatment – often it is unclear exactly which of these interventions is being judged.

Some systematic reviews suggest that the evidence for specific exercises does not indicate they are effective (Koes *et al.* 1991; van Tulder *et al.* 2000a). These reviews include a heterogeneous collection of different types of exercises from which they seek a generalised interpretation of all exercise. Most trials fail to prescribe exercise in a

rational manner to suitable patients, but rather exercises are given in a standardised way. The reviewers show great concern for methodological correctness, but display less understanding of the interventions they are seeking to judge – trials that use extension exercises are considered to be using the McKenzie approach. Hilde and Bo (1998) failed to reach a conclusion regarding the role of exercise in chronic back pain.

Other reviews have been more positive, especially concerning exercises used during the sub-acute and chronic phases (Faas 1996; Haigh and Clarke 1999; Maher *et al.* 1999; Nordin and Campello 1999). Maher *et al.* (1999) concluded that acute back patients should be advised to avoid bed-rest and return to normal activity in a progressive way and that this basic approach could be supplemented with manipulative or McKenzie therapy. For chronic back patients there is strong evidence to encourage intensive exercises.

This brief overview of the literature makes for sobering reading concerning normal physiotherapy practice. For a wide range of passive therapies still being dispensed by clinicians on a regular basis, there is scant supporting evidence. Even for the interventions that receive some support from the literature, namely manipulation, exercise, behavioural therapy and information provision, there is sometimes contradictory or limited evidence.

Informed both by this evidence and by the role that psychosocial factors have in affecting chronic disability, the outlines of an optimal management approach begin to emerge:

- avoidance of bed-rest and encouragement to return to normal activity
- information aimed at making patients less fearful
- seeking to influence some of their attitudes and beliefs about pain
- advising patients how they can manage what may be an ongoing or recurrent problem
- informing patients that their active participation is vital in restoring full function
- encouraging self-management, exercise and activity
- providing patients with the means to affect symptoms and thus gain some control over their problem.

These would appear to be the main themes that should be informing clinical management of back pain.

Conclusions

Our understanding of the problem of low back pain must be guided by certain irrefutable truths:

- Back pain is so common it may be said to be normal. In the way of other endemic problems, such the common cold or dental hygiene problems, resistance to the medicalisation of a normal experience should be allied to a self-management approach in which personal responsibility is engendered.
- The course of back pain is frequently full of episodes, persistence, flare-ups, reoccurrences and chronicity. It is important to remember this in the clinical encounter. Management must aim at long-term benefits, not short-term symptomatic relief.
- Many people with back pain manage independently and do not seek health care. They do this using exercises and postural or ergonomic strategies. Some patients find the adoption of this personal responsibility difficult and may need encouragement. Successful self-management involves the adoption of certain intellectual and behavioural strategies that minimise pain and maximise function.
- The cost of back pain to the health industry and society as a whole is vast. Indirect 'societal' costs absorb the majority of this spending. The direct medical costs are dominated by spending on the chronic back pain population. Therefore, management should be directed to trying to reduce the disability and need for care-seeking in this group by encouraging a self-reliant and coping attitude.
- Back pain is not always a curable disorder and for many is a lifelong problem. No intervention has been shown to alter the underlying prevalence, incidence or recurrence rates. Consequently, management must – and should always – be offering models of self-management and personal responsibility to the patient.

- Passive modalities appear to have no role in the management of back pain. There is some evidence that favours exercise, manipulation, information provision and behavioural therapy.

Given these aspects of back pain, perhaps it should be viewed in light of other chronic diseases in which management rather than curative therapy is on offer. A therapeutic encounter needs to equip the sufferer with long-term self-management strategies as well as short-term measures of symptomatic improvement. It may also be suggested that to do otherwise and treat patients with short-term passive modalities or manipulation, but not equip them with information and strategies for self-management, is ill-conceived and is not in the patients' best interests. If a condition is very common, persistent, often episodic and resistant to easy remedy, it is time patients were fully empowered to deal with these problems in an optimal and realistic fashion. As clinicians, we should be offering this empowerment to our patients.

2: Risk and Prognostic Factors in Low Back Pain

Introduction

Aetiological factors are variables relating to lifestyle, occupation, genetics, individual characteristics and so on that are associated with a higher risk of developing a specific health problem. These factors are identified for study and their occurrence is noted in those who have the outcome of interest (in this case back pain) compared to those who do not. A *risk factor* is a characteristic that is associated with a higher rate of back pain onset. After the onset of symptoms, certain factors may affect the future course of the problem. Again comparisons are made, this time between those who recover quickly and those who have a protracted problem. A *prognostic factor* may be used to predict outcome once an episode has started (Bombardier *et al.* 1994). A poor prognostic factor is suggestive of someone who will have a protracted period of back pain.

Sections in this chapter are as follows:

- risk factors
- individual risk factors
- biomechanical risk factors
- psychosocial risk factors
- all risk factors
- onset
- individual and clinical prognostic factors
- biomechanical prognostic factors
- psychosocial prognostic factors
- all prognostic factors.

Risk factors

Epidemiological studies have generally considered risk factors for the onset of back pain to relate to three dimensions: individual and lifestyle factors, physical or biomechanical factors and psychosocial

factors. Examples of each are given in Table 2.1 (Bombardier *et al.* 1994; Frank *et al.* 1996; Ferguson and Marras 1997).

Table 2.1 Three major classes of risk factors for back pain

Class of risk factor	*Examples*
Individual and lifestyle factors	History of back pain, age, sex, weight, muscle strength, flexibility, smoking status, marital status
Physical or biomechanical factors	Lifting, heavy work, posture, vibration, driving, bending, sitting, twisting
Psychosocial factors	Depression, anxiety, beliefs and attitudes, stress, job satisfaction, relationships at work, control at work

Individual factors have in the past received most scientific attention, but in general their predictive value was low. Ergonomic epidemiology emphasised physical factors, but research has provided only limited evidence of their importance; the focus more recently is upon psychosocial dimensions (Winkel and Mathiassen 1994). This chapter considers the variables that may be risk factors in the onset of back pain, as well as variables that may be prognostic factors in the outcome of an episode of back pain once it has started.

Individual risk factors

The strongest risk factor for future back pain is history of past back pain. This factor is found consistently across numerous studies, indicating its vital predictive role in future episodes (Frank *et al.* 1996; Ferguson and Marras 1997). Frank *et al.* (1996) estimated that an individual with a previous history is three to four times more likely to develop back pain than someone without that history. The epidemiology reviewed in Chapter 1 suggests that more than half of those who have an episode of back pain will have a recurrence.

The association of increasing age and female gender to back pain are less well established. For the majority of other individual factors, such as obesity, smoking or fitness, the evidence is contradictory or scant (Frank *et al.* 1996; Ferguson and Marras 1997; Burdorf and Sorock 1997). In a review of individual risk factors for back pain, the following variables were considered: age, gender, height, weight, strength, flexibility, exercise fitness, leg length discrepancy, posture, Scheuermann's disease, congenital anomalies, spondylolisthesis and

low education (Nachemson and Vingard 2000). There was a striking variability and inconsistency of results when all studies were considered – overall more studies indicated negative or no association between that factor and back pain rather than a positive association. They conclude that none of the variables considered in this review are strong predictors of future back pain.

Biomechanical risk factors

Assessing the role of physical factors in the aetiology of back pain is not straightforward, and as a consequence there have been conflicting reports over its importance. Various problems exist in the studies that have been done (Bombardier *et al.* 1994; Dolan 1998; Frank *et al.* 1996; Burdorf 1992). Much of the literature in this area is cross-sectional in nature; that is, risk factors and prognostic factors are measured at the same time as noting the presence or absence of back pain. This means that it is often difficult to determine if a factor contributed towards onset or towards prognosis. It also means that although a factor may be associated with back pain, we cannot be sure that it caused it. Prospective studies are better at identifying causation.

Furthermore, the measurement of exposure to a possible risk factor, such as frequent lifting, may be imprecise if based on self-report or job title rather than direct, objective measurement. There is also the 'healthy worker' effect, when those who have survived in an occupation without developing back pain will always be over-represented compared to those who had to leave the job because of back pain (Hartvigsen *et al.* 2001). This will tend to downplay the importance of mechanical factors.

In general there has been a failure to measure the different dimensions of exposure to a physical factor – degree of exposure, frequency and duration; thus invalid exposure assessment may fail to expose a relationship between mechanical factors and symptoms (Winkel and Mathiassen 1994).

These methodological problems with the literature on biomechanical risk factors for back pain have probably led to an under-reporting of their role, such that the association between these factors and back pain may well be stronger than was previously imagined (Dolan 1998). Hoogendoorn *et al.* (2000a) conducted a high-quality study using a

prospective design in which exposure levels were actually measured rather than estimated, and psychological variables and other physical risk factors were accounted for in the analysis. Their results showed a positive association between trunk flexion and rotation at work and back pain, with a greater risk of pain at greater levels of exposure.

Taken individually, the reports provide only weak evidence of causation, but the consistency of reporting of certain factors and the strength of association between these factors and back pain is supportive of a definite relationship between biomechanical exposures and the onset of back problems (Frank *et al.* 1996; Burdorf and Sorock 1997).

Individual studies have shown certain mechanical factors to be associated with back pain or disc prolapse:

- repeated bending and lifting at work (Damkot *et al.* 1984; Videman *et al.* 1984; Kelsey *et al.* 1984a; Frymoyer *et al.* 1983; Marras *et al.* 1993; Waters *et al.* 1999; Zwerling *et al.* 1993)
- repeated bending at home (Mundt *et al.* 1993)
- prolonged bending (Punnett *et al.* 1991; Hoogendoorn *et al.* 2000a)
- unexpected spinal loading (Magora 1973)
- driving (Kelsey 1975; Kelsey *et al.* 1984b; Frymoyer *et al.* 1983; Damkot *et al.* 1984; Krause *et al.* 1997; Masset and Malchaire 1994)
- sedentary jobs (Kelsey 1975)
- a high incidence of back pain has been found in those who spend a lot of their working day either sitting or standing, but was much less common in those who were able to vary their working positions regularly during the day (Magora 1972).

Pheasant (1998) summarised the work done by Magora, which identified two distinct groups of people most at risk of back pain. In those whose jobs were physically very demanding and those whose jobs were essentially sedentary, about 20% of individuals experienced back pain. Those whose jobs entailed varied postures, some sitting and some standing, and were moderately physically active were at a much lower risk, with only about 2% of this group experiencing back pain.

Several large-scale reviews of the relevant literature have been conducted (Frank *et al.* 1996; Bombardier *et al.* 1994; Burdorf and

Sorock 1997; Ferguson and Marras 1997; Hoogendoorn *et al.* 1999; Vingard and Nachemson 2000). Ferguson and Marras (1997) included fifty-seven studies investigating risk factors; Burdorf and Sorock (1997) included thirty-five publications. Occupational physical stresses that have been found to be consistently and in general strongly associated with the occurrence of back pain across multiple systematic reviews are as follows:

- heavy or frequent lifting
- whole body vibration (as when driving)
- prolonged or frequent bending or twisting
- postural stresses (high spinal load or awkward postures).

Frank *et al.* (1996) estimated the relative risk of back pain associated with heavy lifting and whole body vibration to be three to four times normal, and that of spinal loading, postural stresses and dynamic trunk motion to be more than five times normal.

Psychosocial risk factors

The role of psychological and social dimensions as prognostic factors for chronic back pain and disability is now well known and is considered later in the chapter. Epidemiological studies addressing psychosocial risk factors as a cause of back pain are far fewer than those investigating physical factors. Low job satisfaction, relationships at work, including social support, high job demand, monotony or lack of control at work, stress and anxiety are factors that have an association with back pain in several studies, although the evidence for these factors is often weak or contradictory (Burdorf and Sorock 1997; Ferguson and Marras 1997). There are equal numbers of studies that are negative and show no relation between these psychosocial variables and back pain (Vingard and Nachemson 2000).

The role of low job satisfaction as a risk factor for back pain may be partly a product of less rigorous study designs that have failed to account for other psychosocial factors and physical work load (Hoogendoorn *et al.* 2000b). One study found that while work dissatisfaction was associated with a history of back pain, it was not related to the onset of back pain (Skovron *et al.* 1994). Two prospective studies indicate that low levels of perception of general health are predictors of new episodes of back pain (Croft *et al.* 1996, 1999).

Severe back pain has been found to be less prevalent among those with a higher socio-economic status, after physical work factors have been accounted for (Latza *et al.* 2000).

A review of psychosocial factors at work concluded that due to methodological difficulties in measuring variables, there is no conclusive evidence for psychosocial variables as risk factors for back pain, but that monotonous work, high perceived work load and time pressure are related to musculoskeletal symptoms in general (Bongers *et al.* 1993).

Frank *et al.* (1996) estimate that psychosocial factors have a weak relative risk for the occurrence of back pain, one to two times more likely than normal. Linton (2000b) made a thorough review of psychological risk factors for neck and back pain. He concluded that there was strong evidence that these factors may be associated with the *reporting* of back pain, and that attitudes, cognitions, fear–avoidance and depression are strongly related to pain and disability; however, there is no evidence to support the idea of a 'pain-prone' personality.

All risk factors

The evidence would suggest that individual, physical and psychosocial factors all could have an influence upon back pain onset. Studies that have included different factors have found that back pain is best predicted by a combination of individual, physical and psychosocial variables (Burton *et al.* 1989; Thorbjornsson *et al.* 2000). One prospective study found that physical and psychosocial factors could independently predict back pain (Krause *et al.* 1998), while another found that distress, previous trivial back pain and reduced lumbar lordosis were all consistent predictors of any back pain (Adams *et al.* 1999).

Most studies, however, have investigated a limited set of risk factors and have not assessed the relative importance of different variables. If risk estimates are not adjusted for other relevant risk factors, the overall effect may be to under- or over-estimate the role of particular variables (Burdorf and Sorock 1997). Research has only recently begun to address the relative contribution to back pain onset of individual, biomechanical and psychosocial factors together.

In terms of the relative importance of these different factors, several studies have shown that a history of trivial or previous back pain is a much stronger predictor of serious or future back pain than job satisfaction or psychological distress (van Poppel *et al.* 1998; Papageorgiou *et al.* 1996; Mannion *et al.* 1996; Smedley *et al.* 1997). After adjusting for earlier history, one study found that risk of back pain in nurses was still higher in those reporting heavier physical workload (Smedley *et al.* 1997). In a review of risk factors for occupational back pain, it was concluded that biomechanical factors are more significant factors of causation than psychosocial ones (Frank *et al.* 1996). In another review it was concluded that whereas the strength of psychosocial factors as risk indicators was strongly affected by sensitivity analysis, the role of physical load factors as risk indicators is more consistent and insensitive to slight changes in analysis (Hoogendoorn *et al.* 2000b).

If risk factors were clinically important, they would explain a large proportion of the predictive variables associated with back pain; however, even at best this is not so. The proportion of new episodes attributable to psychological factors at the most has been found to be 16% (Croft *et al.* 1996); another study found this to be only 3% (Mannion *et al.* 1996). While job dissatisfaction has been highlighted as a risk factor for back pain, in the original study that identified this, most of those who reported never enjoying their job did not in fact report back pain (Bigos *et al.* 1991). When all risk factors have been considered together, only between 5% and 12% of back pain has been explained (Mannion *et al.* 1996; Adams *et al.* 1999).

It is apparent that there are no simple causal explanations for back pain and that individual, physical and psychosocial factors may, to varying degrees, all have a role in aetiology. However, at most these factors, individually
of back pain. A past history of back pain is the factor most consistently associated with future back pain.

Onset

Although mechanical factors are associated with back pain and can therefore be seen as predisposing factors, onset is not always related to a specific event. Patients often report the precipitating factor involved flexion activities, such as lifting and bending. Generally,

however, more patients report back pain that commenced for no apparent reason (Kramer 1990; Videman *et al.* 1989; Kelsey 1975; Laslett and Michaelsen 1991; McKenzie 1979). Both Kramer (1990) and Waddell (1998) found that about 60% of patients in their clinics developed pain insidiously.

Hall *et al.* (1998) examined the spontaneous onset of back pain in a study group of over 4,500 – and 67% could not identify a specific event that triggered their symptoms. By contrast, in a group that was required to report a specific causal event for compensation purposes only, 10% failed to attribute their pain to an incident. The authors considered spontaneous onset to be part of the natural history of back pain. The rate of spontaneous onset was greater in the sedentary employment group (69%) than the heavy occupation group (57%). McKenzie's clinic records also demonstrated the effect of compensation requirements on causal attribution. In 1973, in 60% of patients the onset of back pain was reported as 'no apparent reason'. After the introduction of a national compensation scheme in New Zealand, onset was related to an accident by 60% of patients (unpublished data). Whenever the patient is unable to recollect a cause for the onset of their symptoms, which clearly is common, the role of normal, everyday activities in precipitating the onset of symptoms should be considered.

The degree to which contemporary lifestyles are dominated by activities that involve flexion should thus be borne in mind; this may be sustained as in sitting or often-repeated motions such as bending. From the moment we wake and put on our socks, clean our teeth, go to the toilet, dry ourselves after a shower, sit down to eat breakfast, drive to work, sit at the desk, stoop over a bench or sit to eat lunch until the time in the evening when we 'relax' – either sitting on the sofa to watch television or play computer games, read or sew – we are in flexed postures of varying degrees. It would appear that these normal activities not only predispose people to back pain, but also can precipitate symptoms with no additional strain and can perpetuate problems once they arise (McKenzie 1981).

"Sitting is the most common posture in today's workplace, particularly in industry and business. Three-quarters of all workers in industrial countries have sedentary jobs" (Pope *et al.* 2000, p. 70). About 45% of employed Americans work in offices. Many display poor posture

and report increased pain when sitting, which is more severe the less they are able to change positions. Occupational back pain has long been associated with sedentary work, especially the seated vibration environment when driving (Pope *et al.* 2000). However, the vibration studies fail to discriminate between the effects of vibration and the effects of the sustained seated posture.

Individual and clinical prognostic factors

History of previous back pain is both a risk factor for future back pain and a prognostic factor for prolonged symptoms. Reported leg pain at onset is associated with poor outcomes and a greater likelihood of developing chronic symptoms (Goertz 1990; Lanier and Stockton 1988; Chavannes *et al.* 1986; Cherkin *et al.* 1996a; Carey *et al.* 2000; Thomas *et al.* 1999). Centralisation of leg pain, which is discussed elsewhere, has been shown to be a predictor of good outcomes (Donelson *et al.* 1990; Sufka *et al.* 1998; Long 1995; Werneke *et al.* 1999; Karas *et al.* 1997). The value of centralisation compared to other demographic and psychosocial variables has not been evaluated until recently. Inability to centralise the pain was found to be the strongest predictor of chronicity, compared with a range of psychosocial, clinical and demographic factors (Werneke and Hart 2000, 2001).

Biomechanical prognostic factors

As is seen from the assessment of risk factors, the physical variables that have been analysed do not explain a substantial amount of back pain onset, nor has the ergonomic approach brought dramatic benefits (Hadler 1997). However, once back pain has started, the same physical tasks become difficult and painful to do and will frequently affect symptoms. Even if the role of mechanical factors in onset is obscure, the ability of physical loading strategies to aggravate and relieve symptoms is quite pronounced.

Biomechanical factors are important both in the causation of an episode of back pain and in its perpetuation and aggravation (McKenzie 1972, 1981; Kramer 1990; Adams and Dolan 1995; Dolan 1998). The majority of spinal pain is seen as varying in intensity with the patient's activity and is *almost always aggravated by mechanical factors* (Spitzer *et al.* 1987). Indeed, this important report referred to *activity-related spinal disorders*, with the clear assumption

of the importance of day-to-day activities and postures that influence patients' pain.

Various reports have investigated the role of physical loading strategies in symptom response – these highlight the effect that normal mechanical loads, such as sitting, walking or lying, have on aggravating or relieving symptoms (Table 2.2).

Table 2.2 Aggravating and relieving mechanical factors in those with back pain

	McKenzie 1979	*Painting and Chester 1996*	*Biering-Sorensen 1983b*	*Boisson-nault and Di Fabio 1996*	*Stankovic and Johnell 1990*
1. Aggravating					
Sitting	82%	83%	30%	74%	46%
Rising from sitting					39%
Bending		83%	65%	79%	46%
Driving				55%	
Sedentary		79%			
Walking	16%	48%			18%
Standing		37%	26%		24%
Sneezing or Coughing			22%		
Lying		61%		49%	7%
2. Relieving					
Sitting					7%
Rising from sitting					0%
Bending					2%
Walking	79%	41%	36%	38%	32%
Standing		45%			17%
Changing position / on the move		71%		53%	
Lying		32%	53%	70%	83%

These studies illustrate the mechanical sensitivity that back pain displays to different loading strategies. They reveal a range of different responses to the same loads – either worsening or improving symptoms, or having no effect. However, a common picture is of

symptoms aggravated in positions involving flexion (sitting, rising from sitting, bending, driving) and improvement when walking, or being generally active, which are postures of extension. Alternatively, a smaller group of patients have their pain aggravated by standing and walking.

Common identified physical risk factors that predispose to back pain involve flexion – lifting, bending, driving and sitting; the precipitating event, however, is frequently trivial and unrelated. Once back pain has been initiated, postures involving flexion frequently perpetuate the problem and prevent resolution. This is not the only pattern of response to mechanical loading strategies, but clinically it is extremely common. Several clinical studies have demonstrated the value of avoiding flexion activities and postures (Williams *et al.* 1991; Snook *et al.* 1998; Snook 2000) – see Chapter 11 for detail.

"Controlling early morning lumbar flexion is a form of self-care that can help develop a sense of control or mastery over low back pain, and thereby build confidence and improve outcome" (Snook *et al.* 1998, p. 2606). McKenzie (1981) had previously identified the morning as a time when patients were frequently worse and at risk of suffering a relapse or exacerbation.

Psychosocial prognostic factors

While the evidence implicating psychosocial factors in the onset of back pain is limited, there is considerably more evidence relating these factors to the transition from acute to chronic back pain.

A cutting-edge review on fear–avoidance and its consequences concluded that pain-related fear and avoidance appear to be an essential feature in the development of a chronic problem for a substantial number of patients with musculoskeletal pain (Vlaeyen and Linton 2000).

A systematic review of psychological risk factors in back and neck pain concluded that these factors play a significant role in the transition to chronic problems and also may have a role in the aetiology of acute problems (Linton 2000a). Fuller conclusions based on the evidence from thirty-seven studies and supported by two or more good-quality prospective trials were as follows:

- psychosocial variables are clearly linked to the transition from acute to chronic pain and disability
- psychosocial factors are associated with reported onset of pain
- psychosocial variables generally have more impact than biomedical or biomechanical factors on disability
- cognitive factors (attitudes, cognitive style, fear–avoidance beliefs) are related to the development of pain and disability – especially passive coping, catastrophising, and fear–avoidance
- depression, anxiety and distress are related to pain and disability
- self-perceived poor health is related to chronic pain and disability
- psychosocial factors may be used as predictors of the risk of developing long-term pain and disability.

Emotional, cognitive and behavioural dimensions were specifically identified as being important in these studies; nonetheless, these factors only account for a proportion of the variance. Other factors are known to be important and psychosocial factors must be seen as part of a complex multidimensional view of musculoskeletal problems. Although it is tempting to conclude a causal relationship between these factors and the outcomes, this may be incorrect. The reciprocal nature of psychological factors and spinal pain has created a 'which came first, the chicken or the egg' dilemma – did the individual's depressive nature predispose them to back pain or did the persistent back pain produce depression? Nonetheless, efforts should be made to incorporate this information into clinical practice to enhance assessment and management (Linton 2000a).

All prognostic factors

Numerous factors have been associated with chronic back pain and failure to return to work. Generally these relate to three different aspects of a patient's presentation – clinical, psychological and social factors. Psychosocial factors that may have a role in the development of chronic musculoskeletal pain and disability are known as 'yellow flags'.

Table 2.3 lists the factors that have been associated with chronic back pain, disability or failure to return to work (Abenhaim *et al.* 1995; Klenerman *et al.* 1995; Gatchel *et al.* 1995; Philips *et al.* 1991; Burton *et al.* 1995; Cherkin *et al.* 1996a; Deyo and Diehl 1988a;

Hasenbring *et al.* 1994; Potter and Jones 1992; Potter *et al.* 2000; Goertz 1990; Hellsing *et al.* 1994; Williams *et al.* 1998b; Lanier and Stockton 1988; Pedersen 1981; Chavannes *et al.* 1986; Carey *et al.* 2000; Weiser and Cedraschi 1992; van der Giezen *et al.* 2000; Werneke and Hart 2000).

Table 2.3 Factors associated with chronic back pain and disability

Clinical factors	*Psychological factors*	*Social factors*
Leg pain	Fear–avoidance behaviour	Lower educational level
Nerve root pain	Depression	Lower income
Previous history of back pain	Anxiety about pain	Heavy manual work
Disc herniation	Passive coping strategies	Sitting occupation
Specific diagnosis	Catastrophising	Lack of alternative work duties to return to
Higher levels of reported pain and disability	Low self-efficacy beliefs External health locus of control beliefs	Low job satisfaction
Lack of centralisation	Poor general health Higher levels of reported pain and disability	Over-protective spouse

Factors that cause acute pain to become chronic are clearly complex, multiple and heterogeneous between individuals. The more sophisticated studies, which include a range of potential risk factors, suggest that chronic symptoms are predicted more by psychological than by clinical factors, or a combination of both (Burton *et al.* 1995; Klenerman *et al.* 1995; Gatchel *et al.* 1995; Deyo and Diehl 1988a; Hasenbring *et al.* 1994; Thomas *et al.* 1999). These studies suggest that chronic back pain disability and persistent symptoms are associated with a combination of clinical, psychological and social factors.

It is now widely accepted that psychological and social factors play a role in the maintenance of illness as pain moves from the acute to the chronic stage. It is further proposed that the patient-clinician relationship also has a role to play in the patient's recovery, with inappropriate advice or management preventing or prolonging recovery. Likely iatrogenic factors leading to disability include overemphasis on pain, and over-prescription of rest and time off work (Weiser and Cedraschi 1992). Failure to achieve centralisation has been highlighted as an important clinical prognostic factor that could be more significant than psychosocial ones (Werneke and Hart 2000, 2001).

Conclusions

This chapter has looked at some of the individual, biomechanical and psychosocial factors involved with precipitating and perpetuating episodes of back pain. One of the strongest risk factors for a future episode of back pain is a past history of back pain – such patients need education and information to reduce this risk. Biomechanical variables are risk factors in back pain onset, but also are notable in the perpetuation and aggravation of symptoms. Many of these relate to postures of flexion; the ubiquitous nature of this common posture has been noted.

There are some recent suggestions that psychological factors may predispose to back pain onset in a few individuals, and there is stronger evidence for the role of these factors in perpetuating episodes of back pain. Such factors may confound the effects of treatment. Management strategies need to recognise the possible existence of these 'yellow flags' and develop appropriate responses. The need for active patient involvement in management would appear to be paramount. A thorough explanation of the problem and how they can best manage it, reducing fears about movement, improving control and self-efficacy and avoiding passive interventions help this to happen. See Chapter 18 on patient management for a fuller description.

3: Pain and Connective Tissue Properties

Introduction

Pain is usually the prime concern of the patient, and so some means of understanding and interpreting pain is important. This chapter reviews certain aspects of pain that are relevant to the lumbar spine. The distinction between nociception and the pain experience is made; the most common sources of pain in the lumbar spine are identified; the differences between pain of somatic and neural origin, between local and referred pain, and also between pains initiated by mechanical or chemical mechanisms are made. The distinction between these two mechanisms of pain is an important determinant of the appropriateness of mechanical therapy (McKenzie 1981, 1990). In musculoskeletal problems a common cause of inflammation follows soft tissue trauma; the healing process of inflammation, repair and remodelling is also described. Some consideration is also given to chronic pain.

Sections in this chapter are as follows:

- nociception and pain
- sources of back pain and sciatica
- types of pain
- activation of nociceptors
- mechanical nociception
- chemical nociception
- trauma as a cause of pain
- distinguishing chemical and mechanical pain
- tissue repair process
- failure to remodel repair tissue
- chronic pain states.

Nociception and pain

The means by which information concerning tissue damage is experienced and transmitted to the cortex is termed *nociception*. This has several components (Bogduk 1993):

- the detection of tissue damage (transduction)
- the transmission of nociceptive information along peripheral nerves
- its transmission up the spinal cord
- modulation of the nociceptive signals.

The nerve endings that detect pain are not specialised receptors. Normally they are involved with other sensory functions, but as the stimulus becomes noxious, the graded response of the receptors crosses the threshold from normal mechanical or thermal sensation and triggers the nociceptive process (Bogduk 1993). After tissue damage is detected, this information is transmitted via the peripheral and central nervous system to the cortex; however, en route the nociceptive message is modulated. In this way the central nervous system can exert an inhibitory or excitatory influence on the nociceptive input (Henry 1989; Walsh 1991; Charman 1989). Given the current understanding of pain, the classical concept of pain being a straightforward reflection of specific tissue damage is outmoded (Waddell 1998). Especially with patients who have chronic pain, the factors that influence the clinical presentation are more than simple nociception.

Pain has been defined as *"an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage"* (Merskey 1975). This much-quoted and widely accepted definition recognises that the experience of pain is a cortical phenomenon (Bogduk 1993; Adams 1997) and is influenced by affective and cognitive factors as well as sensory ones (Henry 1989; Johnson 1997; LaRocca 1992; Waddell 1998).

It is important to recognise that the experience of pain involves patients' emotional and cognitive reactions to the process of nociception. Patients' anxieties, fears and beliefs can strongly determine their response to injury, pain and treatment. Fear of pain and re-injury may lead to avoidance of activities that are believed will do more harm. It may lead patients to restrict their actions and movements and to withdraw from their normal lifestyle. An exaggerated fear of

pain coupled with a hypervigilance to every minor discomfort can lead the patient into a perpetual circle of disuse, depression, disability and persistent pain (Vlaeyen and Linton 2000). Such lack of understanding of their condition causes inappropriate action in the face of pain and produces feelings of limited ability to control or affect the condition. Such avoidance in the long term will have a deleterious effect on the patient's recovery (Philips 1987).

We can start to address these factors by providing patients with a thorough understanding of their problem and educating them in the appropriate use of activity and exercise to regain function and reduce pain. Facilitating patients' control over their problem, encouraging active coping strategies and helping them confront their fear of pain should all be part of management (Klaber Moffett and Richardson 1995). Strategies based upon education and patient activity are important as a means of addressing patient responses to a painful condition as well as the condition itself.

Sources of back pain and sciatica

Any structure that has a nerve supply is capable of triggering the nociceptive process. This means that possible sources of pain around the lumbar region are the capsules of the zygapophyseal and sacro-iliac joints (SIJ), the outer part of the intervertebral discs, the interspinous and longitudinal ligaments, the vertebral bodies, the dura mater, nerve root sleeve, connective tissue of nerves, blood vessels of the spinal canal and local muscles (Bogduk 1994a, 1997; Butler 1991; Bernard 1997). The wide distribution of nociceptors around the lumbar spine makes it impossible to devise testing procedures that selectively stress individual components of the spinal segment.

An interesting insight into the most common sources of back pain and sciatica is provided by the progressive local anaesthetic studies performed by Kuslich *et al.* (1991) in patients undergoing surgery for decompression operations for disc herniations or spinal stenosis. In 193 consecutive patients who were awake or lightly sedated, each successive tissue was stimulated prior to anaesthetisation and incision and the area of provoked pain was recorded.

Table 3.1 Pain production on tissue stimulation in 193 patients in order of significance

Tissue	*Number tested*	*Number and % some pain*	*Significant pain*	*Area of provoked pain*
Always painful				
Compressed nerve root	167	166 (99%)	90%	Buttock, leg, foot
Often painful				
AF/PLL* – central	183	135 (74%)	15%	Central back
AF – lateral	144	102 (71%)	30%	Lateral back (buttock)
Vertebral endplate	109	67 (61%)	9%	Back
Dura – posterior				
Anterior	92	21 (23%)	6%	Buttock, leg
	64	15 (23%)	5%	Back, buttock
Rarely painful				
Facet capsule	192	57 (30%)	2.5%	Back (buttock)
Supraspinous ligament	193	49 (25%)	0%	Back
Interspinous ligament	157	10 (6%)	0.5%	Back
Muscle	193	80 (41%)	0%	Back
Never painful				
Fascia	193	32 (17%)	0.5%	Back
Spinous process	193	21 (11%)	0%	Back
Lamina	193	2 (1%)	0%	Back
Ligamentum flavum	167	0	0%	
Facet synovium	186	0	0%	
Nucleus pulposus	176	0	0%	

*AF/PLL = annulus fibrosus / posterior longitudinal ligament

Source: Kuslich *et al.* 1991

This study identifies compressed nerve roots as *the* source of significant leg pain, and the outer annulus fibrosus as *the* source of significant back pain, while all other anatomical sources of pain appear to be much less relevant. Normal nerve roots were rarely painful; it is only once the root has become compressed, stretched or swollen that pain was reproduced. The findings of this study accord with earlier work involving pain provocation studies around the time of surgery that identified the nerve root as the source of patients' limb pain and the intervertebral disc as the source of their spinal pain (Fernstrom 1960; Smyth and Wright 1958; Falconer *et al.* 1948; Wiberg 1949; Cloward 1959).

More recent studies have also shown the major role of the intervertebral disc as a cause of back pain (Schwarzer *et al.* 1995d), while other structures, such as the zygapophyseal and sacro-iliac joints, have a more limited aetiological significance (Schwarzer 1994b, 1995a).

Types of pain

One proposed pain classification system has suggested the following broad categories of pain (Woolf *et al.* 1998):

- transient pain, which is of brief duration and little consequence
- tissue injury pain
- nervous system injury pain.

Tissue injury pain relates to somatic structures, while nervous system injury pain includes neurogenic or radicular and pain generated within the central nervous system. The other source of pain that occasionally must be considered in the differential diagnosis is visceral pain from organs.

Table 3.2 Basic pain types

Pain type	*Structures involved*
Somatic pain	Musculoskeletal tissue
Radicular pain	Nerve root / dorsal root ganglion / dura
Central pain	Central nervous system
Visceral pain	Visceral organs

Somatic pain

Somatic structures include the intervertebral disc, posterior longitudinal ligament, SIJ, zygapophyseal joint capsule, etc. Only pain that originates from cutaneous tissue is felt localised to the area; all pain that stems from deep somatic structures is referred pain to a greater or lesser extent (Bogduk 1993, 1994a). The deeper the structure, the more difficult it is to localise the pain source – therefore most musculoskeletal pain is referred pain to a varying degree. The brain is simply aware of pain signals emanating from those structures that are supplied by a certain segment of the spinal cord. The mechanism for this is known as convergence. Neurons in the central nervous system receive afferents from structures in the lumbar spine and from the lower limb. The brain is unable to determine the true source of

nociceptor signals from the shared neuron (Oliver and Middleditch 1991; Bogduk 1997). The pain is perceived deeply in the area appropriate to the deep segmental innervation of the body. This is more closely related to myotomes, the segmental innervation of muscles, than to dermatomes (Table 3.3).

Table 3.3 The segmental innervation of the lower limb musculature

Major muscle groups	*Segmental innervation*
Anterior	
Hip flexors / adductors	L2, 3, 4
Knee extensors	L3, 4
Foot and ankle extensors / invertors	L4, 5
Posterior	
Hip extensors / abductors	L4, 5, S1
Knee flexors	L5, S1
Foot and ankle flexors / evertors	L5, S1, 2

Source: Bogduk 1993

However, the segmental distribution of referred pain patterns should not be rigidly interpreted. There is considerable overlap between different segments in one individual, and considerable variation between individuals, so these areas should not be thought of as universally consistent locations. Furthermore, dorsal horn cells have the ability to increase their receptive field following injury, a mechanism by which the sensation of pain can spill over segmental boundaries (Gifford 1998).

Referred pain simply reflects the lack of localising information available with nociceptor activity from deep structures. The quality of somatic referred pain is deep and aching in quality, vague and hard to localise. The deeper the structure the more vaguely distributed and widespread is the pain (Bogduk 1993, 1994a). The stronger the noxious stimulus, the further the pain spreads down the limb (Kellgren 1939; Inman and Saunders 1947; Mooney and Robertson 1976).

Somatic pain can originate from any innervated tissues, but unfortunately it is impossible to localise the source of pain by the pattern of referred symptoms. Symptomatic intervertebral discs, zygapophyseal joints and SIJ are all capable of referral below the knee, but there are no clear distinguishing characteristics of the pain pattern or clinical

features that are pathognomonic of any of these conditions (Schwarzer *et al.* 1994b, 1995a, 1995b; Dreyfuss *et al.* 1996).

Radicular pain

Radicular or neurogenic pain is produced when the nerve root or dorsal root ganglia are involved in symptom production. This is the product of pressure on nerve roots that are already inflamed or irritated, not on normal nerve roots. Although sudden onset of sciatica does occur, experimentally tension or pressure has only reproduced radicular pain on sensitised, not normal nerve roots (Smyth and Wright 1958; Kuslich *et al.* 1991).

It is different in quality from somatic pain, and is frequently associated with other abnormalities of nerve conduction such as weakness or numbness and abnormal tension tests (Bogduk 1994a; Cavanaugh 1995). Radicular pain is severe and shooting in quality, felt along a narrow strip, and thus different in quality from the vague, dull aching associated with somatic-referred pain. All nerve root pain will be felt in the leg, and it is always referred pain; often the leg pain will be worse than any back pain that may be present. However, all leg pain is *not* nerve root pain (Rankine *et al.* 1998). Radicular pain tends to be distributed in dermatomal patterns, with the L4, L5 and S1 nerve roots most commonly affected. Typically pain from L4 is felt down the anterior aspect of the thigh and leg, L5 is down the lateral aspect and S1 down the posterior aspect – however, variety exists, and pain patterns are not rigid.

Pain may be distributed anywhere in the dermatome in patches, or in a continuous line. The distal pain is often worse. Motor and sensory abnormalities are not always present; root tension signs are earlier and more common than signs of root compression (Waddell 1998). Signs and symptoms of root compression present as muscle weakness or wasting, absent or reduced reflexes and areas of paraesthesia, pins and needles or numbness. Sensory disturbance, when present, is found in the distal part of the dermatome – thus on the medial shin for L4, the great toe for L5 and the lateral border of the foot for S1.

Combined states

Referred pain is thus either somatic or radicular in origin. These two states may be combined in one individual (Bogduk 1994a). For instance, a patient may have back pain of somatic origin, from

pressure of the annulus fibrosus, and leg pain of radicular origin, which is caused by involvement of the nerve root.

Central pain

Another form of neurogenic pain may arise from cells within the central nervous system, known as central pain. Classic examples of this are phantom limb pain, post-herpetic neuralgia and the pain from a brachial plexus lesion. There is growingngpeculation that in some musculoskeletal pains, especially chronic conditions, central mechanisms may be more important in the maintenance of symptoms than peripheral nociception (Bogduk 1993). Pain in this instance would be the result of abnormalities within the central nervous system.

A barrage of nociceptive input from a peripheral source, either somatic or radicular, can lead to sensitisation of central neurones. This is characterised by reduced thresholds and increased responses to afferent input, heightened responses to repeated stimuli, expansion of receptive fields and spontaneous generation of neuronal activity. Normal mechanical pressure can be interpreted as pain, and pain can be perceived without any appropriate peripheral input (Cavanaugh 1995; Gifford 1998; Johnson 1997; Siddall and Cousins 1997; Dubner 1991).

Visceral pain

Viscera may also refer pain – for example, renal pain may be felt in the loin and inguinal region, and cardiac pain in the arm (Bogduk 1993; Oliver and Middleditch 1991).

Activation of nociceptors

Only three mechanisms are known that can activate nociceptors – thermal, mechanical and chemical (Bogduk 1993; Zimmerman 1992; Cavanaugh 1995; Weinstein 1992). It is the latter two that are our concern here.

Mechanical nociception

Pain may be produced in the absence of actual tissue damage by excessive mechanical strain or tension upon collagen fibres. This is thought to be the result of the deformation of collagen networks so that nerve endings are squeezed between the collagen fibres with the excessive pressure perceived as pain (Bogduk 1993). No damage to

the tissues need have occurred, and when the stress is removed the pain will abate. Mechanical pain can ensue from normal stresses upon weakened, damaged or abnormal tissues. If the excessive strain is so great as to produce actual tissue damage the inflammatory process will be provoked.

A simple example of mechanical articular pain is readily at hand. Bend your left forefinger backwards, using your right forefinger to apply overpressure. Keep applying this pressure until the nociceptive receptor system indicates its enhanced active state by the arrival of pain. This is simple mechanical deformation of pain sensitive structures. If you bend the finger backwards further, the intensity of the pain will increase; and if you maintain the painful position longer, the pain will become more diffuse, widespread and difficult to define. Thus, pain alters with increasing and prolonged mechanical deformation. If you now slowly return the finger to its normal resting position, the pain will disappear. This example has one significant implication: the finger is obviously being moved in one direction as the pain increases, and in another direction as the pain decreases.

Once the finger is returned to its normal position, the pain ceases. In this instance the sensation of pain does not depend on the existence of pathology. Mechanical forces sufficient to stress or deform local nociceptors produced the intermittent pain. The nociceptor system was activated by the application of mechanical pressure, and as soon as this was withdrawn, the nociceptors returned to their normal quiescent state. Intermittent low back pain can be caused in this same manner, by end-range mechanical stress. No chemical treatment will rectify or prevent pain arising from mechanical deformation. When intermittent mechanical pain is the main presenting symptom, drugs should never be the treatment of choice (McKenzie 1981).

"There are no drugs available that can inhibit the transduction of mechanical nociception. It is therefore futile to attempt to treat mechanical nociception with peripherally acting drugs. Mechanical transduction can only be treated by correcting the mechanical abnormality triggering nociception" (Bogduk 1993, p. 80).

Chemical nociception

In this situation pain is produced by the irritation of free nerve endings in the presence of certain chemicals, such as histamine, serotonin, hydrogen ions, substance p and bradykinin. These chemicals are released as a result of cell damage or by cells associated with the inflammatory process. Therefore, except in the case of inflammatory or infective diseases and certain degenerative conditions, chemical pain only occurs following trauma and actual tissue damage.

Trauma as a cause of pain

Pain due to trauma is produced by a combination of mechanical deformation and chemical irritation. Initially, mechanical deformation causes damage to soft tissues, and pain of mechanical origin will be felt. In most instances this is a sharp pain. Shortly after injury chemical substances accumulate in the damaged tissues. As soon as the concentration of these chemical irritants is sufficient to enhance the activity of the nociceptive receptor system in the surrounding tissues, pain will be felt.

In most instances pain of chemical origin will be experienced as a persistent discomfort or dull aching as long as the chemicals are present in sufficient quantities. In addition, the chemical irritants excite the nociceptive receptor system in such a way that the application of relatively minor stress causes increased pain that under normal circumstances would not occur. Thus, at this stage there is a constant pain, possibly a mild aching only, which may be enhanced but will never reduce or cease due to positioning or movement. As the concentration of chemical irritants falls below the critical threshold, this may be replaced by tenderness and increased sensitivity to mechanical stimulation, with intermittent pain with normal stress or periods of constant pain following excessive activity (Bogduk 1993; Saal 1995).

Distinguishing chemical and mechanical pain

As the cause of pain is an important determinant of the appropriateness of mechanical therapy, it is vital to distinguish between mechanical and chemical sources of nociception (McKenzie 1981, 1990). We can begin to distinguish between these types of

pain by certain factors gained during the history-taking and largely confirm this impression during the physical examination. A key characteristic that indicates the possibility of pain of chemical origin is constant pain. Not all constant pain is inflammatory in nature, but chemical pain is always constant. The term ***constant pain*** indicates that the patient is ***never*** without an ache or discomfort from the moment they wake until the moment they fall asleep. The ache may be exacerbated by movements and be less at times, but the dull, relentless ache never goes away entirely. Constant pain may result from chemical or mechanical causes or be due to the changes associated with chronic pain.

Key factors in the identification of pain of an inflammatory nature:

- constant pain
- shortly after onset (traumatic or possibly insidious)
- cardinal signs may be present – swelling, redness, heat, tenderness
- lasting aggravation of pain by all repeated movement testing
- no movement found that reduces, abolishes or centralises pain.

Key factors in identifying constant pain of mechanical origin:

- certain repeated movements cause a lasting reduction, abolition or centralisation of pain
- movements in one direction worsens symptoms, whereas movements in the other direction improves them
- the mechanical presentation improves with the symptoms.

Intermittent pain is almost certainly mechanical in origin and is generally easier to treat than constant pain. During normal daily activities the patient is causing sufficient mechanical stresses to trigger nociceptive signals, which may persist after that activity has ceased. They may also be performing certain activities or sustaining certain postures that reduce mechanical deformation sufficiently to abolish their symptoms temporarily. This sensitivity to mechanical forces, in which different activities and postures both aggravate and reduce symptoms, is a notable characteristic of most back pain – consequently the terms ***mechanical backache*** (CSAG 1994) and ***activity-related spinal disorders*** (Spitzer *et al.* 1987).

Tissue repair process

Following tissue injury, the process that in principle leads to recovery is divided into three overlapping phases – inflammation, repair and remodelling (Evans 1980; Hardy 1989; Enwemeka 1989; Barlow and Willoughby 1992). *"No inflammation / no repair is a valid dictum"* (Carrico *et al.* 1984). In fact, each part of this process is essential to the structure of the final result. Connective tissue and muscle do not regenerate if damaged, but are replaced by inferior fibrous scar tissue (Evans 1980; Hardy 1989). To produce optimal repair tissue, all phases of this process need to be completed in the appropriate time.

Stages of Healing:

1. Inflammation
2. Tissue repair
3. Remodelling

Inflammation

In response to tissue damage, a host of inflammatory cells with specialist functions are released and attracted to the damaged area. There is increased local blood supply, leaking of plasma proteins and leukocytes from the blood vessels, and accumulation of white cells at the site of the injury (Enwemeka 1989; Evans 1980). These cells will be involved in the clearance of dead and dying cells and any foreign matter prior to the regrowth of new vascular channels and nerves into the damaged area. The cardinal signs of inflammation, heat, redness, pain, swelling and lack of function may be displayed (Evans 1980) and are a result of the inflammatory exudate. Swelling, heat and redness are products of the vascular activity; the pain results from the presence of noxious inflammatory chemicals and heightened mechanical sensitivity.

Just as tissue damage always causes inflammation, so inflammation always causes the tissues to become hypersensitive (Levine and Taiwo 1994). The inflammatory irritants sensitise the local pain receptor system and lower the thresholds at which the system is triggered, creating a state of 'peripheral sensitisation' (Cousins 1994; Woolf 1991). In this situation, the application of relatively minor mechanical stresses causes pain that under normal circumstances would not occur – allodynia; noxious stimuli create exaggerated responses – primary hyperalgesia; and there may be a spread of hyper-responsiveness to

non-injured tissue – secondary hyperalgesia (Cousins 1994; Levine and Taiwo 1994). At this stage, there will be aching at rest, tenderness and exaggerated pain on touch and movement (Levine and Taiwo 1994). Movement can superimpose mechanical forces on an existing chemical pain and increase it, but it will never reduce or abolish chemical pain. This is significant in the differentiation between chemical and mechanical pain. Repeated movements will cause a lasting worsening of symptoms (McKenzie 1981).

Because of this heightened sensitivity, there is a lack of correlation between mechanical stimuli and the intensity of the pain response – it hurts much more than it should (Woolf 1991). When acute, this response is normal and it encourages protective, immobilising actions that are appropriate immediately after injury and during the inflammatory stage. Rest at this point has the important effect of reducing exudate and protecting the injured tissue from further damage. The same response at a later stage of the healing process does not serve any useful purpose, and is in fact detrimental. Only during the inflammatory period are rest and relative rest required; this must be followed by early mobilisation to optimise tissue healing. It is at this stage, however, when individuals learn the habit of avoiding activities because they hurt. If this habit is prolonged and individuals develop the habit of avoidance of painful movements, the repair process will be retarded, remodelling will not occur, normal function will not be restored and persistent symptoms are likely.

The aching will progressively lessen and healing and repair begin during the first seven to ten days after injury. Inflammatory cells, which are the source of chemically mediated pain, decrease in numbers until by the third week after injury none are present (Enwemeka 1989). *The patient will experience constant pain and tenderness until such time as the healing process has sufficiently reduced the concentration of noxious irritants.* The situation can occur during healing in which the level of chemicals falls below the threshold that triggers nociception, although tenderness would still be present. Normal mechanical loads may sufficiently irritate the tissues so as to re-trigger a constant chemical ache. Thus aching that abates, but is easily reproduced, represents an interface between mechanics and a resolving inflammatory state. If this is the case, tenderness should still be present. By two to three weeks, the constant pain due to chemical irritation should have abated and be replaced by a pain felt intermittently only when the repair itself is stressed.

In this initial stage a mesh of fibrin forms from the protein fibrinogen in the inflammatory exudate and seals the injury. During this time the application of ice, compression, elevation and gentle muscle movements are indicated to reduce the inflammatory exudate (Evans 1980). The greater the amount of exudate, the more fibrin will be formed and the more inextensible will be the repair. Ice, if applied in the first few days following injury, can reduce pain and oedema. Ice is of little value after the fifth day as the inflammatory cells are replaced by fibroblasts. These soon begin to lay down fibrils of collagen.

Tissue repair

The fibroplastic or repair stage commences as the acute inflammatory stage subsides and lasts about three weeks (Enwemeka 1989). It is during this phase that the collagen and glycosaminoglycans that will replace the dead and damaged tissue are laid down. There is cellular proliferation, which results in a rapid increase in the amount of collagen, and damaged nerve endings and capillaries 'sprout' and infiltrate the area (Cousins 1994). The cellular activity is stimulated by the physical stresses to the tissue. With inactivity, collagen turnover occurs and new collagen is made, but it is not oriented according to stress lines. At the end of this phase fibrous repair should be established, collagen mass is maximal, but the tensile strength of the new tissue is only 15% of normal (Hardy 1989).

To encourage good quality repair with collagen fibres oriented according to stress lines, gentle natural tension should be applied to recent injuries, commencing at about the fifth day (Evans 1980). Gentle tension applied early in the healing process promotes greater tensile strength in the long-term. From the first week a progressive increase in movement should be encouraged so that full range is possible by the third or fourth week. *It is within this period that appropriate education and movement provides the optimal climate for uncomplicated repair.* An experimental animal model showed that the application of stress during this repair phase was able to change the length of scar tissue and thus remodel it according to function. The same stresses applied to scar tissue that was three months old had little effect on its length (Arem and Madden 1976).

It should be noted, however, that at this stage if an over-enthusiastic approach to treatment is adopted the repair process can be delayed or disrupted, and the presence of inflammatory chemical irritants and exudate will be prolonged or re-stimulated. During this early

stage of healing, movements should be just into stiffness and pain and entirely under the patient's control. Any discomfort provoked by the movement should abate as soon as the movement is released. If lasting pain is provoked, it is likely that re-injury has occurred, the inflammatory phase has been re-triggered and resolution of the problem will be further delayed.

Remodelling

Wound repair is only optimal if remodelling of the scar tissue occurs. This involves increases in strength and flexibility of the scar tissue through progressive increased normal usage and specific loading. Remodelling is the process of turning weak, immature and disorganised scar tissue into a functional structure able to perform normal tasks. The repair is unlikely to achieve the strength of the original tissue, but progressive loading and mechanical stimulation enhances the tensile strength and improves the quality of the repair. This occurs over several months after the original injury. Tensile strength is increased by stabilisation of the fibres through cross-linking, alignment of the fibres along the lines of stress and synthesis of type I collagen (Barlow and Willoughby 1992; Witte and Barbul 1997).

An animal model of healing following an induced rupture of a medial collateral ligament illustrates the role of scarring in tissue repair (Frank *et al.* 1983). All ligaments healed by scar tissue bridging the gap; this healing occurred quickly, with granulation tissue filling the rupture by ten days and signs of remodelling being noted after three weeks. Histologically collagen cross-links were significantly abnormal in the scar area, with increasing cross-links between ten days and six weeks, and return to normal values only seen at forty weeks. The scar started to contract three weeks after injury. At forty weeks scarring was still obvious to the naked eye; local hypertrophy and adhesions between the injury site and surrounding tissues were still present, but less than previously. Scar tissue was mechanically inferior to normal tissue, with lower failure properties, and persisting changes in quantitative and qualitative collagen and non-collagen matrix.

Several factors can operate to promote a less than optimal repair. The granulation tissue, which repaired the damage, can now act as glue to prevent movement between tissue interfaces. During the period when collagen turnover is accelerated, there is also increased molecular cross-linkage – these processes can produce adhesion formation and impair collagen gliding (Hunter 1994; Donatelli and

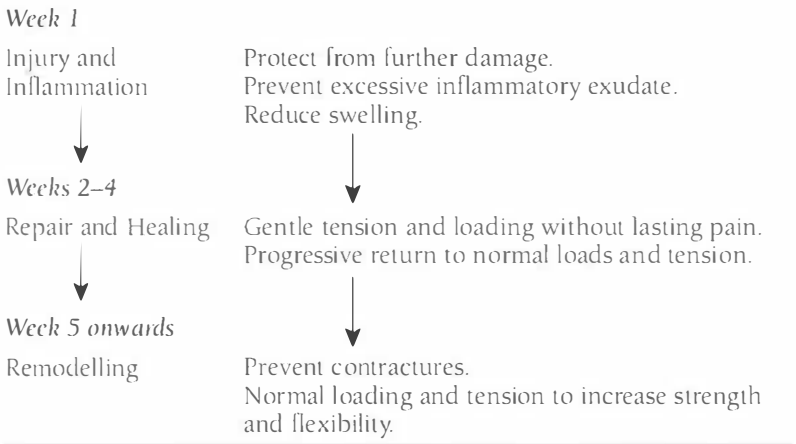
Owens-Burkhart 1981). Newly synthesised collagen will tend to contract after three weeks; this naturally occurring shrinkage is said to continue for at least six months (Evans 1980). Thus, recently formed scar tissue commences shortening unless it is repeatedly stretched. Provided the stretching process is commenced in the early stages following injury and continued well after full recovery, no soft tissue shortening is likely to develop. Low load regular application of stress also helps to increase the tensile strength of the repair tissue (Hardy 1989). Failure to perform the appropriate tissue loading will leave the repair process complete, but the remodelling stage incomplete – the individual may still be bothered by pain and limited function and the tissue weak and prone to re-injury. The nerves, which infiltrated the tissue during repair, can now be sources of pain each time the scar is stretched or loaded. This is a common cause of persistent symptoms in many patients.

The regular application of *intermittent* stress or loading to bone and normal soft tissue enhances structural integrity through the process of remodelling. During the healing process, loading for *prolonged* periods must be avoided as this may disrupt the repair process. *Prolonged stress damages, intermittent stress strengthens* (McKenzie 1981). Thus the proper rehabilitation of tissue damage involves progressive, incremental loading and activity to restore the structure to full function and to restore the patient's confidence to use it. This is the essential management strategy during the repair and remodelling stages.

In summary, no injury can be made to heal faster than its natural rate; whenever there has been tissue damage, the processes of inflammation, repair and remodelling have to occur to allow full restoration of normal function. *"Failure of any of these processes may result in inadequate or ineffectual repair leading to either chronic pathological changes in the tissue or to repeated structural failure"* (Barlow and Willoughby 1992). These processes are essentially the same in tendons, muscles, ligaments and all soft tissues; however, intrinsic factors may be more likely to impair the recovery process in tendon injuries, especially if the onset is through overuse rather than trauma (Barlow and Willoughby 1992). Early, progressive, active rehabilitation is essential to optimise repair and function. No passive modality used within physiotherapy has yet been shown to reduce the time for the completion of natural healing. We can avoid delay to

the healing process and ensure that the climate for repair is favourable (Evans 1980). Strenuous mechanical therapy applied when the pain from the injury is essentially chemical will delay recovery. The integrity of the repair must be established before more vigorous procedures are applied. However, of equal importance is the use of a progressive, controlled programme of loading the tissues at the appropriate time during the repair process in order to promote a fully functional structure *that the patient is confident to use*.

Figure 3.1 Matching the stage of the condition to management



Failure to remodel repair tissue

Following tissue damage, an important factor in the physiology of repair is the phenomenon of contracture of connective tissues. A characteristic of collagen repair is that it will contract over time. Recently formed scar tissue will always shorten unless it is repeatedly stretched, this contracture occurring from the third week to the sixth month after the beginning of the inflammation stage. Contracture of old scar tissue may in fact occur for years after the problem originated (Evans 1980; Hunter 1994). Cross-linkage between newly synthesised collagen fibres, at the time of repair, can act to prevent full movement. Nerve endings infiltrate this area during the repair process and thus can make the scar tissue a sensitised nodule of abnormal tissue (Cousins 1994).

In some patients contracture resulting from previous injury can now prevent the performance of full range of motion. These patients will have been unwilling to stretch the recent injury, perceiving the 'stretch'

pain as denoting further damage, and they will not have received appropriate rehabilitation advice around the time of the injury. They will present later with restricted range of movement and pain provoked by stressing the scar tissue. The tissue will become progressively more sensitised and deconditioned for normal function with lack of use. A similar functional impairment may affect contractile tissues, and although this may restrict end-range flexibility, it is most commonly exposed with resisted movements that stress the muscle or tendon.

In such cases the remodelling of collagen by applying a long-term structured exercise programme will be necessary. By applying regular stress sufficient to provide tension without damage, collagen undergoes chemical and structural changes that allow elongation and strengthening of the affected tissue. Because tissue turnover is slow, one must recognise it may be a slow process. If the contracture has been present for some time, the remodelling programme will have to be followed for several months; Evans (1980) reports that some patients may have to exercise for the remaining years of their life. Applying tension to old injuries should be routinely practised, especially prior to participation in sporting activities (Hunter 1994). The animal experiment of Arem and Madden (1976) showed that 'old' scar tissue might be unresponsive to a remodelling programme. Well-established contractures, especially where the original healing process has been interrupted by repeated re-injury, causing the production of more inflammatory exudate, can be resistant to improvement.

Chronic pain states

Chronic pain is different in quality, as well as time, from acute pain. In the latter, biomechanical and biochemical factors may be the dominant influences on the pain experience and there is a more straightforward relationship between pain and nociception. With the passage of time, neurophysiological, psychological and social factors may come to dominate the maintenance of pain, and the link to the original tissue damage may become minimal (Waddell 1998; Adams 1997). The plasticity of the central nervous system following a barrage of peripheral input can cause pathological changes that maintain the pain state in the absence of peripheral pathology (Johnson 1997; Siddall and Cousins 1997). Psychological and behavioural attitudes

and responses, as well as the process of nociception, shape individuals' experience of pain (Waddell 1998).

The acute and sub-acute model of tissue injury and healing described earlier is not an appropriate model for an understanding of chronic pain. If pain persists beyond the normal healing time, other factors can exist that complicate the picture (Johnson 1997). Persistent peripheral nociceptive input can induce changes in the central nervous system (Woolf 1991; Melzack 1988). This can lead to the sensitisation of neurones in the dorsal horn – a state characterised by reduced thresholds and increased responses to afferent input, such that normal mechanical stimuli is interpreted as pain. As well, there may be heightened responses to repeated stimuli, expansion of receptor fields, and spontaneous generation of neuronal activity (Johnson 1997; Siddall and Cousins 1997; Dubner 1991; Cousins 1994). This is known as *central sensitisation*.

Nociceptive signals can also be initiated in altered parts of the peripheral or central nervous system, which can produce the effect of localised 'phantom' pain in a part of the periphery where tissue damage no longer exists (Bogduk 1993). Pain can radiate to be felt in uninjured areas adjacent to the original problem (secondary hyperalgesia), normal movement can be painful (allodynia), repeated movements can exaggerate pain responses and pain signals can fire off without any appropriate stimulus (ectopic pain signals).

Psychosocial factors certainly have a role in peoples' response to a painful experience and can also be important in maintaining chronic pain (Bogduk 1993; Johnson 1997). Factors affecting pain responses are cultural, learned behaviour, meaning of pain, fear and anxiety, neurotocism, lack of control of events, passive coping style and focus on the pain (Cousins 1994). A recent systematic review of psychological risk factors in back and neck pain concluded that these factors play a significant role in the transition to chronic problems and can also have a role in the aetiology of acute problems (Linton 2000b). Psychosocial and cognitive factors are closely related to the development of chronic back disability. Depression, anxiety, passive coping and attitudes about pain are related to pain and disability. Catastrophising, hyper-vigilance about symptoms and fear–avoidance behaviour are attitudes and beliefs that have been highlighted as being particularly significant in this context. These psychosocial factors, which can have prognostic significance, are termed 'yellow flags'.

These psychological characteristics are thought to be key factors in the chronic pain experience. Chronic pain patients often feel little or no control over the pain, a helplessness that tends towards anxiety and depression, which in its turn can make people more concerned about symptoms (Adams 1997). The fear–avoidance model proposes that some individuals react to a pain experience by continued avoidance of any activity that they think might hurt, long after rest is of any therapeutic value, leading ultimately to disability and exaggerated pain behaviour (Lethem *et al.* 1983). The value of this model in predicting chronicity in back pain patients has been demonstrated (Klenerman *et al.* 1995; Waddell *et al.* 1993). It is proposed that this avoidance of pain is driven by a concept that pain equals further damage, leading the patient to further rest and avoidance of activity (Hill 1998).

There are thus neurophysiological and psychological reasons that are capable of maintaining painful states beyond the normal time-scale (Meyer *et al.* 1994; Cousins 1994). The patient with a chronic condition can not only be experiencing persistent pain, but also be distressed, inactive, deconditioned and have unhelpful beliefs about pain. They can be overly passive and reliant on others and possibly suffering economic and social deprivations due to the impact of the condition on their lifestyle (Nicholas 1996). The prevalence of this chronic pain syndrome is unknown; it possibly is a factor in those whose pain has persisted for months or years (Johnson 1997). Such a state may cloud the diagnostic and therapeutic usefulness of mechanically produced symptom responses (Zusman 1994). Therefore, there exist in some patients with chronic pain conditions various factors that can confound attempts to resolve the problem and can muddy the waters of diagnosis and symptom response.

Although these complicating factors can undermine treatment attempts, many patients with persistent symptoms will respond to mechanical therapy and a mechanical assessment should never be denied patients according to the duration of their symptoms. However, in patients with persistent symptoms there is a need to recognise the possible importance of non-mechanical pain behaviour. This can involve peripheral sensitisation, central sensitisation or psychosocially mediated pain behaviour, or any combination of these factors, which will obscure or complicate any purely mechanical approach. The causes of chronic pain are different from the causes of

acute pain. Although both problems can encourage reduction of normal activities and produce disability, in the acute stage this can be proportionate and appropriate whereas in the chronic stage this is inappropriate and irrelevant.

Clinicians' behaviour towards patients at all stages of a condition should guard against encouraging any passive responses to pain – especially so in the chronic patient. It is hardly surprising that patients get depressed, anxious, fearful and focussed on their persistent pain. Often health professionals seem unable to deal with it, some of whom imply it is primarily 'in their heads', as the pain is *"apparently discordant with discernible abnormalities"* (Awerbuch 1995). Maladaptive or inappropriate behaviour in the face of ongoing pain states does not represent malingering; it should be remembered that on the whole, the emotional disturbance is more likely to be a consequence of chronic pain rather than its cause (Gamsa 1990).

Although only a very small proportion of back pain patients develop chronic intractable pain (Waddell 1998), given the complexity of the pain experience, treatment in the acute stage should defend against chronic disability and in the chronic stage should be cognisant of psychological and behavioural dysfunction.

Conclusions

This chapter has considered aspects of pain that are relevant to a consideration of musculoskeletal pathology. It must be recognised that pain and nociception are different entities and that an individual's pain experience can be affected by cognitive, emotional or cultural as well as somatic factors. The multiplicity of factors that can affect the pain experience is especially relevant in chronic pain states when psychosocial and/or neurophysiological factors can dominate the patient's pain experience and militate against easy resolution of the problem.

In terms of pathology, the source of most back and radiating pain is one of the various innervated structures in or around the lumbar spine, with the intervertebral disc probably the most important. Less frequently, radicular pain is the product of nerve root involvement also. Nociceptors are activated by mechanical and/or chemical mechanisms, a differentiation between which is crucial in the use of mechanical diagnosis and therapy. An understanding of the stages of

the repair process that follows tissue trauma is essential. When patients present with painful musculoskeletal problems, this can be due to different conditions in peripheral or central structures, with the pain maintained by different mechanisms (Table 3.4). Within several states a distinction can be made between pains of somatic or radicular origin.

Table 3.4 Pain-generating mechanisms

State of tissues	*Pain mechanism*
Normal	Abnormal stress – mechanical
Inflamed (acute)	Predominantly chemical – somatic and/or radicular
Healing (sub-acute)	Chemical / mechanical interface
Abnormal (contracted / scar tissue)	Mechanical – somatic and/or radicular
Abnormal (derangement)	Mechanical – somatic and/or radicular
Persisting hypersensitivity (chronic)	Peripheral / central sensitisation
Barriers to recovery (acute to chronic)	Psychosocial factors

An understanding of the different pain mechanisms that can pertain in different patients allows a broader perspective of the different factors that might need to be addressed in management.

4: The Intervertebral Disc

Introduction

This chapter presents aspects of anatomy and pathology that are relevant to an understanding of discogenic pain. It examines morphological changes that occur in the intervertebral disc and their relevance to back pain. This focuses chiefly on radial fissures through the annulus and disc herniations. The study of *biomechanics*, a term introduced by Breig (1961), is closely related to functional anatomy; it means the study of changes in anatomical structures occurring during movements of the body. Of most relevance to the concept presented here are the biomechanics of the intervertebral disc, the effects that abnormal morphology have on these biomechanics and the combined role that biomechanics and structural disruptions have in the creation of pathology.

The chapter is divided into the following sections:

- structural changes
- innervation
- mechanical or chemical pain
- diagnosing a painful disc
- the mobile disc
- discogenic pain
- radial fissures
- disc herniation
- stress profilometry.

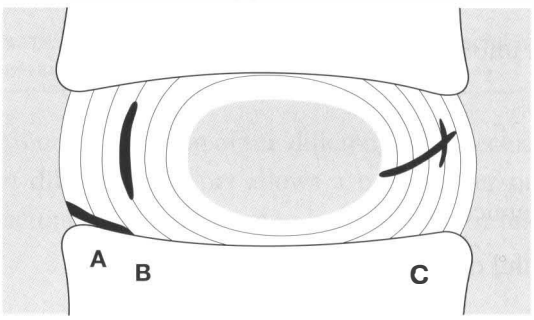
Structural changes

As ageing occurs, the morphology of the i
certain normal structural changes that make the disc more vulnerable to symptomatic pathology (Kramer 1990). Biochemical changes in the disc start early and continue throughout life – these changes involve the drying out of the disc, an increase in collagen and decrease in elastin. The net result is that the disc as a whole becomes more

fibrous. Cells exhibiting necrosis increase; the distinction between the annulus fibrosus and the nucleus pulposus becomes blurred. The nucleus functions less efficiently at distributing radial pressure evenly to the annulus. In turn, the annulus fibrosus comes to bear increasing vertical loads. This has an effect on the structural integrity of the disc (Bogduk 1997). Distortion, disruption and fissuring occur in the layers of the annulus fibrosus. Three types of fissures (Figure 4.1) are commonly found in the annulus fibrosus (Hirsch and Schajowicz 1953; Yu *et al.* 1988a; Osti *et al.* 1992):

- transverse tears or rim lesions, with rupture of Sharpey's fibres in the periphery of the annulus near the ring apophysis, or in the outer wall of the annulus
- circumferential tears between the lamellae of the annulus
- radial fissures cutting across the layers of the annulus.

Figure 4.1 Commonly found fissures of the annulus fibrosus



Key:
A. Rim lesions
B. Circumferential tears
C. Radial fissures

There is desiccation and loss of coherence in the nucleus pulposus (Yu *et al.* 1989). The homogenous structure of the disc may be disrupted as the nucleus becomes more fibrous, desiccated and disintegrated, and discrete fibrous lumps of nucleus or annulus may appear (Adams *et al.* 1986; Brinckmann and Porter 1994; Yu *et al.* 1988b; Kramer 1990). The degenerative changes are frequently visible in both parts of the disc together, with the drying out and disintegration of the nucleus pulposus often associated with radial fissures and disruption of the annulus fibrosus (Yu *et al.* 1989).

Much of this altered morphology, including quite gross changes in structure, will be asymptomatic as the inner two-thirds of the annulus fibrosus and the whole of the nucleus pulposus is without innervation.

Innervation

There is ample evidence going back many years that the intervertebral disc is innervated; this is reviewed by Bogduk (1994b, 1997). In general it has been found that the nucleus pulposus and the inner two-thirds of the annulus fibrosus are without nerve endings, which only exist in the outer third, or less, of the annulus. For instance, in samples obtained from patients undergoing back operations, receptors have been found in the outer half and the outer 3mm of the annulus (Yoshizawa *et al.* 1980; Ashton *et al.* 1994). Nerve endings are present in all aspects of the outer annulus, but not uniformly – nerve endings are found most frequently in the lateral region of the disc, a smaller number in the posterior region and the least number anteriorly (Bogduk 1997). Nerve endings are also found in the anterior and posterior longitudinal ligaments (Bogduk 1997).

There is evidence that in painful and degenerated discs the innervation can be much more extensive (Coppes *et al.* 1997; Freemont *et al.* 1997). In eight out of ten severely degenerated and painful discs, the innervation extended into the inner two-thirds of the annulus, and in two out of ten to the periphery of the nucleus pulposus (Coppes *et al.* 1997). Freemont *et al.* (1997) found considerable variety in the extent of innervation of the discs they studied, which were from patients with chronic back pain. Nerves extended into the inner third of the annulus in nearly half and into the nucleus pulposus in nearly a quarter.

Mechanical or chemical pain

It has been suggested that either mechanical or chemical mechanisms could initiate discogenic pain (Bogduk 1997). Plenty of evidence exists for mechanical disc problems; two possible means by which pain is produced are discussed below. These relate to radial fissures and internal disc derangements. In the presence of radial fissures, with or without a displacement, excessive mechanical stress would be placed upon the remaining intact portions of the annulus. The fissures would disrupt the normal even distribution of load-bearing on the annulus fibrosus and disproportionate loads would be borne by the residual, innervated outer lamellae. The stress peaks recorded by stress profilometry (see later section) could be examining the same phenomenon. Alternatively, internal displacements of discal material, whose position could be influenced by spinal postures, could exert

pressure on the intact outer, innervated part of the annulus. Such displacements if unchecked could progress to full-blown disc herniations. In both instances pain is the result of excessive mechanical loads on weakened tissue.

An alternative model suggests a chemical rather than a mechanical mechanism of disc pain (Derby *et al.* 1999; Bogduk 1997). Chemical nociception may occur if nerve endings in the annulus are exposed to inflammatory cells. With severe back pain, patients' cells associated with chronic inflammation have been found in the anterior annulus (Jaffray and O'Brien 1986). It is proposed that chemical discogenic pain can be detected when concordant pain is provoked at very low pressures on discography (Derby *et al.* 1999). In seventy-eight chronic back pain patients undergoing discography and surgical fusion, a chemical mechanism detected in this way was believed to be responsible for symptoms in about half of the sample.

Pain from a nerve root may also be caused by mechanical or chemical mechanisms, or a combination (Garfin *et al.* 1995; Olmarker and Rydevik 1991; Rydevik *et al.* 1984). Disc herniations or stenosis may cause compression or tension leading to oedema, impairment of nutritional transport and subsequent intraneural damage and functional changes in nerve roots. This may result in inflammation of the nerve or produce nutritional compromise and ischaemia. In patients undergoing surgery for disc herniations, inflammatory cells have been harvested from around the nerve root (Gronblad *et al.* 1994; Spiliopoulou *et al.* 1994; Doita *et al.* 1996; Takahashi *et al.* 1996). Experiments using animal models have indicated the inflammatory effect of nucleus pulposus beyond the annular wall (McCarron *et al.* 1987; Olmarker *et al.* 1993).

However, the presence of inflammatory cells is variable. In patients investigated at surgery, such cells were found abundantly in about 60 – 70% of individuals (Gronblad *et al.* 1994; Doita *et al.* 1996) and a complete absence of inflammatory cells at surgery has also been noted (Cooper *et al.* 1995). Furthermore, animal experiments using only mechanical factors have been shown to produce histological and physiological abnormalities consistent with radicular pain following compression of the nerve root and dorsal root ganglion (Howe *et al.* 1977; Triano and Luttges 1982; Rydevik *et al.* 1989; Hanai *et al.* 1996; Yoshizawa *et al.* 1995), the dorsal root ganglion

being especially sensitive to abnormal loads, which rapidly induce heightened mechanical sensitivity.

Another mechanism that may explain whether radicular pain is mechanical or chemical in origin relates to the type of disc herniation. One study found some inflammatory cells were present in up to 50% of patients with sequestrations. In patients with extrusions and protrusions, about 30% and 25%, respectively, had some inflammatory cells (Virri *et al.* 2001). Inflammatory cells were also more common when a positive straight leg raise was present, especially if bilaterally positive.

The literature would thus suggest that either mechanical or chemical mechanisms might be the source of patients' symptoms. The prevalence of each at present is unknown. These different mechanisms will respond differently to therapeutic loading strategies. An appropriate mechanical evaluation in the presence of a mechanical problem should generate a favourable response, while in the presence of a chemically maintained problem symptomatic response will be unfavourable.

Diagnosing a painful disc

It is not entirely clear why discs become painful; there are several models that have been used to describe the cause of internal disc pain (Bogduk 1997; Kramer 1990; McNally *et al.* 1996; Crock 1970, 1986). One of the key confounding factors in the debate about the cause of back pain is the existence of morphological abnormality in asymptomatic populations.

A systematic review of studies about radiographs and back pain concluded that although radiographic findings indicating disc degeneration are associated with back pain, this does not indicate a causal relationship (van Tulder *et al.* 1997c). More detailed imaging studies with magnetic resonance imaging (MRI), found 'abnormal discs' (bulging or herniated) in 20 – 76% of asymptomatic populations that were studied (Boden *et al.* 1990; Jensen *et al.* 1994; Weinreb *et al.* 1989; Boos *et al.* 1995). Patterns of disc disruption, including fissures and herniations, have been seen as commonly in volunteers as in patients with back pain (Buirski and Silberstein 1993). In a particularly thorough study, in which patients with sciatica were

matched with volunteers without back pain by age, sex and physical risk factor, 76% of those with no symptoms had a disc herniation and 22% had one that involved the nerve root (Boos *et al.* 1995). However, the proportion of patients with symptoms who had nerve root compression was significantly greater – this was 83%.

In fact, MRI is often not particularly good at determining what is a painful disc when compared to invasive methods such as discography. This actually seeks to reproduce the patient's pain by injecting into the disc (Horton and Daftari 1992; Brightbill *et al.* 1994; Ricketson *et al.* 1996; Simmons *et al.* 1991). Discography involves physical stimulation of the disc through needle placement, which is correlated with morphological abnormalities and pain response (Sachs *et al.* 1987). In volunteers without back pain, discography is not particularly painful (Walsh *et al.* 1990). It has been an essential tool in revealing the significance of radial fissures in the annulus fibrosus as a cause of chronic back pain (Vanharanta *et al.* 1987; Moneta *et al.* 1994). However, extensive radial fissures, which are strongly associated with back pain, are also found not to be a cause of pain in some individuals and at some segmental levels (Smith *et al.* 1998).

Despite continuing controversy, discography is still seen by many authorities to be the only certain way of identifying symptomatic discogenic pain as long as stimulation of a control disc at an adjacent level does not reproduce their pain (Bogduk 1997; Schwarzer *et al.* 1995d).

The study by Donelson *et al.* (1997) has shown the reliability of a mechanical assessment of patients' pain response to predict the presence of discogenic pain and the competency of the annular wall. The assessment process was superior to MRI scanning in distinguishing painful from non-painful discs – this study is described in more detail later.

The mobile disc

Asymmetrical loading of the disc tends to displace the nucleus pulposus to the area of least pressure (McKenzie 1981; Kramer 1990; Bogduk 1997). Thus the anterior compression caused by flexion 'squeezes' the nucleus backwards, and conversely extension forces it forwards. This effect has been confirmed in cadaveric experiments (Shah *et al.* 1978; Krag *et al.* 1987; Shepperd *et al.* 1990; Shepperd

1995) and in living subjects using various imaging techniques (Schnebel *et al.* 1988; Beattie *et al.* 1994; Fennell *et al.* 1996; Brault *et al.* 1997; Edmondston *et al.* 2000). All these studies have shown a posterior displacement of the nucleus pulposus with flexion and an anterior displacement accompanying extension of the lumbar spine. In vivo experiments have been almost entirely conducted in asymptomatic volunteers. In the one attempt to study nuclear movement in a symptomatic population, pain changes were not found to correlate with movement of the nucleus (Vanharanta *et al.* 1988). It would seem that movement of the nucleus becomes less predictable when the disc becomes more degenerated (Schnebel *et al.* 1988; Beattie *et al.* 1994).

Based on experimental work carried out by his team, and clinical experience, Kramer (1990) has written in some detail about the mobile disc. Displacement occurs most rapidly in the first three minutes of asymmetrical loading, but will continue for several hours at a slower rate if the asymmetrical compression is maintained. Because of the more fibrous nature of the nucleus pulposus with advancing age, it is displaced less easily in older individuals. The nucleus pulposus that has been displaced by asymmetrical loading returns to its original position once the loading is released. If the loading on the disc is sustained, the displaced nucleus has a tendency to remain in its abnormal position, but its return can be facilitated by *compression in the other direction.*

"Postures of the spine which result in decentralization of the nucleus pulposus due to asymmetrical loading of the intervertebral segment play an important role in the pathogenesis and in the prophylaxis of intervertebral disc diseases" (Kramer 1990, p. 29).

There may come a point when the natural resilience of the disc to recover from asymmetrical loading is undermined by structural changes within the disc. *"The intervertebral disc becomes vulnerable when tears and attritional changes cause the annulus fibrosus to lose its elasticity and allow the central gel-like tissue of the nucleus pulposus to be displaced beyond its physiological limits"* (Kramer 1990, p. 29).

If the internal architecture of the disc is intact, displacement is soon reversed on returning to a symmetrical posture. However, the changes that occur during ageing make the disc more vulnerable to symptomatic pathology (Kramer 1990). In the presence of radial

fissures, displacements can exert pressure on the outer annulus, which is innervated. As long as this holds, the displacement can be reversed, but if it weakens sufficiently or ruptures, the displacement herniates through the outer annulus. An intact hydrostatic mechanism in the disc is thus essential to influence any displaced tissue. If the outer annular wall is intact, the hydrostatic mechanism is also intact and displaced tissue can be affected by loading. However, once the outer wall is ruptured or so attenuated as to be incompetent, then movements and positions will have no lasting effect on displaced discal tissue.

Discogenic pain

As an innervated structure, the intervertebral disc is capable of being a source of pain in its own right. Studies involving discography have shown that internal disc disruption, with intact outer annular walls and no mass effect beyond the disc wall, can be a painful entity (Bernard 1990; Park *et al.* 1979; Milette *et al.* 1995, 1999; McFadden 1988; Schellhas *et al.* 1996; Grubb *et al.* 1987; Horton and Daftari 1992; Fernstrom 1960; Wiley *et al.* 1968; Wetzel *et al.* 1994; Colhoun *et al.* 1988; Ohnmeiss *et al.* 1997). These studies show that discogenic pain, without nerve root involvement, can be the cause of back and leg pain.

Direct stimulation of the disc carried out during surgical procedures also demonstrates the entity of discogenic pain (Wiberg 1949; Kuslich *et al.* 1991; Smyth and Wright 1958). In these studies, back pain only was produced; sciatic leg pain could only be reproduced by stimulation of swollen, stretched or compressed nerves. Buttock pain was reproduced, with difficulty, on simultaneous stimulation of the nerve root and annulus (Kuslich *et al.* 1991). However, other studies involving mechanical stimulation of discs have been able to reproduce leg pain (Fernstrom 1960; Murphey 1968), although only in a minority (Fernstrom 1960).

The site of the referred pain depended on the site where the annulus was being stimulated. The central annulus and posterior longitudinal ligament produced central back pain, while stimulation off centre produced lateral pain to the side being stimulated (Kuslich *et al.* 1991). Cloward (1959) found the same direct correlation between the site of stimulus and the site of referred pain, central or lateral, in his experiments with cervical disc patients. Murphey (1968) found that on stimulation of the lateral part of the disc, patients reported leg pain.

Kuslich *et al.* (1991) found considerable variability in the sensitivity of the annulus. Although they were unable to explain why, they suggested this could be the result of differing innervation or levels of chemical irritants. One-third of patients were exquisitely tender upon stimulation of the annulus, one-third were moderately tender and one-third were insensitive. Various other tissues were stimulated in this study involving 193 sedated but awake patients, from which the authors concluded that the intervertebral disc is *the* cause of back pain (see Table 3.1 for more detail). Fernstrom (1960), also found disc sensitivity to be variable with just over half of 193 discs responding painfully to pressure. A possible cause of this symptomatic variability is the inconsistency that is present relative to the extent and presence of innervation in the disc.

Radial fissures

When discography is combined with computerised axial tomography (CAT) scans, it permits four separate categories of information (Sachs *et al.* 1987). These relate to generalised degeneration, annular disruption, pain response (pressure sensation, dissimilar pain, similar pain or exact reproduction of pain), volume of contrast medium injected into the disc and other comments. The extent of fissures in the annulus is gauged from the spread of the contrast medium, which is assessed by CAT scans. Originally four grades of ruptures or fissuring were listed (Sachs *et al.* 1987); later authors have suggested additions (Table 4.1, Figure 4.2).

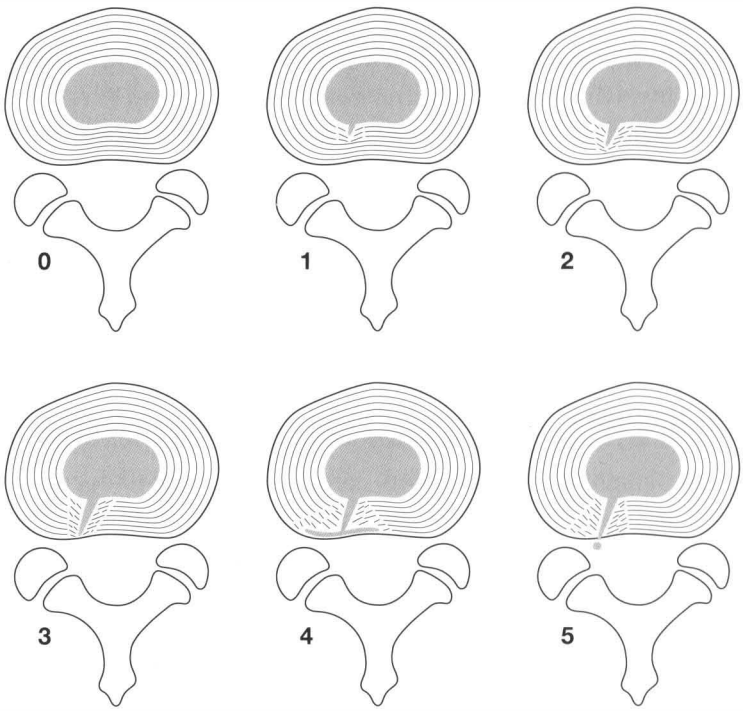
Table 4.1 Grading of radial fissures in annulus fibrosus

Grade	*Description*	*Pain status*
0	None	No
1	Into inner annulus	No
2	Into outer annulus	Yes / No
3	To outer annulus	Yes / No
4	3 + circumferential spread between lamellae in both directions (Aprill and Bogduk 1992)	Yes / No
5	Complete tear with leakage beyond annulus (Schellhas *et al.* 1996)	Yes / No

Some discrepancy exists over the definition of grade 3 fissures. Some authors state that this is when the annular disruption extends *beyond* the outer annulus (Sachs *et al.* 1987; Ninomiya and Muro 1992),

while others believe that this is a radial fissure that extends *into* the outer annulus (Schellhas *et al.* 1996; Aprill and Bogduk 1992).

Figure 4.2 Grades of radial fissures according to discography



Key:
0. None
1. Inner annulus
2. Outer annulus
3. To outer annulus

It is the presence of radial fissures into the outer third of the annulus that are most closely associated with painful discs, rather than general degeneration of the disc (Vanharanta *et al.* 1987; Moneta *et al.* 1994). Although the higher grade radial tears are found in asymptomatic individuals, the correlation between grade 3 and 4 fissures and back pain is very strong, and these are commonly found in chronic back pain populations that receive invasive imaging (Vanharanta *et al.* 1987; Moneta *et al.* 1997; Aprill and Bogduk 1992; Smith *et al.* 1998; Ricketson *et al.* 1996; Milette *et al.* 1999; Ohnmeiss *et al.* 1997). Indeed, so strong is the association between grade 3/4 fissures and exact reproduction of patient's pain that *"no other demonstrable morphological abnormality has been shown to correlate so well with back pain"* (Bogduk 1997, p. 205).

The studies of Milette *et al.* (1999) and Ohnmeiss *et al.* (1997) make clear that grade 2 radial fissures are as potent a source of symptoms as grade 3 fissures and even protrusions. In one sample of patients with chronic back and leg pain, the presence of radial fissures into the outer annulus was shown to be a more important predictor of symptomatic discs than the outer contour of the annular wall; that is, disc bulges and protrusions (Milette *et al.* 1999).

Grade 4 radial fissures with circumferential spread of contrast medium are strongly correlated with an MRI feature known as a ***high-intensity zone*** (HIZ). This was recognised by Aprill and Bogduk (1992) who, along with others (Schellhas *et al.* 1996), found it highly predictive of painful discs. However, other authors have found it to be a poor predictor of painful discs (Ricketson *et al.* 1996; Smith *et al.* 1998). It is suggested that the HIZ represent an irritated or inflamed outer annular fissure, which is different from a disc herniation (Aprill and Bogduk 1992; Schellhas *et al.* 1996).

Disc herniation

Although radial fissures can be a source of pain in their own right, the fissure may also act as a conduit for displaced discal material (Porter 1993; Bogduk 1997). These displacements are termed disc herniations.

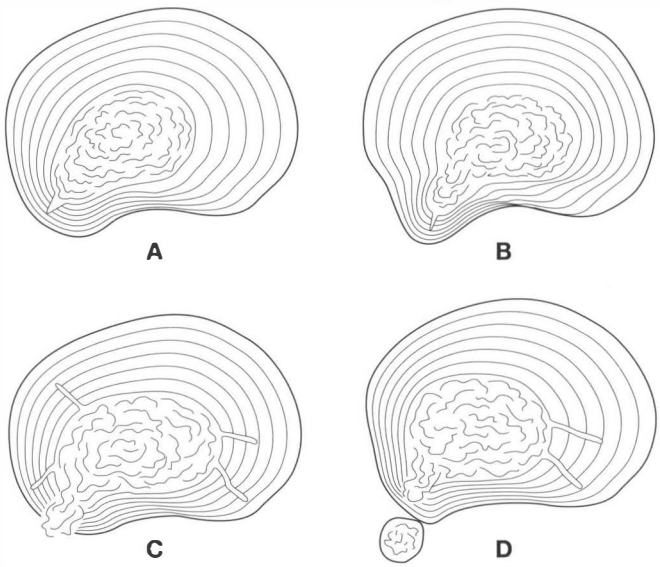
Definitions

There has been a lack of standardisation of terminology used to describe disc herniations, and synonyms are many and varied. There have been recent attempts to standardise the nomenclature and classification of lumbar disc pathology (Milette 1997; Fardon *et al.* 2001). This distinguishes annular fissures, herniations and degenerative changes, as well as disc infections and neoplasia. Different types of herniation are further delineated as protrusion, extrusion and sequestration, and intravertebral, when aspects of size, containment, continuity and location are considered. In relation to mechanical therapy, a key consideration is the state of containment – when ***contained*** the outer annular wall is intact, when ***non-contained*** disc material is displaced beyond the annular covering.

Kramer (1990) distinguishes four stages of discal displacement (Figure 4.3):

- intradiscal mass displacement – non-physiological displacement of tissue within the disc
- protrusion – the displaced material causes a bulge in the intact wall of the annulus
- extrusion – the disc material is displaced through the ruptured annular wall
- sequestration – a discrete fragment of disc material is forced through the ruptured annular wall into the spinal canal.

Figure 4.3 Four stages of disc herniations – in reality there will be many sub-stages



Key:
A. Intra-discal displacement
B. Protrusion
C. Extrusion
D. Sequestration

In this text the term *disc herniation* is used as a non-specific term that includes any of the more specific terms that carry with them clear-cut pathological and prognostic meaning (Table 4.2). If the hydrostatic mechanism is intact and the herniation is contained, then forces exerted on it can affect a displacement – it is reducible. If the hydrostatic mechanism is no longer intact, the outer wall is breached or incompetent and the herniation is non-contained, then the displacement cannot be affected by forces – the displacement is now irreducible.

Table 4.2 Disc herniations: terms and pathology used in this text

Term	*Pathology*	*Hydrostatic mechanism*
Herniation	Non-specific term including any of below	Non-specific term
Protrusion	Intact and competent annular wall	Intact
Protrusion	Intact annular wall, but so attenuated as to be incompetent	Not intact
Extrusion	Annular wall breached by intra-discal mass that protrudes through, but remains in contact with disc	Not intact
Sequestration	Annular wall breached by intra-discal mass that has separated from disc	Not intact

Routes and sites of herniations

The majority of fissures and herniations occur posteriorly or postero-laterally, the direction that causes greater symptoms, as displacement beyond the annular wall can involve the nerve root. A smaller proportion of herniations are directly lateral or anterior, and some go in a cephalic or caudad direction into the endplate of the vertebral body above or below. Lateral or far-lateral herniations may also involve the nerve root, as these can extrude into or lateral to the intervertebral foramen. The clinical importance of anterior and vertebral herniations, or Schmorl's nodes, is less well established.

An understanding of the pathogenesis of displacements can suggest movements and positions that could be utilised in their treatment. Some studies (Bernard 1990; Fries *et al.* 1982; Maezawa and Muro 1992; Ninomiya and Muro 1992; Fuchioka *et al.* 1993) have described the routes of displacements or existing fissures and the final point of herniation (see Table 4.3 and Figure 4.4). The findings from these different studies, involving over 2,000 patients who were surgical candidates, are striking in their similarities. Because they were so similar, and for simplicity, the mean from the four studies is shown.

Table 4.3 Herniation routes/fissures and sites of final herniation*

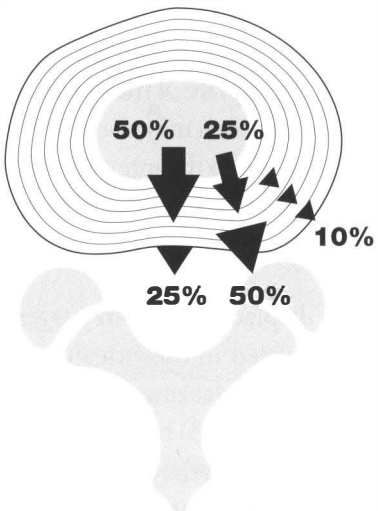
Site	*Fissures*	*Protrusions*	*Extrusions*
Central	57%	28%	14%
Postero-lateral	20%	59%	79%
Far lateral	11%	8%	4%
Multiple	19%	9%	

* % shown = mean from four studies with over 2,000 patients

Source: calculated from original data in Bernard 1990; Fries *et al.* 1982; Maezawa and Muro 1992; Ninomiya and Muro 1992; Fuchioka *et al.* 1993

Over half of all displacements and fissures appear to start centrally in the disc, while about a quarter start postero-laterally. However, well over half end up herniating postero-laterally on the dura and/or nerve root, with another quarter herniating centrally. The majority of all displacements thus occur in the sagittal plane, implicating flexion/extension movements both in their pathogenesis and treatment (Ninomiya and Muro 1992). Less than 10% of all displacements commence and herniate far laterally into, or lateral to, the intervertebral foramen. These run obliquely to the sagittal plane and implicate torsional or lateral forces both in their pathogenesis and their treatment. Herniation routes, however, do not follow straight lines and on occasion underwent complex twists and turns, even crossing the mid-line.

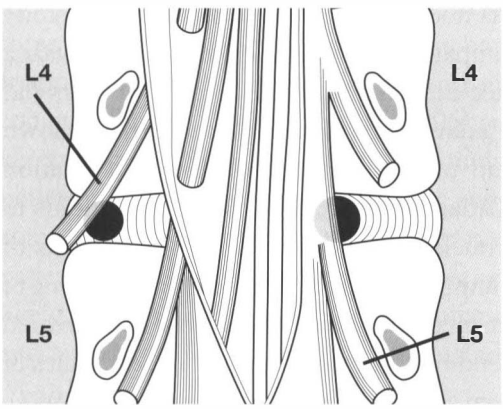
Figure 4.4 Routes and extrusion points of herniations
(see Table 4.2 for detail and references)



The prevalence of lateral disc herniations, known as extreme or far lateral, varies from 6% to 12% of all surgically treated herniations in different studies (Abdullah *et al.* 1988; Kunogi and Hasue 1991; Jackson and Glah 1987; Patrick 1975; O'Hara and Marshall 1997; Postacchini *et al.* 1998). These tend to occur at slightly higher segmental levels than the more common postero-lateral herniation, about 75% occurring at L3 – L4 and L4 – L5, and nearly 10% occurring at L2 – L3. This compares with 98% of postero-lateral herniations occurring at L4 – L5 and L5 – S1.

Postero-lateral herniations involve the descending nerve root, which is situated nearer the mid-line. Far lateral disc herniations affect the nerve root exiting at that segmental level, which is the nerve above. Thus, an L4 – L5 lateral herniation would affect the L4 nerve, while a postero-lateral herniation would affect the L5 nerve root (see Figure 4.5). Therefore, lateral herniations are more likely to be involved when signs and symptoms point to upper lumbar nerve root compression (Abdullah *et al.* 1988).

Figure 4.5 At L4 – L5, a lateral disc herniation (left) affects the exiting nerve root (L4); a postero-lateral disc herniation (right) affects the descending nerve root (L5)



Reports of anterior herniations are much less frequent, but do appear in the literature as case reports and MRI studies (Buirski 1992; Jinkins *et al.* 1989; Brooks *et al.* 1983; Cloward 1952). Jinkins *et al.* (1989), in a retrospective review of 250 MRI examinations, listed the directional differentiation of disc extrusions, the clinical significance of which is unproven (see table). Just as posterior herniations are frequently found in asymptomatic populations (Boden *et al.* 1990; Jensen *et al.* 1994; Weinreb *et al.* 1989; Boos *et al.* 1995), it is likely that anterior and vertebral herniations are also frequently incidental findings of unknown clinical significance.

Table 4.4 Directional differentiation of disc extrusions on MRI

Type of extrusion	*Proportion*
Posterior/ Postero-lateral	57%
Anterior	29%
Vertebral	14%

Source: Jinkins *et al.* 1989

Of the anterior herniations, about half were in the mid-line and the rest were antero-lateral or directly lateral. Both anterior and vertebral herniations were much more common at upper lumbar levels (L1 – L2 to L3 – L4). Anterior and vertebral disc herniations are reported to cause back and diffuse non-specific limb pain, and non-specific paraesthesia (Jinkins *et al.* 1989; Cloward 1952; Brooks *et al.* 1983). Straight leg raise and neurological examination are negative.

Intravertebral disc herniation, also known as Schmorl's nodes, can be an asymptomatic and incidental finding (Bogduk 1997). They have been reported with varying frequencies in several studies of cadaveric spines with greater occurrence in the thoracic and upper lumbar spines (Resnick and Niwayama 1978; Hilton *et al.* 1976). The incidence of Schmorl's nodes in back pain patients in one study was found to be 19% compared to 9% in a control group, with a particularly high incidence in those between 10 and 40 years old (Hamanishi *et al.* 1994). Cadaveric experimental studies have shown that endplate damage can unleash a chain of disc degeneration affecting the whole disc (Adams *et al.* 2000b). The damage leads to reduced pressure in the nucleus pulposus and increased peaks of compressive stress in the annulus fibrosus. Buckling and fissuring of the annulus and displacement of the nucleus can follow. An increased density of sensory nerve endings has been found in the endplates of patients with severe back pain and disc degeneration (Brown *et al.* 1997).

Schmorl's nodes are reported to occur acutely with significant trauma such as motorcycle accidents and falls, particularly in adolescents and young adults, and can be associated with severe back pain and significant disability (McCall *et al.* 1985; Fahey *et al.* 1998). One study using discography noted leakage of contrast material into the vertebral body in fourteen of 692 discs injected (Hsu *et al.* 1988). Pain on injection was concordant with the patient's pain and severe or moderately severe in thirteen of the fourteen (93%), compared to 42% in the remaining discs. This statistically significant difference suggests that endplate disruptions can be a source of symptoms.

In summary, the primary source of symptomatic disc herniations is posterior or postero-lateral. Postero-lateral and the much less common lateral disc herniations are the cause of radicular pain. However, anterior or antero-lateral herniations may also be a more unusual cause of symptoms. The role of intravertebral disc herniations or Schorl's nodes in symptom production is less well established, but

they appear capable of producing back pain and possibly unleashing a degenerative process leading to degradation of the whole disc (Bogduk 1997).

Herniated material

When discs do actually herniate, there is no consensus about the material that is involved in this pathological process. Histological analysis of disc herniations from different studies shows that extrusions can consist predominantly of nucleus pulposus, endplate or annulus fibrosus (Brock *et al.* 1992; Yasuma *et al.* 1986, 1990; Gronblad *et al.* 1994). Combinations of the different material are also found – although 34% of extrusions in one study were nucleus only, the rest were mixtures of nucleus, annulus and endplate (Moore *et al.* 1996). Harada and Nakahara (1989) also found combinations of the three different tissues, and occasionally bone, in their samples, with fragments of annulus or annulus and endplate being the most common finding. Takahashi *et al.* (1996) suggested that most of the herniated material was nucleus and/or annulus, but that distinguishing between the two was difficult. It is suggested that herniations comprising predominantly nucleus pulposus are common in younger patients, whereas in older patients the extruded material is more likely to be annular and endplate (Yasuma *et al.* 1986, 1990; Harada and Nakahara 1989). Clearly the herniated material is variable.

Stress profilometry

In this procedure a stress transducer is drawn through the disc, monitoring the vertical and horizontal stress profiles through the whole disc. It was developed and tested on cadavers, which revealed distinct patterns of stress associated with degenerative changes (McNally and Adams 1992; McNally *et al.* 1993; Adams *et al.* 1996a, 1996b). Comparing degenerated to non-degenerated discs, there is a 50% reduction in the diameter of the 'functional nucleus' and a 30% fall in its pressure. This is accompanied by an 80% increase in the width of the 'functional annulus', and an increase of 160% in compression 'stress peaks' in the annulus (Adams *et al.* 1996a). In degenerated discs, greater loads fall on the annulus.

These measurements reveal the increased stresses that fall on the annulus fibrosus as a consequence of the degenerative changes that affect the nucleus. These stresses were most marked at lower lumbar levels and in the posterior annulus. Stress peaks in the posterior annulus

were exaggerated after creep loading (Adams *et al.* 1996b) and may predispose to annular failure or disc prolapse (McNally *et al.* 1993). High peaks of compressive stress may predispose to further damage and may elicit pain from innervated parts of the annulus or from the vertebral endplates (Adams *et al.* 1996a). It is suggested that multiple stress peaks may represent an early painful stage of disc pathology, when the annulus is failing, but still functioning (Adams *et al.* 1996a). This is consistent with the concept of discogenic pain from grade 2 annular fissures.

Stress peaks also vary according to the posture of the motion segment being tested. In 'degenerated discs' exposed to extension, there was a generalised increase in stress peaks in the posterior annulus, while flexion tended to equalise the compressive stress. However, in seven of the nineteen motion segments tested, lumbar extension decreased maximum compressive stress in the posterior annulus by a considerable amount (Adams *et al.* 2000a).

McNally *et al.* (1996) investigated stress profilometry and discography in a small group of patients. Patterns of stress distribution varied widely between discs, but anomalous loading of the posterolateral annulus was highly predictive of a painful disc. Discogenic pain was most associated with single and multiple stress peaks in the annulus, broadening of the 'functional annulus' and depressurisation of the nucleus.

Conclusions

In summary, the intervertebral disc is a common source of pain in its own right. It undergoes certain morphological changes that make it susceptible to becoming symptomatic. Considerable degeneration of an asymptomatic nature can occur. It has nerve endings in its outer layers, and in the diseased state this innervation can be much deeper in the disc. Even radial fissures and disc herniations can be found in asymptomatic populations, but these findings are frequently symptomatic. By direct stimulation at surgery and by exerting pressure with injection using discography, patients' familiar back and leg pain has been reproduced.

The structural abnormality that appears most closely linked to discogenic pain is the radial fissure. Numerous studies have shown that it is this particular disruption of the outer lamellae of the annulus that correlates closest with painful discs. Indeed, no other morphological

abnormality is so clearly associated with back pain. Pain may be the result of excessive mechanical loads on innervated, weakened tissue. Alternatively, the fissure may act as a conduit for displaced tissue, which is affected by positions and movements.

Additionally, but much less commonly, the disc can be a source of radicular pain by causing tension or compression of lumbar nerve roots. In this pathology, a radial fissure and displaced discal tissue are necessary to exert pressure on the outer annular wall. If the annulus remains intact, movements or positions can influence the displacement. If the outer annular wall is ruptured or weakened sufficiently, then the displacement may herniate through it, and loading is no longer able to affect its location. The clinical presentations of these different entities are examined in the next chapter.

5: Disc Pathology – Clinical Features

Introduction

The intervertebral disc is a common cause of back pain and the most common cause of radiculopathy or sciatica (Schwarzer *et al.* 1995d; Spitzer *et al.* 1987; AHCPR 1994). It has been proven that the disc is innervated. Although this may be partial and variable between individuals, it is a potential source of pain in its own right (Bogduk 1994b). Schwarzer *et al.* (1995d) found the disc to be the source of pain in 39% of a sample of chronic back pain patients. However, the gross and most renowned representation of discal pathology, the 'disc herniation' causing sciatica, is by most estimates comparatively rare – occurring in less than 5% of the back pain population (CSAG 1994) – although in one population survey 12% of those with back pain described symptoms of sciatica (Deyo and Tsui-Wu 1987). The clinical presentation of discogenic pain and of sciatica will be outlined.

Sections in this chapter are as follows:

- discogenic pain – prevalence
- discogenic pain – clinical features
- sciatica – prevalence
- sciatica – clinical features
- state of the annular wall
- natural history of disc herniation.

Discogenic pain – prevalence

Schwarzer *et al.* (1995d) found in a sample of ninety-two consecutive chronic back pain patients undergoing invasive imaging in tertiary care that, according to their strict criteria, 39% could be diagnosed as suffering from *internal disc disruption*. Pain and guarded movements are present; there is normal radiology and computer tomography (CT) imaging. The definitive diagnosis relies on two tests: the reproduction of the patient's pain with discography and the use of CT discography to reveal internal disc disruption. As a control, stimulation of at least one other disc should fail to reproduce

pain, and to prove disruption a grade 3 radial fissure should be present on CT discography.

Using discography to reproduce patients' symptoms has resulted in the classification of 75%, 57% and 33% respectively of the populations studied as having discogenic symptoms (Ohnmeiss *et al.* 1997; Donelson *et al.* 1997; Antti-Poika *et al.* 1990). It will only ever be a select group who receive this invasive imaging, namely chronic back pain patients in hospital settings who have failed to improve with previous conservative care and in whom clear indications for surgery have not been found – that is, no definite nerve root involvement. Nonetheless, these studies suggest that the intervertebral disc is the most common single source of back pain.

Discogenic pain – clinical features

In patients who have nerve root involvement, direct stimulation of the annulus fibrosus has either been unable to provoke leg pain (Kuslich *et al.* 1991) or has done so in only a minority (Fernstrom 1960). However, in patients who have not had clear signs or symptoms of nerve root involvement, leg pain has been commonly provoked by discographic stimulation (Park *et al.* 1979; McFadden 1988; Milette *et al.* 1995; Donelson *et al.* 1997; Ohnmeiss *et al.* 1997; Colhoun *et al.* 1988; Schellhas *et al.* 1996). Ohnmeiss *et al.* (1997) found that pain referred into the thigh or calf was as common in those patients with a grade 2 disruption of the annulus as a grade 3 disruption. In their sample, those without internal disc disruption were significantly less likely to have lower limb pain than those who had a discogenic source of symptoms. Referral of pain into the leg can clearly be a feature of discogenic pain; in those with nerve root involvement, it appears that the leg pain is primarily a result of nerve root compression.

Schwarzer *et al.* (1995d) compared those who had the diagnosis of internal disc disruption to those who did not, according to various aspects of their clinical presentation. There was no statistically significant association between historical or examination findings and whether patients had a positive discography. Sitting, standing, walking, flexion, extension, rotation and straight leg raise were neither more likely to aggravate nor relieve pain in patients who had discogenic pain than in those whose pain was non-discogenic, nor

could pain patterns distinguish the two groups, both having buttock, groin, thigh, calf and foot pain. Those with bilateral or unilateral pain distribution were more likely to have discogenic pain than those with central symptoms.

Rankine *et al.* (1999) examined the clinical features of patients with an HIZ with no evidence of neural compromise. On simple history-taking and clinical examination, they were unable to differentiate those with this sign from those without it. Features examined were pain referral above or below the knee, aggravation of pain by standing, walking, sitting, bending, lying, lifting and coughing, and neurological symptoms and signs. None of these variables were more common in those with an HIZ, and so they could not define particular clinical features that predicted this outer annular disruption.

A dynamic mechanical examination is much more successful at detecting symptomatic discs and determining the state of the outer annular wall (Donelson *et al.* 1997). Sixty-three chronic patients, the majority experiencing pain below the knee with no neurological deficits and no clear surgical indications on MRI, underwent discography and a McKenzie mechanical evaluation. The experienced McKenzie clinicians who conducted the examination were blinded to the outcomes from the discography. The clinicians used the movement of pain proximally or distally during the examination to classify the patients as centralisers, peripheralisers or no symptomatic change. Their classification was then correlated with the outcomes from discography. The criteria for a positive discogram were exact pain reproduction and an abnormal image, as long as no pain was reproduced at an adjacent level.

Thirty-one patients were classified as centralisers, sixteen as peripheralisers and sixteen as 'no change'. About 70% of centralisers and peripheralisers had a positive discogram, whereas only two patients (12.5%) in the 'no change' group had a positive discogram. Among the centralisers with a positive discogram, 91% had a competent annular wall on discography, whereas among peripheralisers with a positive discogram, only 54% had a competent annular wall. All these differences were significant ($P < 0.05$).

Thus most centralisers had discogenic pain with a competent annular wall, and most peripheralisers also had discogenic pain with a much

higher prevalence of outer annular disruption. Symptoms that did not change during the mechanical assessment were very unlikely to be discogenic in origin. The authors conclude *"a non-invasive, low-tech, relatively inexpensive clinical assessment using repeated end-range lumbar test movements can provide considerably more relevant information than non-invasive imaging studies. Namely, it can distinguish between discogenic and nondiscogenic pain and provides considerable help in distinguishing between a competent and incompetent annulus"* (Donelson *et al.* 1997, p. 1121).

According to this study, if pain centralises or peripheralises, the probability of discogenic pain is 72%, while if pain remains unchanged the probability of non-discogenic pain is 87% (positive and negative predictive values recalculated from original data). Centralisation of pain has been recorded in about 50 – 90% of populations studied (Donelson *et al.* 1990, 1991, 1997; Long 1995; Delitto *et al.* 1993; Erhard *et al.* 1994; Werneke *et al.* 1999; Sufka *et al.* 1998). It is a very common occurrence in acute and chronic backs, and strongly suggests diagnostic implications.

Sciatica – prevalence

Disc herniations are the most common cause of nerve root involvement in back pain, commonly known as sciatica (Spitzer *et al.* 1987; AHCPR 1994). It has been estimated that this involves less than 5% of all those who have back pain (CSAG 1994; Heliovaara *et al.* 1987); some studies give higher estimates. When a definition was used of pain that radiated to the legs and that increased with cough, sneeze or deep breathing, 12% of those with back pain fit into this category (Deyo and Tsui-Wu 1987). A study conducted in Jersey in the Channel Islands recorded the frequency of diagnoses given by physicians for absences from work because of back pain (Watson *et al.* 1998). In this group, over 7% were diagnosed as having sciatica and a further 5% as having a prolapsed intervertebral disc. Dutch GPs diagnosed 14% of over 1,500 patients with radicular pain, and most of the rest (72%) with non-specific back pain (Schers *et al.* 2000). In tertiary care, the prevalence of neurological symptoms is greater; in a study of nearly 2,000 patients, 21% were found to have neurological signs and a further 41% had distal leg pain (Ben Debba *et al.* 2000).

Sciatica – clinical features

The classical criteria that need to be present to make the diagnosis of a symptomatic disc herniation with nerve root involvement are shown in Table 5.1 (Porter 1989; Porter and Miller 1986; Kramer 1990).

Table 5.1 Criteria for identifying symptomatic disc herniation with nerve root involvement

- unilateral leg pain in a typical sciatic root distribution below the knee
- specific neurological symptoms incriminating a single nerve
- limitation of straight leg raising by at least 50% of normal, with reproduction of leg pain
- segmental motor deficit
- segmental sensory change
- hyporeflexia
- kyphotic and/or scoliotic deformity
- imaging evidence of a disc protrusion at the relevant level.

Lumbar disc herniations occur most commonly among young adults between the ages of 30 and 40 (Deyo *et al.* 1990). However, it is reported that 1 – 3% of operations for lumbar disc herniations are performed on patients who are under 21 years of age (Silvers *et al.* 1994), and 4% on those over sixty (Maistrelli *et al.* 1987).

Typically the imaging study is done on suspicion of a disc herniation because of the clinical presentation of a patient. Variability of signs and symptoms is considerable. Over 95% of disc herniations occur at the L4 – L5 and L5 – S1 levels, thus the nerves most commonly affected are L5 and S1 (Andersson and Deyo 1996). Kramer (1990) states that about 50% of all herniations may be clearly assigned to a single segmental level, predominantly L5 and S1. The other cases are either not specific enough to be assigned a definite level or else more than one root is involved. Another study locates over 97% of just over 400 disc herniations at L4 – L5 and L5 – S1 interspaces (Kortelainen *et al.* 1985) (see Table 5.2).

Table 5.2 Distribution of single nerve root involvement in disc herniations

Segmental level	*Proportion of single nerve root involvement*	*Interspace*	*Disc herniations (%)*
L2	0.5%		
L3	0.5%	L2 – L3	<1%
L4	1.0%	L3 – L4	<2%
L5	44%	L4 – L5	57%
S1	54%	L5 – S1	41%

Source: Kramer 1990; Kortelainen *et al.* 1985

Typically the pain is referred down the lateral (L5) or posterior (S1) aspect of the thigh and leg below the knee into the dorsum of the foot and the big toe (L5), or the heel and outer aspect of the foot (S1) (see Table 5.3 and Figure 15.1). Nerve root tension signs are present – if L4, L5 or S1 are involved, this is the straight leg raise test; if upper lumbar (L1 – L3), this is the femoral nerve stretch test. Weakness may be present and is found in tibialis anterior (L4/L5), extensor hallucis longus (L5) or the calf muscles (S1/S2). If sensory deficit occurs, this is most common in the big toe (L5) or the outer border of the foot (S1). However, the radicular pain pattern or location of sensory deficit is not a definite means of identifying the nerve root involved. A disc herniation at L4 – L5, although more likely to produce symptoms of an L5 lesion, may also produce symptoms of an S1 lesion. Likewise, a disc herniation at L5 – S1, although more likely to produce symptoms of an S1 lesion, may produce symptoms of an L5 lesion (Kortelainen *et al.* 1985). Herniations at both levels may affect both nerve roots.

Table 5.3 Typical signs and symptoms associated with L4 – S1 nerve roots

	L4	*L5*	*S1*
Distribution of pain and *sensory loss*	(Anterior thigh) Anterior / *medial leg* (Great toe)	(Lateral thigh) Lateral leg Dorsum of foot *Great toe*	Posterior thigh Posterior leg *Lateral border of foot* Sole
Motor weakness	Quadriceps *Dorsiflexion*	*Big toe extension* *Extension of the toes*	*Plantarflexion* Eversion
Reflex	Knee		Ankle

Source: Waddell 1998; Nitta *et al.* 1993; Smyth and Wright 1958; Butler 1991; Kramer 1990

Some patients may present with small, isolated patches of distal pain rather than the typical dermatomal pattern. Root tension signs, due to irritation of the nerve root, occur earlier and more commonly than motor, sensory and reflex signs, which only occur once the function of the root is disturbed. These findings are variable. Flexion in standing, as described in this book, is also a form of root tension sign.

Flexion increases the compressive force acting on the nerve root complex and aggravates symptoms (Kramer 1990; Schnebel *et al.* 1989). *Patients will generally be made worse in positions of flexion.* However, sometimes temporary relief may be gained during sitting, while the intervertebral foramina are enlarged, but upon returning to an upright position symptoms return to their former intensity or are worse. Disc-related symptoms are also affected by other activities that increase intra-discal pressure, such as coughing, sneezing or straining (Kramer 1990)

Unfortunately, none of the questions or tests that are part of the history and physical examination has a high diagnostic accuracy by itself (Andersson and Deyo 1996; Deyo *et al.* 1992; van den Hoogen *et al.* 1995; Deville *et al.* 2000). The history-taking and physical examination in patients with suspected lumbar nerve root involvement have been shown to involve considerable disagreement – Kappa value 0.40 after the history and 0.66 after the examination (Vroomen *et al.* 2000).

The presence of sciatica has a high sensitivity (0.95) and specificity (0.88), but poor diagnostic accuracy in identifying disc herniations (Andersson and Deyo 1996; van den Hoogen *et al.* 1995). The straight leg raise test has a high sensitivity, but low specificity, while the crossed straight leg raising test is less sensitive, but much more specific (Andersson and Deyo 1996; van den Hoogen *et al.* 1995; Deyo *et al.* 1992; Deville *et al* 2000). The sensitivity of other neurological signs tends to be less good, while the specificity is somewhat better. In particular, muscle weakness tests have a higher specificity. Patients with disc herniations have significantly less range of forward flexion compared to patients with no positive findings, and also significantly more pain distributed in to the legs on extension in standing (Stankovic *et al.* 1999).

Most of the studies that these papers review involve surgical cases at the severe end of the pathological spectrum, and so their results are based on biased samples that do not correspond to the true range of patients; those with definite disc herniations will tend to be over-represented. When the prevalence of a disease in a population is high as in these studies, the predictive value of tests will be over-inflated. In the back pain population as a whole, in which the prevalence of disc herniations is much lower, the predictive value of these tests will be poorer. This means a substantial probability of false-positive test results (Andersson and Deyo 1996; Deville *et al.* 2000). The accuracy of individual tests is likely to be improved by considering combinations of responses and tests.

Upper lumbar disc herniations are relatively rare compared to herniations in the low lumbar spine, but they do occur. One series of about 1,400 patients identified 73 with herniations that affected L1, L2, and L3 nerve roots (Aronson and Dunsmore 1963). This represented only 5% of the total, of whom 70% had involvement of L3, 25% involvement of L2 and 5% involvement of L1. Radiation of pain was primarily over the lateral and anterior aspect of the thigh, and some cutaneous sensory loss was present in about 50% of patients in the same area. Muscle weakness mostly affected quadriceps or psoas, but extensor hallucis longus was occasionally affected. The knee jerk was reduced or absent in 50% of patients.

Part of the clinical presentation of acute back pain patients may be a deformity of kyphosis and/or scoliosis or lateral shift. The aetiology of the shift for a long time has been thought to relate to disc herniations (O'Connell 1943, 1951; Spurling and Grantham 1940; Falconer *et al.* 1948). Conceptually it was imagined that the shift occurred to avoid pressure on the nerve root. The widely quoted theory suggested that a contralateral shift was an attempt to reduce pressure on a nerve root from a disc herniation that was lateral to the root, while an ipsilateral shift was an attempt to reduce pressure on a nerve root from a herniation that was medial to it (Kramer 1990; Weitz 1981). These theories have now been disproved. Although it is described in the literature, clear-cut definitions and standardised terminology have not been used. Lateral shifts are still generally believed to relate to disc pathology (see Chapter 9).

State of the annular wall

Displacement great enough to cause significant deformity can with further displacement cause rupture of the annulus and perhaps even extrusion of disc material. Deformity is a sign of major displacement, as are the other criteria of a significant disc lesion such as constant radicular pain, constant numbness or myotomal weakness. If no position or movement can provide *lasting* improvement of symptoms, we can surmise an incompetent annular wall in which the hydrostatic mechanism of the disc has been lost. The annular wall may have been breached by herniated discal material (extrusion or sequestration), or else the outer annular wall has become so attenuated and weakened as to be incompetent (protrusion). This presentation is associated with a poor chance of rapid improvement under conservative care as it is at the extreme end of the pathological continuum.

However, displacements develop from an embryonic stage when only minor symptoms of spinal pain will be experienced. Being well contained by a relatively healthy annulus, minor displacements are short-lived and rapidly reversible, being at the minor end of the continuum. A less extreme clinical presentation on the continuum is intermittent leg pain and neurological symptoms that are influenced by movements and positions. In this instance we may surmise an intact annular wall, a functioning hydrostatic mechanism, and a displacement which loading can either push to the periphery or to the centre of the disc. Positions and movements may be found that have an effect on the displacement with a consequent increase or decrease of pressure on the symptom-generating annulus and/or nerve root.

"The symptoms caused by a disk protrusion vary because the protruding disk tissue is still part of an intact osmotic system and participates in the pressure-dependent changes of volume and consistency of the disk. As long as the protruding tissue is covered by strong intact lamellae of the annulus fibrosus, the displaced fragment can relocate back into the center of the disk.... In some cases the protruded tissue can displace further and rupture the annulus fibrosus as a disc extrusion" (Kramer 1990, p. 128).

Disc herniations thus represent a continuum, at the severe end of which the annulus is ruptured and breached by an extrusion or a sequestrum is extruded from the disc into the spinal or vertebral canal. In such a case recovery will only occur slowly with the passage

of time if treated conservatively or else the patient is a likely case for surgery. At the less extreme end of the continuum a protrusion may be the source of symptoms, held in place by a competent annulus. The hydrostatic mechanism of the disc is still intact, and with the use of repeated movements and sustained postures the displacement may be reduced and the symptoms resolved. Therefore a key clinical decision arises as to whether the annulus is competent and the displacement still responds to mechanical forces, or it is incompetent or ruptured and no longer amenable to lasting changes. Pathologically and clinically, the distinction is between a protrusion with a competent annular wall and an extrusion/sequestration (Table 5.4). It is those with an incompetent or ruptured annular wall who are possible surgical candidates – see next section.

Table 5.4 Differences between sciatica due to a protrusion or an extrusion/sequestration

Disc protrusion	*Disc extrusion/sequestration*
LBP =/> thigh / leg pain	Leg pain >> LBP / No LBP Distal pain ++
Gradual onset leg pain	Sudden onset leg pain
Onset leg pain LBP remains the same	Onset leg pain LBP eases or goes
Postural variation ++	Less postural variation
Intermittent / constant pain	Constant pain
Intermittent / constant tingling	Constant numbness
Variable deformity	Constant deformity
Variable weakness	Motor deficits
Moderate / variable tension signs	Major, constant tension signs Crossed straight leg raise positive
Movements able to decrease, abolish or centralise symptoms	Movement increases distal symptoms No movement able to decrease, abolish or centralise symptoms in a lasting way Severe restriction walking capacity
Possible neck pain	

LBP = low back pain

Source: Kramer 1990; Brismar *et al.* 1996; Beattie *et al.* 2000; Pople and Griffith 1994; Vucetic *et al.* 1995; Uden and Landin 1987; McKenzie 1981; Jonsson *et al.* 1998; Jonsson and Stromqvist 1996b

Natural history of disc herniation

Given the difficulties of case definition, heterogeneity of pathology and presentation, recruitment bias, inadequate follow-up and variable interventions, identifying the natural history of disc herniations is difficult. Considerable variability in the history of those with disc herniations will be seen. Some will make a speedy and spontaneous recovery, while others will run a protracted course despite multiple conservative treatment interventions. Just as the underlying pathology varies, so too does the potential for easy resolution.

It should also be borne in mind that the correlation between the morphological abnormality and symptoms is not straightforward. Disc herniations may exist in the asymptomatic population (Boos *et al.* 1995), symptoms may resolve with little regression of a herniation and symptoms may show little change with a substantial reduction in herniation size (Matsubara *et al.* 1995).

Nonetheless, it is generally considered that the natural history of disc herniation if left untreated is positive, if rather protracted (Saal 1996; Kramer 1995; Weber 1994). The worst pain from discogenic sciatica is in the first three weeks, when any inflammatory response is most intense and the mechanical effect of the extruded disc material is greatest (Kramer 1995). It is recommended, because of the positive natural history within the first three months, that surgery is rarely indicated before six to twelve weeks (Saal 1996). It is also suggested that neither the failure of passive conservative care nor imaging test results and the presence of neurological deficit should be used as sole criterion for proceeding with surgical intervention. The only specific indicators for early surgery are (Saal 1996):

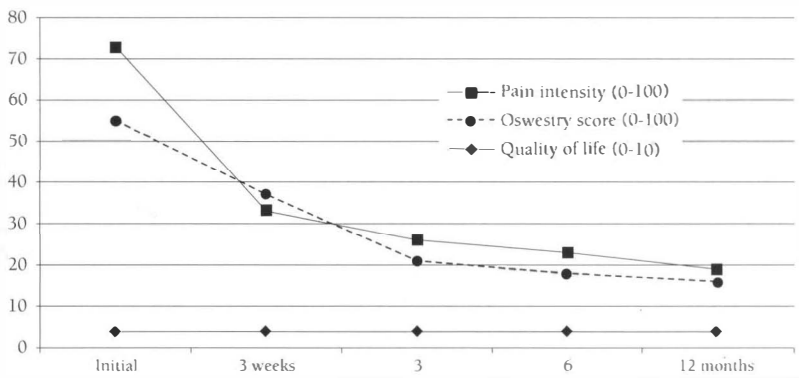
- cauda equina syndrome
- progressive neurological deficit
- profound neurological deficit (e.g. foot drop) showing no improvement over six weeks.

A trial evaluating the effect of NSAIDs in the management of sciatica used a placebo control group, which allows a reasonably true assessment of natural history. Weber *et al.* (1993) compared piroxicam to a placebo in over 200 patients with acute sciatica; there was no difference in outcomes between the two groups. Over the first four weeks, average pain on the visual analogue scale improved from about

five and a half out of ten to two out of ten, function improved markedly and 60% were back to work. There was no further improvement in leg symptoms at three months or one year, while back pain was reported to be worse at three months but the same at one year as at four weeks. About 40% of patients still complained of back and/or leg pain, and 20% were still out of work at one year. Previous episodes of sciatica were associated with poorer prognosis.

The natural history and clinical course of patients with nerve root signs and symptoms may be poor. Of eighty-two consecutive patients followed for a year following in-patient conservative therapy, only 29% were fully recovered and 33% had come to surgery (Balague *et al.* 1999). Most recovery occurred in the first three months, after which there was little further improvement (Figure 5.1). A positive neurological examination was associated with failure to recover at one year. However, surgery is not a simple panacea; 5 – 15% of surgical candidates have poor outcomes and further operations (Hoffman *et al.* 1993). In a Finnish study, 67% of 202 patients, whether having had surgery or not, continued to have significant problems as long as thirteen years after the onset of severe sciatica (Nykvist *et al.* 1995).

Figure 5.1 Recovery from severe sciatica

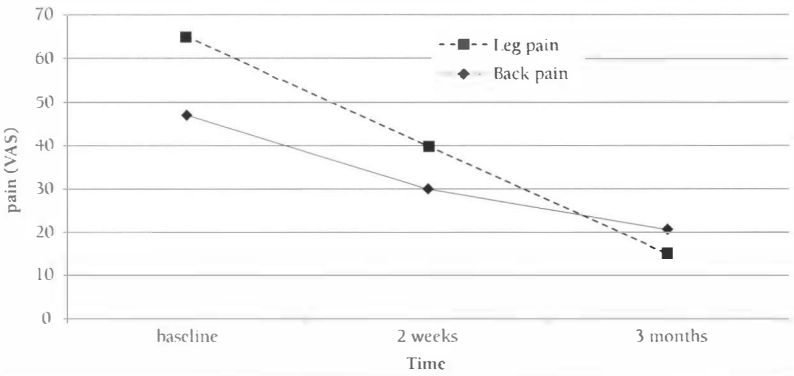


Source: Balague *et al.* 1999

Bed-rest has been shown to be no more effective than 'watchful waiting' in acute sciatica (Vroomen *et al.* 1999). The latter group was instructed to be up and about whenever possible, but to avoid straining the back or provoking pain, and were allowed to go to work. Both groups used NSAIDs and analgesics. In both groups at two weeks about 70% reported some improvement and 35% a great

improvement; on the visual analogue scale average pain in the leg was reduced from sixty-five to forty, and average pain in the back from forty-seven to thirty. At twelve weeks, 87% of both groups reported improvement and pain scores had fallen to fifteen in the leg and twenty in the back. The leg pain was worse initially, but overall improved more than the back pain (Figure 5.2). About 18% of both groups received surgery ultimately.

Figure 5.2 Recovery from sciatica in first three months



Source: Vroomen *et al.* 1999

Numerous studies have noted the regression of disc herniation when patients have been exposed to repeat imaging studies and conservative treatment. Unfortunately these studies are unable to define the time-scale of recovery, only its occurrence. Regression of herniation and improvement or resolution of symptoms usually occurred within six months or a year, although some follow-up studies were done up to two years after initial assessment. Maigne *et al.* (1992) performed repeat CT scans on forty-eight patients, all of whom showed a reduction in herniation, in eight of which this was between and 50% and 75% and in thirty-one between 75% and 100% reduction in size. Regression was seen in fourteen out of twenty-one patients on repeat CT scans (Delauche-Cavallier *et al.* 1992), and twenty-five out of thirty-six herniations (Ahn *et al.* 2000b). Larger herniations, and extrusions or sequestrations rather than protrusions, have repeatedly shown a greater tendency to decrease in size (Maigne *et al.* 1992; Matsubara *et al.* 1995; Delauche-Cavallier *et al.* 1992; Ahn *et al.* 2000b; Bozzao *et al.* 1992; Komori *et al.* 1996).

Regression of the disc herniation is generally associated with an improvement in symptoms, although not always exactly. In thirteen

patients whose leg symptoms resolved with conservative treatment, eleven demonstrated resolution or improvement in the disc herniation, but in two the size of herniation was unchanged (Ellenberg *et al.* 1993). The absolute area and sagittal and transverse measurements of the disc herniation have been shown to correlate with symptoms (Thelander *et al.* 1994). Constant symptoms were associated with larger herniations than intermittent symptoms, which were associated with larger herniation compared to those with no pain. The area of herniation decreased markedly over time, mirroring an improvement in symptoms. Teplick and Haskin (1985) reported on eleven patients with regression or disappearance of herniation, in nine of whom the associated radicular symptoms resolved. Bush *et al.* (1992) followed up over a hundred patients treated with epidural injections. At one year 14% had undergone surgery, and in the remaining patients pain was reduced by 94%. Complete or partial regression had occurred in 75% of the disc extrusions and 26% of the disc protrusions. Eleven patients with extrusions who had a repeat MRI at a median time of twenty-five months all had a regression of the herniation and resolution of their sciatica (Saal *et al.* 1990).

It is suggested that recovery from neurological deficit is variable, again depending upon the initial insult to the nerve root (Saal 1996) (see Table 27.2). Spontaneous recovery, when likely to occur, will generally show initial signs of improvement in the first three to six weeks.

Table 5.5 Recovery from neurological deficit

Possible pathology	*Degree of nerve damage*	*Presentation*	*Pattern of recovery*
Neurapraxia	Mild	Mild sensory loss, with/without mild motor deficit	Recovers in 6 – 12 weeks
Axonotmesis	Moderate	Absent reflex, moderate motor deficit, numbness	Recovers in 3 – 6 months
Axonotmesis	Severe	Absent reflex, Severe motor deficit, numbness	May take up to one year to improve, or may not recover fully at all

Source: Saal 1996

The natural history of disc herniations is generally good. Major improvements in symptoms and function happen in the first three or four weeks, while recovery from neurological deficit occur more slowly. After three months further recovery is less certain, so patients who still have symptoms at this point are not guaranteed the normal good natural history. Patients who have more severe pathology, such as extrusions and sequestrations, are as likely to have a good recovery, and may in fact do better than those with protrusions. However, despite the generally good prognosis, a substantial minority will have persistent symptoms at one year, and many will improve but not become fully symptom-free.

Conclusions

Recognition of symptomatic discogenic pain is problematic, as no specific signs or symptoms exist. However, a mechanical evaluation may accurately detect discogenic pain from assessment of symptom location change or lack of it. The signs and symptoms denoting sciatica include pain patterns, paraesthesia, muscle weakness and tension signs; these are variably present. Lateral shifts may be present and may be associated with poor prognosis if correction is not possible. Definite and proven sciatica due to an irreducible disc herniation is one of the few indicators for possible surgical intervention.

The primary source of symptomatic disc herniations is posterior or postero-lateral, with the latter being the most important cause of radicular pain. Anterior and vertebral herniations have a much more limited role in symptoms. The first key clinical decision concerns the postural loading that may reduce the disc displacement: should the patient be flexed or extended, or moved laterally? The second clinical decision concerns the ability to affect the disc displacement in a lasting way: is this a contained lesion with the hydrostatic mechanism intact, or has the annular wall been breached or become incompetent? Factors in the history may help us to determine these issues, which should be confirmed by the patient's response to a full mechanical evaluation.

6: Biomechanics

Introduction

Certain principle movements are available at the lumbar vertebrae. Range of movement varies considerably between individuals and may be affected by age and by the presence of back pain. Postures alter the sagittal angle of the lumbar vertebrae. Different movements and positions have various mechanical effects on the spine. Sustained movements have a different effect to single movements. Due to the biomechanical properties of collagenous tissue, the loading history on the spine may be significant. Experimental studies provide in vitro information about the effects of loading strategies.

This chapter considers some of the effects and characteristics of common postures and movements as revealed by physiological, clinical and experimental data. For a more detailed consideration of biomechanics, it is recommended that a clinical anatomy text be consulted (Bogduk 1997; Oliver and Middleditch 1991; Twomey and Taylor 1994a, 1994b; Adams 1994).

Sections in this chapter are as follows:

- movements at the lumbar spine
- range of movement
- lumbar lordosis
- loading strategies and symptoms
- effect of postures on lumbar curve
- biomechanics of the lumbar spine
- time factor and creep loading
- creep in the lumbar spine
- optimal sitting posture
- effect of time of day on movements and biomechanics
- effect of posture on internal intervertebral disc stresses.

Movements at the lumbar spine

The principal movements available at the lumbar spine as a whole and its individual motion segments are axial compression and distraction, flexion, extension, axial rotation and lateral flexion. Horizontal translation does not occur as an isolated or pure movement, but is involved in axial rotation (Bogduk 1997). There is considerably more sagittal movement available in the lumbar spine than rotation or lateral flexion, especially at the lowest segments. Flexion is substantially greater than extension.

Range of movement

Mobility of the lumbar spine varies considerably between different individuals. It may also be influenced by the following factors: age, sex, ligamentous laxity, genetics and pathology (Oliver and Middleditch 1991). In individuals, age and back pain are the most significant causes of variable movement patterns over time.

Age

Age causes increased stiffness of the motion segment and a decline in total range. From childhood to 60-year-olds there is nearly a halving of sagittal and frontal plane movements. During adulthood the change is less marked, but still it declines by about a quarter (Twomey and Taylor 1994a, 1994b). However, standard deviation accounts for up to 23% of the mean range of movement – there is a considerable range of what is 'normal'. This means a stiff and sedentary 40-year-old may display less mobility than a flexible and active 60-year-old.

Back pain

In general, patients with back pain are less mobile than asymptomatic individuals; however, there is such a wide spectrum of mobility that assigning people to diagnostic groups on the strength of movement loss is very difficult (Adams and Dolan 1995). Several studies have found differences in the range of movement between back pain patients and controls. Patients with back pain have been found to have significantly less flexion than control groups without pain (Pearcy *et al.* 1985; McGregor *et al.* 1995), and patients with tension signs showed significantly less flexion and extension (Pearcy *et al.* 1985). Groups with back pain have been found to have a significantly diminished range of spinal extension compared to controls without back pain (Pope *et al.* 1985; Beattie *et al.* 1987).

Thomas *et al.* (1998) found a statistically significant reduction in all planes of movement in a back pain group compared to a control group. About 90% of patients had at least one restriction of the seven tested, while 40% of controls had at least one restriction. The presence of three or more restrictions was found in 50% of patients, but only in 3% of those without symptoms. The largest differences between back patients and asymptomatic controls were in standing extension (12 degree difference) and finger-to-floor flexion (10 centimetre difference). Waddell *et al.* (1992) found measures of total flexion and extension, among other measurements, to successfully discriminate patients from controls.

Range of movement has also been shown to improve with patient recovery (Pearcy *et al.* 1985; Magnusson *et al.* 1998a). Improvements in impairment and disability can clearly discriminate those who are successfully treated and those who fail treatment (Waddell and Main 1984).

Because of the high degree of variability between individuals, detecting impairment due to back pain is problematic (Sullivan *et al.* 1994). For example, if an individual's normal range was above average, a loss of movement due to back pain may go undetected. Another individual, whose mobility is well below average, may give the appearance of impairment but be asymptomatic. The key contrasts are between the patient's present ability to move compared to normal and how this changes over time.

Time of day

Time of day affects an individual's flexibility, with increased range later in the day. Other aspects of spinal mechanics also change (see later section – *Effect of time of day on range of movement and biomechanics*).

Lumbar lordosis

The relationship between back pain and the lumbar lordosis has been evaluated in several studies with contradictory findings. Several studies have found that the lumbar lordosis of back pain subjects was significantly less than a control group without back pain (Jackson and McManus 1994; Simpson 1989; Magora 1975; Magora and Schwartz 1976). However, other studies have found no differences between symptomatic and asymptomatic groups and did not correlate

loss of lordosis with back pain (Hansson *et al.* 1985; Pope *et al.* 1985; Torgerson and Dotter 1976; Frymoyer *et al.* 1984).

Burgin *et al.* (2000) undertook a systematic literature review of postural variations and back pain, for which they identified six further studies. These demonstrated an association between an increased or decreased lumbar lordosis and back pain. However, as no study used a longitudinal prospective study design, a cause-and-effect relationship could not be established.

The range of what should be considered a normal lumbar lordosis is considerable (Torgerson and Dotter 1976; Jackson and McManus 1994; Hansson *et al.* 1985; Dolan *et al.* 1988). In a study in which measurements were made in 973 pain-free individuals, the mean lumbar lordotic angle was 45 degrees, with most of the sample falling somewhere in the range 23 – 68 degrees (Fernand and Fox 1985).

Given that the range of normal lordosis is so wide, identification of abnormality by observation only, as in the radiography studies above, is clearly difficult, if not impossible. An individual may have always had a small lordotic angle but no back pain; alternatively, in an individual who normally has a large lordotic angle, the advent of back pain may be accompanied by a reduced but still normal angle. Simply using observation, only very severe alterations should be given clinical significance. For instance, a recent onset inability to extend and absent lordosis is clinically relevant. Ultimately, the only way to test the correlation between the lumbar angle and symptoms is to alter the posture and record the symptomatic response. Most studies that have observed the role of posture in spinal problems have failed to make a direct correlation between posture and symptoms *at the same time*.

Loading strategies and symptoms

A few studies have made direct correlations between postures assumed and symptoms. Some studies have looked at the effect of different seating positions and comfort in asymptomatic populations. These studies are mentioned in more detail in the chapter on postural syndrome. The consistent finding is that seating that helps maintain the lumbar lordosis is generally found to be most comfortable, while more flexed postures were much more likely to produce discomfort

or pain (Harms 1990; Eklund and Corlett 1987; Mandal 1984; Knutsson *et al.* 1966).

The study of Harms-Ringdahl (1986) of healthy volunteers has shown the effect of sustained loading in the cervical spine. They maintained a protruded head posture and began to feel pain within two to fifteen minutes, which increased with time until they were eventually forced to discontinue the posture.

Mechanical diagnosis and therapy uses the concept that sustained postures and movements cause symptoms to decrease, abolish, centralise, produce, worsen or peripheralise. Certain therapeutic loading will have a favourable effect on symptoms and should be encouraged, while other loading has an unfavourable effect on symptoms and should be temporarily avoided. The next chapter discusses the phenomenon of centralisation at length. In the subsequent chapter, which reviews relevant literature, a section looks at studies that have investigated directional preference. This is the concept that patients with symptoms find their pain worsens with certain postures or movements, often but not always with flexion, and improves with the opposite posture or movement, often but not always with extension.

This is illustrated in the study by Williams *et al.* (1991) that compared the effects of two sitting postures on back and referred pain over a twenty-four- to forty-eight-hour period. There was a significant reduction in back and leg pain at all test points in the group that had been encouraged to maintain their lordosis and were provided with a lumbar roll. There was no change in severity of leg symptoms, but there was a worsening of back pain in the group who had been instructed to maintain a flexed posture when sitting. Centralisation above the knee occurred in over half the lordotic group, while peripheralisation occurred in 6%. Centralisation was reported in 10% of the flexion group, and peripheralisation in a quarter of the group.

The role of posture in predisposing to back pain incidence and then in perpetuating or aggravating it once present is considered in Chapter 2, and studies into directional preference are referred to in Chapter 11. Different postures clearly have different effects on symptoms, and consequently a good understanding of the biomechanics of posture on the lumbar spine is important.

Effect of postures on lumbar curve

Everyday positions, movements and activities affect the lumbar spinal curve. These positions, whose physiological effect is well known, are the ones asked about during the patient interview. In the sagittal plane certain activities are fundamentally activities of flexion, some activities of extension and some may be either (Table 6.1 and Figure 6.1).

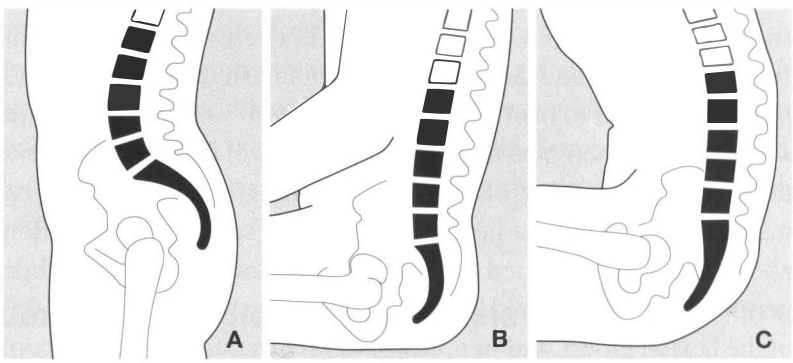
Table 6.1 Effect of different postures on the spinal curve

Postures of flexion	*Postures of extension*	*Variable postures*
Sitting	Standing	Side lying
Bending	Walking	
	Supine lying (legs extended)	
	Prone lying	

Standing / walking

Upright postures, such as standing and walking, are primarily activities of extension. When standing straight the lumbar lordosis is emphasised; comparatively, when sitting it is reduced considerably, and the spine becomes more flexed (Lord *et al.* 1997; Andersson *et al.* 1979; Dolan *et al.* 1988; Keegan 1953). Walking increases extension as the hind leg anteriorly rotates the pelvis, accentuating the lordosis (McKenzie 1981).

Figure 6.1 The effect of different postures on the lumbar curve



Key:
A. Standing – lordosis and anterior pelvic rotation
B. Sitting upright – reduced lordosis
C. Sitting slouched – kyphosis and posterior pelvic rotation

Sitting

When moving from standing to unsupported sitting, the lumbar lordosis decreases by on average 38 degrees, most of this movement occurring with the rotation of the pelvis, which on average accounts

for 28 degrees (Andersson *et al.* 1979). Sitting is primarily an activity of flexion; however, the amount is dependent upon numerous other factors. Sitting relaxed produces the most lumbar flexion, crossing the legs flexes the spine and sitting erect produces less extension than upright standing (Dolan *et al.* 1988). Thus, although sitting is a more flexed posture than standing, several factors may influence the degree of flexion that is attained (Table 6.2).

A backrest has some affect on lessening flexion in sitting, but a lumbar support has a more significant influence with increased support causing increasing lordosis, although the exact position of the support is less important (Andersson *et al.* 1979). A significant factor in the angle of the lumbar spine when sitting is the rotation that occurs at the pelvis. As the pelvis rotates posteriorly, as in slumped sitting, the lumbar spine is made to flex; as it rotates anteriorly, as in erect sitting, the lumbar spine is made to extend (Black *et al.* 1996; Andersson *et al.* 1979; Majeske and Buchanan 1984). The use of a lumbar roll facilitates a direct increase in the lordosis as well as ensuring a more anteriorly rotated pelvis (Andersson *et al.* 1979; Majeske and Buchanan 1984).

The angle between the thighs and the trunk has an effect on the lumbar curvature due to tension in the posterior thigh muscles (Harms 1990). Increasing hip flexion rotates the pelvis posteriorly and has the effect of flattening or flexing the spine (Keegan 1953). Thus, sitting with the knees above the hips, as is common on many settees/lounge chairs or car seats, flattens the spine.

Table 6.2 Factors that affect the spinal curve in sitting

Factors that accentuate the lordosis	*Factors that increase flexion*
Anterior rotation of pelvis	Posterior rotation of pelvis
Hip extension	Hip/knee flexion
Backrest inclined backwards	Crossing legs
Lumbar support/roll	

Bending

Leaning forward is obviously an activity of flexion. Bending fully causes more flexion of the lumbar spine than sitting (Keegan 1953). Flexion moments are exerted on the lumbar spine when a person leans forward; the further they lean, the greater the resulting moment. The magnitude of the flexion moment is a product of the weight of the trunk above the spine and the distance from the spine to the line

of gravity acting through the trunk, known as the *moment arm*. The greater the moment arm, for instance if a person were to lean forward and hold a weight in outstretched hands, the greater the force acting on the spine (Bogduk 1997). Lowering one's height by squatting produces less flexion of the spine than purely bending forward.

Lying

The shape of the spinal curve in lying is dependent upon the position adopted. Three basic postures are available: side, prone or supine. In side lying the spine may be either flexed or extended depending on the position of the legs. Increasing hip flexion, with its concomitant posterior rotation of the pelvis, flattens the lordosis, while increasing amounts of hip extension accentuate it. Lying in the foetal position is one of extreme flexion, while lying with hip or hips extended tends to extend the spine. Side lying also causes a degree of lateral translation towards the side the individual is lying on (McKenzie 1981). In supine lying, the spinal curve is dependent upon the position of the legs. With knees and hips extended, the anterior thigh muscles anteriorly rotate the pelvis and increase the lordosis, while with hip and knee flexion the pelvis rotates posteriorly and the spine flattens. Prone lying for most people is a position of relative lumbar extension.

Biomechanics of the lumbar spine

Biomechanics (Breig 1961) is the study of changes in anatomical structures occurring during movements of the body. Flexion and extension involve two components – sagittal rotation and sagittal translation. For instance, in flexion there is a combination of anterior sagittal rotation and anterior translation of the lumbar vertebrae (Oliver and Middleditch 1991).

With flexion, the intervertebral disc is compressed anteriorly and the posterior annulus is stretched. Flexion causes a posterior displacement of the nucleus pulposus (Shah *et al.* 1978; Krag *et al.* 1987; Shepperd *et al.* 1990; Shepperd 1995; Schnebel *et al.* 1988; Beattie *et al.* 1994; Fennell *et al.* 1996; Brault *et al.* 1997; Edmondston *et al.* 2000). The movement causes a lengthening of the vertebral canal and places tension on the spinal cord and the peripheral nervous system. Intradiscal pressure, measured in the nucleus pulposus, increases by up to 80% in full flexion (Adams 1994).

With extension, the intervertebral disc is compressed posteriorly and the anterior annulus is stretched. The movement is associated with impacting of the spinous processes, or the inferior articular processes, on the lamina below. Loading may be concentrated in the area of the pars interarticularis (Oliver and Middleditch 1991). Extension causes an anterior displacement of the nucleus pulposus (Shah *et al.* 1978; Krag *et al.* 1987; Shepperd *et al.* 1990; Shepperd 1995; Schnebel *et al.* 1988; Beattie *et al.* 1994; Fennell *et al.* 1996; Brault *et al.* 1997; Edmondston *et al.* 2000). Extension reduces the size of the vertebral canal and intervertebral foramen. Nuclear pressure is reduced by up to 35% in extension (Adams 1994).

For a detailed analysis of movement, coupled movements, the control and restraint of movement and the effects of testing spinal segments to failure, readers are referred to clinical anatomy texts (Bogduk 1997; Oliver and Middleditch 1991; Twomey and Taylor 1994a, 1994b; Adams 1994).

Time factor and creep loading

Various studies in asymptomatic volunteers have demonstrated the role of sustained loading in the generation of spinal pain (Harms 1990; Harms-Ringdahl 1986; Eklund and Corlett 1987). It is not the act of slouched sitting or, in the cervical spine, protrusion of the head that causes the ache to appear, but rather the maintenance of this end-range position for a sustained period. With muscular activity reduced, the mechanical stress falls mostly on non-contractile articular and peri-articular structures such as ligaments, joint capsules and the intervertebral disc. The effect of sustained or repeated loading on collagenous structures has an important role in the pathogenesis and maintenance of musculoskeletal problems.

Insidious onset back pain is more common in life than sudden injury. Experimental findings offer supportive evidence that explain this phenomenon by fatigue damage, which occurs at low loads with accumulative stress (Dolan 1998; Adams and Dolan 1995; Wilder *et al.* 1988). This highlights the role of loading history in spinal mechanics and the aetiology of back pain – for instance, sustained loading generates stress concentrations in the posterior annular fibres (Adams *et al.* 1996b), which may be a cause of pain in vivo (McNally *et al.* 1996). As the largest avascular structure in the body, the

intervertebral disc is particularly prone to fatigue failure as it has a very limited capacity for repair or remodelling (Adams and Dolan 1995, 1997). Creep loading in flexion, together with anterior translation with time, may be a cause of distortion or structural damage to any collagenous spinal structure. Attenuation and fissuring in the lamellae of the annulus, or weakening of ligaments and joint capsule, are all possible with sustained loading (Adams *et al.* 1980; Twomey *et al.* 1988).

Creep, hysteresis and set

If a constant force is left applied to a collagenous structure for a prolonged period of time, further movement occurs. This movement is very slight; it happens slowly, is imperceptible and is known as *creep* (Bogduk 1997). Creep is the result of rearrangement of collagen fibres and proteoglycans and of water being squeezed from the tissue. Brief stress does not act long enough on the tissue to cause creep, whereas sustained force allows displacement to occur so that elongation of the structure occurs.

Upon release from the force, as long as this has not been excessive, the structure begins to recover. However, restoration of the initial shape of the structure occurs more slowly and to a lesser extent than the initial deformation. The rate at which recovery happens between loading and unloading is known as *hysteresis* (Bogduk 1997). Initially the structure may not return to its original length, but remain slightly longer. This difference between initial and final length is known as *set*. This often occurs after creep, but if the interval between creep loading is sufficient, full recovery may occur and the structure eventually returns to its original shape. Depending upon the tissues and the forces applied, structures may be temporarily lengthened if loading is tensile or compacted if loading is compressive.

However, if the collagen fibres are not given enough time to recover before creep loading occurs again, or if creep loading has caused the bonds between and within collagen fibres to be broken, the set may persist indefinitely. Thus normal forces applied over lengthy and repeated periods of time may cause an alteration of the mechanical properties of collagenous structures. Not only may ligaments, capsules or parts of the disc become lengthened and less capable of fulfilling their normal mechanical functions, but also the structure may become vulnerable to injury. In this way tissues may become susceptible to fatigue failure.

After sustained or repetitive normal mechanical stresses, structures may fail at loads that are substantially less than that needed to cause damage with a single application of force. While one loading has no deleterious affect upon the tissue, the same loading, within normal bounds, prolonged or frequently applied may eventually lead to disruption of the tissue. *"The clinical importance of fatigue failure is that damage to tissues may occur without a history of major or obvious trauma"* (Bogduk 1997, p. 77); hence 'no apparent reason' for the onset of musculoskeletal problems is so common.

Creep in the lumbar spine

Flexion creep loading

Creep has a profound effect on the mechanical properties of the motion segment (Adams 1994). Experimentally the effects of creep in the lumbar spine have been studied relative to flexion, extension and axial loading (reviewed by Twomey and Taylor 1994a, 1994b). During creep loading, in flexion the anterior part of the disc is compressed, the posterior part is stretched and the zygapophyseal joint surfaces are compressed. Fluid is expressed from the soft tissues so that there is relative deprivation of nutrients. There is progressive anterior movement, so that the range of flexion increases. During sustained flexion, creep causes an increase in the flexion angle of 10% in twenty minutes (McGill and Brown 1992). Sustained flexed postures also have the effect of reducing the resistance of the spinal ligaments, therefore making the spine weaker and more susceptible to injury – holding a flexed posture for five minutes reduces resistance by 42%, holding it for an hour reduces resistance by 67% (Bogduk 1997).

"If the amount of 'creep' involved after prolonged load bearing in flexion is considerable, then recovery back to the original starting posture (hysteresis) is extremely slow. It takes considerable time for the soft tissues to imbibe fluid after it has been expressed during prolonged flexion loading. Many occupational groups (e.g. stonemasons, bricklayers, roofing carpenters and the like) regularly submit their lumbar spines to this category of insult. They work with their lumbar column fully flexed and under load for considerable periods of time. There is often little movement away from the fully flexed position once it has been reached, and little opportunity for recovery between episodes of work in this position" (Twomey and Taylor 1994a, p. 144).

Sustained loading in flexion causes creep deformation of the lumbar vertebral column that progresses with time, and from which there is not immediate recovery, especially in older specimens. This predisposes the spine to be more susceptible to flexion injuries (Twomey and Taylor 1982; Twomey *et al.* 1988). Disc mechanics depend upon loading history as well as the load that is applied (Adams *et al.* 1996b). Flexion and fatigue loading to simulate a vigorous day's activity (Adams and Hutton 1983) and one hour of sitting (Wilder *et al.* 1988) have been shown to produce distortions, weakening and radial fissures in the lamellae of the annulus. Static loading to simulate extended and flexed sitting postures found that the latter generated considerably greater tensile force in the region of the posterior annulus (Hedman and Fernie 1997).

The role of repeated and sustained flexion postures in the aetiology of structural damage to spinal tissues has been explored experimentally. A modelling experiment has shown that bending may cause annular failure as the strain is highly localised in the posterior disc and if increases in fibre length exceed 4%, the annulus would be damaged (Hickey and Hukins 1980). Computer-generated disc models predict posterior annular fissuring will occur with flexion and compression (Natarajan and Andersson 1994; Shirazi-Adl 1989, 1994; Lu *et al.* 1996). Sustained flexion loading may lead to distortion and rupture of the annulus, which may be followed by extrusion of disc material (Adams and Hutton 1983, 1985a; Gordan *et al.* 1991; Wilder *et al.* 1988). Flexion and compression, with or without lateral bending or rotation, may cause disc prolapse, which may be sudden or gradual (Adams and Hutton 1982, 1985a; McNally *et al.* 1993; Gordan *et al.* 1991). However, these events are not easily produced and structural failure in the intervertebral disc may involve internal damage to the annulus rather than prolapse of disc material (Adams and Dolan 1995).

In contrast to the above effects, flexed postures have several physiological and mechanical advantages. Flexion is said to improve the transport of metabolites in the intervertebral disc and reduce the stress on the zygapophyseal joints and on the posterior half of the annulus fibrosus. It gives the spine a high compressive strength and reduces the stress peaks in the posterior annulus fibrosus (Adams and Hutton 1985b; Adams *et al.* 1994).

Extension creep loading

Compared to flexion, prolonged maintenance of an extended posture when working is unusual, although prolonged standing tends to increase the lordosis. However, high peaks of repetitive extension loading occur in certain sports, such as fast bowlers in cricket, gymnasts and high jumpers. The forces involved are considerable as the inferior articular process impacts on the lamina of the vertebrae below – with the highest concentration on the pars interarticularis. Repetition of extension and flexion movements may cause fatigue fractures of the pars interarticularis – which is the site at which spondylolysis occurs (Twomey and Taylor 1994b) (see section in Chapter 13 for more detailed consideration).

Axial creep loading

Axial creep loading occurs each day after the recumbent posture during sleep. The pressure sustained by the intervertebral discs causes a loss of fluid, amounting to a 10% loss in disc height. The fluid loss means that the individual is 1 – 2% shorter at the end of the day, and the loss is made up during sleep when the discs are rehydrated due to the osmotic pressure of the proteoglycans. Rehydration occurs more rapidly in the flexed than in the extended position (Bogduk 1997). The average change in human stature throughout the day is about 19mm (Adams *et al.* 1990). In effect, the disc swells during the night and is compressed during the day. The changes in disc height occur rapidly: 26% of the loss over eight hours upright occurs in the first hour and 41% of recovery over four hours occurs in the first hour of rest (Krag *et al.* 1990).

Optimal sitting posture

Two recent studies (Harrison *et al.* 1999, 2000; Pynt *et al.* 2001) have reviewed the evidence relating to the optimal sitting and driving posture. Harrison *et al.* (1999), in a thorough review of the biomechanical and clinical literature, concluded that the consensus on the optimal sitting position included maintenance of the lordosis with a lumbar support, seat inclination backwards, arm rests and seating that allowed freedom of movement. Flexion in sitting was shown to cause several disadvantages, and the consensus was in favour of a lordosis when sitting.

Pynt *et al.* (2001) reviewed the advantages and disadvantages of the lordotic and kyphotic sitting posture, drawing mostly on cadaveric

and a few clinical studies. They summarise the main arguments of proponents of both postures (Table 6.3). They found many of the arguments previously used by those who advocate the flexed posture to be flawed and unsubstantiated, and some of the data they re-evaluated. They conclude that the lordotic sitting posture, if regularly interrupted with movement, is the optimal seating position for spinal health and for preventing low back pain.

"In summary, then, a sustained lordosed sitting posture decreases disc pressure and thereby disc degeneration, exhibits less injurious levels of ligament tension, and although it increases zygapophyseal loading, this is not of itself considered hazardous to spinal health. A sustained kyphosed sitting posture, on the other hand, increases intradiscal pressure leading to increased fluid loss, decreased nutrition, and altered cell synthesis and biomechanics of the disc, appearing to culminate ultimately in disc degeneration that is a cause of low back pain" (Pynt *et al.* 2001, p. 14).

Table 6.3 Proposed advantages and disadvantages of kyphotic and lordotic sitting postures

Advantages of flexed position	*Disadvantages of flexed position*
	Increase intradiscal pressure
	Increase tensile stress posterior AF
	Increase compressive load posterior AF
Unload ZJ, but increase loading on IVD	Compressive force born entirely by IVD, ZJ only resists shear
	Poor position to resist creep
	Increased creep
	Dehydrates IVD
	Decreased nutrition
Advantages of lordotic position	*Disadvantages of lordotic position*
Decrease intradiscal pressure	
Reduce compressive forces on IVD	Increase compressive load posterior AF
Balance of forces acting on ZJ	Increases load on ZJ

AF = annulus fibrosus
IVD = intervertebral disc
ZJ = zygapophyseal joint

Source: Pynt *et al.* 2001

Effect of time of day on range of movement and biomechanics

Time of day affects not only the water content of the disc, and thus disc height, but this in turn affects range of movement and spinal biomechanics. Range of motion increases during the day (Ensink *et al.* 1996; Adams *et al.* 1987). There is a significant change in flexion and a smaller change in extension. From morning to evening one study found an average gain of eleven degrees of flexion, but only three degrees of extension (Ensink *et al.* 1996).

The axial creep loading that occurs during the day causes the disc to lose height, bulge more, and become stiffer in compression and more flexible in bending. Disc tissue becomes more elastic as the water content is reduced and disc prolapse becomes less likely. Maximal stress is thus exerted on the disc and posterior longitudinal ligament in the morning (Adams *et al.* 1990). Creep causes an increased stress on the annulus, a reduction of pressure on the nucleus pulposus and closer contact between the zygapophyseal joints (Adams 1994).

Because of the increased fluid content of the disc in the early morning, it is more resistant to flexion. Compared to later in the day, the stresses caused by forward bending are about 300% greater on the disc and 80% greater on the ligaments of the neural arch (Adams *et al.* 1987). Consequently, it is concluded that there is an increased risk of damage occurring to the disc when bending in the early morning. An experimental model using bovine discs has demonstrated that both flexion loading and high hydration rates were key factors in causing the break up and displacement of fragments of nucleus (Simunic *et al.* 2001).

Effect of posture on internal intervertebral disc stresses

Nachemson and colleagues in the 1960s (Nachemson and Morris 1964; Nachemson and Elfstrom 1970) performed the earliest measurements of disc pressures in vivo in a variety of normal postures (reviewed in Nachemson 1992). A needle attached to a miniature pressure transducer was placed in the nucleus pulposus of the L3 disc and measurements made in some common static positions, as well as some dynamic activities.

If the pressure in upright standing was 100%, in lying it was about 20 – 50%, and in sitting upright or leaning forward was about 150%. When sitting, the pressure in the disc is reduced by an inclined backrest, a low lumbar support, and the use of arm rests. Bending forward and lifting weights, whether sitting or standing, causes substantial increases in pressure – for instance, from 500 Newtons when standing at ease to 1700N when standing bending forward, weights in each hand, arms extended (Nachemson 1992).

Nachemson (1992) refers to six other reports that generally verified their preliminary findings. Quinnell and Stockdale (1983) confirmed the same relative disc pressures in lying, sitting and standing; however, the absolute values they recorded were less. Since these earlier studies of intradiscal pressure little new work has been done until recently. Wilke *et al.* (1999) performed a single case study of an individual with a non-degenerated L4 – L5 disc with pressure recordings over a twenty-four-hour period in a range of different activities. Their results generally correlated with Nachemson's data, except in one critical area. While the pressure in lying was 20% of relaxed standing, sitting slouched in a chair was also less, about 60% of standing. Turning over in bed, bending forward in sitting or standing, lifting and standing up from a chair all had the effect of causing substantial increases in pressure. Lifting with a rounded back caused considerably more pressure than lifting with knees bent and a straight back. Over a period of seven hours rest at night, pressure increased 240%.

On the finding that the intradiscal pressure in relaxed sitting may in fact be less than that in relaxed standing, the authors comment that both muscle activity and lumbar curvature affect the pressure. Slouched sitting may reduce pressure due to minimal muscle activity. The finding contradicts most other work that has been done in this field.

Another recent intradiscal pressure study reported findings from a group of patients and volunteers (Sato *et al.* 1999). Again, the pressure in lying was found to be much less than that in standing or sitting, and although the pressure in sitting was more than in standing, the difference was not substantial. Lying was about 20% of standing, sitting was about 20% more than standing, and the angles of flexion/extension had a considerable affect. While bending backwards in sitting or standing increased pressure by 10 – 20%, bending forward increased it by 100 – 150%. The degree of disc degeneration correlated linearly with reduced intradiscal pressure – more degeneration, less pressure.

In summary, the magnitude of pressure in the disc is influenced by various factors, such as trunk muscle activity, posture, body weight, size of disc, disc degeneration and externally applied loads (Sato *et al.* 1999). Posture is only one component in the equation; however, these studies show that it has a potent affect upon the pressure within the disc. All the studies report a substantial decrease in intradiscal pressure when non-weight bearing. Sitting is generally, but not universally, found to cause a higher intradiscal pressure than standing. Extension raises intradiscal pressure, but considerably less than does flexion.

Nachemson's original findings were used to justify ergonomic concepts used in back school. Before extrapolating these findings to the clinical situation, some limitations ought to be recognised. If the intervertebral disc is severely degenerated, the hydrostatic property is lost; therefore all pressure measurements can only be done in individuals with relatively normal discs (Nachemson 1992). Painful discs are likely to be morphologically abnormal, in which instance pressures may be substantially different. Most of the early studies were done at L3 disc, while the majority of symptomatic pathology occurs at the lower two discs. Perhaps most importantly, these measurements are made in the nucleus pulposus, which is not the site of discogenic pain. This most commonly is in the outer annulus (Moneta *et al.* 1994).

A more recent technique, stress profilometry, has sought to evaluate stresses in the intervertebral disc in both the nucleus and the annulus. To date, most work on stress profilometry has been conducted in vitro, with only one in vivo study. Stress peaks vary according to the posture of the motion segment being tested. In a cadaver study, 'degenerated discs' exposed to extension showed a generalised increase in stress peaks in the posterior annulus, while flexion tended to equalise the compressive stress. However, in seven of the nineteen motion segments tested, lumbar extension decreased maximum compressive stress in the posterior annulus by a considerable amount (Adams *et al.* 2000a). See Chapter 4 on intervertebral disc for more detail.

Conclusions

The chief movements available at the lumbar spine are flexion and extension. Over time, an individual's mobility may vary because of the ageing process and because of back pain. Because there is a

considerable range of normal values in mobility between people, movement loss must be judged against the individual's normal range and be correlated with pain response. Movement cannot be solely judged against some theoretical normative database.

With the use of everyday positions, the spine adopts movements of flexion, extension, and so on. These commonly adopted postures have clear effects on the lumbar curve, and patients' symptomatic responses to these loading strategies help us to understand their directional preference or lack of it. This understanding has important diagnostic and management implications.

Movements have different physiological affects on the lumbar spine, and in particular on the intervertebral disc. Some of this data comes from cadaveric studies, and extrapolation from in vitro studies to real life should not be taken too far. Nonetheless, it is apparent that different postures and movements influence internal disc dynamics and disc pressures. The role of loading history in causing fatigue damage to collagenous structures, such as the disc, is clearly significant in morphological change and in back pain. The frequency with which patients report that their musculoskeletal pain developed for 'no apparent reason' becomes understandable in this context.

7: Diagnosis and Classification

Introduction

Despite the technological advances that have been made in recent years, we are still unable to identify the origin of back pain in the majority of patients. Even with the advent of advanced imaging technology, such as computerised axial tomography (CAT) scanning and magnetic resonance imaging (MRI), our ability to identify the precise structure that generates symptoms and the exact nature of the pathology affecting it remains extremely limited.

Fordyce *et al.* (1995) lists the following as known causes of specific back pain with our present state of knowledge:

- disc herniation
- spondylolisthesis, usually in the young
- spinal stenosis, usually in the older age group
- definite instability, exceeding 4 – 5mm on flexion-extension radiographs
- vertebral fractures, tumours, infections and inflammatory diseases.

He goes on to state: *"The best evidence suggests that fewer than 15% of persons with back pain can be assigned to one of these categories of specific low back pain"* (Spitzer *et al.* 1987). However, ambiguities exist even about some of these conditions – in middle-aged patients the association between spondylolisthesis and back pain is weak and only found in women (Virta and Ronnemaa 1993). *"There is lack of scientific*
1994), and sagittal translation exceeding 4mm is found in those without back pain (Woody *et al.* 1983; Hayes *et al.* 1989). Furthermore, even within these groups, the most efficacious treatment has not been clearly defined. The vast majority of the back pain population, the other 85% at least, belong in the realm of non-specific back pain where the ambiguity of their diagnosis rests on the particular 'expert' that they consult (Deyo 1993).

While both clinicians and patients await the elucidation of this diagnostic ambiguity, management must be offered to those who seek

health care for the problem of back pain. If a diagnosis cannot be clearly made, classification systems may be a clinically useful way to characterise different sub-groups and their management strategies.

This chapter considers some of the issues concerning diagnosis and back pain. It describes the most widely adopted classification systems in use, those described by the Quebec Task Force (Spitzer *et al.* 1987), and the US and UK guidelines for back care (AHCPR 1994; CSAG 1994). The categories within these systems will be used to indicate those patients who are contraindicated for mechanical diagnosis and therapy and those who may be selected for a mechanical evaluation.

Sections in this chapter are as follows:

- identification of specific pathology
- classification of back pain
- Quebec Task Force classification
- classification by pain pattern
- other classification systems
- diagnostic triage
- indications for mechanical diagnosis and therapy
- factors in history that suggest a good response
- contraindications for mechanical diagnosis and therapy.

Identification of specific pathology

Although some specific diagnoses such as spinal stenosis or disc herniation may be suspected from clinical examination, to confirm such a diagnosis requires paraclinical investigations. Sophisticated imaging studies, blood tests or biopsies are examples of tests used to confirm a diagnosis. One way of interpreting clinical tests is their ability to relate an abnormal finding to the presence of disease. *Sensitivity* is the term used to describe those who have the 'disease' that are correctly identified as 'disease positive' by the test. *Specificity* is the term used to describe those who do not have the 'disease' that are correctly identified as 'disease negative' by the test (Altman 1991). A key failing of many types of spinal imaging, however sophisticated, is their inability to relate pathology to symptoms. Abnormal morphology may be found in individuals who have no symptoms,

thus the specificity of that test is poor. In effect, many people may be told, for instance, that they have degenerative disease of the lumbar spine, when in fact these radiographic findings are not related to their symptoms – these are false-positive findings. To base diagnosis and management upon these findings alone is seriously questionable.

A recent systematic review of studies looking at the association between radiographic findings and non-specific back pain concluded that there was no firm evidence for a causal relationship between the two (van Tulder *et al.* 1997c). Spondylolysis, spondylolisthesis, spina bifida, transitional vertebrae, spondylosis and Scheuermann's disease did not appear to be associated with back pain. Degeneration, defined by the presence of disc space narrowing, osteophytes and sclerosis, appears to be associated with back pain, but not in any causal way (van Tulder *et al.* 1997c). When any of these abnormalities are found on radiography, 40 – 50% will be false-positive findings; that is, found in those with no back pain (Roland and van Tulder 1998). The authors suggest that a finding of advanced disc degeneration on radiography should have this information inserted in any report: *"Roughly 40% of patients with this finding do not have back pain, so finding may be unrelated"*. They advise similar caveats to accompany the reporting of any of the other morphological abnormalities listed above.

The more sophisticated imaging studies are also associated with poor specificity. Computer-assisted tomography (CAT) found abnormalities, mostly disc herniation, in about 20% of asymptomatic individuals younger than 40 and in 50% of asymptomatic individuals older than 40 (Wiesel *et al.* 1984).

Numerous studies have identified the very high rate of false-positive findings on magnetic resonance imaging (MRI) of the lumbar spine. Bulging or protruded discs have been found in over 50% of asymptomatic individuals (Jensen *et al.* 1994; Weinreb *et al.* 1989), and in those over 60 years 36% of subjects had a disc herniation and 21% had spinal stenosis (Boden *et al.* 1990). When patients were matched to controls by age, sex and physical risk factors, 76% of the asymptomatic controls had protrusions or extrusions of the disc, which in 22% even included neural compromise. In the patients, the respective proportions were 96% and 83% (Boos *et al.* 1995). Patterns of disc disruption have been seen as commonly in volunteers without back pain as in patients (Buirski and Silberstein 1993), and MRI has

failed to reliably predict symptomatic discs that have been identified by discography (Brightbill *et al.* 1994; Horton and Daftari 1992).

These studies clearly show that imaging studies *by themselves* have very little value in identifying abnormal morphology of symptomatic significance, and thus should not be used to formulate diagnosis or treatment in isolation from the patient's clinical presentation. Imaging studies can identify abnormal morphology, but this may not be relevant. It is well recognised that in the case of disc herniations imaging studies should be used to confirm the findings of a clinical evaluation – the diagnosis can only be confirmed by MRI or CAT, but in the absence of the clinical presentation false-positive findings are likely (Deyo *et al.* 1990).

Some authors argue that with the use of intra-articular or disc stimulating injections, a source of pain may be found in over 60% of back pain patients (Bogduk *et al.* 1996). According to their criteria, the prevalence of discogenic pain is about 39%, the prevalence of zygapophyseal joint pain is about 15% and the prevalence of sacro-iliac joint pain is about 12%. These diagnoses, however, rely upon invasive procedures involving significant exposure to x-rays, which are costly and require high levels of skill – they are not a realistic alternative for the majority of back pain patients. Furthermore, while injections may identify these diagnostic groups, no clinical criteria have been revealed that would allow their identification by simpler means, and at this stage no effective treatments exist for such diagnoses.

Mechanical evaluation can identify and affect the mechanism of symptom generation. The McKenzie assessment process has been found to be superior to MRI in distinguishing painful from non-painful discs. *"A non-invasive, low-tech, relatively inexpensive clinical assessment using repeated end-range lumbar test movements can provide considerably more relevant information than non-invasive imaging studies"* (Donelson *et al.* 1997).

Our desire as clinicians to diagnose and label back pain should be circumspect with a natural humility in light of the above. Using unproven pathological labels may not only be a fraudulent attempt to augment our professional credibility, it may also lead to exaggerated illness behaviour by patients and abnormal treatment patterns by clinicians.

"We use unproven labels for the symptoms of back pain; our ability to diagnose 'facet
trials. 'Degenerative disc disease' is common among all of us above 30 years of age. 'Isolated disc resorption' is a common diagnosis presumed to require fusion operation on in some parts of the world. 'Segmental instability' is also generally undefined. These are diagnostic 'waste baskets' into which we sort our patients. Abnormal diagnostic behaviour leads some patients into sick role behaviour. Patients become afraid, they ask, 'Can you cure degenerative disc disease?' Ill-defined labels help to produce a person who cannot cope, leading to illness behaviour, which in turn might lead physicians and surgeons to perform 'abnormal' treatment" (Nachemson 1999a, p. 475).

Classification of back pain

In the absence of clear diagnoses, classification systems provide several advantages (Spitzer *et al.* 1987; Fairbank and Pynsent 1992; Delitto *et al.* 1995). They help in making clinical decisions, may aid in establishing a prognosis, and are likely to lead to more effective treatment if patients are treated with regard to classification. They aid communication between clinicians and offer an effective method of teaching students. Classification systems also further our understanding of different sub-groups and should be used in the conduct of audit and research.

Unfortunately, there exists a wide variety of back pain classifications from which to choose (Fairbank and Pynsent 1992; Riddle 1998), and more systems continue to appear. Three classification systems based on extensive research reviews will be briefly mentioned (Spitzer *et al.* 1987; AHCPR 1994; CSAG 1994). These highlight the fact that most back pain is non-specific, but also that we must be aware of certain specific pathologies that are far less common – nerve root pathology and serious spinal pathology.

Quebec Task Force classification

After an exhaustive review of the literature, the Quebec Task Force (QTF) reported on Activity-Related Spinal Disorders (Spitzer *et al.* 1987), within which they addressed the problem of diagnosis. They highlighted the fact that in the vast majority pain is the only symptom – which, although initially nociceptive in origin, can be influenced by

psychological and social factors during the progression into chronicity. Although pain may develop due to irritation of bones, discs, joints, nerves, muscles and soft tissues, the identification of the precise origin of pain is difficult. Pain characteristics are generally non-specific for different structures, and clinical observations cannot easily be corroborated through objective methods. *"Non-specific ailments of back pain....with or without radiation of pain, comprise the vast majority of problems"* (Spitzer *et al.* 1987).

The QTF determined that their classification system must be compatible with present knowledge, universally applicable, involve mutually exclusive categories, be reliable between clinicians, be clinically useful and be simple to use. Using these criteria as a guide, the QTF recommended the following classification be universally adopted.

Table 7.1 QTF classification of back pain

1. Back pain without radiation
2. Back pain with radiation to proximal extremity
3. Back pain with radiation to distal extremity
4. Back pain with radiation to distal extremity and positive neurological signs (i.e. focal muscle weakness, asymmetry of reflexes, sensory loss in a dermatome, or loss of bladder, bowel or sexual function)
5. Presumptive compression of a spinal nerve root on radiographs (i.e. instability or fracture)
6. Compression of a spinal nerve root confirmed by special imaging techniques (i.e. as category 4 with moderate or severe findings on neuroradiological review at appropriate level)
7. Spinal stenosis
8. Post-surgical status, 1 – 6 months after intervention, asymptomatic (8.1) or symptomatic (8.2)
9. Post-surgical status, > 6 months after intervention, asymptomatic (9.1) or symptomatic (9.2)
10. Chronic pain syndrome
11. Other diagnoses (i.e. metastases, visceral disease and fracture).

Source: Spitzer *et al.* 1987

Their first four categories represent *most* cases and are determined by history-taking and clinical assessment, categories 5 – 7 depend upon paraclinical investigations and categories 8 – 10 on response to treatment. Each of the first four classifications is subdivided by a temporal division into acute (< 7 days), sub-acute (7 days – 7 weeks) and chronic (> 7 weeks), as well as work status (working or idle).

Categories 1, 2 and 3 describe disorders of somatic structures, while QTF 4 and 6 describe disorders affecting the nerve root as well. QTF 4 includes the classic radicular syndromes most frequently caused by disc herniations; if this is confirmed by an imaging study, this becomes QTF 6.

Classification by pain pattern

The QTF uses pain pattern as a means of classification of non-specific back pain. Pain pattern is certainly an indicator of severity. Patients with sciatica or referred symptoms are substantially more disabled (Leclaire *et al.* 1997) and have a more protracted rate of recovery and return to work than patients with back pain alone (Andersson *et al.* 1983; Hagen and Thune 1998). Leg or sciatic pain is a factor that is commonly recognised as having a poorer prognosis for recovery and a greater likelihood of developing chronic symptoms (Goertz 1990; Lanier and Stockton 1988; Chavannes *et al.* 1986; Cherkin *et al.* 1996a; Carey *et al.* 2000; Thomas *et al.* 1999), and as a risk factor for predicting future episodes of back pain (Smedley *et al.* 1998; Muller *et al.* 1999).

When the QTF classification system or a similar system has been used, higher categories are associated with increasing severity of symptoms and reduced functional ability (Atlas *et al.* 1996a; Selim *et al.* 1998; BenDebba *et al.* 2000). The hierarchical classification demonstrated progressive increases in the intensity of pain, associated disability, the use of medical services and a gradual reduction in health-related quality of life. *"Patients with equivocal evidence of radiculopathy tend to have intermediate impairment, compared with the impairment in those with sciatica and with the impairment in those with LBP alone"* (Selim *et al.* 1998). Patients with distal leg pain and positive neural tension signs were nine times more likely to receive an advanced imaging study than patients with back pain only, and thirteen times more likely to come to surgery (BenDebba *et al.* 2000). The natural history and clinical course of patients in QTF categories 4 and 6 is frequently poor. Of 82 consecutive patients followed for a year following in-patient conservative therapy, only 29% were fully recovered and 33% had come to surgery. Most recovery occurred in the first three months, after which there was little further improvement (Balague *et al.* 1999).

The QTF recommendations support the concept of classification of non-specific spinal disorders by utilising pain patterns. The first four categories of the QTF are very similar to the pain pattern classification first proposed by McKenzie (1981). The classification offers a way of monitoring deteriorating or improving spinal disorders. Movements or positions that produce increasing peripheral symptoms are to be avoided. The centralisation of pain results from a reduction in the deformation or compression of the nerve root and articular structures, and thus movements or positions that cause this abolition of peripheral symptoms are to be encouraged. By causing tingling in the outer toes to cease and pain felt below the knee to change location to the buttock and thigh, the severity of the condition is reduced and the classification changes from QTF 4 to QTF 2. *This simple way of monitoring symptoms provides clinicians with a reliable way to judge a worsening or improving clinical situation and thus the appropriateness of certain procedures.*

Rather than representing different categories within the back pain population, the different pain patterns actually represent stages of the same problem that commonly change during the natural history of the episode as it waxes and wanes. They represent a way of monitoring the status (improving, worsening or unchanging) of a condition and the response to therapeutic loading strategies. Any loading that reduces, abolishes or centralises distal pain should be pursued, just as alternatively any loading that produces, increases or peripheralises pain should be avoided. It is hoped that we will change a patient with QTF category 3 to QTF 1, prior to complete abolition of pain. *The value of pain pattern classification is thus not in representing distinct categories, but as a means of monitoring symptom severity and response to therapeutic loading strategies.*

Other classification systems

Two national guidelines published in the USA (AHCPR 1994) and the UK (CSAG 1994) have recommended an even simpler classification system based upon a hierarchy of pathological risk. After determining that it is a musculoskeletal problem, the initial focused assessment should classify patients into one of three groups:

- serious spinal pathology – cauda equina syndrome, cancer, neurological disorder, inflammatory disease, etc.

- nerve root problems – disc herniation, spinal stenosis
- mechanical backache – non-specific back and radiating leg pain representing the majority of patients, in which symptoms vary with different physical activities and time.

Diagnostic triage

Within this diagnostic triage, the majority of all patients will be in the 'simple backache' group, with true nerve root problems said to affect less than 5%, and less than 2% due to serious spinal disease (such as tumour or infection) and inflammatory conditions (CSAG 1994). There is some overlap between the three classification systems. Respectively these groups represent QTF categories 1, 2, 3 (mechanical backache), QTF categories 4, 5, 6, 7 (nerve root pathology) and QTF category 11 (serious spinal pathology).

The first category, mechanical backache, describes the patients most commonly referred for conservative physiotherapy treatment by physicians. The second category, nerve root pathology, describes a much smaller group who are also seen regularly and are often suitable for conservative treatment. The last category, serious spinal pathology, is unsuitable for conservative treatment. It is hoped that most patients attending physiotherapy will have been screened by medical practitioners and those with unsuitable pathologies excluded. However, in case unsuitable patients are referred, and as physioclinicians are more commonly becoming first-line practitioners in assessing back pain patients, *an awareness of the 'red flags' indicating serious spinal pathology is imperative.* The first task is to screen out patients who have 'red flags', which indicate serious spinal pathology.

Serious spinal pathology

These conditions are very unusual; in a cohort of over 400 patients with acute back pain in primary care, six (1.4%) had 'red flag' conditions (McGuirk *et al.* 2001). Three of these had fractures and three had carcinomas. A few key questions during the medical history could alert clinicians to 'red flag' pathology and ensure that serious underlying conditions, such as cancer, inflammatory diseases or significant neurological disorders are not missed (AHCPR 1994; CSAG 1994; Deyo *et al.* 1992):

Cauda equina syndrome/widespread neurological disorder:

- bladder dysfunction (usually urinary retention or overflow incontinence)
- loss of anal sphincter tone or faecal incontinence
- saddle anaesthesia about the anus, perineum or genitals
- global or progressive motor weakness in the lower limbs.

Possible serious spinal pathology (cancer, infection, fracture):

- age (>55)
- history of cancer
- unexplained weight loss
- constant, progressive, non-mechanical pain, worse at rest
- systemically unwell
- persisting severe restriction of lumbar flexion
- widespread neurology
- systemic steroids
- history of intravenous drug use
- history of significant trauma enough to cause fracture or dislocation (x-rays will not always detect fractures)
- history of trivial trauma and severe pain in potential osteoporotic individual
- no movement or position centralises, decreases or abolishes pain.

Possible inflammatory disorders:

- gradual onset
- marked morning stiffness
- persisting limitation of movements in all directions
- peripheral joint involvement
- iritis, psoriasis, colitis, uretheral discharge
- family history
- no movement or position centralises, decreases or abolishes pain.

Management

It is imperative (CSAG 1994) that patients with symptoms indicating spinal cord damage, cauda equina syndrome or a widespread neurological disorder are referred to a specialist immediately. *For these patients, mechanical therapy is absolutely contra-indicated.* If there is not a direct referral system to a specialist, you must send these patients directly to the emergency department. Although very rare, it is extremely important that patients who are suspected of having these conditions are sent to the appropriate specialist straight away.

Failure to react promptly to a patient who reports loss of bladder control can result in permanent loss of bladder, bowel and sexual function. A recent retrospective review of patients who had had surgery for cauda equina syndrome highlighted the need for urgency of referral in such cases (Shapiro 2000). Patients who had the diagnosis made and surgery performed within 48 hours of onset were compared to those who had surgery more than 48 hours after onset. Those who had delayed surgery were significantly more likely to have persistent bladder and bowel incontinence, severe motor deficit, sexual dysfunction and persistent pain.

Patients with other possible serious spinal pathology or inflammatory disorders should also be referred to the appropriate specialists. *For these patients, mechanical therapy is absolutely contra-indicated.* If there are suspicious clinical features or if acute pain has not settled in six weeks, an erythrocyte sedimentation rate test and plain radiograph should be considered (CSAG 1994).

Detailed descriptions of specific examples of serious spinal pathology are given in a Chapter 12. Assessment for 'red flag' pathology is also included in the chapter on history-taking (14).

Nerve root problems

The following aspects of the clinical presentation gained during the history-taking can indicate nerve root pain (CSAG 1994; AHCPR 1994):

- unilateral leg pain > back pain
- pain radiating to foot or toes, especially in dermatomal pattern
- numbness or paraesthesia in the same distribution
- history of weakness in the legs

- history of neurogenic claudication (limitation of walking distance due to leg pain).

The following signs, gathered during the physical examination, will heighten suspicion of neurological involvement (CSAG 1994; AHCPR 1994):

- weakness of ankle dorsiflexion, or great toe or calf and hamstring muscles, suggesting involvement of L4, or L5 or S1 nerve roots
- loss of ankle reflex, suggesting involvement of S1 nerve root
- loss of sensation in area of medial ankle, big toe or lateral foot, suggesting involvement of L4, or L5 or S1 nerve roots
- reduced straight leg raise
- cross straight leg raise (in which straight leg raising the opposite leg increases symptoms in the painful leg)
- in patients with irreducible disc herniations or spinal stenosis, no movement or position will be found that will centralise, decrease or abolish pain.

This abbreviated neurological examination will detect most clinically significant nerve root pathology at the lower lumbar levels (L4 – L5 and L5 – S1), where over 90% of all disc herniations occur (AHCPR 1994; Deyo *et al.* 1990). It will miss the much less common lesions involving upper lumbar levels. These may be suspected with anterior thigh pain and reduced sensation, quadriceps weakness and reduced quadriceps reflex – present in less than 5% of patients with proven disc herniations (Deyo *et al.* 1990). Patients with nerve root involvement usually do not display all the above signs and symptoms. Patients with stenosis generally present with fewer neurological signs, are much less likely to have the marked root tension signs found in those with disc herniation and complain of intermittent claudication.

Established musculoskeletal causes of nerve root problems, which may be suspected clinically but need paraclinical investigations to be confirmed, are:

- disc herniations
- spinal stenosis
- malignant and non-malignant tumours (rare).

Management

The two main causes of nerve root pathology are disc herniations and spinal stenosis. Disc disease is discussed in more detail in Chapter 4, and the clinical features of sciatica in Chapter 5. It is important to be aware that this diagnosis represents a continuum from reducible protrusions through to non-contained sequestrations, whose prognosis is very different. For many the prognoses with conservative management is reasonable, and this is the recommended approach at least in the first six weeks – during which period 50% are said to recover from the acute attack (CSAG 1994; Deyo *et al.* 1990), although this seems rather over-optimistic. A minority, with the more extreme pathology, may need surgery. Stenosis, although irreversible, is usually not progressive and is discussed in more detail later.

Management decisions must be made with awareness of the greater pain and disability often associated with nerve root problems compared to simple backache. As a consequence, these patients may respond more slowly, and some may not respond at all to conservative treatment. In pathological terms, the two entities of backache and nerve root problems represent different conditions affecting different structures with different natural histories. See Chapter 3 for a summary of somatic and radicular pain. *However, there is no reason to differentiate these groups as far as initial management is concerned.* These were the patients referred to previously (McKenzie 1981) as those with derangement 5 or 6. Greater caution should be exercised when testing patients with nerve root problems.

Simple or mechanical backache

The criteria for this group are as follows (CSAG 1994):

- mostly aged 20 – 55 years at onset
- lumbosacral region, buttocks and thighs
- 'mechanical' in nature; that is, the pain varies with physical activity and over time
- patient is generally well.

In essence, after those with specific serious pathology or nerve root involvement, this is all the rest – that is, the majority of those who have back problems.

This group includes those previously (McKenzie 1981) referred to as having derangement 1, 2, 3 and 4, dysfunction and postural syndrome and also those with other entities such as sacro-iliac joint, hip problems or those with symptomatic spondylolisthesis.

Management

The initial management pathway for both simple backache and nerve root problems should be the same. A mechanical evaluation should follow the history-taking, and details from both elements of data gathering should be used in patient classification. Following the mechanical evaluation, which is described later, many patients are classified as mechanical responders and management using extension, lateral, flexion, or some combination of forces can be instigated. Some patients with nerve root pathology display signs of non-contained disc lesions – that is, not amenable to conservative therapy – irreducible derangements. Other mechanical non-responders may belong in other categories such as stenosis, sacro-iliac joint (SIJ) problems, spondylolisthesis or chronic back pain.

Indications for mechanical diagnosis and therapy

The majority of back pain patients, with or without referred symptoms, thus include those ideally suitable for a mechanical evaluation either by repetitive end-range motion and/or static loading. The effect of repeated or static end-range loading on pain patterns can determine, often on day one, the potential of that patient to respond to mechanical therapy. Treatment response indicators are looked for during the mechanical evaluation when a directional preference or other consistent mechanical response is sought, thus indicating the presence of one of the three mechanical syndromes (derangement most commonly, followed by dysfunction and then posture). This will include the majority of patients with non-specific spinal pain. By using such an assessment, we can classify sub-groups within the non-specific spectrum of mechanical spinal disorders – that is derangement, dysfunction, or posture syndromes. Thus we are able to identify those patients who may be helped and, just as importantly, those who are unlikely to respond to mechanical therapy.

Some patients in QTF classifications 3 and 4 may turn out to have irreducible derangements or present clinically as spinal stenosis (QTF 7). Patients who fit into QTF category 4, with significant motor deficit and severe constant pain due to nerve root irritation, are less likely

to respond because of the severity of their pathology. However, a trial of mechanical therapy is always valuable as there are exceptions, and those who have intermittent symptoms of nerve root interference should certainly be evaluated. Once nerve root compression has been confirmed with imaging studies (QTF 6), the likelihood of surgical intervention is much greater (Atlas *et al.* 1996a).

In others in whom a consistent mechanical response is not forthcoming when mechanical therapy has been tested for several days, other classifications may need to be considered. Chapter 13 gives descriptions of conditions not encompassed within the three mechanical syndromes described previously (McKenzie 1981). These may need to be considered in the differential diagnosis, ***but only if the response is atypical to one of these syndromes***. The history and mechanical evaluation, which is described later, allows classification into one of the mechanical syndromes (derangement, dysfunction or posture). Classification is confirmed or questioned by the patient's response to mechanical therapy, which can involve testing over several days. If classification into a mechanical syndrome is not confirmed, differential diagnosis should be considered. It is thus essential to conduct a full mechanical evaluation in all suitable cases before proceeding to include non-mechanical differential diagnoses. ***Secondary classifications should only be considered once the extended mechanical evaluation has ruled out a consistent mechanical response.***

Once this has been done, the specific and non-specific categories (see Chapter 13) are those commonly considered in the literature; this includes spinal stenosis, hip joint problems, SIJ, back pain in pregnancy, zygapophyseal joint problems, spondylolisthesis, instability, mechanically inconclusive, post-surgical and chronic pain syndromes. The descriptions given make clear that while the existence of some of these categories are both substantiated by the literature and putative recognition is clinically feasible, for other categories the evidence fails to endorse their existence as a clinical entity and/or their recognition through physical examination.

The classification algorithm and the accompanying criteria and operational definitions are detailed in the appendix.

Factors in history that suggest a good response

An episodic history of back pain

Several aspects of history indicate factors that are frequently associated with a good response to mechanical diagnosis and therapy. Patients who have experienced recurring episodes of back and referred pain can do very well on the protocols outlined in this book. These patients describe long periods – weeks or months at a time – when they are completely symptom-free and can move fully and freely, and then unexpectedly they develop another episode of back pain. Patients with such a history can be very receptive to ideas on better self-management of their condition, especially if they have received passive or manipulative therapy in the past that has provided short-term relief, but given them no better long-term control. When taught appropriate exercises, these patients feel much more able to self-manage their problem by reducing the rate of recurrences and by resurrecting the exercises if symptoms return (Udermann *et al.* 2000, 2001; McKenzie 1979; Laslett and Michaelsen 1991). Not providing patients with the ability to manage their recurrent problem better is clearly poor practice.

Intermittent back pain

A second and perhaps more significant factor denoting those patients who will be most responsive is the group who feel their symptoms intermittently; that is, there are times during the day when, as a result of being in certain positions or performing certain activities or for no apparent reason, the patient has no pain. Even in those patients who have had symptoms for years and may be deemed chronic, intermittent symptoms indicate the likelihood of a good prognosis. Back pain that behaves in this way is demonstrating mechanically responsive pain – certain positions or movements are causing strain upon spinal tissues that generates pain, while other positions or movements reduce deformation of spinal tissues and relieve the pain. Frequently patients are very aware of postures that aggravate or relieve their symptoms, and educating them to temporarily avoid aggravating factors and make use of reductive factors is very straightforward.

If patients have pain and paraesthesia below the knee on an intermittent basis, they should respond well to the appropriate procedures. However, should they have constant pain below the knee, constant paraesthesia or numbness and motor or reflex deficit, rapid resolution is much less likely, and failure to respond to conservative care is common.

Variability in pain pattern

Another factor that can be a good predictor of a patient who responds well to mechanical therapy is when the patient reports that their pain changes location. It might be on the left sometimes, or at times on the right of their spine. Alternatively, a patient might report that the distal spread of their pain varies during the day and with different physical activities. Sometimes they only have back pain, and sometimes it radiates into their thigh or leg. They could report that in maintaining certain postures such as sitting they experience leg pain, but this is abolished when they walk about. This variability of pain pattern often indicates a patient who will do well with the management strategies outlined in this book.

A good indication of patient suitability for this approach to treatment is often obtained on day one during the mechanical assessment. If, during the initial testing procedures, pain centralisation or reduction of pain intensity occurs, this is invariably indicative of a good prognosis. However, it is sometimes necessary to conduct the mechanical evaluation over several days in order to ensure exposure of response.

Contraindications for mechanical diagnosis and therapy

Patients whose history suggests 'red flag' pathology are absolutely unsuitable for treatment. Those with suspected fractures, metastases, cauda equina, bone weakening disease or progressive neurological disease should be *immediately* referred on for further investigation (see Chapter 12). Usually a full mechanical evaluation is unnecessary as the relevant information can be gained during the initial interview. *A full mechanical assessment might be contraindicated in such individuals*. Patients with suspected but as yet undiagnosed inflammatory joint diseases, such as rheumatoid arthritis, ankylosing spondylitis, Reiter's syndrome, etc. should be referred for rheumatological assessment.

Conclusions

This chapter has described the initial algorithm for evaluation of those with back pain. In very general terms, patients either present with mechanical low back pain, nerve root pathology or serious spinal pathology. The latter, if detected, is unsuitable for mechanical diagnosis and therapy and any patient with the features outlined

above should be referred on to a specialist – these are considered in more detail in the chapter on serious spinal pathology (Chapter 12).

Ninety-eight percent or more of patients with back pain are suitable for a mechanical evaluation, including those with and without signs of nerve root involvement. The full mechanical assessment, which is described later, seeks to identify those patients whose conditions are mechanically responsive and fit into one of the mechanical syndromes. These are described in the chapters on derangement, dysfunction and posture syndromes. Testing for them should be carried out over several days.

Not all patients fit neatly into one of the mechanical syndromes. During the period of mechanical evaluation, atypical or inconclusive responses may arise. In this instance one of the specific or non-specific categories described in Chapter 13 should be considered.

Table 7.2 gives an outline of initial clinical categories. The anatomical definitions for specific categories, their criteria and operational definitions are detailed in the appendix – *this is essential reading*. The clinical reasoning algorithm focusing on the mechanical syndromes is given in more detail in the next chapter. Descriptions of serious spinal pathology, the mechanical syndromes and other categories are given in later chapters.

Table 7.2 Initial management pathway – key categories, estimated prevalence in back pain population

Serious spinal pathology <2%	*Nerve root pathology <10%*	*Simple backache >90%*
Specialist referral	Mechanical evaluation	Mechanical evaluation
	Mechanical responders	Mechanical non-responders
		Irreducible derangements
		Other

Patients with either simple back pain or that involving nerve root signs or symptoms can be considered for initial mechanical evaluation. Most of these will prove to be positive mechanical responders. A few will be non-responders due to irreducible derangements or other pathology. A very small number of patients present with 'red flags' indicating serious spinal pathology – for such patients mechanical therapy is contraindicated and urgent appropriate referral is required.

8: Mechanical Diagnosis

Introduction

As discussed in the chapter on diagnosis and classification, specific diagnoses within the field of spine care are still largely illusory. For this reason non-specific classifications have been suggested, except in the instance of serious spinal pathology (Spitzer *et al.* 1987; CSAG 1994; AHCPR 1994). McKenzie (1981, 1990) proposed three non-specific mechanical syndromes – posture, dysfunction and derangement – which are now widely used in musculoskeletal care.

A syndrome is a characteristic group of symptoms and pattern of happenings typical of a particular problem (*Chambers Dictionary*). It describes an entity that is recognisable by its typical pattern of symptoms, which can be used to guide treatment as it also describes a distinguishing pattern of responses. Syndrome recognition is achieved through a mechanical evaluation – that is, a focused history-taking and physical examination.

The three separate mechanical syndromes can be recognised by certain features of the clinical presentation and by applying a structured sequence of loading. The characteristic of each in response to repeated and/or sustained end-range loading is completely different. Correct identification allows the application of the appropriate mechanical therapy. Within these syndromes can be found the vast majority of non-specific spinal problems.

The history-taking and physical examination that is required in order to explore each clinical presentation is given in later chapters. This chapter briefly defines the three mechanical syndromes and their accompanying conceptual models. Their clinical presentations and more detail will be given in the chapters relevant to each syndrome.

Sections in this chapter are as follows:

- derangement syndrome
- dysfunction syndrome
- postural syndrome

- the role of mechanical diagnosis and therapy in the management of back pain.

Derangement syndrome

This is the most common of the three mechanical syndromes encountered in spinal problems. The clinical pattern in derangement is much more variable than in the other two syndromes. Pain from derangement can arise gradually or suddenly. Pain can be constant or intermittent, it may move from side to side, and proximally and distally; repeated movements and sustained postures can rapidly and progressively worsen or improve the severity and spread of pain. Signs and symptoms may be either somatic, radicular or a combination of the two, depending on the severity of the condition. Derangement syndrome is also characterised by a mechanical presentation, which usually includes diminished range or obstruction of movement and may include temporary deformity and deviation of normal movement pathways. Because both the symptomatic and mechanical presentations are influenced by postural loading strategies during activities of daily living, they may vary during the day and over time. Inconsistency and change are characteristic of derangement.

Internal derangement causes a disturbance in the normal resting position of the affected joint surfaces. Internal displacement of articular tissue of whatever origin will cause pain to remain constant until such time as the displacement is reduced. Internal displacement of articular tissue obstructs movement.

The conceptual model that has been used to explain derangement syndrome relates the presentation to internal intervertebral disc displacements (McKenzie 1981, 1990). These may present in a variety of different ways, as derangements are a continuum. At its embryonic stage, individuals may suffer from brief bouts of back pain and minor limitations of function that last only a few days and resolve spontaneously. At its most extreme, the internally displaced tissue overcomes the restraining outer wall of the annulus fibrosus and extrudes into the spinal or intervertebral canal, causing predominantly radicular signs and symptoms. The conceptual model is discussed at length in Chapter 9.

The derangement syndrome is clearly distinguishable from the other mechanical syndromes, both by its presentation and its response to loading strategies. A unique characteristic of the derangement syndrome is the ability of therapeutic loading strategies to bring about lasting changes in the symptoms and mechanics of back pain. Certain loading patterns may cause pain to worsen or peripheralise, while opposite loading strategies cause a reduction, abolition or centralisation of symptoms and a recovery of movement. These types of changes are only found in derangement syndrome. Many derangements respond to extension and some to lateral or flexion loading – these would be the principles applied to reduce the derangement, restore mobility and improve the symptoms.

In some instances of more severe derangements, no loading strategy is able to exert a lasting change on symptoms. All treatment principles either have no effect or else only produce a worsening or peripheralisation of symptoms. In this instance the mechanical evaluation has detected an irreducible derangement. When related to the conceptual model, this concerns an incompetent or ruptured outer annular wall that is not amenable to resolution by loading strategies and is at the extreme end of the pathological continuum.

Derangement syndrome is characterised by a varied clinical presentation and typical responses to loading strategies. This includes worsening or peripheralisation of symptoms in response to certain postures and movements. It also includes the decrease, abolition or centralisation of symptoms and the restoration of normal movement in response to therapeutic loading strategies.

Dysfunction syndrome

In the dysfunction syndrome, pain is never constant and appears only as the affected structures are mechanically loaded. Pain will stop almost immediately on cessation of loading. When affecting articular structures, the dysfunction syndrome is always characterised by intermittent pain and a restriction of *end-range movement*. When affecting contractile structures, functional impairment is demonstrated when the muscle or tendon is loaded at any or certain points during the physiological range. Movements and positions consistently cause pain to be produced, but symptoms cease when the position or loading is ended.

It is relatively straightforward to distinguish these separate types of dysfunction in extremity problems, whereas in the spine the distinction is not so clear. In the spine the syndrome presents as articular dysfunction, with pain at limited end-range.

Pain from the dysfunction syndrome is caused by mechanical deformation of structurally impaired tissues. This abnormal tissue may be the product of previous trauma or inflammatory or degenerative processes. These events cause contraction, scarring, adherence, adaptive shortening or imperfect repair. Pain is felt when the abnormal tissue is loaded.

Dysfunction syndrome arises from a past history of some kind, such as trauma or a previous episode of back pain, or it can arise insidiously, resulting from years of poor posture or degenerative changes. There may have been a previous episode of back pain, the original cause of which has recovered by fibrous repair. Six to eight weeks later the individual is left with persistent symptoms each time they stretch the affected tissue, and full function does not return, or persisting poor postural habit could have the effect of overstretching ligamentous and capsular structures, causing minor but recurrent micro-trauma and repair. Eventually this may lead to a loss of elasticity, a restricted range of movement and pain when the affected tissues are stretched. Whatever the initial cause, adaptive shortening of tissues now causes a painful restricted end of range; pain is produced each time the affected tissue is stretched or compressed, but abates as soon as the position is released. In each instance tissues have gone through the repair process, but have not been adequately remodelled to return to full function.

When structural changes and or impairment affect joint capsules or adjacent supporting ligaments, painful *restriction* of *end-range* movements in one or more directions will be experienced. Pain from the dysfunction syndrome persists until remodelling of the affected structures occurs. Alternatively, abnormal tissue may persist from an unreduced derangement, in which case there will be a painful *blockage* to *end-range* and symptoms are produced on compression of the joint.

Generally, the exact tissue at fault in dysfunction syndrome is not known. In spinal problems pain is always produced at end-range, when tissues are stretched and/or compressed. Thus in the spine

dysfunction presents as articular, but involvement of contractile structures cannot be ruled out. In one instance, adherent nerve root, the source of symptoms is known. In this form of dysfunction a past derangement causing an episode of sciatica has resolved, but the repair process has left some tethering or adherence that now inhibits full movement of the nerve root/dural complex. The syndrome is also a common consequence of spinal surgery id appropriate rehabilitation is not instigated. In the case of an adherent nerve root, flexion is markedly restricted and each attempt to flex fully reproduces the patient's pain, which can be felt in the back or the leg. This is the only dysfunction that can produce peripheral pain; all other examples cause spinal pain only. Most commonly these are caused by dysfunctions affecting movements into extension and flexion.

Pain from dysfunction will not go away by itself, but persists as long as the adaptive shortening or blockage to movement exists, and is consistently reproduced every time the affected tissue is stressed. The only way to resolve dysfunction is a regular remodelling programme that repeatedly stresses the tissue in order to return it to full function.

It should be noted that the most common classification is derangement, and if this is suspected it is not possible at the outset to make a diagnosis also of 'underlying dysfunction'. The derangement is always treated first as the main source of symptoms, which can present with end-range pain, and it is not possible to know if there is an underlying dysfunction until the derangement is reduced. On most occasions, once the derangement is reduced there is no 'dysfunction' to treat.

Dysfunction is classified by the direction of impairment. For instance, if the patient lacks extension range and end-range extension produces symptoms, this is an extension dysfunction. If patients have a limited and painful range of flexion with end-range pain on repeated flexion, which is no worse on cessation of movement, this is a flexion dysfunction, etc.

Postural syndrome

The postural syndrome is characterised by intermittent pain brought on only by prolonged static loading of normal tissues. Time is an essential causative component, with pain only occurring following *prolonged loading*. However, the loading period required to induce

symptoms may decrease with repeated exposure over time. Patients with the postural syndrome experience *no pain with movement or activity*. Neither do they suffer restriction of movement. No pathological changes occur in this syndrome. Once the aggravating posture is changed, the symptoms cease. The most common posture to provoke pain in this syndrome is slumped sitting.

Pain from the postural syndrome in the spine is caused by mechanical deformation of normal soft tissues arising from prolonged end-range loading affecting the peri-articular structures.

Clinically, patients with pain of postural syndrome rarely present for treatment, as they learn how to abolish symptoms by changing their position. Occasionally concerned parents accompany their teenage children to the clinic with this problem. Often they are individuals who lead a reasonably sedentary lifestyle and their posture is very poor. Although the syndrome is only occasionally seen in the clinic, the role of postural stresses on the genesis and persistence of musculoskeletal conditions is very important. Postural syndrome is not a discrete entity, but part of a continuum. These patients, if they do not alter their postural habits, can progress on to the more clinically common syndrome of derangement. A postural component is invariably present in derangement, which must be addressed to ensure resolution and prevent recurrence.

In the spine, postural pain arises mostly from *joint capsules or adjacent supportive ligaments* and is the result of *prolonged end-range positioning*. Moving from the end-range is sufficient to relieve pain immediately. Only appropriate education in postural correction will remedy pain in this syndrome.

Conclusions

In this chapter an introduction to the three mechanical syndromes and their conceptual models has been made. They describe three separate entities, which present in quite distinct ways and respond very differently to the mechanical evaluation outlined later. Details gained during the history-taking and symptomatic responses to repeated movements and sustained postures would be completely different. This means the three mechanical syndromes are clearly differentiated from each other, allowing the distinct management strategy necessary for each syndrome to be implemented.

Each syndrome must be treated as a separate entity in completely different ways. In the postural syndrome, postural correction must be performed to relieve the development of painful prolonged mechanical loading in normal tissue. In the dysfunction syndrome, structurally impaired tissue must be remodelled by repeatedly stressing the abnormal tissue. In the derangement syndrome, reductive forces must be applied to relocate displaced tissue, and loading strategies are applied that reduce, abolish or centralise symptoms. Appropriate mechanical therapy cannot be applied without correct recognition of these different entities. For instance, treatment of dysfunction requires the regular reproduction of the patient's pain, whereas treatment of derangement is by regular movements that reduce the displacement and cause the reduction, abolition or centralisation of pain.

It must be emphasised that the most common reason for patients to seek assistance is as the result of derangement – this is the entity that is most commonly seen in the clinic. Treatment of derangement is more complex and varied and will be discussed at length; however, the key management decision is to determine the direction of loading that is necessary to reduce the displacement. The means of reduction is identified by a loading strategy that decreases, abolishes or centralises symptoms. The most common derangements are posterior, and thus extension is the most common reductive force used. Lateral and some postero-lateral derangements require lateral forces or lateral forces combined with sagittal ones, and anterior derangements need flexion forces. The means by which these sub-groups can be identified and then treated are discussed in the chapters on management of derangement.

If at first assessment two syndromes are suspected, namely derangement and dysfunction, it is always the derangement that is treated first. Frequently what appeared to be a dysfunction disappears once the derangement is reduced. Once the derangement is reduced, a secondary dysfunction may be present; this should be addressed once the reduction of the derangement is stable.

These non-specific mechanical syndromes include the majority of patients with spinal pain. Failure to clearly identify a mechanical response or an atypical response may require further classification in a limited number of patients. In these instances, various non-mechanical or specific categories of back pain may need to be

considered. These are described elsewhere in the book. *Other categories should never be considered without first conducting a thorough mechanical evaluation over several days.* Recognition of these other categories is based on factors in the history-taking, failure to respond in a typical manner to a mechanical loading evaluation pursued over several days and certain responses to mechanical testing.

Figure 8.1 Mechanical and non-mechanical diagnosis – relative roles

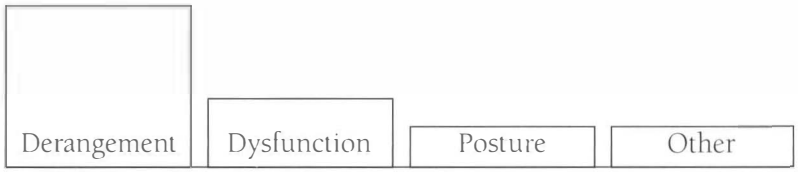


Figure 8.2 Classification algorithm

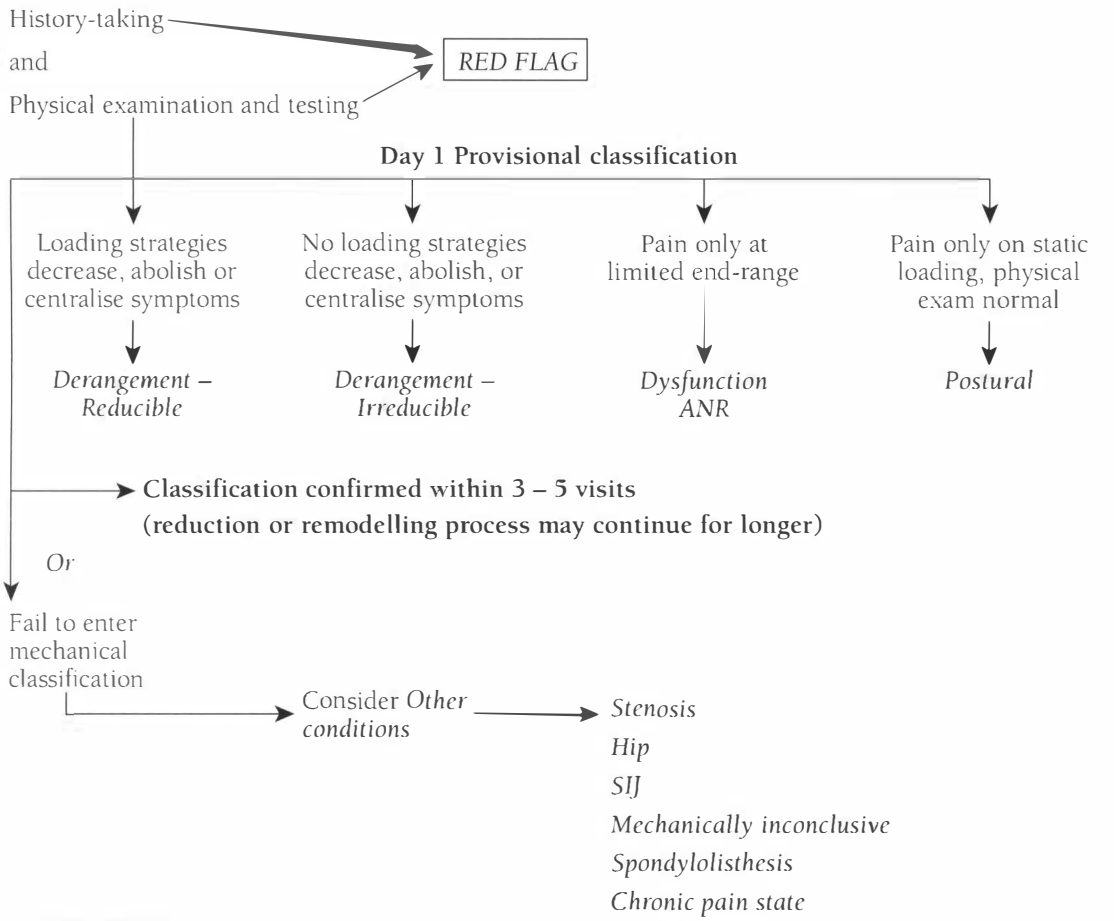


Figure 8.2 displays the clinical reasoning process for determining the mechanical or non-mechanical diagnosis. Suspicion of red flag pathology should mostly be determined by history-taking. Everyone else, about 99%, should been given a thorough physical examination as described later. From this most patients can be classified by a mechanical diagnosis, although initially in some this will be provisional. By five visits the mechanical diagnosis will be confirmed, or, due to an atypical response, one of the 'other' categories may be considered. To be entered for consideration, the patient displays no symptom response that suggests a mechanical diagnosis, as well as displaying signs and symptoms appropriate for that 'other' diagnosis. *The algorithm must be used in conjunction with the criteria and operational definitions in the Appendix – this is essential reading.*

9: Derangement Syndrome – the Conceptual Model

Introduction

The conceptual model that has been used to explain derangement syndrome relates the presentation to internal intervertebral disc displacements (McKenzie 1981, 1990). As derangements are a continuum, these may present in various ways. At its embryonic stage, with minor internal disc displacement, individuals may suffer from brief bouts of back pain and trivial limitations of function that last only a few days and resolve spontaneously. At its most extreme, the internally displaced tissue overcomes the restraining outer wall of the annulus fibrosus and extrudes into the spinal or intervertebral canal, causing predominantly radicular signs and symptoms. The continuum of back pain may start with posture syndrome and later proceed to minor and then major derangement. Back pain may proceed to back and leg pain and then on to sciatica. With the passage of time, dysfunction and nerve root adherence may occur. Irreducible derangement and entrapment are at the extreme end of the continuum, and spinal stenosis may be the culmination of a long history of back pain.

In this chapter a description of the clinical model is presented with the associated signs and symptoms. Evidence that supports this conceptual model is then presented, some of which is considered in more detail in the chapter on intervertebral discs. An understanding of the model allows, in most cases, a reliable prediction to be made regarding the preferred direction of applied forces and the likely response. The conceptual model has diagnostic and pathological implications; however, mechanical diagnosis and therapy are not totally dependent on the model as exceptions to the norm do occur. Ultimately it is a system that utilises repeated movements and loading strategies to treat signs and symptoms.

Sections in this chapter are as follows:

- conceptual model
- loading strategies
- dynamic internal disc model

- lateral shift
- place of the conceptual model.

Conceptual model

Innervation of the disc is absent in the inner part of the annulus fibrosus, and thus considerable occult changes can occur without symptomatology. Over many years of everyday postural stresses, strains and minor trauma, the integrity of the disc is impaired. The annulus fibrosus develops fissures, first circumferentially, then radially, and the homogenous nature of the nucleus pulposus is compromised. At some stage during this normal degenerative process, internal disc disruption and displacement can occur and abnormal morphology can become symptomatic.

Prior to actual disc displacement, pain from prolonged or poor posture may arise from excessive loading of any soft tissues. Early on, brief episodes of back pain may be caused by minor displacements of disc tissue that exert pressure on the outer innervated wall of the annulus fibrosus. Typically these arise following activities involving sustained or repeated flexion; sometimes quite trivial forces can trigger an episode, and this should be viewed against the background of lifestyles in which the ubiquitous posture is flexion. The majority of such episodes arise from minor well-contained posterior or postero-lateral displacements. These may cause back pain, which can be felt centrally or to the right or left of the spine, depending on the site of the pressure within the annulus. Some limitation of function may occur and pain may be experienced during movement, but just as the derangement is minor, so too is the symptomatology. At an early stage displacements are rapidly reversible, and more often than not individuals spontaneously become symptom-free and fully functional.

With the passage of time more persistent episodes may be experienced. There may be a progressive increase in internal disc disruption and displacement posteriorly, and attenuation or rupture of the lamellae of the annulus fibrosus. Symptoms thus become more severe and may radiate into the leg, and functional impairment is more marked – movements and activities are restricted, and after a period of sustained flexion the patient struggles to reverse the spinal curve against an obstructive displacement that prevents extension. Episodes now take longer and more effort to resolve. Failure to fully

reduce the displacement leads to residual symptoms and a restriction in the range of motion. As the most common derangements are posterior or postero-lateral; typically there is a failure to regain full extension, and individuals in future refrain from prone lying as this position is painful.

Internal derangements alone may produce symptoms that radiate into the leg; however, peripheral symptoms may also be caused by irritation of the nerve root/dura complex. As long as the outer annular wall is intact and pressure from the displacement is intermittent, the derangement and the symptoms of sciatica are reversible. The phenomena of peripheralisation and centralisation relate to increasing and decreasing stress on the source of pain generation. This may be the outer innervated annular wall or may include the irritated nerve root.

Larger displacements can cause such a disturbance in the normal resting position of the affected motion segment that it forces the body into asymmetrical alignment. The displacement obstructs movement in the opposite direction and fixes the patient in a temporary deformity of kyphosis, in the case of a posterior displacement; lateral shift, in the case of a postero-lateral displacement; and lordosis, in the case of an anterior displacement. The inability of patients to reverse the spinal curve at this stage provides a clue as to the underlying mechanical deformation that is the common aetiological factor in these apparently different disorders.

Ultimately, the outer restraining wall of the annulus fibrosus may be ruptured completely or so attenuated as to become incompetent. At this point displaced or sequestered disc material has interrupted the outer contour of the annulus and posterior longitudinal ligament or invaded the spinal canal. There is constant pressure on the nerve root and/or dura mater and signs and symptoms are consistent with a radiculopathy. A non-specific reversible mechanical backache has progressed into an irreversible and identifiable pathology, thus indicating the likely pathology that exists in many patients with so-called 'non-specific' back pain. With time there will be absorption, fibrosis or adhesions and symptoms will subside or change in nature, but at this stage only surgical intervention will produce a rapid resolution of the pain.

Deformities

Kyphosis The patient can be locked in a position of *lumbar kyphosis and is unable to extend*. Conceptually, the patient has developed an obstruction to extension caused by excessive posterior internal displacement. The *displacement obstructs curve reversal and locks the patient in a flexed posture they cannot easily correct*.

Lateral shift The patient can be locked in a position of *lateral shift*. For example, their trunk and shoulders are shifted to the right, a right lateral shift. They are unable to straighten or laterally glide to the left, or, if they can do so, they cannot maintain the correction. Conceptually, the patient has developed an obstruction to left lateral glide caused by excessive postero-lateral internal displacement to the left. *The displacement obstructs curve reversal and locks the patient in a lateral shift deformity that they are unable to correct themselves.*

Lordosis The patient can be locked in a position of *extension and is unable to flex*. Conceptually, the patient has developed an obstruction to flexion caused by excessive anterior internal displacement. *The displacement obstructs curve reversal and locks the patient in extension they cannot self-correct.*

In all three of these situations, the excessive internal displacement in one direction *locks* the segment in that position and prevents *voluntary* curve reversal or movement in the opposite direction. This is akin to the locked knee joint arising from internal derangement within that joint. These deformities are easily recognised and are the result of *significant* displacements. The greater the displacement, the greater is the deformity. Lesser displacements cause obstruction to movement and problems of curve reversal, but not deformity.

Loading strategies

In earlier stages of derangement, different postural loads will have a marked effect upon symptoms and movement. Unfavourable loading increases the displacement and worsens or peripheralises pain and makes movement more difficult. In contrast, favourable loading decreases the displacement and lessens symptoms and improves movement. Typically patients report a worsening of pain when sitting and an easing of pain when they walk about. Other patterns of pain behaviour occur. Sometimes movements that open the joint space may temporarily reduce the pain, but promote greater displacement

and more pain when the person returns to a normal posture. Thus, certain positions can be found that alleviate the pain while the position is maintained, but that aggravate or perpetuate the pain afterwards. For instance, in major postero-lateral derangements, patients find temporary relief in positions of flexion, but afterwards struggle to regain extension and are no better.

During the assessment of patients with spinal disorders, clinicians should be aware of these tendencies for certain favoured and unfavoured postures and movements. Knowledge of these should be used in management, with temporary avoidance of unfavoured loading strategies, and regular use of favoured loading strategies. However, the ability to affect these disorders is related to the state of the annulus fibrosus. In the early stages of derangement, the displacement is well contained by intact lamellae and properly identified repeated movements or sustained postures are easily able to reduce the displacement. At the end stage of derangement, the annular wall has either ruptured (extrusion or sequestration) or become incompetent and is no longer able to restrain displacements (protrusion). As long as the hydrostatic mechanism of the disc is intact with the integrity of the outer wall of the annulus maintained, it is still possible to exert an effect upon the internal displacement using mechanical forces. Once this has been compromised, however, the derangement is not reversible, and no lasting symptomatic changes can be achieved.

The conceptual model as outlined by McKenzie (1981, 1990) makes clear that derangements form a continuum with progressively larger derangements causing more mechanical deformation and consequently more signs and symptoms. For this reason a sub-classification of derangements one to six was outlined that described progressions of the same disturbance within the intervertebral disc. These presented clinically as increasing peripheral pain with or without deformity. These derangements affected the posterior or postero-lateral aspect of the disc, and thus were also capable of causing deformation of the nerve root, thereby producing radicular signs and symptoms. A separate sub-classification (derangement 7) described anterior displacements.

The conceptual model allows determination of therapeutic pathways. It not only describes a pathology and rationale for the origin of many

non-specific spinal pains, but also indicates the treatment direction required. Posterior derangements need extension forces in their reduction, anterior derangements need flexion forces and postero-lateral derangements need lateral or extension/lateral forces. Acceptance of the conceptual model allows us to determine, with good reliability, the direction of the required therapeutic motion.

Dynamic internal disc model

Various studies validate the conceptual model. There is now ample evidence concerning the innervation of the disc, and therefore its ability to be a pain-generating source in its own right (Bogduk 1994b, 1997). Pain provocation studies have commonly demonstrated exact reproduction of patients' symptoms with discography (Vanharanta *et al.* 1987; Moneta *et al.* 1994; Aprill and Bogduk 1992; Smith *et al.* 1998; Ricketson *et al.* 1996; Milette *et al.* 1999; Ohnmeiss *et al.* 1997). The disc is the most common cause of mechanical back pain (Schwarzer *et al.* 1995d; Milette *et al.* 1995; Ohnmeiss *et al.* 1997) and the most common cause of back pain and sciatica (Kuslich *et al.* 1991; AHCPR 1994).

Symptomatic presentation

Pain provocation studies at surgery have shown that the site of pressure on the annular wall is reflected in the site of perceived pain (Kuslich *et al.* 1991; Cloward 1959; Murphey 1968). Stimulation centrally produces symmetrical pain, and stimulation laterally produces unilateral pain. This would account for pain that changes site.

Some studies have found that leg pain could only be reproduced by stimulation of an already sensitised nerve root (Kuslich *et al.* 1991; Fernstrom 1960), but discography studies have commonly been able to reproduce leg symptoms in disorders without nerve root involvement (Park *et al.* 1979; McFadden 1988; Milette *et al.* 1995; Donelson *et al.* 1997; Ohnmeiss *et al.* 1997; Colhoun *et al.* 1988; Schellhas *et al.* 1996). Discogenic pain alone can cause radiating symptoms.

The most significant factor in painful discs appears to be radial annular disruptions. Those discs with little or no fissuring of the annulus are rarely painful, but when fissures extend to the outer edge of the disc they frequently are (Vanharanta *et al.* 1987, 1988). Only the presence of outer annular ruptures predicts a painful disc; neither inner annular tears nor general disc degeneration are associated with painful discs

(Moneta *et al.* 1994). In these instances, the extent of pain referral may reflect the degree of mechanical pressure to which the ruptured and weakened annular fibres are subjected.

The degree of radiation of somatic symptoms can be a reflection of the intensity of the stimulation of the pain-generating mechanism. Several experimental studies have shown this to be the case (Kellgren 1939, 1977; Feinstein *et al.* 1954; Mooney and Robertson 1976; Moriwaki and Yuge 1999). More mechanical pressure is associated with more distal referral of symptoms.

Mechanical stimulation of intervertebral discs in patients with radicular syndromes produces their back pain, while their leg pain could only be produced by stimulation of a sensitised nerve root (Kuslich *et al.* 1991; Fernstrom 1960; Smyth and Wright 1958). The distal extent of the radicular pain, its severity and frequency all appear to be a function of the amount of pressure exerted on the nerve root (Smyth and Wright 1958; Thelander *et al.* 1994). Thus increased discal pressure on the nerve results in more distal pain and a reduction of pressure causes the pain to move proximally.

Pople and Griffith (1994) found that the pain distribution pre-operatively was highly predictive of findings at surgery in 100 patients (Table 9.1). When the leg pain was predominant this usually indicated a disc extrusion, whereas if the back pain was worse than the leg pain this was more likely to indicate a protrusion. When pressure was still being exerted on the disc back pain was dominant, and when pressure was mostly on the nerve root leg pain dominated. Furthermore, when an extrusion was present, back pain tended to decrease or go completely.

Table 9.1 Pre-operative pain distribution and operative findings

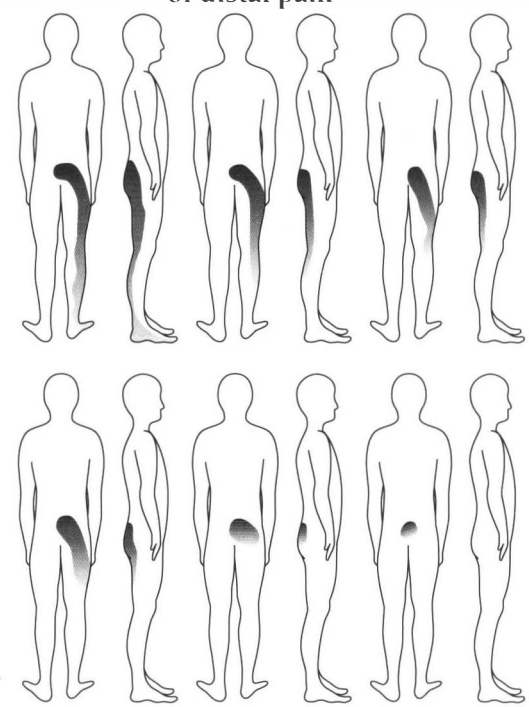
Pain	*Extrusion*	*Protrusion*	*Total*
Leg pain only	96%	4%	27
90 – 99% leg pain	58%	42%	12
50 – 90% leg pain	37%	63%	49
Back > leg pain	17%	83%	12
Total	53	47	100

Source: Pople and Griffith 1994

The study by Donelson *et al.* (1997) correlated findings from a mechanical assessment and discography. Whereas 70% of those whose pain centralised or peripheralised had a positive discography, only 12% of those whose symptoms did not change had disc-related pain. Among those who centralised their pain, 91% had a competent annular wall according to discography, compared to 54% among those whose pain peripheralised.

These studies demonstrate that the site of pain from internal disc lesions is reflected in the symmetry or unilateral nature of the pain perceived, and that these are capable of causing radiating symptoms. More extensive radiation of symptoms can be caused by more mechanical pressure. If the nerve root is involved then pain is referred down the leg, and neurological signs and symptoms may also occur. The accumulative evidence to date attests to the importance of discogenic pain in the back pain population, and also provides the theoretical background for an understanding of the phenomenon of peripheralisation and centralisation. Increased displacement or pressure on the outer annulus or nerve root produces more peripheral symptoms, while reduced pressure relieves these symptoms.

Figure 9.1 Centralisation of pain – the progressive abolition of distal pain



Pathological model

Numerous studies have shown the internal disc to be mobile. This effect has been demonstrated in cadaveric experiments (Shah *et al.* 1978; Krag *et al.* 1987; Shepperd *et al.* 1990; Shepperd 1995) and in living subjects (Schnebel *et al.* 1988; Beattie *et al.* 1994; Fennell *et al.* 1996; Brault *et al.* 1997; Edmondston *et al.* 2000). These have shown a posterior displacement of the nucleus pulposus with flexion and an anterior displacement accompanying extension of the lumbar spine. These studies support McKenzie's (1981) proposal that anterior to posterior displacement resulted in an obstruction to extension in a majority of patients with low back pain.

Kramer (1990) hypothesised that a combination of factors trigger pathological displacement of disc tissue:

- high loading pressure on the disc
- high expansion pressure of the disc
- structural disruption and demarcation of disc tissue, such that internal loose fragments of disc tissue can become displaced down existing fissures as a result of asymmetrical loading
- pushing and shearing forces encountered in ordinary activity.

Kramer (1990) suggests that external mechanical forces act as a trigger on tissue that may be predisposed to symptoms because of the other factors. Therefore, minor additional postural stresses can lead to deformation, tearing of annulus fibres or displacement of disc tissue. This displacement may be internal or may exceed physiological dimensions and lead to protrusions and extrusions.

There is thus a continuum between asymptomatic disc degeneration and symptomatic structural changes to the annulus. The process develops in a sequential manner, with the distortion, then failure of the annulus leading to the formation of radial fissures, which are a prerequisite of displacement. In its turn the displacement can be checked by the outer annular wall or this can be ruptured also and a complete herniation results. Once the annular wall has been completely breached and the hydrostatic mechanism of the disc is impaired, it is no longer possible to influence the displaced tissue (Kramer 1990).

Radial fissures are a common finding in cadaveric studies (Hirsch and Schajowicz 1953; Yu *et al.* 1988a; Osti *et al.* 1992). Various experimental and clinical studies describe the sequential way in which radial fissures develop, which may culminate with disc herniation (Adams and Hutton 1985a; Adams *et al.* 1986; Bernard 1990; Buirski 1992). For instance, Adams *et al.* (1986) describe the stages of disc degeneration as shown on discograms in cadavers; with fissures and clefts in the nucleus and inner annulus, leading to outer annular tears and complete radial fissures. In vivo discography studies (Bernard 1990; Buirski 1992) show the stages of disc disruption, with early annular fissuring and later radial tears sometimes associated with discal protrusion.

In vitro experiments have demonstrated that fatigue flexion loading of discs can lead to distortion and rupture of the annulus, which may be followed by extrusion of disc material (Adams and Hutton 1983, 1985a; Gordan *et al.* 1991; Wilder *et al.* 1988). Computer-generated disc models predict annular fissuring will occur with flexion loads (Natarajan and Andersson 1994; Shiraz-Adl 1989). These models also predict that failure is most likely to occur in the posterolateral section of the annulus fibrosus (Hickey and Hukins 1980; Shiraz-Adl 1989).

Other experimental and clinical studies (Brinckmann and Porter 1994; Moore *et al.* 1996; Cloward 1952) support this ***dynamic internal disc model*** because in the presence of fissures and disc fragments, the effects of normal loading can lead to the non-physiological displacement of discal material, protrusions and extrusions. The development of radial fissures would seem to be the key factor in the pathology of disc problems. These entities can be painful in themselves, but in some patients these fissures may act as conduits for intradiscal material to be displaced, to protrude or to be extruded beyond the contours of the annulus. The study by Milette *et al.* (1999) shows that in patients with discogenic pain, radial fissures may be more significant than protrusions. This study also found that bulging and protruded discs were significantly associated with grade 2 or grade 3 fissures (see Table 4.1). This also would indicate a continuum between these entities, with abnormal disc contour and possible nerve root involvement being impossible without pre-existing disruption of the annulus.

Although some posterior herniations can be asymptomatic, many do cause somatic and radicular pain. It is in the posterior aspect of the disc that the majority of pain-generating pathology has been identified. This relates both to radial fissures and actual herniations. It is primarily postero-central or postero-lateral herniations that are the cause of nerve root symptomatology. These are likely to be worsened with flexion loading, which experimentally has been shown to cause disruption and displacement (Adams and Hutton 1983, 1985a; Gordan *et al.* 1991; Wilder *et al.* 1988).

Although the end result may be actual disc herniation with nerve root involvement, this only represents the extreme end of the continuum and a minority of patients. The majority of patients present at an earlier stage in this continuum with the outer annular wall still intact, when the displaced tissue can be influenced by movement and positioning and when the symptom-generating mechanism is reversible. At this stage the mechanism of symptom generation is primarily from the disc, although there may be intermittent irritation of a nerve root.

Lateral shift

What will be referred to in this text as a lateral shift has also been described as a (gravity-induced) trunk list or (acute) lumbar, lumbosacral or sciatic scoliosis. The Scoliosis Research Society recognises the lumbosacral list as a non-structural shift caused by nerve root irritation from a disc herniation or tumour (Lorio *et al.* 1995). Longstanding scoliosis may be the result of a primary structural deformity in the vertebrae of the lumbar spine, while a secondary curve can develop to compensate for a morphological abnormality, such as a leg length inequality or contracture around the hip joint. In contrast to such entities, the lateral shift is an acute and temporary occurrence that accompanies the onset of an episode of back and leg pain. However, it should be noted that very rare causes of non-mechanical back pain, such as osteoid-osteoma and discitis, are also associated with rapid onset scoliosis (Keim and Reina 1975; Greene 2001).

Typically the patient has an asymmetrical alignment of the spine. With the onset of this episode of back, and usually referred leg pain, they develop a shift to one side. If they have had previous episodes of back pain, a history of previous s`afts is not uncommon. The shift

is temporary and resolves as the episode of back pain resolves. The shift is gravity-induced and often worsens the more the patient stands or walks. When they lie down the shift is abolished.

The prevalence of lateral shifts within the back pain population is unclear and there is considerable variability in the reported proportion that present with this sign (Table 9.2). As clear-cut definitions are usually not included in these reports, the variability may simply reflect different operational definitions.

Table 9.2 Prevalence of lateral shift

Reference	*Patient population*	*Total sample*	*N (%) with shift*	*% shift who had surgery*
Porter and Miller 1986	Back pain clinic in hospital	1,776	100 (6%)	20%
O'Connell 1951	Surgical cases; DH	500	244 (49%)	100%
Falconer *et al.* 1948	Surgical cases; DH	100	50 (50%)	100%
Khuffash and Porter 1989	Back pain clinic in hospital; DH	113	32 (28%)	41%
Matsui *et al.* 1998	Surgical cases; DH	446	40 (9%)	100%

DH = symptomatic disc herniation for which patient was treated surgically

Lateral shifts are strongly associated with symptomatic disc herniations. In Porter and Miller's (1986) sample of 100, 49% fulfilled three or more of the criteria for a symptomatic disc herniation. Shifts also appear to be particularly associated with disc herniations at the extreme end of the pathological continuum and to augur a poor prognosis requiring surgical intervention (O'Connell 1943, 1951; Falconer *et al.* 1948; Porter and Miller 1986; Khuffash and Porter 1989). Compared to patients without a shift and without cross leg pain, patients with a shift were three times more likely to come to surgery, and those with a shift and cross leg pain six times more likely (Khuffash and Porter 1989). The pressure on the nerve root from the disc herniation in patients undergoing surgery has been found to be significantly higher in those with a shift compared to those without (Takahashi *et al.* 1999).

The evidence makes clear that some of the previous assumptions that had been made about lateral shifts are incorrect. Although the shift most commonly occurs in those with leg or radicular pain, it has also been reported to occur in those with back pain only (Falconer *et al.* 1948; Porter and Miller 1986; Gillan *et al.* 1998). Gillan *et al.*

(1998) reported 55% of forty patients to have back pain only. Porter and Miller (1986) reported back pain in 16%, thigh pain in 13% and nerve root pain in 71% of 100 patients.

Multiple studies have found no consistency between the direction of the shift and the topographical relationship of the disc herniation to the nerve root found during surgery (Falconer *et al.* 1948; Porter and Miller 1986; Lorio *et al.* 1995; Laslett *et al.* 1992; Suk *et al.* 2001; Matsui *et al.* 1998). Traditional concepts relating the shift to a certain topographical relationship between the herniation and the nerve root are no longer tenable.

Different terminology has been used to describe the direction of the shift (Table 9.3). Earlier studies relate the convexity or concavity to the side of the pain, while more recent studies mostly use the terms 'contra' and 'ipsilateral'. Several reports mention the existence of alternating shifts – that is, patients whose shift might change sides.

Table 9.3 Sidedness of lateral shifts

Reference	*Convex to side of pain*	*Concave to side of pain*	*Contra-lateral shift*	*Ipsila-teral shift*	*Alter-nating shift*
O'Connell 1951	73%	17%			10%
Falconer *et al.* 1948[1]	53%	36%			11%
McKenzie 1972[2]			91%	9%	Some present
Porter and Miller 1986[3]			54%	46%	
Suk *et al.* 2001			67%	33%	
Matsui *et al.* 1998	80%	20%			
Tenhula *et al.* 1990[4]			68%	32%	

1 = 47/50 with leg pain
2 = 526 patients
3 = 67/100 with unilateral leg pain
4 = 22/24 with unilateral symptoms

Only a few authors have discussed conservative management of the lateral shift. McKenzie (1972) reported on 526 patients treated with lateral shift correction followed by restoration of extension. A further sixteen patients had increased pain on test movements and were rejected as unsuitable for conservative treatment, and 470 (89%) patients were symptom-free at the end of one week with no residual deformity, with the majority greatly improved within forty-eight hours. Of the remaining twenty-four (5%), eighteen were symptom-

free, but with residual deformity at the end of one week. The other six patients took several weeks to resolve or still had minor symptoms or residual deformity. Of the thirty-two (6%) who failed to respond to treatment, 84% had had symptoms for more than twelve weeks and 66% had root compression with neurological deficit.

Gillan *et al.* (1998) conducted a randomised controlled trial to compare lateral shift correction by a McKenzie-trained therapist with massage and standard back care advice. Disability scores improved in both groups at twenty-eight and ninety days' follow-up, with no significant difference between the groups. After twenty-eight days the shift had resolved in 64% of the McKenzie group and 50% of the control group. At ninety days shift resolution was significantly different, at 91% and 50% respectively. Unfortunately there was considerable loss to follow-up, with only twenty-five of forty patients being available at ninety days. Patients included in this trial had had symptoms for less than twelve weeks; outcomes in the control group demonstrate that the natural history for many patients with a lateral shift is towards resolution.

Place of the conceptual model

This conceptual model (McKenzie 1981, 1990), when applied to the clinical situation, becomes an effective and reliable diagnostic and therapeutic tool (Donelson *et al.* 1997; Kopp *et al.* 1986; Alexander *et al.* 1991; Nwuga and Nwuga 1985). Using it during a mechanical evaluation enables the prediction of discogenic pain and the state of the annular wall (Donelson *et al.* 1997).

Patients' response to repeated movements enables the prediction of suitability for conservative care (Kopp *et al.* 1986; Alexander *et al.* 1992). Patients presenting with signs and symptoms of disc herniations with nerve root involvement were given extension exercises as long as this did not increase radicular pain. Thirty-five (52%) of them responded to conservative therapy, of which thirty-four (97%) achieved full extension, mostly in the first few days of extension exercises. Thirty-two failed to improve with conservative treatment or rest, and went to surgery. Of these, twenty-four (75%) had sequestrations or evidence of nerve root displacement, but only two (6%) achieved full extension pre-operatively (Kopp *et al.* 1986). Failure to achieve extension in this study had clear predictive

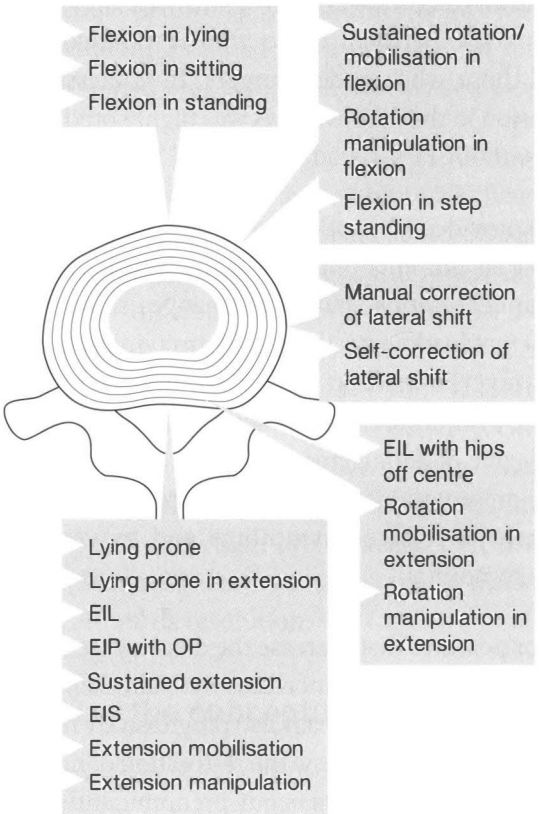
implications, and a larger study replicated the same findings (Alexander *et al.* 1992). Neurological signs and symptoms, straight leg raising and abnormal imaging studies in those with disc herniation were unable to differentiate between those who responded to a McKenzie regime and those who needed surgery. In contrast, the ability to achieve extension in the first five days was highly predictive of treatment group ($P = 0.0001$).

The conceptual model provides a hypothetical pathology to explain various presentations that are encountered in the clinic such as centralisation and peripheralisation, pain that changes sides, pain that fluctuates with different loading strategies, deformity, obstruction to movement, curve reversal and so on. There is an intimate connection between the symptomatic presentation, the mechanical presentation and the degree of derangement. Greater displacements produce more extreme presentations of pain and altered mechanics, and as the derangement is reduced symptoms and movement, aberrations will return to normal.

Repeated movements or postures that increase the displacement also increase the obstruction, which in turn increases the pain. Repeated movements that progressively reduce the pain also progressively reduce the obstruction and derangement and allow the restoration of normal pain-free movement. Disc displacements occur predominantly in a posterior or postero-lateral direction; according to the conceptual model, this type of derangement requires the extension principle in its reduction, or a combination of extension and lateral forces. An anterior displacement requires the flexion principle, and lateral displacements require the lateral principle of treatment.

The dynamic internal disc model allows the clinician to determine the direction of therapeutic motion that needs to be employed to reduce the displacement, as well as the direction of movement that can worsen the displacement and therefore needs to be temporarily avoided. Clinical findings suggest that a large proportion of displacements are primarily affected by sagittal plane procedures and will be reduced by extension forces and aggravated by flexion forces (McKenzie 1981). A smaller proportion of displacements occur in the frontal plane and require lateral or torsional forces in their reduction. A small proportion of displacements are anterior and will need flexion forces in their reduction (McKenzie 1981). Figure 9.2 relates direction of displacement to specific mechanical procedures.

Figure 9.2 Conceptual model and procedures; relating procedures to direction of derangement



This model not only provides a useful indicator of appropriate management, it is also useful as a teaching tool for patients. Most patients are more satisfied attending clinicians who provide logical and utilisable models of pathology. It is important for the patient to know that the disc is a source of pain generation and is a mobile structure influenced by everyday postures and movements. This enables them to achieve and improve compliance with their posture and exercise. Understanding the model teaches self-reduction and preventive techniques.

A better explanation may eventually be found for some of the features of back pain, but until that time this is a reasonable and reliable model upon which to base mechanical therapy. Since the model was first suggested (McKenzie 1981), numerous studies have been conducted that have increased our knowledge concerning disc disease, many of which endorse an internal dynamic disc model of pathology as noted above.

Although the definition of derangement relates to internal articular displacement, it could also be defined by its characteristic symptomatic and mechanical presentations. This mechanical syndrome is present when, for example, there is sudden onset of pain, peripheralisation, centralisation, spontaneous resolution of pain, improvement and worsening with loading strategies, deformity, sudden loss of range of movement and so on. It is most clearly defined by its response to the appropriate loading strategies, which is a rapid and lasting change in pain intensity and location. This only occurs in derangement syndrome – a syndrome being a collection of commonly observed signs and symptoms. Management of derangement is based upon symptomatic and mechanical responses to loading strategies.

Conclusions

This chapter describes the pathophysiological model that may be the explanation for derangement. It presents some of the clinical and experimental studies that support this explanation. The conceptual model suggests that derangement is related to internal disc dynamics and is initially a form of discogenic pain that may later, in a minority, involve the nerve root. The model embraces a continuum, which would account for the varied presentation of derangement, and offers an explanatory model for such clinical phenomenon as acute spinal deformities, blockage to movement, centralisation and peripheralisation. At the end of the pathological continuum is the irreducible derangement in which the hydrostatic mechanism of the disc is no longer intact and internal disc mechanics can no longer be influenced. When the outer annular wall is intact, posture and movement can influence disc displacement, and thus the conceptual model allows for the logical formulation of therapeutic loading. The model is a possible explanation for clinical events, but ultimately the treatment of derangement is dependent upon symptomatic and mechanical responses.

10: Centralisation

Introduction

Centralisation describes the progressive reduction and abolition of distal pain in response to therapeutic loading strategies. It is one of the key symptomatic responses that denotes derangement, the others being reduction or abolition of pain. Centralisation occurs during the reduction of a derangement. This chapter presents a detailed description of this phenomenon, as well as outlining its characteristics.

Sections in the chapter are as follows:

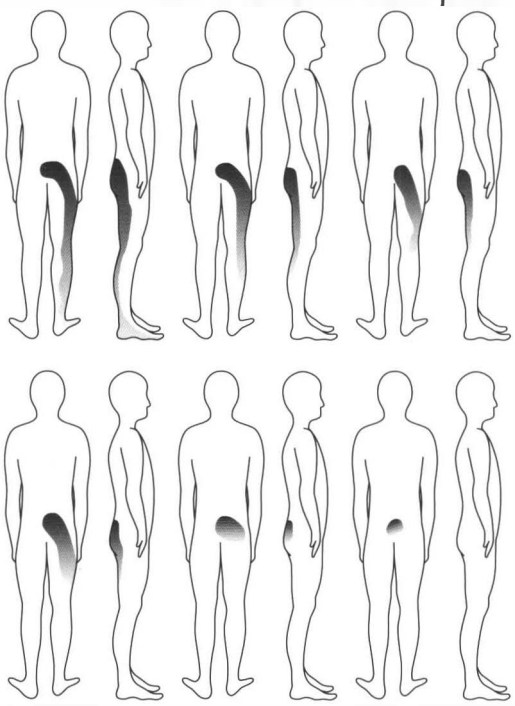
- definition
- description of the centralisation phenomenon
- discovery and development of centralisation
- characteristics of centralisation
- literature on centralisation
- reliability of assessment of symptomatic response.

Definition

- in response to therapeutic loading strategies, pain is progressively abolished in a distal-to-proximal direction with each progressive abolition being retained over time until all symptoms are abolished
- if back pain only is present, this moves from a widespread to a more central location and then is abolished.

Description of the centralisation phenomenon

Figure 10.1 Centralisation of pain – the progressive reduction and abolition of distal pain



Centralisation describes the phenomenon by which distal limb pain emanating from the spine, although not necessarily felt there, is immediately or eventually abolished in response to the deliberate application of loading strategies. Such loading causes reduction, then abolition of peripheral pain that appears to progressively retreat in a proximal direction. As this occurs there may be a simultaneous development or increase in proximal pain.

The perceived movement of pain either distally or proximally can occur during the natural history of an episode of pain and during the different activities of daily function. The identification of this pain behaviour during the history-taking provides an indication of the stage of the disorder and helps to identify appropriate management strategies. Centralisation specifically describes the abolition of distal pain that occurs *in response to clinically prescribed repeated end-range movement, static end-range loading or maintenance of corrective postural habits.*

The retreat of distal pain can occur immediately during the first assessment on the first day, and centralisation and final reduction can be a rapid process. Alternatively, it may be apparent from an initial assessment that a particular loading strategy is having a centralising effect, which may, if pursued over a longer time period, result in the abolition of distal symptoms and a more gradual process of centralisation.

The term 'centralisation' also applies if pain felt only in the back localises to the centre of the spine. Continuing application of the appropriate loading results in decrease and finally, abolition of pain.

The phenomenon only occurs in derangement syndrome (McKenzie 1981, 1990). Reduction describes the process by which the derangement is progressively lessened. During this process symptomatic and mechanical presentations are gradually improved, thus centralisation occurs and movement is restored. The process of reduction and centralisation are intimately related and occur together. When the derangement is fully reduced, pain is abolished and full-range, pain-free movement is regained. Maintenance of reduction is highly variable. Some reductions are stable in a short period of time and with a limited application of loading strategies, while others need a strict application of loading strategies over a more protracted period to bring about and maintain reduction. Some reductions are so unstable that simply a change in loading causes re-derangement. On occasion the derangement may be reduced, but pain on end-range movement, which may be limited, persists because of dysfunctional tissue.

Centralising means that in response to the application of loading strategies, distal symptoms are decreasing or being abolished. Symptoms are in the *process* of becoming centralised, but this will only be confirmed once the distal symptoms are abolished. This process can be rapid or may occur gradually over time with repeated exposure to the appropriate loading. The centralising phenomenon indicates that reduction of the derangement is in progress. The reductive process is continuing when pain is reported to be progressively centralising, decreasing or has ceased distally, or if pain located in the back is centralising, decreasing or ceasing.

Reduction is complete only when the patient reports no back or referred pain when undertaking normal daily activities and pain-free movement is fully restored. During the process of reduction the patient may undertake certain activities that impede or reverse the process and cause distal symptoms to reappear. With cessation of the aggravating positions and performance of the appropriate end-range movements, symptoms should once again start centralising.

Centralised means that as a result of the application of the appropriate loading strategies, the patient reports that all of the distal radiating or referred symptoms are abolished and have not recurred during normal activity. They may be left with back pain. The reductive process has been stabilised, and further end-range movements will decrease and then abolish the remaining spinal symptoms.

Pain that is centralising during the application of loading strategies may be a stable or an unstable phenomenon. If, following repeated end-range movements performed in lying, pain has centralised and remains better on resuming the upright position and being normally active, the centralisation process is stable (but not necessarily complete). ***Stable symptomatic improvement resulting from end-range loading indicates the stable nature of the reduction of the derangement and generally offers a good prognosis.***

Stability of reduction process is evidenced when any symptomatic improvement achieved *from end-range loading applied in lying* is maintained on and after the resumption of weight-bearing and normal activity. If symptomatic improvement is stable, further reduction will occur with a continuation of the same management.

Although symptoms may return if aggravating postures are maintained, any increase in intensity or peripheralisation of pain will cease and be reversed by more rigorous application of the appropriate loading strategies. Centralisation of symptoms occurring during loading applied *in standing* is usually stable.

Temporary cessation or centralisation of pain in response to end-range loading performed in the lying position is an *indication* that reduction *may* be occurring. Should pain *immediately* reappear on weight-bearing, the reduction process indicates of loading strategies and complete avoidance of aggravating postures.

Unstable reduction may indicate that a good prognosis can be achieved over a protracted period if rigorous application of management is applied; however, frequently it indicates a derangement that is not amenable to lasting reduction, and prognosis in these cases is poor. It can generally be determined over a test period of a few days whether or not stability of reduction and a lasting centralising effect are being achieved.

Spontaneous abolition of pain achieved by adopting the lying position is not an indication that the derangement has been reduced. Pain in this case has ceased because of removal of compressive loading and will return with the resumption of weight-bearing. In this situation it is inappropriate to consider that centralisation has been achieved or that reduction has occurred.

Peripheralisation describes the phenomenon when pain emanating from the spine, although not necessarily felt in it, spreads distally into or further down the limb. This is the reverse of centralisation. Loading strategies may produce temporary or lasting distal pain. In response to repeated movements or a sustained posture, if pain is produced and remains in the limb, spreads distally or increases distally, that loading strategy should be avoided. In some situations an instant but short-lived production of distal pain may occur with a particular loading strategy. This is not peripheralisation.

Centralisation

- only occurs in derangement syndrome
- occurs with the reduction of the derangement
- involves lasting abolition of peripheral or radiating pain
- may occur rapidly or gradually
- is accompanied by improvements in mechanical presentation
- occurs in response to loading strategies (repeated movements or postural correction).

Peripheralisation

- only occurs in derangement syndrome
- distal symptoms are produced and remain or distal symptoms are made more severe

- occurs in response to loading strategies (repeated movements or postures).

Discovery and development of centralisation

McKenzie's first experience with what he was to call the 'Centralisation Phenomenon' occurred in 1956. A patient, 'Mr Smith', who had pain extending from his back to his knee, had undergone treatment for three weeks without any improvement. He could bend forward, but his extension was painful and limited. He was told to undress and lie face down on the treatment table, the end of which had been raised for a previous patient. Without adjusting the table, he lay in a hyperextended position unknown to staff in the clinic. On being found five minutes later, he reported that he was the best he had been all week – the pain had disappeared from his leg, the pain in his back had shifted from the right to the centre, and his restricted range of extension had markedly improved. When he stood up he remained better, with no recurrence of his leg pain. The position was adopted again the following day and resulted in the complete resolution of his remaining central back pain.

During the following two or three years, every patient with back or referred leg pain was placed in either the extended position or was asked to repeat extension movements ten or fifteen times while lying in the prone position. There emerged a consistency of response to these exercises that could not have been coincidental.

Patients with certain referred pain patterns would become symptom-free within two or three days. Whenever this rapid resolution occurred, recovery was preceded by a change in the location of the pain from a referred to a near central midline position. Referred symptoms were seen to rapidly disappear at the same time as localised central back pain appeared or increased. Once symptoms centralised, referred symptoms would not reappear as long as patients avoided flexed postures. Continuation of the centralising manoeuvre caused rapid resolution of the central back pain. Consistently, concomitant restriction of extension mobility improved and patients *remained better as a result* of performing the exercises.

Some individuals with unilateral pain would not experience improvement as a result of sagittal plane extension movements, but did after applying lateral flexion in a loaded position – after which

centralisation occurred. In others, if lateral flexion was too vigorously applied, the pain would disappear from one side, but appear on the other. It became clear that by performing certain movements one could influence the site of pain radiation. This suggested that when pain changed location, something with the segment had also changed location and when pain centralised, reduction of displacement was occurring. If centralisation of pain occurred, the prognosis was invariably excellent and a rapid response would usually follow.

Patients whose pain extended below the knee and never abated reacted in an unpredictable manner, many being significantly aggravated rather than improved by these manoeuvres. Referred pain and neurological symptoms were sometimes exacerbated or produced by repeated movements, both in the sagittal and frontal planes. If extension was maintained for an excessive period of time or if the exercise was forced to an excessive degree, some of these patients, in the experimental years, remained worse as a result of the procedures. Many of these patients did not respond to mechanical therapy.

Characteristics of centralisation

With the realisation that movements that cause pain to centralise are therapeutic and cause a good outcome, the *prognostic significance* of centralisation became apparent. Movements that caused centralisation also indicate the direction in which any mobilising or manipulation procedures should be applied when an *increase of force* is necessary because of incomplete or partial responses to self-treatment exercises. Likewise, it became clear that movements that caused symptoms to peripheralise were undesirable and therefore contraindicated.

The phenomenon of centralisation most commonly occurs in patients who also demonstrate *significant obstruction* to full range of extension. When these patients are subjected to repeated end-range unloaded extension movements, centralisation of pain develops in conjunction with and directly proportional to the rapid recovery of extension that follows.

Although many patients with back pain experience centralisation with the performance of *extension exercises* carried out from the prone lying position, there are others, identified during mechanical evaluation, who must perform extension from a prone hips off-centre position. Some patients respond to lateral movements, and a further

group must repeat flexion movements in order to cause centralisation of pain.

The prognostic value of centralisation derives partly from the fact that the change in pain location that it describes is of a *lasting nature*. Furthermore, while one direction – often but not always extension – produces this desirable change in pain location, very often the opposite movement – often flexion – causes the peripheral pain to return and the condition to worsen. Patients frequently exhibit this *directional preference* in which one direction improves and the opposite worsens the symptoms.

Literature on centralisation

More detail about many of the studies mentioned in this section are provided in Chapter 11, which includes a literature review of centralisation and relevant reliability studies. Centralisation has been commonly identified during repeated movement tests (Donelson *et al.* 1990, 1991, 1997; Williams *et al.* 1991; Long 1995; Sufka *et al.* 1998; Erhard *et al.* 1994; Karas *et al.* 1997; Delitto *et al.* 1993; Kilby *et al.* 1990; Kopp *et al.* 1986; Werneke *et al.* 1999; Werneke and Hart 2000, 2001). This has occurred in between half and three-quarters of the patient groups evaluated. Studies that have examined centralisation have done much to confirm the characteristics of the phenomenon as outlined above.

Centralisation has been associated with good outcomes in both acute and chronic back pain populations (Donelson *et al.* 1990; Sufka *et al.* 1998; Long 1995; Rath and Rath 1996). Centralisation has been associated with improved functional disability scores and better return-to-work rates compared to individuals whose symptoms did not centralise (Werneke *et al.* 1999; Sufka *et al.* 1998; Karas *et al.* 1997). Donelson *et al.* (1990) found it to be an excellent predictor of outcome in 87 patients with acute and chronic referred pain; in 87% centralisation occurred with sagittal or frontal plane repeated movements. There was a correlation between the occurrence of centralisation and better outcomes.

Table 10.1 Prognostic significance of centralisation

Outcome	*No. of patients with each outcome (%)*	*Occurrence of centralisation in each outcome group (N)*
Excellent	59 (68%)	100% (59)
Good	13 (15%)	77% (10)
Fair	7 (8%)	57% (4)
Poor	8 (9%)	37% (3)
Total	87 (100%)	87% (76)

Source: Donelson *et al.* 1990

Centralisation occurred in over 80% of all patients, regardless of how long the symptoms had been present. However, centralisation was more definitely associated with a good or excellent outcome in those with acute symptoms (88%) compared to those with symptoms that had been present for over one month (67%) (Donelson *et al.* 1990). Centralisation readily occurs in those with more recent onset of symptoms, but it can also be gained in many patients with chronic back and referred pain (Donelson *et al.* 1990; Sufka *et al.* 1998). In one study in which centralisation occurred in twenty-five out of thirty-six patients (69%), the rates on centralisation decreased with the longevity of symptoms (Sufka *et al.* 1998).

Table 10.2 Occurrence of centralisation in acute, sub-acute and chronic back pain

Duration of back pain	*Occurrence of centralisation %*
< 7 days	83%
7 days to 7 months	73%
> 7 months	60%
Total	69%

Source: Sufka *et al.* 1998

In studies of chronic populations, about 50 – 60% of patients describe centralisation of their pain (Long 1995; Donelson *et al.* 1997; Sufka *et al.* 1998), again associated with a better outcome (Long 1995). It is thus independent of the duration of symptoms, but tends to be observed somewhat less frequently in those with chronic back pain.

Just as centralisation tends to be strongly associated with greater improvements in pain severity and perceived functional limitations, failure of centralisation to occur is strongly associated with poor

overall response (Donelson *et al.* 1990; Karas *et al.* 1997; Werneke *et al.* 1999; Werneke and Hart 2000). *"Failure to centralize or abolish pain rapidly indicates a lack of response to mechanical treatment and presages a poor result"* (Karas *et al.* 1997). Werneke *et al.* (1999) found that some patients experienced centralisation rapidly (average four visits), while in some it occurred more gradually or partially (average eight visits) and was not directly related to observed therapeutic loading in the clinic. If patients had failed to show a decrease in pain intensity by the seventh visit, no significant improvements in pain or function were found.

Failure to achieve centralisation as a prognostic factor was compared to other historical, work-related and psychosocial variables in predicting outcomes at one year (Werneke and Hart 2001). This included Waddell's non-organic physical signs, depression, somatisation and fear–avoidance beliefs. In a multivariate analysis that included all the significant independent variables, only leg pain at intake and non-centralisation significantly predicted outcomes at one year. This study is of great importance; for the first time it identifies a clinical variable that is more predictive of outcome than a psychosocial one.

When using sagittal or frontal plane repeated movements, 87% of patients experienced centralisation (Donelson *et al.* 1990). In a single testing protocol when only sagittal plane movements were used, centralisation occurred in 40% of patients with extension and 7% with flexion (Donelson *et al.* 1991). Movements in the opposite direction can worsen pain, and thus patients' conditions are deemed to have a preferential direction of movement. Centralisation can occur rapidly and be lasting in nature. It occurs with end-range repeated movements, and can demonstrate paradoxical responses in that a single movement may increase symptoms, but repeated movements leave the patient better overall. Sometimes it is associated with a concomitant increase in spinal pain (Donelson *et al.* 1990, 1991).

Centralisation can occur with posture correction only. Those adopting a lordotic sitting posture over a twenty-four- to forty-eight-hour period experienced a 56% reduction in leg pain and 21% decrease in back pain. Those who adopted a flexed posture over the same period experienced an increase in back pain and no change in leg symptoms (Williams *et al.* 1991).

Although centralisation by its very nature seems more likely to be described when peripheral symptoms are present, in fact there is some indication that it is more likely to occur with back, buttock and thigh pain rather than leg pain (Werneke *et al.* 1999; Sufka *et al.* 1998).

Table 10.3 Occurrence of centralisation according to site of referred pain

Referral of symptoms	*Occurrence of centralisation %*
Back	80%
Thigh	73%
Calf	43%
Total	69%

Source: Sufka *et al.* 1998

Different studies have used slightly different operational terms to define centralisation. Most have termed it ***abolition*** of distal pain during repeated end-range movements, with classification usually made during the initial assessment (Long 1995; Donelson *et al.* 1990, 1997); some studies have included ***reduction*** of distal pain also (Karas *et al.* 1997; Erhard *et al.* 1994; Delitto *et al.* 1993). In these studies, the rate of centralisation varied from 47% to 87%. Sufka *et al.* (1998) defined centralisation as reduction to central pain only within fourteen days, which occurred in 69% of their sample. The consensus from these studies suggests that the important qualitative distinction is that changes in pain status are rapid and occur over a period of days to a week or two, and are lasting in nature.

Werneke *et al.* (1999) employed a much stricter definition of centralisation in which symptoms had to retreat during the initial assessment, remain better, and at each subsequent session display further progressive abolition of symptoms. They found that 31% fitted these criteria, while a further 46% centralised fully or partially in between treatment sessions or during some sessions only. Although the full centralisation group required significantly fewer treatment sessions (four sessions compared to eight in the partial centralisation group), both groups had significant improvements in pain and function compared to the non-centralisation group. There were no significant differences in outcomes between the partial and fully centralising groups except the number of treatment sessions. If symptoms had not centralised by the seventh treatment session, any improvement was unlikely.

In summary, centralisation can thus occur or start to occur on the first day; however, in other patients it occurs over a period of a few weeks. It can occur both during treatment sessions and gradually in the time between sessions. However, the key distinction is between those who fail to centralise at all and those who may experience centralisation rapidly or more slowly. Outcomes are likely to be good in those experiencing centralisation – abolition of distal symptoms that remain better afterwards. After a thorough trial of up to seven therapy sessions, failure to alter symptoms is associated with a poor outcome.

Table 10.4 Characteristics of centralisation

- refers to the immediate or eventual abolition of distal pain in response to therapeutic loading strategies
- may be accompanied by increase in spinal pain
- usually a rapid change in pain over a few treatment sessions
- always a lasting change in pain
- occurs in acute and chronic patients
- often occurs in patients with obstruction to movement
- occurs most commonly with extension
- occurs with end-range repeated movements or postural correction
- occurs less commonly with lateral movements or flexion
- indicates directional preference
- indicates good prognosis
- failure to achieve indicates poor prognosis.

Reliability of assessment of symptomatic response

As the phenomenon of centralisation is entirely based upon the patient's report of pain location and behaviour, it is important to know that this subjective response can be reliably assessed. The Kappa value is a numerical expression of agreement between testers that seeks to exclude the role of chance (see Glossary).

The ability of different clinicians to concur on the existence of centralisation occurring in an individual has been found to be good to excellent, with rates of agreement of about 90% and Kappa scores of 0.92 – 1.0 (Sufka *et al.* 1998; Werneke *et al.* 1999). In one study involving eighty physical clinicians and physical therapy students who were evaluated on their ability to assess pain changes during movement from a video, agreement was 88% and Kappa value 0.79 (Fritz *et al.* 2000a).

Several studies have examined how much agreement there is between clinicians when interpreting pain responses in general to the performance of movements. These studies have shown that judgements about the site of pain and the behaviour of pain on movement can be reliably assessed (Spratt *et al.* 1990; Donahue *et al.* 1996; Kilby *et al.* 1990; McCombe *et al.* 1989; Strender *et al.* 1997). Tests involving pain responses are invariably more reliably assessed than tests involving visual or palpatory queues (Donahue *et al.* 1996; Kilby *et al.* 1990; Strender *et al.* 1997; Potter and Rothstein 1985).

"In lieu of the common limitation of imaging and other diagnostic studies in identifying the underlying disorder, pain and, in particular, its location would seem to be useful as a reflection of the nature of that underlying disorder" (Donelson *et al.* 1991).

Conclusions

This chapter has considered the phenomenon of centralisation, which refers to the lasting abolition of distal, referred symptoms in response to therapeutic loading. Various studies have demonstrated its frequent occurrence in the back pain population and its use as a favourable prognostic indicator. This clinically induced change in pain location has been reported in both acute and chronic patients with back, and back and leg, symptoms. This occurs with repeated end-range movements, particularly but not only with extension, and postural correction. As it can be consistently assessed, it is a reliable occurrence upon which to base treatment. The failure to alter the site of distal symptoms is conversely associated with poor outcomes. A description of this phenomenon and its characteristics have been presented in this chapter, while the following chapter provides a more detailed analysis of the articles mentioned here.

11: Literature Review

Introduction

Since the publication of the first edition of this book (McKenzie 1981), there has been a considerable amount of research into different aspects of the approach. Different types of study design that relate to mechanical diagnosis and therapy are considered in this chapter. Within the hierarchy of evidence, systematic reviews and randomised controlled trials (RCTs) are considered the strongest study design when evaluating interventions (Gray 1997). The relevant research is described as well as some of its limitations.

Other study designs must be considered when investigating other issues, such as the reliability of assessment process or the value of prognostic factors. It is also important to consider the evidence that relates to other aspects of the McKenzie approach. Key elements are the use of symptomatic response to guide treatment, the phenomenon of centralisation and the concept of directional preference. Some of these other issues are also considered and the available published literature presented.

The chapter considers the evidence under the following headings:

- systematic reviews and guidelines
- controlled trials and randomised controlled trials
- other efficacy trials
- studies into directional preference
- reliability studies
- reliability of palpation studies
- studies into the prognostic and diagnostic utility of centralisation.

Systematic reviews and guidelines

Various systematic reviews have evaluated the efficacy of exercise in general for back pain, some of which have included an analysis of McKenzie trials, and also some reviews have specifically focused on the McKenzie approach. For systematic reviews an electronic database and hand search is conducted, and only RCTs are included in the

analysis. There are predefined inclusion criteria, quality control standards and outcome measures. The methodological quality of the studies is considered, and often a method score for the different trials is given in an attempt to rate their quality. These show the modest methodological quality of most research, with scores from three reviews averaging 50% or less (Koes *et al.* 1991; Faas 1996; Rebbeck 1997). Common weaknesses in the literature include small sample sizes, lack of a placebo control group, inadequate follow-up, patient attrition, failure to measure compliance, use of other interventions and insufficient description of interventions (Koes *et al.* 1991; Faas 1996). However, an improvement over time has been noted, with a recent review noting high quality in 41% of studies compared to 17% in 1991 (van Tulder *et al.* 2000a).

Although the methodological scoring system is meant to objectify analysis of the different trials, qualitative judgements have to be made in defining aspects of the methods. Comparison between different reviews reveals a lack of agreement over the quality of certain trials. Rebbeck (1997) adopted a slightly modified version of the scoring system proposed by Koes *et al.* (1991), yet their scores for the same trials reveal considerable disparity.

Table 11.1 Comparison of method scores for the same trials

Reference	*Score: Rebbeck 1997*	*Score: Koes et al. 1991*
Nwuga and Nwuga (1985)	46%	28%
Stankovic and Johnell (1990)	61%	42%

Koes *et al.* (1991) reviewed sixteen RCTs into exercise for back pain, from which they decided that no conclusion could be drawn about whether exercise therapy is better than other conservative treatments for back pain or whether a specific type of exercise is more effective. Belanger *et al.* (1991) found three 'scientifically admissible' trials into the McKenzie approach, all of which favoured the approach for acute back pain, but these were criticised for lack of randomisation, blinding and use of a control group. Faas (1996) reviewed eleven RCTs from the literature between 1991 and 1995 to update the earlier review by Koes *et al.* (1991). In patients with acute back pain exercise was deemed to be ineffective, but two trials favoured McKenzie therapy compared with the reference therapy. As both had low method scores, the necessity of additional trials to clarify the efficacy of the system were indicated. For sub-acute and chronic back pain there is some

evidence for the benefits of exercise therapy, but conclusions about which type of exercise is most suitable could not be made.

Rebbeck (1997) located twelve clinical trials in the literature that used the McKenzie regime. Seven were excluded from the review, five of which found the system superior to the comparison regime. Failure to be included resulted from lack of a pure McKenzie approach or lack of publication in a peer-reviewed journal. Of the five acceptable trials, four demonstrated statistically significant improvements compared to the reference therapy. As the trials in acute patients did not include a control group, given the tendency for many to recover quickly, it cannot be definitively known that the McKenzie regime is superior to the natural history. Evidence for a positive effect is more apparent in chronic patients. In an overview of all twelve trials, the McKenzie regime was shown to be significantly better in reducing back and leg pain than flexion regimes, a mini back school, traction, an NSAID or a non-specific exercise programme. However, it was not better than a combination of extension, flexion and manipulation, or chiropractic manipulation. Overall trials were too few and methodologically of poor quality to make absolute recommendations.

Maher *et al.* (1999) reviewed sixty-two trials in the attempt to answer the question: Prescription of activity for low back pain: what works? Relative to acute and sub-acute back pain, few of the relevant trials demonstrated that exercises were more effective than the control treatment. The only clinical trial that did note an improvement used the McKenzie approach, with exercises being supplemented by posture correction and postural advice (Stankovic and Johnell 1990, 1995), the benefits of which were quite substantial in certain outcomes. The review recommends that patients with acute back pain be advised to avoid bed-rest and return to normal activity using time rather than pain as a guide. This advice may be supplemented by the provision of McKenzie therapy or manipulative therapy. For chronic back pain, there is strong evidence to support the use of general intensive exercises. They also found convincing evidence that exercise has a preventative effect on future back pain.

Van Tulder *et al.* (2000a) identified thirty-nine trials for their systematic review of exercises for back pain in the Cochrane Library. Their conclusions were similar to earlier reviews – for acute back pain exercises appear to be no more effective than other treatments,

whereas for chronic back pain exercises appear to be helpful. They also reported specifically on flexion and extension exercises, including the McKenzie approach. Three low-quality studies evaluated flexion exercises for acute back pain, which showed they were ineffective or produced worse outcomes than comparison treatments. Four studies evaluated extension exercises in acute back pain, two of good quality (Cherkin *et al.* 1998; Malmivaara *et al.* 1995), and two of low quality (Stankovic and Johnell 1990, 1995; Underwood and Morgan 1998). Three of them failed to show a significant difference in favour of the extension exercises, and one of these showed they were significantly less effective than comparison treatments. They concluded, somewhat confusingly, that extension exercises are more effective than flexion exercises, but that both are not effective in the treatment of acute back pain. For chronic back pain, no trials were found exploring the role of flexion or extension exercises compared to other treatments, and the three comparisons between the two types of exercise produced conflicting results.

Two guidelines about the general management of back pain, which use a thorough and systematic review of the literature, include mention of exercise therapy according to McKenzie (DIHTA 1999; Philadelphia Panel 2001a). The Danish Institute for Health Technology Assessment (DIHTA 1999) in a chapter on *Treatments that could generally be recommended* included the following summary. They separated the approach into a treatment and a diagnostic method. As a treatment method they concluded that "*McKenzie exercises can be considered as a treatment method for both acute and chronic low-back pan*". A few studies showed a positive clinical effect in both patient groups, with or without radiating symptoms. This meant that this recommendation was weighted as strength C – "*Limited research based documentation such that there is at least one relevant medium quality study, which supports the usefulness of a particular technology*".

As a diagnostic method they concluded that several studies indicate the method has value as both a diagnostic tool and prognostic indicator. They recommended that the approach could be used for both acute and chronic back pain. This recommendation was weighted as strength B – "*Moderate research based documentation such that there is at least one relevant high quality study or several medium quality studies, which support the usefulness of a particular technology*".

The Philadelphia panel evidence-based clinical practice guidelines have been developed using a structured and rigorous methodology (Philadelphia Panel 2001b). A whole edition of *Physical Therapy* records their findings according to back, neck, shoulder and knee conditions (*Physical Therapy 2001*, volume 81, number 10). They compare their findings with other guidelines and also include practitioner comments. For acute back pain, they find no evidence for therapeutic exercise. For sub-acute and chronic back pain, they recommend that there is good evidence to include extension, flexion and strengthening exercises, which include the McKenzie Method (Philadelphia Panel 2001a).

In summary, there is no straightforward consensus concerning McKenzie therapy from these systematic reviews. In some the evidence is seen as quite supportive, while in others the evidence is seen to be absent. Its apparent benefit is undermined by the low quality of the supportive trials and insufficient high-quality trials. The evidence concerning exercise in general is more positive in chronic rather than acute back pain. Part of the problem with evaluating the McKenzie Method is the fact that it does not fit neatly into one type of treatment. It uses exercise and postural instruction, but also can employ mobilisation and manipulation. While in some reviews a lot of effort is expended on determining the methodological quality of a trial, often the quality and type of intervention is not considered. Ultimately systematic reviews are only as useful as the trials on which they are based, so it would be helpful next to consider the individual trials included by the reviews, as well as other studies not included.

Controlled trials and randomised controlled trials

Some earlier reports of exercise therapy for back pain that utilised extension involved active backwards bending (Kendall and Jenkins 1968; Davies *et al.* 1979; Zylbergold and Piper 1981). As this is different from extension in lying, the procedure advocated by McKenzie (1981), these studies are not included in the literature review. Included is research that includes the extension exercises proposed by McKenzie (1981), as well as studies that sought to replicate the McKenzie approach in a more thorough manner. Some of the main outcomes are summarised in Table 11.2. To give some idea of the strength of the different studies, where available, the

method score for that trial is given, as well as the source of that score. All of these studies are randomised controlled trials (RCTs), excepting two stated instances.

Buswell (1982) compared a programme of extension exercises and postural advice, incorporating some of McKenzie's ideas, with one of flexion exercises and advice in fifty patients with an acute exacerbation of back pain. Both groups improved significantly with no important difference between them. Method score – 30% (Koes *et al.* 1991).

Ponte *et al.* (1984) assigned, not randomised, twenty-two acute patients to Williams' flexion exercises and postural instruction or a McKenzie exercise and posture protocol in which extension, lateral or flexion exercises were selected. Improvements in pain, sitting tolerance, forward flexion and straight leg raise were significantly better in the McKenzie group, of whom 67% were pain-free at the post-treatment evaluation compared to 10% in the Williams group. Patients in the McKenzie group received an average of 7.7 treatment sessions compared to 10.4 in the other group; this difference was also significant. Method score – 43% (Rebbeck 1997).

Nwuga and Nwuga (1985) used a sample of sixty-two women with disc protrusions and root compression of recent onset, which had been confirmed by investigations. These were assigned, not randomised, to McKenzie extension exercises and posture instruction, or Williams' flexion exercises. Re-evaluation of patients occurred at six weeks and was conducted by a blinded assessor. There were significant improvements in pain, sitting endurance and straight leg raising in the McKenzie group, but not in the Williams group, and mean treatment time was significantly less in the McKenzie group. Method score – 28% (Koes *et al.* 1991), 46% (Rebbeck 1997).

Stankovic and Johnell (1990) randomised 100 patients with acute back and leg pain to a McKenzie protocol involving extension exercises, preceded by lateral correction if necessary, and then flexion exercises, or a 'mini back school'. This involved education, advice on resting positions and keeping as active as possible, but refraining from exercises. Follow-ups were performed at three weeks and one year; there were significant differences between the groups at various points. All patients in the McKenzie group had returned to work within six weeks, as opposed to eleven weeks in the other group. There was significantly less pain in the McKenzie group at three and fifty-two

weeks, there were fewer recurrences and fewer had to seek medical help. Method score – 42% (Koes *et al.* 1991), 61% (Rebbeck 1997).

Eighty-nine patients from this trial were followed up five years later (Stankovic and Johnell 1995). Differences were much less than previously, but were still significant as far as recurrences of back pain and sick leave were concerned. There were no differences between the groups in seeking health care or in ability to self-help. Pain was present in 64% of the McKenzie group and 88% of the other group. Method score – 41% (Faas 1996).

Unlike all the trials mentioned so far, Elnaggar *et al.* (1991) chose to explore the effects of flexion and extension exercises in patients with chronic back pain. Postural instructions were not given, exercises were performed only for one session a day for two weeks and a pure McKenzie regime was not adopted. Both groups had a significant reduction in pain post-treatment, but no significant difference between the groups. Method score – 36% (Koes *et al.* 1991).

Spratt *et al.* (1993) explored the use of extension and flexion exercises and postures, incorporating braces and a no-treatment control group in fifty-six patients with chronic back pain and specific radiographic findings. These were spondylolisthesis, retrodisplacement or normal sagittal translation. Patients were reviewed after a month, at which point the extension group pain score was significantly better than the other two groups, and was the only one that showed a significant improvement across time. The pattern of treatment response was similar across all translation sub-groups. Method score – 45% (Faas 1996).

Delitto *et al.* (1993) and Erhard *et al.* (1994) investigated exercises in small groups of patients who were classified as extension responders by showing reduction or centralisation of symptoms with extension and worsening of symptoms with flexion. Once so-classified, twenty-four patients were then randomised to either a manipulation procedure followed by extension exercises or a flexion exercise regime. There was a significantly greater improvement in Oswestry disability score in favour of the manipulation/extension group (Delitto *et al.* 1993). Method score – 30% (Faas 1996). In the second trial (Erhard *et al.* 1994), twenty-four patients were randomised to an extension group or a group who received a manipulation and then performed a spinal flexion/extension exercise.

At a week, only two of the first group met the discharge criteria, while nine of the second group did so. Follow-up at one month was only 50%, but also favoured the manipulation group. Method score – 52% (Rebbeck 1997).

Dettori *et al.* (1995) recruited 149 soldiers with acute back and leg pain. These were randomised to extension, flexion and control groups, but then at the end of week two, half of each of the active exercise groups also performed the other exercise. Exercises were done three times daily and patients were instructed in the appropriate postural advice according to their group. The control group lay prone with an ice pack over the lumbar spine. All groups improved rapidly over the eight weeks of the trial with no statistically significant differences in pain or function over this period. There was a tendency for both exercise groups to show a better return of function in the first week, at which time there was very little change in the control group; when the two exercise groups were combined and compared with the control group, this was significant at this point. In the six- to twelve-month follow-up, recurrences of back pain were similar in all groups, at over 60%. However, control group patients were more likely to require medical care than those who had exercised, and those who had been in the extension group, particularly, were less likely to need medical care and work limitation.

Malmivaara *et al.* (1995) did not refer to the McKenzie approach; however, backward bending and lateral bending exercises were used in one of the treatment arms; other patients were randomised to either a bed-rest or a normal activity control group. It is not indicated if exercises were performed in lying or standing, and they were done only three times a day. One hundred and eighty-six patients with acute back and leg pain were entered in the trial. At three weeks there were significant improvements in favour of the control group over the exercise group in terms of sick days, duration of pain and Oswestry scores. At twelve weeks some of the outcomes still favoured the control group, but these were not stated to be significant. Method score – 63% (Faas 1996).

Underwood and Morgan (1998) randomised seventy-five patients with acute back pain to either a single back class lasting up to one hour with one to five patients in which the 'teaching was as described by McKenzie', or to receive conventional management. At no point during the follow-up year were there any statistical differences

between the two groups in terms of pain or Oswestry score. There was a statistically significant difference at one year when 50% of the class group reported 'back pain no problem' in the previous six months compared to 14% of the control group.

Gillan *et al.* (1998) attempted to study the natural history of lateral shift and the effect of McKenzie management. Forty patients were recruited to the trial and randomised to the McKenzie group or a non-specific back massage and standard back advice group. Patients were followed up at twenty-eight and ninety days, but 37% of patients were lost by the last follow-up. Resolution of shift occurred more frequently in the McKenzie group, with a significant difference at ninety days. However, there was no difference in functional outcome at any point.

Cherkin *et al.* (1998) randomised 323 acute back pain patients to one of three groups: a McKenzie regime, chiropractor manipulation or a control group who were given an educational booklet. This was the first study to recognise the importance of using trained clinicians, but rather than using experienced McKenzie clinicians, they were trained prior to the study. The trial, because of exclusion criteria, ultimately recruited only 8.5% of those who attended their primary care physician with back pain. At four weeks the chiropractic group (P= 0.02) and the McKenzie group (P=0.06) had less severe symptoms than the booklet group, but not different Roland-Morris disability scores. At twelve weeks there were no significant differences in symptoms or function between the three groups, and there had been no further improvement in outcomes. In the subsequent two years recurrences were similar in all groups, as was care-seeking. Costs were substantially lower in the booklet group, but satisfaction with care was significantly worse than in the two other interventions.

In summary, several trials are supportive of the McKenzie approach (Nwuga and Nwuga 1985; Ponte *et al.* 1984; Stankovic and Johnell 1990,
trials are of poor or moderate quality, which can have the tendency to exaggerate treatment effects (Gray 1997). Many of the trials have small numbers, which can mean the trial has insufficient power and therefore is unable to detect important clinical differences, although in fact all did. Two trials appear to show parts of the McKenzie system perform less well against comparison (Erhard *et al.* 1994; Malmivaara *et al.* 1995); however, the interventions bear so little resemblance to the approach if used properly that such a conclusion would be erroneous.

Several trials have ambivalent conclusions; for instance, that neither extension nor flexion exercises are necessarily better (Buswell 1982; Elnaggar *et al.* 1991; Dettori *et al.* 1995), and that a single 'McKenzie class' is no better than usual care in the short-term (Underwood and Morgan 1998). Again, with these trials the approach is not rigorously mechanical diagnosis and therapy; for instance, there is lack of attention to patient selection. The study by Cherkin *et al.* (1998) also has an ambivalent outcome. That mechanical diagnosis and therapy performed as well as chiropractic manipulation is very positive, given the support for manipulation by numerous systematic reviews. Only 10% of patients had pain below the knee, thus it is likely that there was a preponderance of patients with back pain only without referred symptoms. It is precisely this group, acute simple back pain, which is supposed to be the optimal group to receive manipulation (AHCPR 1994). However, neither intervention was more than marginally better than a cheap booklet.

Table 11.2. Main outcomes from published randomised controlled trials using extension exercises or purporting to use McKenzie regime (see text for more detail)

Reference	*Group 1*	*Group 2*	*Group 3*	*Outcomes Statistically significant improvements supporting McKenzie intervention.* Not supportive.
Buswell 1982	Extension	Flexion		Improvements both groups NS difference
Ponte *et al.* 1984	McKenzie protocol	Flexion		*Pain (10):* *1: -4.9* *2: -3.2 (P =0.001)*
Nwuga and Nwuga 1985	McKenzie protocol	Flexion		*Pain (10):* *1: -5.3* *2: -2.7 (P<0.01)*
Stankovic and Johnell 1990	McKenzie protocol	Education Normal activity		*Sick leave (days):* *1: 11.9* *2: 21.6 (P<0.001)* *Recurrences:* *1: 22* *2: 37 (P<0.001)*
Elnaggar *et al.* 1991	Extension	Flexion		Improvements both groups. NS difference

Continued next page

Reference	*Group 1*	*Group 2*	*Group 3*	*Outcomes*
Spratt *et al.* 1993	Extension	Flexion	Control	*Pain: Only 1 improved post treatment (<0.004)*
Delitto *et al.* 1993	Manipulation Extension	Flexion		*Oswestry:* *1: -23%* *2: -10%*
Erhard *et al.* 1994	Extension	Manipulation Flex / Ext		Discharge criteria: 1: 2/12 2: 9/12 (P<0.05)
Stankovic and Johnell 1995	See 1990 study			*Sick leave:* *1: 51%* *2: 74% (P<0.03)* *Recurrences:* *1: 64%* *2: 88% (P<0.01)*
Dettori *et al.* 1995	Extension (+flexion)	Flexion (+extension)	Control	Improvements all groups NS difference
Malmivaara *et al.* 1995	Extension + side-bending	Usual activity	Bed-rest	Sick days: 1: 5.7, 2: 4.1, 3: 7.5 Oswestry: 1: -15, 2: -22, 3: -19
Underwood and Morgan 1998	'McKenzie class'	Usual management		Improvements both groups. NS difference *Chronic back pain:* *1: 50%* *2: 14% (P<0.007)*
Gillan *et al.* 1998	McKenzie lateral shift protocol	Massage and advice		*Resolution of shift > 5mm:* *1: 91%* *2: 50% (P = 0.04)* Oswestry NS
Cherkin *et al.* 1998	McKenzie regime	Chiropractor manipulation	Booklet control	Improvements all groups

NS = any differences are non-significant

It should be emphasised that nearly every trial makes no selection of patient appropriateness for a given exercise regime. Exercise programmes are invariably standardised, are prescribed routinely or implemented in groups, and by clinicians of unknown skill or experience in the McKenzie approach. No attempt is made to assess for suitability, which is a key component of the approach. The only trials that attempt patient selection are those by Delitto *et al.* (1993) and Erhard *et al.* (1994). These suffer from very small numbers, considerable loss to follow-up and confusion as to exactly which component of the interventions was responsible for the effects observed.

The importance of individual assessment of suitability for exercise regimes is highlighted by the study by Donelson *et al.* (1991) – method score 57% (Rebbeck 1997). This showed that back pain frequently responds differently to different movements – nearly one-half of this group had a clear directional preference, most for extension, but a few for flexion. Not only did one direction clearly centralise symptoms, but also the opposite movement typically intensified and/or peripheralised it. This study was only short-term, but illustrated the importance of directional preference as a key to the management of mechanical back pain. Other studies have shown the good prognostic significance of identifying centralisation early on (Donelson *et al.* 1990; Sufka *et al.* 1998; Long 1995; Werneke *et al.* 1999; Karas *et al.* 1997). More patients may have demonstrated centralisation or a decrease in symptoms if testing had been pursued over a longer time period, and if other movements, besides sagittal ones, had been employed. For instance, in a study of eighty patients in which frontal and sagittal plane movements were used, 87% of them demonstrated centralisation (Donelson *et al.* 1990). If this directional preference is not taken into account and exercises are dispensed to all comers, then some in that group might respond, but some may be made worse and overall such a trial would show no value in a particular exercise.

Most of these trials have been conducted in patients with acute back pain. In this group there is a marked tendency for spontaneous recovery with whatever intervention is used, or if none is used. This is well-illustrated in the study by Cherkin *et al.* (1998). Disability is seen to fall rapidly from a starting point of twelve out of a twenty-four-point scale to seven at week one, and about four at week four *in all groups*. After this at weeks twelve, fifty-two and 104 the scores remain virtually unchanged, except for some minor further improvements in the physical therapy group. There is, in other words, a minor level of functional disability after recovery from the acute episode that remains largely unchanged two years later.

Various other shortcomings, which are common characteristics of these trials, limit their generalisability for mechanical diagnosis and therapy. A distinction is often not made between those with back pain only and those with referral of symptoms or with sciatica. Frequently interventions are inadequately described, performed with inadequate regularity and with adherence to exercise programmes not monitored. None of the trials excluded patients in whom no movement or position could be found to abolish, reduce or centralise

symptoms. Such patients should be excluded from treatment groups (McKenzie 1981). Randomisation should be made after a mechanical evaluation – if a patient is intolerant of penicillin, they don't get it! The level of skills and experience of the participating clinicians is rarely considered, but this affects clinical efficiency as seen in the section on reliability studies. Lack of understanding of the McKenzie approach has a deleterious effect on its application. Trials that need to be performed include the effects of mechanical diagnosis and therapy, using suitably trained clinicians, involving patients with chronic and recurrent back pain and also to distinguish its effects in patients with back pain and in those with referred symptoms.

Other efficacy trials

Besides the evidence reviewed above, there are also a number of studies that have either not been published in peer-reviewed journals, and therefore have not gone through the critical appraisal process that is necessary prior to publication, or else lack a control group. Despite weaknesses, it is still worth considering this other literature, which on the whole is supportive of the approach. Principle findings are summarised in Table 11.3.

Kopp *et al.* (1986) included sixty-seven patients with acute disc prolapse, displaying radicular pain and at least one sign of nerve root irritation, and evaluated their response to an extension exercise protocol. If extension exercises worsened radicular pain, further attempts were abandoned. If extension was limited and produced back pain without worsening the leg pain, gradual extension procedures after the method of McKenzie (1981) were implemented. Thirty-five of these patients responded to the extension programme, and 97% of them achieved full-range extension within a matter of days. Thirty-two patients failed to respond and came to surgery, and of these only two (6%) were able to achieve extension. At surgery 75% had either a sequestered or protruding disc with nerve root displacement or deformity. There was no difference between the two groups in referred pain, positive straight leg raise or neurological signs and symptoms. The authors coined the phrase the 'extension sign' – being the inability to achieve extension – as an early predictor of the need for surgical intervention. At long-term follow-up, average six years, the extension sign was able to predict a favourable response to non-operative treatment in 91% of cases (Alexander *et al.* 1991).

Alexander *et al.* (1992), in a further report dealing with a total of 154 patients with disc herniation, reported on seventy-three patients who were selected for conservative management based on their ability to achieve full-range extension in lying. The decision to proceed with a McKenzie approach was made by the fifth day, by which time most had achieved extension if they were going to. These patients were then discharged and instructed to continue with extension exercises. Those in whom the extension sign remained positive were managed surgically. Thirty-three (45%) of the conservatively managed patients were traced about five years later. Symptoms were resolved or slight in 82%, functional limitations nil or minor in 85%, and 94% were satisfied with their treatment.

In those who initially had a positive extension sign that became negative, complete resolution was reported in 47%, compared to 21% in those who had a negative extension sign at admission and at five days. Patients (nineteen of thirty-three) whose extension sign changed from positive to negative (achieving extension) within five days had consistently better outcomes, and this mechanical presentation was a strong predictor of successful conservative management. This ability to regain extension in the acute stage was highly significant in predicting the treatment group, conservative or surgical. Other factors, such as neurological signs and symptoms, straight leg raising or abnormal imaging studies, were unable to differentiate between the two groups.

Numerous studies have only been published as abstracts (Vanharanta *et al.* 1986; Adams 1993; Kay and Helewa 1994; Goldby 1995; Fowler and Oyekoya 1995; Udermann *et al.* 2000, 2001; Schenk 2000; Borrows and Herbison 1995a) or as a dissertation or separate publication (Roberts 1991; Borrows and Herbison 1995b); detailed evaluation of these is not always available.

Vanharanta *et al.* (1986) allocated 138 patients to back school, McKenzie exercises or a home traction device according to date of birth. In the McKenzie group 97% had improved after one week, while less than 50% improved in the other two groups. After two weeks 36% of the back school group and 37% of the traction group had to change treatment because of lack of improvement; no changes were necessary in the McKenzie group. The McKenzie and traction groups recovered more quickly, with a statistically significant difference at one month, but no group differences at six months.

Roberts (1991) compared McKenzie therapy to treatment with a non-steroidal anti-inflammatory drug (NSAID) in patients with acute back pain, all of whom were encouraged to mobilise actively. At seven weeks the McKenzie group was less disabled, a difference that was significant in the sub-group of patients who were classified according to the mechanical syndromes at the first assessment. However, sick leave was greater in the McKenzie group.

Adams (1993, Adams *et al.* 1995) gave twenty-three chronic back pain patients a standardised six-week treatment programme of McKenzie extension procedures. Post-treatment values showed a significant reduction in pain scale rating. While prior to treatment patients showed a higher psychological involvement, reduced range of movement and increased EMG activity compared to matched non-pain controls, after treatment these differences were no longer significant.

Fowler and Oyekoya (1995) did a retrospective note review of twenty-seven subjects, twenty (74%) of whom had excellent recovery using McKenzie treatment within a shorter time period than other therapies previously or concurrently applied.

Kay and Helewa (1994) randomly assigned twelve patients with acute back pain to a McKenzie or Maitland protocol. At three weeks the McKenzie group showed an eighteen-point reduction on the pain scale, while the Maitland group reported a sixteen-point increase ($P=0.029$). There were no significant differences in range of movement or disability. Longer-term follow-up was not reported.

Goldby (1995) conducted a double blind randomised controlled trial on fifty patients with chronic back pain, of whom complete data existed on thirty-six. One group was treated along the McKenzie principles and one group received a non-specific exercise programme. There were improvements in both groups that were significant. Comparisons between the two groups found significant differences in favour of the McKenzie regime and significant changes in health locus of control that were not found in the non-specific exercise group.

Borrows and Herbison (1995b) reported on the Accident Rehabilitation and Compensation Insurance Corporation (ACC) evaluation of the effectiveness of four treatment programmes for chronic compensated back pain patients in New Zealand. All programmes used different exercise and rehabilitation regimes, three

on an outpatient basis, while the McKenzie regime was a fourteen-day residential programme. Nearly 800 patients with an average of twenty months on compensation were allocated, not randomised, to the different programmes. The main outcome was 'Fitness to Work'; at one month this had improved by 35% in the McKenzie programme compared to 20% in the next best intervention, and by less than 4% in the other two. Secondary outcomes showed a similar picture, with the best two interventions producing substantially greater improvements in functional disability scores (about eight points on a twenty-four-point scale) and depression (five- to six-point improvement) than the other programmes (about three points and less than two points respectively). Long-term outcomes were missing in this study, and all programmes achieved a 20% return to work rate at three months. Nonetheless, two of the programmes, including the McKenzie one, showed significant and clinically meaningful greater improvements. The authors made various attempts in their analysis of the results to ensure against bias or confounding as a randomisation process was not used, and felt confident that the improvements were the true effect of treatment. While the McKenzie residential programme lasted nine days, the other programmes had an average duration of 103 to 127 days. This programme is described in more detail in the section on treatment of chronic pain.

Udermann *et al.* (2000) reported on the value of a purely educational approach, using *Treat Your Own Back* (McKenzie 1997) in sixty-two volunteers with chronic back pain, of whom 81% were available for follow-up nine months after reading the book. At this point 87% were still exercising regularly, 91% still used good posture, 82% noted less back pain and 60% were pain-free. Mean pain severity had dropped from 1.3 on a four-point scale to 0.44, and mean number of episodes from 4.1 to 1.0 per annum. Over 70% had found extension exercises to be most beneficial. Although there was no control group in this study, with a mean length of duration of back pain of over ten years prior to the intervention, this chronic sample served as its own control. At eighteen months fifty-four (87%) were contacted again (Udermann *et al.* 2001). Over 92% still claimed to be exercising regularly and focusing on posture. Pain severity had decreased to 0.33 and episodes per annum to 0.15.

From thirty-four patients recruited with lumbar radiculopathy, Schenk (2000) classified twenty-five as derangements, who were then

randomly assigned to McKenzie exercises or joint mobilisation. The McKenzie group demonstrated significantly greater improvements in pain and function after three sessions.

Table 11.3 Other literature – abstracts, uncontrolled trials, etc.
(see text for more detail)

Reference	*Group 1*	*Group 2*	*Results* *Statistically significant improvements supporting McKenzie intervention.*
Kopp *et al.* 1986	Negative extension sign: McKenzie	Positive extension sign: Surgery	*Achieved full extension:* *1: 97%* *2: 6% (P<0.005)*
Alexander *et al.* 1992	Negative extension sign: McKenzie	Positive extension sign: Surgery	*Mechanical response predicted treatment group (P = 0.0001)*
Vanharanta *et al.* 1986	McKenzie (extension)	2. Back school 3. Back Traction	*Significant difference in pain at one month*
Roberts 1991	McKenzie	NSAID	*Significant difference in disability at seven weeks in those classified by mechanical syndrome*
Adams 1993	Extension		*Pain reduction (P<0.001)* *Increased ROM (P<0.05)*
Kay and Helewa 1994	McKenzie	Maitland	*Pain:* *1: -18* *2: +16 (P=0.029)*
Goldby 1995	McKenzie	Non-prescriptive exercise	*Significant differences in pain, Oswestry, HLC*
Fowler and Oyekoya 1995	McKenzie	Other therapies	*74% responded quicker to McKenzie*
Borrows and Herbison 1995a, 1995b	McKenzie residential rehabilitation programme	2. 3. and 4. Gym-based exercise and rehabilitation programmes	*Impairment:* *1: -7* *2: 0* *3: -1* *4: -4 (P=0.0005)* *Oswestry:* *1: -7%* *2: -3%* *3: -3.5%* *4: -9% (P= 0.0005)*

Continued next page

Reference	*Group 1*	*Group 2*	*Results*
Udermann *et al.* 2000	*Treat Your Own Back*		*Pain: -0.9 (4 point scale) Episodes: -3 (P<0.0001)*
Schenk 2000	McKenzie	Mobilisation	*Significant differences: pain (P<0.014) function (P<0.032)*

Positive extension sign = increase in radicular pain on extension in lying
HLC = health locus of control

Studies into directional preference

Directional preference describes the propensity of mechanical back and referred pain to lessen if movements or positions in one direction are performed and to worsen if movements or postures in the opposite direction are performed. Likewise, opposite postures or movements may centralise or peripheralise patients' symptoms. Studies included in this section have specifically investigated the phenomenon of directional preference. This has been done by randomly exposing patients to repeated movements or postural practises with different loading strategies and examining their symptomatic response. Studies have been conducted on the effects of extension and flexion and into control or limitation of flexion – main findings are summarised in Table 11.4.

Donelson *et al.* (1991) examined the effects of flexion and extension on symptoms in the short-term by randomising 145 patients to two different protocols. In one group flexion movements were performed first and then extension movements, first in standing and then in lying; in the other group the order of movements was reversed. Whichever protocol was performed, flexion generally had the effect of increasing symptoms and extension generally had the effect of decreasing symptoms. Individually, back pain decreased in fourteen subjects (10%) during flexion and in thirty-one subjects (21%) during extension. Individually distal leg pain decreased in eleven subjects (8%) during flexion and in fifty-six subjects (39%) during extension. Interestingly, only one patient reported improvement with both flexion and extension movements. An analysis model that assumed different responses to flexion and extension in central and peripheral pain and centralisation/peripheralisation was tested out, which found significant differences in pain behaviour to the different movements (P<0.0001). Methods score – 57% (Rebbeck 1997).

Williams *et al.* (1991) compared the effects of two sitting postures on back and referred pain over a twenty-four- to forty-eight-hour period. Two hundred and ten patients with acute and chronic symptoms were randomised to a kyphotic or lordotic sitting group. Patients' response to the different sitting postures was assessed while in the clinic, and then over the next day or two they were instructed, when they sat, to assume a particular posture. The lordotic group was provided with a lumbar roll and instructed to maintain their lordosis; the kyphotic group with a portable cushion and instructed to sit with the spine in flexion. Back and referred symptoms were again assessed on return to the clinic.

There was a significant reduction in back and leg pain at all test points in the lordotic group compared to baseline, but no change in the kyphotic group. Whereas at baseline there was no significant difference between the two groups after the intervention, they differed significantly in terms of back ($P = 0.009$) and leg ($P = 0.018$) pain. There was a 21% and 56% reduction in intensity of back and leg pain respectively in the lordotic group, while in the kyphotic group back pain increased by 14%, and there was no change in leg pain intensity. Pain peripheralised to below the knee in 6% of the lordotic group and in 24% of the kyphotic group ($P = 0.017$). Conversely, pain centralised above the knee in 56% of the lordotic group and 10% of the kyphotic group ($P = 0.001$).

Snook *et al.* (1998) tested the effect of controlling early morning flexion in a group of patients with chronic back pain whose mean duration of symptoms was seventeen years. After recruitment symptoms were monitored for six months, patients were then randomised to the intervention or a control group who performed flexion exercises, which a previous study had found to be ineffective. The intervention group received instructions and help in a strict regimen of abstaining from flexion in the first two hours after rising, and relative restriction on flexion activities thereafter. After six months the control group was instructed in the intervention.

At six months there were significant improvements in pain intensity ($P < 0.01$), days in pain ($P < 0.05$) and medication use ($P < 0.05$) for the intervention group that were not found in the control group. At one year there were further improvements in pain for the intervention group and a number of significant changes in both groups relating to pain and disability compared to baseline.

The drop-out rate from this study was high, especially from the intervention group, with a 30% attrition rate following randomisation. This perhaps attests to the difficulty of making such behavioural changes; the postural rules expected of the patients were extremely strict and demanding. Fifty-three of the sixty patients who completed the trial were followed up at three years (Snook 2000). Sixty-two percent of this group were still finding the intervention useful and restricting their flexion, and 74% reported a further reduction in days in pain.

Table 11.4. Studies into directional preference

Reference	*Intervention*	*Effects of extension*	*Effects of flexion*	*Difference between groups*
Donelson *et al.* 1991	Repeated movements in single assessment. Randomised: 1. extension/ flexion 2. flexion/ extension	LBP better: 21% Leg pain better: 39%	LBP better: 10% Leg pain better: 8%	*P = 0.0001*
Williams *et al.* 1991	Two-day period Randomised: 1. lordotic sitting 2. kyphotic sitting	LBP better: 21% Leg pain better: 56% Centralisation: 56% Peripheralisation: 6%	LBP worse: 14% Leg pain: no change Centralisation: 10% Peripheralisation: 24%	*P = 0.009* *P = 0.018* *P = 0.001* *P = 0.017*
		Control of morning flexion		*Difference from baseline*
Snook *et al.* 1998	One year study Randomised: 1. flexion control / flexion control 2. sham / flexion control	Pain intensity reduced Pain days reduced Impairment days reduced Medication days reduced		*1 and 2: P < 0.001* *1: P < .001* *2: P < 0.05* *1: P< 0.05* *2: P < 0.01* *1 and 2: P<0.05*

LBP = low back pain

These trials and those mentioned in other sections in this chapter (for instance, Kopp *et al.* 1986; Alexander *et al.* 1992; Donelson *et al.*

1990) illustrate the effect that different loading strategies can have on back pain. All movements are not the same. Commonly in these studies, extension or control of flexion is the direction of preference. Donelson *et al.* (1991) demonstrated that in a single session without the use of force progressions, 40% showed a clear preference for extension, and Williams *et al.* (1991) states that nearly 60% showed a preference for an extended posture when sitting. In the long-term follow-up conducted by Snook (2000), about 60% of those who completed the trial still found limitation of flexion helpful. However, a minority of patients demonstrate other directional preferences, with 7% showing a clear preference for flexion in one trial (Donelson *et al.* 1991). Patients at all stages of the natural history of back pain show these responses, both those with acute and chronic symptoms. The fact that different patients show preferences for different movements should be considered in the construction of future trials. In the past, individual assessment of suitability for exercise regimes has rarely been conducted.

The importance of directional preference in management strategies has been recognised in other classification systems: Fritz and George (2000) include a flexion and an extension syndrome, Sikorski (1985) includes an anterior and a posterior element category, Wilson *et al.* (1999) also include patterns that are based on directional preferences or antipathies. One pattern is worse with flexion, another worse with extension. The largest group were those made worse by flexion activities, which represented about 65% of the population sample. The other classification systems categorised 36% to 50% of their samples as having directional preferences for extension or flexion (Fritz and George 2000; Sikorski 1985).

As different mechanical back pain problems display different directional preferences, all back pain cannot be viewed as a homogeneous entity, nor can it be presumed that all patients will respond in the same way to the same exercises.

Reliability studies

Certain studies have sought to evaluate the reliability of the McKenzie system as a whole, whereas other studies have examined the reliability of components of the whole approach. The Kappa coefficient is commonly used in reliability studies (see Glossary). Principle findings are summarised in Table 11.5.

Kilby *et al.* (1990) developed a clinical algorithm to test out the reliability of the syndrome classification system. The behaviour of pain with repeated movements and sustained positions was the key factor in the determination of the syndrome. Two clinicians, with limited attendance on McKenzie courses, assessed forty-one patients. One assessed the patient while the other one observed; no communication was allowed. Inter-clinician agreement was assessed by Kappa, with percentage agreement being used where numbers were insufficient for Kappa analysis. The answers were within 10% of perfect agreement in all but three questions. There was perfect or near perfect agreement on questions about centralisation (Kappa 0.51), constant pain, referred pain, pain on static loading and central or symmetrical pain. There was poorer agreement about the presence of a kyphotic and lateral shift deformity and pain at end-range. Agreement was less good by diagnosis, with less than 60% agreement on the classification recorded, although this improved to 74% if derangements three and four, and five and six were amalgamated (McKenzie 1981).

The strong point of the system revealed by this study is the level of agreement on interpreting pain behaviour on repeated movements. Centralisation, reduction or abolition of pain may be reliably interpreted. Visual observation, such as the presence of a lateral shift, has a weaker level of agreement. When this decision-making process was taken out of the equation, and derangement three and four and five and six amalgamated, agreement on derangement classification increased substantially. In thirteen of the forty-one (32%) of the sample, the diagnosis was uncertain or the problem had resolved.

Riddle and Rothstein (1993) conducted a multi-centred study involving 363 patients and forty-nine clinicians evaluating the reliability of the classification system. Information for the clinicians on the criteria for classification into syndromes was summarised by the authors. Only sixteen of the clinicians had attended at least one postgraduate course in the use of the McKenzie approach, so for most of them this four-page pamphlet, which contained inaccuracies, was the only information available. Patients were assessed first by one clinician and then within fifteen minutes by the second clinician, meaning that patients were put through two lengthy assessments that may have had the effect of changing symptoms. For all clinicians, agreement on classification was 39% (Kappa 0.26) and ranged from 22% to 60% in the different clinics (Kappa 0.02 to 0.48). Agreement was even less in those with some training at 27% (Kappa 0.15).

This study did not assess any component parts of the assessment procedure, but only the final mechanical syndrome classification. The study reveals a considerable lack of understanding of factors involved in this classification process by the participating clinicians. For instance, the different derangements, one and two, three and four, and five and six, are differentiated by the site of the pain. However, pain that one clinician reported to be referred to the knee or foot was reported in another area on 50% or more of occasions. Classification of postural, dysfunction and derangements syndromes was completely muddled – of the thirty-eight patients, one clinician reported postural syndrome, the other clinician reported 29% dysfunction and 29% derangement. Of twenty-eight patients that one clinician reported to be derangement five, the other clinician reported 25% dysfunction, 18% derangement one and two and 21% derangement three and four. The study certainly shows that in the hands of untrained clinicians, the system cannot be reliably used as basic errors in interpretation of pain site and pain behaviour are being made.

Razmjou *et al.* (2000b) reformulated some of the data reported by Riddle and Rothstein (1993) to compare the effect of education. When it comes to differentiating between the different syndromes, although percentage agreements are not much better between the untrained and the partially trained clinicians, there is far less variability. In particular, there is no disagreement between postural and derangement categories, and the most common mistake is between dysfunction and derangement syndromes.

The patient population studied were a very chronic group, with a mean duration of symptoms of seventy-four weeks. In the instructions given to clinicians, only ten of each repeated movement was allowed. Despite this, rather surprisingly in a group that would be expected to require lengthier periods of testing, clinicians failed to give a diagnosis in only sixteen (4%) patients. The study reveals the importance of experience and education in the use of a classification system, which allows interpreters to gain an understanding of the significance of different aspects of the assessment. Lack of experience permits multiple erroneous decisions during the diagnostic process.

Donahue *et al.* (1996) evaluated the reliability of the identification of a lateral shift and relevant lateral component. Forty-nine patients were examined separately by two clinicians drawn from a pool of ten clinicians, all with no postgraduate McKenzie training. Reliability

was studied for a two-step process – first the agreement on presence of a lateral shift using a spirit level, second on the relevance of the lateral component by performing repeated movements. Relevance was determined by a change in the location or intensity of pain during or immediately following side glide testing. Overall there was 47% agreement (Kappa = 0.16). However, when the two steps of the process were evaluated separately, the results were very different. The inter-tester reliability of the presence and direction of lateral shifts using the spirit level was 43% agreement (Kappa = 0.00). The inter-tester reliability of the relevance of the lateral component as determined by symptom response to repeated movements was 94% agreement (Kappa 0.74).

Another study demonstrated that larger lateral shifts could be reliably observed. Tenhula *et al.* (1990) examined twenty-four patients with 'an observable lateral shift', with apparently those with an equivocal shift not being admitted to the study. However, an operational definition of what is 'an observable lateral shift' was not given. One clinician judged the presence and direction of a shift while the second determined the same factors from a slide image of the patients. Perfect agreement (Kappa 1.00) was found. This study also found a statistically significant relationship between the shift and a positive side bending movement when it was seen to alter symptoms. This indicates the usefulness of repeated movements to identify the presence of a lateral shift.

Razmjou *et al.* (2000a) examined components of the assessment as well as the classification system itself. One clinician examined forty-five patients while a second clinician observed the interaction; both had considerable postgraduate McKenzie training. Various elements of the assessment process were shown to have good to excellent reliability, including the relevance of the lateral component and lateral shift, the presence of a sagittal plane deformity, syndrome identification and derangement sub-classification. The presence of a lateral shift had moderate reliability. There were three disagreements over classification; one clinician classified these patients as 'other', and one clinician as dysfunction and derangement.

It is interesting to contrast the proportion of syndrome classification made by these experienced McKenzie practitioners with the untrained and partially trained clinicians in Riddle and Rothstein (1993). While Razmjou *et al.* (2000a) diagnosed derangement in about 88% of

patients, dysfunction in about 7% and posture in 2%, in the other study classification was respectively about 55%, 35% and 10%. Clinicians unfamiliar with the system do not recognise the preponderance of derangements in clinical practice and overestimate the prevalence of the other two mechanical syndromes.

Fritz *et al.* (2000a) used a different method to evaluate the inter-tester reliability of centralisation involving video footage of patient assessments. This was then shown to forty clinicians and forty student clinicians, who were also given clear operational definitions of centralisation, peripheralisation and status quo. Agreement over symptomatic response among all clinicians was 88% (Kappa 0.79), with students only slightly less reliable than qualified clinicians.

Werneke *et al.* (1999), as part of a descriptive study of centralisation, carried out a reliability check. Clinicians had near perfect agreement both in location of most distal pain and in categorisation of patients into centralisation, partial centralisation or non-centralisation groups (Kappa 0.917 to 1.00).

Kilpikoski *et al.* (2002) evaluated two clinicians and thirty-nine patients on inter-tester agreement on certain aspects of a McKenzie assessment. One examiner questioned the patient with the other examiner present; they then took it in turns to examine the patient independently. As in other studies, observation of the presence (Kappa 0.2) and direction (Kappa 0.4) of a lateral shift was less reliable than the relevance of the shift (Kappa 0.7). Reliability of classification into McKenzie main syndromes (Kappa 0.6), sub-classification (Kappa 0.7), centralisation (Kappa 0.7) and directional preference (Kappa 0.9) were all good to very good.

Some other studies examining the reliability of different aspects of spinal assessment include evaluations of relevant tests, and these are included in the table below (11.5). Nelson *et al.* (1979) and Strender *et al.* (1997) examined lateral tilt or sagittal configuration. Strender *et al.* (1997) and McCombe *et al.* (1989) examined pain production during single sagittal plane test movements, and Spratt *et al.* (1990) examined repeated movements and pain location and aggravation.

In summary, several studies attest to the good reliability of assessment of symptomatic response, including centralisation (Kilby *et al.* 1990; Fritz *et al.* 2000a; Donahue *et al.* 1996; Razmjou *et al.* 2000a; Werneke

et al. 1999). Decision-making based on observation, such as the presence or not of a lateral shift, has a tendency to be less reliable (Kilby *et al.* 1990; Donahue *et al.* 1996); however, it can be reliable in substantial shifts (Tenhula *et al.* 1990). Although the McKenzie classification system has been shown to be very unreliable when used by clinicians who are naïve to it (Riddle and Rothstein 1993), the system has been shown to have very good reliability in those who are experienced in the approach (Razmjou *et al.* 2000a).

Table 11.5 Studies evaluating the reliability of different aspects of the McKenzie system (see text for more detail)

Component	*Agreement*	*Kappa*	*Reference*
Centralisation	90 –100%	0.51	Kilby *et al.* 1990
	88%	0.79	Fritz *et al.* 2000a
	94%		Sufka *et al.* 1998
		0.96	Werneke *et al.* 1999
	95%	0.7	Kilpikoski 2002
Relevant lateral component – by symptom response	94%	0.74	Donahue *et al.* 1996
	98%	0.85 – 0.95	Razmjou *et al.* 2000
	89%	0.6	Kilpikoski 2002
Directional preference	90%	0.9	Kilpikoski 2002
Constant pain	95%		Kilby *et al.* 1990
Site of pain	93 –100%		Kilby *et al.* 1990
		0.92 – 1.0	Werneke *et al.* 1999
Kyphotic deformity	80%		Kilby *et al.* 1990
	100%	1.0	Razmjou *et al.* 2000
Lateral shift – by observation	55%		Kilby *et al.* 1990
	43%	0.0	Donahue *et al.* 1996
		1.0	Tenhula *et al.* 1990
	78%	0.52	Razmjou *et al.* 2000
	76%	0.39	Strender *et al.* 1997
	70%		Nelson *et al.* 1979
Presence	76%	0.2	Kilpikoski 2002
Direction	78%	0.5	
Classification	58 – 74%		Kilby *et al.* 1990
	39%	0.26	Riddle and Rothstein 1993
	93 – 97%	0.7 – 0.96	Razmjou *et al.* 2000
	74 – 95%	0.6 – 0.7	Kilpikoski 2002
Pain production: – single test movements	82 – 88%	0.63 – 0.76	Strender *et al.* 1997
		0.31 – 0.57	McCombe *et al.* 1989
Repeated movements:			Spratt *et al.* 1990
– pain location	100%		
– pain aggravation	53 – 59%		

Comparison with other classification systems and assessment procedures

It may be instructive at this point to compare the reliability of the McKenzie approach, which has been reviewed above, with examples of other systems of classification and also other assessment procedures that are commonly used in physical therapy.

Wilson *et al.* (1999) investigated the inter-tester reliability of a classification system for back pain that has similarities with the McKenzie approach, as it uses pain patterns and response to movement testing and posture. For instance, one group is worse with flexion, another worse with extension. Overall agreement on classification was moderately good at 79% (Kappa = 0.61). Fritz and George (2000) investigated a classification system based upon a mixture of history, findings from physical examination and pain response to extension and flexion. Overall reliability was again moderately good (Kappa value 0.56).

Reliability of palpation studies

Mechanical diagnosis and therapy primarily uses movement, pain responses and function for assessment purposes. In general, palpation adds very little to this interpretation. A key failing of tests that are dependent upon palpation or observation is their very poor history of reliability. Across a wide range of studies, as illustrated below (Table 11.6), these procedures have been shown to be of limited use in identifying objective or stable markers. Although frequently the same clinician is reasonably reliable in reaching the same conclusion on different occasions, the reliability of palpatory tests between clinicians is consistently poor. To use such insubstantial factors to predict treatment would seem to be unwise.

Most studies are performed on volunteers without symptoms; sometimes the study was conducted on a spinal model. Different statistical measures have often been used in these studies, which have also been conducted with different methods, so results are not always directly comparable. A number of studies have used Kappa values (see Glossary), but not all studies have used this statistical analysis. The conclusion of some studies can only be given in a qualitative judgement. Some studies use intraclass correlation coefficients (ICC), in which the maximum is 1.00, indicating perfect agreement. Mean values are given where possible, sometimes obtained by calculation from original data.

Included in the following tables are results from studies that have also investigated the reliability of tests using pain responses (indicated in bold). Although comparisons between diverse studies using dissimilar statistical analyses may be problematic, the results across different studies consistently reinforce the same conclusions. At a glance it can be seen that intra-tester reliability is considerably better than inter-tester reliability, which is consistently poor. However, procedures that use pain response are far more reliable than those using palpation.

Table 11.6 Reliability of palpation examination procedures in the lumbar spine compared to reliability of pain behaviours

Reference	*Assessment procedure (mean)*	*Intra-tester reliability (mean)*	*Inter-tester reliability*	*Intra-/Inter-tester reliability of pain behaviour*
Mootz *et al.* 1989	Fixations, present or absent	K 0.17*	K 0.00	
McKenzie and Taylor 1997	Spinal level	K 0.74*	K 0.28	
Lindsay *et al.* 1995	AM PIM		K -0.1* K 0.05*	
Binkley *et al.* 1995	AM Spinal level		K 0.09 K 0.30	
Carty *et al.* 1986	AM	K 0.58	K 0.30	
Gonnella *et al.* 1982	PIM	Dependable	Not dependable	
Billis *et al.* 1999	Spinal level	Good	Poor	
Simmonds *et al.* 1995	Grade of accessory motion on spinal model		Large force variability: e.g. 2 – 131 N, 16 – 259 N	
Hardy and Napier 1991	Grade of accessory motion on spinal model	Significant variability, P< 0.01	Significant variability, P < 0.001	
Maher and Adams 1995	Stiffness in human spines		ICC 0.19	

Continued next page

Reference	*Assessment procedure (mean)*	*Intra-tester reliability (mean)*	*Inter-tester reliability*	*Intra-/Inter-tester reliability of pain behaviour*
Maher and Adams 1994	Stiffness in human spines		ICC 0.17*	
	Pain response		ICC 0.66*	
Matyas and Bach 1985	Review:	Reliability coefficients*:	Reliability coefficients*:	Reliability coefficients*:
	AM	0.30	0.26	
	Pain on accessory movements			0.78 / 0.68
	Pain on SLR			0.96 / 0.86
	Pain on flexion			0.96 / 0.73
Van Dillen *et al.* 1998	25 items – alignment and movement			
	28 items – pain response		K 0.46*	K 0.96*

SLR = straight leg raise
ICC = intraclass correlation coefficient
K = kappa
* = calculated from original data
AM = accessory movements
PIM = passive intervertebral movements

The overwhelming evidence from the table is that while intra-tester reliability can be good, inter-tester reliability is consistently poor, or at best fair. Experience does not particularly appear to affect clinicians' ability to be consistent with their peers. Studies that have used experienced clinicians (Vincent-Smith and Gibbons 1999; van Deursen *et al.* 1990; Mootz *et al.* 1989; Simmonds *et al.* 1995; Mior *et al.* 1990) have not shown better results than studies involving student physical clinicians or chiropractors (Carmichael 1987; Mior *et al.* 1990; Matyas and Bach 1985). The poor reliability of judgements about spinal mobility raises the possibility that *"this information provides a false impression of meaningfulness that hinders rather than helps treatment selection and patient management"* (Maher and Adams 1994, p. 807).

Furthermore, the clinical utility of basing treatment on stiffness levels may be unwarranted. *"The large amount of variability in spinal stiffness values between subjects, or at different levels within the one subject, makes the determination of areas of abnormally increased*

stiffness difficult. Increased stiffness may in fact be a normal variant and bear no relationship to the patient's presenting symptoms" (Maher and Adams 1992, p. 259).

The other irresistible conclusion from these studies is that pain response is a more reliable indicator than perceptions of stiffness (Matyas and Bach 1985; Maher and Adams 1992, 1994). *"Studies have consistently shown that manual assessment of factors such as bony anomalies, tissue texture, muscle tension, joint compliance, and range of motion are unreliable whether performed by physioclinicians, physicians, or chiropractors. Tests which relied solely on patient response such as pain and tenderness were found to be more reliable"* (Maher and Adams 1992, p. 258).

It is instructional to compare the Kappa values given in Table 11.6 with those in Table 11.5 of studies evaluating the reliability of different aspects of the McKenzie system. These reinforce the same point: pain response is considerably more reliable than palpation or observation.

Studies into the prognostic and diagnostic utility of centralisation

One of the key symptomatic responses employed in mechanical diagnosis is centralisation. Several studies have investigated this phenomenon, and these will be outlined below. Centralisation is discussed in more depth in Chapter 8. In all of the following studies, the level of training in the McKenzie approach of the involved clinicians is at least considered and displays considerable variability. Main findings are summarised in Table 11.7.

Donelson *et al.* (1990) were the first to describe in the literature the phenomenon that had been observed initially by McKenzie in 1956 (McKenzie 1981). Out of 225 consecutive patients with back pain, eighty-seven patients with radiation of pain to the buttock, thigh or calf were included in this study. Patients had a range of acute and chronic symptoms. Mechanical evaluation and treatment using end-range repeated sagittal and frontal plane movements was conducted, using the movement that abolished distal pain. Outcomes, which were reviewed independently, were said to be excellent if there was complete relief of symptoms, and good if there was partial relief and improvement in three secondary criteria: patient satisfaction, improvement in physical examination and return to work. A fair

result was defined as partial relief, but with failure to improve in some of the secondary criteria and a poor outcome defined as no relief.

Centralisation occurred mostly on the initial visit and sometimes in the subsequent few days. The opposite movement to the one that centralised symptoms always exacerbated them. Seventy-six (87%) patients reported centralisation and seventy-two (83%) reported good or excellent outcomes. In those who had an excellent or good outcome 100% and 77% had centralisation of symptoms, while in those with fair or poor outcomes centralisation occurred in 57% and 37%. Centralisation occurred regardless of the length of time symptoms had been present – 89% in those with symptoms of less than four weeks, 87% in those with symptoms for four to twelve weeks and 84% in those with symptoms for over twelve weeks.

Long (1995) looked at centralisation in a chronic low back pain population. Two hundred and twenty-three patients were classified as centralisers or non-centralisers depending on their response to an initial mechanical evaluation – the most distal, but not all lower limb symptoms had to be abolished. Patients were then entered into a work-hardening programme, after which outcome measures were collected by staff blind to classification. Both groups reported significant reductions in pain intensity measures, but centralisers reported a greater improvement and also a higher return to work rate (68% compared to 52%). There were significant improvements in lifting ability and Oswestry disability scores, but no differences between the groups.

Karas *et al.* (1997) studied a back pain population who were out of work and compared the predictive value of centralisation and Waddell's non-organic signs regarding return to work. One hundred and seventy-one patients were examined, of whom 126 were used in the final calculations. Centralisation was defined as proximal movement or decrease of symptoms in response to movements in one direction within two treatments. Treatment consisted of exercises in the patients' direction of preference, recovery of function and physical conditioning. Low Waddell score (P=0.006) and centralisation (P=0.038), both separately and together, were associated with higher return to work rates. Failure to centralise or abolish symptoms rapidly and high Waddell scores are both associated with a lack of response to mechanical therapy and predict a poor outcome.

Sufka *et al.* (1998) compared, in twenty-four patients, those who completely centralised symptoms within two weeks and those who did not. Poorer outcomes were found in those with chronic symptoms. Centralisation occurred more frequently in those with acute compared to chronic symptoms (83% vs 60%) and in those with back pain only compared to those with pain below the knee (80% vs 43%). Two functional outcome measures were used – both showed greater improvements in the centralisation group, one of which was significant.

Donelson *et al.* (1997) conducted a one-off mechanical evaluation on sixty-three chronic back patients and compared the clinicians' findings with those from diagnostic disc injections. Following the mechanical assessment, patients were classified as centralisers, peripheralisers or no change. Following discography, classification was made as to positive discogenic pain and competency of the annulus. The investigator performing the discogram studies was blinded to the findings from the mechanical assessment. In those in whom pain centralised or peripheralised, 74% and 69% had positive discogenic pain, compared to 12% in the no-change group ($P<0.001$). Ninety-one percent of those who centralised had a competent annulus, compared to 54% of those who peripheralised ($P<0.042$).

Werneke *et al.* (1999) conducted a study involving 289 acute back and neck patients. Patients were classified into three groups: centralisation, non-centralisation and partial reduction. Centralisation was defined much more strictly than previous studies, as a lasting abolition of pain from the initial assessment, with further proximal movements of pain on all subsequent visits until all pain is abolished. The partial reduction group allowed a more gradual decrease in distal pain over a period of time and between clinic visits. With this stricter definition centralisation occurred less frequently (31%) than other studies, but partial reduction also happened regularly (44%). The complete centralisation group averaged fewer visits than both other groups, four compared to eight ($P<0.001$). However, concerning outcomes of pain and function, both the centralisation and partial reduction groups had greater improvements than the non-centralisation group ($P<0.001$). Thus the partial centralisation took longer but achieved the same outcome; this happened in two distinct patterns. About a third demonstrated a proximal change in pain on the initial visit, which was maintained, while 71% showed no change on the

initial visit, but gradually centralised over time. About half had showed this improvement by the third visit, 74% by the fifth visit and 93% by the seventh visit. The authors speculate that improvements in this group were due to the natural history of acute problems, although equally they could have resulted from the prescribed exercise therapy. If patients had not demonstrated an improvement by the seventh visit, no significant changes were noted.

Werneke and Hart (2001) looked at the power of centralisation and non-centralisation to predict outcomes one year after patients were recruited to the study above in the 223 patients with back pain; 84% were contacted. The centralisation and partial reduction group were analysed together and compared to the non-centralisation group. Other demographic, historical, work-related and psychosocial factors were also considered. These included factors previously found to be of important prognostic value, such as pain intensity, duration of symptoms, prior spinal pain, workers' compensation, work satisfaction, Waddell's non-organic signs, depressive symptoms, somatisation and fear–avoidance. The outcomes considered were pain intensity, return to work, sick leave, function at home and health care usage. Nine of the twenty-three independent variables had an individual prognostic influence on certain outcomes at one year. However, in a multivariate logistic regression analysis that included all the significant factors from the univariate analyses, only two factors remained significant. Only centralisation / non-centralisation classification and leg pain at outset were predictive, with pain pattern classification predicting four out of the five outcomes.

Skytte (2001) studied sixty patients who were classified into centralisers (twenty-five) and non-centralisers (thirty-five) and followed them for one year. Forty-six percent of the non-centralisation group received surgery, compared to 12% of the centralisation group (P=0.01). Significant differences were also seen in reported disability and leg pain favouring the centralisation group, but no differences were seen in medication use, sick leave or back pain.

In summary, centralisation is a common occurrence in acute and chronic spinal pain. Various studies have demonstrated that, compared to patients who fail to centralise, the phenomenon is associated with significantly better outcomes relating to pain, function and return to work (Donelson *et al.* 1990; Long 1995; Karas *et al.*

1997; Sufka *et al.* 1998; Werneke *et al.* 1999). The converse is also very apparent from these studies; non-centralisation is significantly associated with a poor outcome. The study by Werneke *et al.* (1999) suggests that if a decrease in pain location score is not apparent by the seventh visit, no improvements are likely. Werneke and Hart (2001) further investigated the predictive value of centralisation or partial reduction compared to non-centralisation along with twenty-three other psychosocial, somatic and demographic variables. Non-centralisation was the most powerful independent predictor of poor outcomes. This is the first study in which a clinical variable has been shown to be of more significance than psychosocial factors in predicting chronic pain and disability.

Studies addressing the reliability of assessment of centralisation are summarised in a section above. Williams *et al.* (1991) demonstrated the use of the lordotic sitting posture to bring about centralisation, and this study is also summarised above in the section on directional preference. The data from the study by Donelson *et al.* (1997) has been re-analysed to determine the diagnostic utility of mechanical diagnosis and assessment (Delaney and Hubka 1999). They determined that using the McKenzie system assessment for discogenic pain had a sensitivity of 94% and specificity of 82%, while assessment for an incompetent annulus had a sensitivity of 100% and specificity of 86%. Compared to nine other tests used in assessment of low back pain, none were more sensitive, but three were more specific.

Table 11.7 Studies investigating centralisation (see text for detail)

Reference	*N*	*Patient description*	*% C*	*Outcomes relative to centralisation (Significant differences)*
Donelson *et al.* 1990	87	Acute 61%, sub-acute 17%, chronic 22% Symptoms below knee 51%	87%	*Correlation between centralisation and good/ excellent outcome (P<0.001), non-centralisation and poor/fair outcome (P<0.001)*
Long 1995	223	Chronic 100% Symptoms below knee 49% Out of work 100%	47%	*Greater reduction in pain intensity (<0.05), higher return to work rate (P=0.034)*

Continued next page

Reference	*N*	*Patient description*	*% C*	*Outcomes relative to centralisation (Significant differences)*
Karas *et al.* 1997	126/171	Acute and chronic Out of work 100%	73%	*More frequent return to work (P=0.038)*
Donelson *et al.* 1997	63	Chronic 100% Majority pain below the knee Not working 70%	49%	*74% positive discogram (P<0.007), of whom 91% competent annular wall (P<0.001)*
Sufka *et al.* 1998	36/48	Acute 16%, sub-acute 42%, chronic 42% Symptoms below the knee 39%	69%	*Greater functional improvement (P=0.015)*
Werneke *et al.* 1999	289	Back pain 77% Acute 100% Symptoms below knee/ elbow 31% Not working 37%	1: 31% 2: 44%	*1: Fewer visits (P<0.001) 1+2: Greater improvements in pain (P<0.001), and function (P<0.001)*
Werneke and Hart 2001	187	Acute 100%, reviewed at one year	77%	*Non-centralisation predicted work status, function, health care use (P<0.001) and pain intensity (P=0.004)*

% C = proportion in which centralisation occurred.

Werneke *et al.* 1999: 1 = centralisation, 2 = partial centralisation (see text).

Conclusions

This chapter has presented the current literature that is relevant to the McKenzie approach. New research is continuously being conducted, and no doubt some new studies will have been missed in this review. The literature has been described by intervention studies, by directional preference, by reliability studies and by studies investigating centralisation.

Intervention studies in the shape of RCTs are deemed to be the 'gold standard' measurement of effective treatment. To date, several studies attest to the efficacy of the McKenzie approach, but more high-quality studies are needed. In particular, there is the suggestion from several

studies that patients with chronic back pain may find the approach especially helpful, and yet this is an area that has been little explored. Most studies have been conducted with patients with acute/sub-acute back pain, a group who often have a good prognosis whatever is done to them.

In most of the literature to date there has been no attempt to classify patients before treatment. It is assumed that all patients respond equally to extension or flexion exercises regardless of their problem. However, back pain is a symptom, not a diagnosis. Several studies attest to the fact that all back pain does not respond equally to the same exercise, but that individual patients have directional preferences for particular exercises (Donelson *et al.* 1990, 1991; Wilson *et al.* 1999; Fritz and George 2000). Some of this work has been done by different groups of clinicians who have, independently of the McKenzie classification system, identified specific sub-groups based upon directional preference. Failure to incorporate this into intervention studies could produce a situation in which some patients improve with, for instance, extension exercises, some worsen, and the net result for the group *as a whole* is no change.

Reliability studies show that a core component of the system of mechanical diagnosis and therapy, evaluation based upon symptomatic response, generally has good to excellent reliability. By way of comparison a section also looks at the reliability of palpation techniques, which the literature shows to be a far less reliable means of examination. The classification system as a whole has been shown to be reliable in the hands of experts, but not when tested by clinicians who are naïve to the system. Centralisation can both be reliably evaluated and has been shown to be a significant prognostic factor; its presence strongly associated with good outcomes and its absence strongly associated with poor outcomes. Several studies suggest that failure to achieve this symptomatic change within seven treatment sessions indicates a failure to respond.

12: Serious Spinal Pathology

Introduction

Other chapters give descriptions of the mechanical syndromes as described by McKenzie (1981, 1990). These will encompass the majority of back pain patients, most of whom will have derangements, a few dysfunction, and even fewer present with pain of postural origin. Only a small number of patients are not grouped in one of the mechanical syndromes. This includes a few patients who have serious spinal pathology, which is the subject of this chapter. The next chapter deals with other conditions.

Within specific conditions that must be considered are the serious spinal conditions that need early identification and onward referral. A brief description is given here of cancer, infection, fractures, cauda equina and cord signs; these are given as the most common examples of serious spinal pathology. Identification of these patients is also considered in the section about *'red flags'* in the chapter on history-taking (14). A brief description is also given here of ankylosing spondylitis, as an example of one of the inflammatory arthropathies that affect spinal joints. Again, patients who are suspected of having this condition need appropriate referral so diagnosis can be clarified, although this is not the referral emergency represented by cauda equina and similar spinal problems.

It is always important to have an index of suspicion concerning specific serious spinal pathology and to use the initial assessment to triage patients (CSAG 1994; AHCPR 1994):

- serious spinal pathology
- nerve root problems
- non-specific 'mechanical' backache.

However, it must always be remembered that the vast majority of all patients fit into the latter category of non-specific, mechanical back pain. *Serious spinal pathology accounts for less than 1% of all back pain; inflammatory arthropathies also account for less than 1% of all back pain* (Waddell 1998; CSAG 1994). 'Red flag' conditions are very unusual; in a cohort of over 400 patients with acute back pain

presenting to doctors in primary care, six (1.4%) had fractures or carcinomas (McGuirk *et al.* 2001).

The sections in this chapter are as follows:

- cancer * *RED FLAG*
- infections * *RED FLAG*
- fractures * *RED FLAG*
- osteoporosis
- cauda equina syndrome * *RED FLAG*
- cord signs * *RED FLAG*
- ankylosing spondylitis.

Cancer * *RED FLAG*

In a retrospective review of radiographs of 782 patients with back pain, 0.84% had metastatic disease (Scavone *et al.* 1981a). In over 400 patients with acute back pain in primary care, 0.7% had a carcinoma: one of the kidney, one of the liver and one of the prostate (McGuirk *et al.* 2001). In nearly 2,000 walk-in patients with a chief complaint of back pain, thirteen patients (0.66%) proved to have cancer as the cause of their back pain (Deyo and Diehl 1988b).

Tumours of the lumbar spine can be clinically silent, cause back pain only, or may cause neurological deficit as well (Macnab and McCulloch 1990; Findlay 1992). They may be either benign or malignant, with a high incidence of neurological involvement in both. Neurological damage may involve the spinal cord or nerve roots or plexus, thus producing upper or lower motor neurone signs and symptoms (Rodichok *et al.* 1986; Ruff and Lanska 1989).

Primary tumours are extremely rare in the spine, while secondary tumours are less so. The breast, lung and prostate are the most common sources of spinal metastases, being the origin of over 60% of spinal metastases (Schaberg and Gainor 1985; Rodichok *et al.* 1986; Bernat *et al.* 1983). The thoracic spine is the most common site of metastases (50% or more), and about 20 – 30% occur in the lumbar spine (Ruff and Lanska 1989; Bernat *et al.* 1983). Back pain may be the presenting finding in about 25% of patients with malignant lesions. However, back pain may be absent; in one profile of 179

patients with spinal metastases, 36% were free of back pain (Schaberg and Gainor 1985). Although all tumours become visible on radiographs, 30% of the bone mass may be destroyed before a lesion is evident.

If the vertebral body is affected, pain is generally produced by pressure on, and then destruction of, the richly innervated periosteum. As the tumour spreads, the vertebra may collapse and soft tissues become involved. Severe pain may be accompanied by paralysis as tumour invasion causes collapse of the vertebra, deformity and neural encroachment (DeWald *et al.* 1985). With intradural tumours back pain occurs later, and muscle spasm and neurological involvement are a more common presentation. As symptoms are the result of a space-occupying lesion, which will only continue to grow and will certainly not shrink or vary over time, once pain commences it will become progressively more severe and intractable.

Findlay (1992) describes the clinical presentation thus: a deep-seated, boring constant pain, which is persistent and worsens as the pathology progresses. Unlike normal musculoskeletal pain, there is a lack of variability over time, and frequently, especially in children, the pain is worse at night. Musculoskeletal pain can also occur at night, but is usually relieved by a change in position; cancer pain is much more severe, may drive the patient from bed and can lead to frequent disturbances all night long (Cadoux-Hudson 2000). Certain tumours trigger considerable paravertebral muscle spasm. Neurological deficit and radicular pain may accompany back pain or may follow it. Tumours may produce localised nerve root or cauda equina syndromes, cord signs, or multiple root level signs if the plexus is damaged (Findlay 1992; Rodichok *et al.* 1986; Ruff and Lanska 1989).

While none of the physical signs were significantly associated with cancer, various elements of the history were (Deyo and Diehl 1988b). Findings that were significantly more common in cancer patients: age fifty years or over, previous history of cancer, sought medical care in last month and not improving, duration of episode greater than one month (Table 12.1). Although not significant, unexplained weight loss was also associated with cancer. Various laboratory findings were also significantly associated with cancer: erythrocyte sedimentation rate (ESR) of more than 50mm/hour (likelihood ratio 19.2), ESR more than 100mm/hour (likelihood ratio 55.5), anaemia (likelihood ratio 4). Radiographic findings of lytic or blastic lesions were excellent discriminators of cancer patients (likelihood ratio 120).

The individual sensitivity and specificity of many of these factors was poor; thus, a constellation of warning factors and an algorithmic approach to diagnosis were proposed (Deyo and Diehl 1988b). Those patients with a history of previous cancer should undergo ESR and x-ray investigation; in this group the prevalence of cancer is 9%. Those aged over 50, or with failure to improve with conservative therapy or unexplained weight loss/systemic signs should undergo ESR tests, and an x-ray should be considered – in this group cancer prevalence is 2.3%. In the rest – 60% of the original sample – no testing strategy is necessary, and the prevalence rate of cancer is 0%.

Table 12.1 Significant history in identification of cancer

History	*Sensitivity*	*Specificity*	*Likelihood ratio*
> 50 years	0.77	0.71	2.7
Previous history of cancer	0.31	0.98	14.7
Unexplained weight loss	0.15	0.94	2.7
Failure to improve after one month of therapy	0.31	0.90	3.0
No relief with bed-rest	>0.90	0.46	
Duration of pain > one month	0.50	0.81	2.6

Source: Deyo *et al.* 1992

The importance of a previous history of cancer as a risk factor for back pain that is caused by metastases is amply illustrated by a series of known cancer patients investigated for spinal pain. In these patients, 54% and 68% were discovered to have epidural, vertebral or nerve root metastases (Ruff and Lanska 1989; Rodichok *et al.* 1986).

Infections * *RED FLAG*

Spinal infections are extremely rare causes of back pain (Macnab and McCulloch 1990). An estimation of incidence is one per 250,000 of population (Digby and Kersley 1979). A survey in Denmark found an incidence of five cases of acute vertebral osteomyelitis per million of population per year – a rate of 0.0005% (Krogsgaard *et al.* 1998). The lumbar spine was affected in 59% and the thoracic spine in 33%. The highest incidence of the disease was in the 60 – 69-year-old age group, with over two-thirds of cases occurring in those between 50 and 80. However, osteomyelitis can occur in adults or children. An impaired immune system is common, and risk factors include insulin-dependent diabetes mellitus, treatment with

corticosteroids, chemotherapy, and renal or hepatic failure (Carragee 1997; Krogsgaard *et al.* 1998).

Back pain may be the main symptom in most patients (Carragee 1997). Patients have severe, progressive back pain of a non-mechanical nature, leading to spinal rigidity; tension signs are common (Macnab and McCulloch 1990). Patients are often unwell, with raised temperature, and suffer from general malaise, night pain, night sweats and raised erythrocyte sedimentation rate (Wainwright 2000). However, fever is not always present, varying between 27% and 83%, depending on the type of infection (Deyo *et al.* 1992).

Spinal infections are usually blood-borne from other sites. An unequivocal primary source of infection is found in about 40% of patients with osteomyelitis. The most common source is from the genitourinary tract, and secondly skin and respiratory infections; other relevant infections include spinal tuberculosis, brucellosis, epidural space infections and, reportedly, injections sites from illegal intravenous drug use (Deyo *et al.* 1992; Carragee 1997; Krogsgaard *et al.* 1998; Waldvogel and Vasey 1980).

A report on thirty patients with non-tuberculous pyogenic spinal infection found urinary tract infection to be the most common source of infection (30%), although in a few patients disease appeared to have been precipitated by spinal trauma (Digby and Kersley 1979). There was a preponderance of two age groups, adolescents and the elderly. Localised back pain was the predominant symptom; this was not always severe, but tended to be constant and unrelated to posture or movement. A febrile episode frequently preceded the onset of back pain, and the erythrocyte sedimentation rate was raised in all cases.

A case report documents a history of acute onset back pain with symptoms referred to the lateral border of the foot with lateral shift and kyphotic deformity, gross limitations of all movements, and limited straight leg raise who was found to have discitis (Greene 2001). He was unable to tolerate shift correction due to pain. Other features provoked suspicion of 'red flags'. The patient reported severe unremitting pain, for which no position of ease could be found, and the pain was getting worse. He was unable to sleep because of the pain and reported symptoms of nausea. He looked unwell and had a raised temperature. In another case report a previously healthy 51-year-old woman presented with acute back pain, restricted range of

movement and loss of motor function in both lower extremities (Poyanli *et al.* 2001). She had a high fever and raised ESR, and in this instance a pneumococcal osteomyelitis led to impaired consciousness in a matter of days.

Table 12.2 Significant history in identification of spinal infection

- recent or present febrile episode
- systemically unwell
- severe constant unremitting pain, worsening
- no loading strategy reduces symptoms.

Fractures * *RED FLAG*

Fractures tend to occur in two groups of patients – those involved in major trauma of any age, more commonly men, and females over 70 years old involved in minor trauma (Scavone *et al.* 1981b). One retrospective review of over 700 radiographs identified acute fractures in less than 3% of patients (Scavone *et al.* 1981a). In over 400 patients with acute back pain in primary care attending their GP, 0.7% had a fracture: two osteoporotic fractures and one crush fracture (McGuirk *et al.* 2001).

A fracture of the transverse process typically leaves patients with a persistent grumbling backache and considerable loss of function in spite of relatively insignificant signs on x-ray. Compression or wedge fractures of the vertebral body may be caused by major traumatic events or by lesser trauma in those at risk of osteoporosis. Those at risk include older post-menopausal women, those who have had hysterectomies and those on long-term corticosteroid therapy. The most common site of such injuries is between T10 and L1 (Macnab and McCulloch 1990).

Table 12.3 Significant history in identification of compression fracture

History	*Sensitivity*	*Specificity*
Age >50	0.84	0.61
Age >70	0.22	0.96
Trauma	0.30	0.85
Corticosteroid use	0.06	0.995

Source: Deyo *et al.* 1992

Osteoporosis

Osteoporosis is the most common metabolic disorder affecting the spine. The suggested World Health Organisation definition is bone mineral density more than 2.5 standard deviations below the mean of normal young people (Melton 1997). According to this definition, approximately 30% of postmenopausal white women in the US have the condition, and 16% have osteoporosis of the lumbar spine. Prevalence is less in non-white populations. Bone density decline begins in both sexes around forty years of age, but accelerates after fifty, especially in women (Bennell *et al.* 2000).

Low bone density leads to increased risk of fracture with no trauma or minimal trauma. The most common fracture sites are the lumbar spine, femur and radius. Vertebral fractures affect about 25% of postmenopausal women; however, a substantial proportion of fractures are asymptomatic and never diagnosed, and so the true rate could be higher. Despite widespread belief that osteoporosis primarily affects women, recent data shows that in fact vertebral fractures are as common in men as women. Because women live longer, the lifetime risk of a vertebral fracture from 50 onwards is 16% in white women and only 5% in white men (Melton 1997; Andersson *et al.* 1997).

Although it occurs predominantly in the elderly and in postmenopausal women, there are important secondary causes of osteoporosis not related to age. These include history of anorexia nervosa, smoking, corticosteroid use, inadequate intake or absorption of calcium and vitamin D, amenorrhea, low levels of exercise, lack of oestrogen and coeliac disease (Smith 2000; Bennell *et al.* 2000).

Low bone mass (osteopenia) is in itself asymptomatic and individuals may be unaware that they have the condition until a fracture occurs. Although pain can be absent, it can be severe, localised and difficult to treat and take many weeks to settle; the fractures also cause a loss of height (Smith 2000).

The condition, or suspicion of it, is an absolute contraindication to manipulation and mobilisation techniques. However, exercise is not only not contraindicated, but should be included as part of the management strategy for primary and secondary prevention. The effects of exercise on skeletal strength vary at different ages (Bennell

et al. 2000). Gains in bone mass are much greater in childhood and adolescence than in adulthood. The adult skeleton is very responsive to the adverse effects of stress deprivation and lack of exercise, which tends to exacerbate the natural decline in bone density that occurs with ageing. Trials of exercise have consistently shown that loss of bone mass is reduced, prevented or reversed in the lumbar spine and femur (Bennell *et al.* 2000; Wolff *et al.* 1999).

Exercise that has a higher ground impact is most effective at bone strengthening. Non-weight-bearing exercises such as cycling or swimming do not strengthen bones, whatever other benefits they may provide (Bennell *et al.* 2000). Exercise programmes have included stair-climbing, aerobics, skipping, jumping, dancing and jogging. More impact and loading is appropriate in primary prevention, but a less vigorous programme should be used in frailer groups. Programmes should be progressed in terms of intensity and impact, and maintained indefinitely, as the positive effects are reversed when regular exercise is stopped. Physiotherapy management and exercise guidelines have been recently reviewed in considerable detail (Bennell *et al.* 2000; Mitchell *et al.* 1999). Exercise therapy is complementary to but not a substitute for medical management, which includes hormone replacement therapy, calcium, vitamin D, calcitonin, biphosphonates and fluoride (Lane *et al.* 1996).

Posture is an important factor in osteoporosis. Flexion should be minimised as this can trigger damage to the vertebra; extension exercises and an extended posture should be encouraged. A group of fifty-nine women with postmenopausal osteoporosis were allocated to different exercise groups, performing extension, flexion, a combination of both or a no-exercise group. At follow-up at least sixteen months later the extent of further fractures in the different groups was compared. Further deterioration was significantly less in the extension group (16%) than the flexion group (89%), the combined group (53%) and the no-exercise group (67%) (Sinaki and Mikkelsen 1984).

Cauda equina syndrome * *RED FLAG*

Cauda equina syndrome results from compression of sacral nerve roots, although lumbar nerve roots are usually also involved. The most common causes are massive central or lateral disc herniations,

sometimes associated with spinal stenosis or spinal tumours – each responsible for about half the total (Kramer 1990). It only occurs in about 1 – 2% of all lumbar disc herniations that come to surgery, so its estimated prevalence rate among all back pain patients is about 0.0004% (Deyo *et al.* 1992). In an earlier series of 930 disc protrusions, cauda equina occurred in 0.6% (O'Connell 1955). It has been reported that there will be one new case each year for every 50,000 patients seen in GP surgeries, an incidence of 0.002% (Bartley 2000).

Principal findings in the history and physical examination that should alert clinicians to the possibility of cauda equina syndrome are in Table 12.4.

Table 12.4 Significant history and examination findings in identification of cauda equina syndrome

- bladder dysfunction, such as altered urethral sensation, urinary retention, paralysis, overflow incontinence and difficulty in initiating micturation
- loss of anal sphincter tone or faecal incontinence
- 'saddle anaesthesia' about the anus, perineum or genitals, or other sensory loss (buttocks, posterior thigh)
- impairment of sexual function
- absence of Achilles tendon reflex on both sides
- foot drop, calf muscle or other motor weakness
- unilateral or bilateral sciatica
- reduced lumbar lordosis and lumbar mobility.

Source: Kramer 1990; Tay and Chacha 1979; Kostuik *et al.* 1986; Choudhury and Taylor 1980; Shapiro 2000; Fanciullacci *et al.* 1989; Gleave and Macfarlane 1990

The most consistent finding is urinary retention, with a sensitivity of 0.90; sciatica, abnormal straight leg raise, sensory (especially 'saddle anaesthesia') and motor deficits are all common, with sensitivities of over 0.80. Anal sphincter tone is diminished in 60% to 80% of cases (Deyo *et al.* 1992). However, not all these signs and symptoms are present in all cases. A combination of features is most pathognomonic, with the constellation of bowel and bladder disturbance, bilateral sciatica and neurological signs and symptoms, especially around the 'saddle area' being most characteristic.

Roach *et al.* (1995) evaluated the use of a series of questions to identify serious back problems and found that most had poor sensitivity, but that several had a high specificity. Questions about sleep disturbance and control of urination were very specific; combining questions

improved sensitivity. However, urinary disturbance of frequency may be reported in cases of back and nerve root pain not due to cauda equina syndrome (Bartley 2000).

Two types of onset of cauda equina are described (Tay and Chacha 1979; Kostuik *et al.* 1986; Shapiro 2000). A sudden onset of cauda equina compression without previous symptoms or a history of recurrent back pain and sciatica, the latest episode resulting in or progressing to a cauda equina lesion. Trauma is only reported in a minority. The most common levels of disc herniations are generally reported to be L4 – L5 and L5 – S1 (> 90%), with the average age about 40 years old. However, in a review of over 300 patients disc herniations were reported at all lumbar levels, with 38% at the two lowest levels and 27% at L1 – 2 (Ahn *et al.* 2000a). Cauda equina compression caused by tumours tends to progress in a slower fashion.

Haldeman and Rubinstein (1992) tell a cautionary tale of cauda equina syndrome onset being associated with lumbar manipulation – with twenty-six cases of such being reported in the world literature between 1911 and 1989. The most disturbing aspect was the failure to recognise the classic features of the syndrome by treating chiropractors and initial medical contacts, leading ultimately to delayed diagnosis. *A delay in diagnosing cauda equina syndrome may have alarming implications.*

Those who have surgery delayed more than forty-eight hours are significantly more likely to have persistent bladder and bowel incontinence, severe motor deficit, sexual dysfunction and persistent pain (Shapiro 2000). Ahn *et al.* (2000a) conducted a met-analysis of 322 patients from forty-two surgical series and confirmed this. Significant differences were found in resolution of urinary and rectal function, and sensory and motor deficits in patients treated within forty-eight hours compared to those treated after forty-eight hours from the onset of symptoms. *The bottom line is, suspicion of cauda equina syndrome demands urgent referral.*

Cord signs * *RED FLAG*

In the upper lumbar region whether a large disc herniation or other space-occupying lesion causes cauda equina syndrome or cord signs and symptoms is a product of variable anatomy. The spinal cord terminates in general at about the level of the L1 – L2 intervertebral disc, but individual differences range from termination at about T12

– L1 to L2 – L3 (Bogduk 1997). Below these levels the lumbar, sacral and coccygial nerve roots run freely in the cauda equina. If the cauda equina is compressed, a lower motor neurone lesion is produced as described above; if the spinal cord is involved, an upper motor neurone lesion is produced.

With a lower motor neurone lesion signs and symptoms are essentially segmental, although several segments can be involved. This involves the combination of dermatomal pain patterns and areas of sensory deficit, myotomal weakness and absent or reduced reflexes that have been listed under cauda equina syndrome and disc problems reviewed in the relevant chapter. Upper motor neurone lesions involve the central nervous system and thus signs and symptoms are extra-segmental.

Spinal cord compression can result from bony or discal protrusions into the spinal canal, especially in those with congenitally narrow spinal canals, or can result from spinal neoplasms (Berkow *et al.* 1987). There may be gradual or rapid progress from back pain to signs and symptoms of corticospinal tract involvement (Table 12.5). These patients should be referred to the appropriate specialist.

Table 12.5 Significant history and examination findings in identification of upper motor neurone lesions

- non-dermatomal sensory loss (for instance, bilateral 'stocking' paraesthesia)
- non-myotomal muscle weakness (for instance, several segments)
- hyper-reflexia
- positive Babinski sign or extensor plantar response
- ankle clonus
- positive Lhermitte sign – neck flexion produces a generalised 'electric shock'
- generalised hypertonicity
- generalised flaccidity
- bladder and/or bowel dysfunction.

Source: Butler 1991; Berkow *et al.* 1987

Ankylosing spondylitis

Ankylosing spondylitis (AS) is one of the inflammatory arthropathies that may affect the spine. These are systemic, multi-system diseases that include a primary musculoskeletal component. AS is

characterised by chronic inflammation and tissue damage affecting principally the spine and sacro-iliac joints, but also peripheral joints and entheses, and non-articular structures such as the uvea (Goodacre *et al.* 1991; Berkow *et al.* 1987). Onset is usually insidious between the ages of 20 and 35, and rare after 40 (Macnab and McCulloch 1990). The disease, as with many conditions, represents a continuum of involvement from mild to severe. In later stages the disease process leads to ossification of spinal ligaments; the characteristic changes are clearly visible on radiographs, and severe restriction of movements and spinal deformity may occur.

Early in the disease there may be little to see, and a diagnosis of AS may be missed. Many people with ankylosing spondylitis remain unaware of their diagnosis, their symptoms of early morning stiffness and backache accepted as 'normal' and no investigations or health care are sought (Little 1988; Gran *et al.* 1985). Recognition of the disease has improved so that diagnosis has come to be made more quickly, although still involving several years' delay (Calin *et al.* 1988).

Prevalence

It has been estimated that AS is ten times more common in men than women (Calin and Fries 1975); however, in the latter the disease may present in a milder form and therefore not be recognised. Population-based epidemiological studies with definite diagnosis based on radiographic findings estimated overall prevalence as around 1%, with higher rates in men and lower rates in the older population (Gran *et al.* 1985; Carter *et al.* 1979; Braun *et al.* 1998). It is estimated that about 10 – 15% of ankylosing spondylitis cases begin during childhood years, with symptoms commencing in lower limb peripheral joints in about half of this group (Schaller 1979).

The antigen HLA-B27 is present in about 95% of patients with the condition, and this antigen is present in about 7% of the healthy white population. The disease is rare among black populations. Possibly about 10% of HLA-B27 positive adults have AS, but probably nearer 2% (van der Linden and Khan 1984). Higher prevalence rates have been suggested (Calin and Fries 1975), but this idea has been rejected (Rigby 1991). However, it is suggested that up to 5% of back pain sufferers in primary care may represent a non-specific or mild form of inflammatory joint pain (Underwood and Dawes 1995; Dougados *et al.* 1991; Braun *et al.* 1998).

Natural history

From several reviews of large numbers of patients with ankylosing spondylitis published in the 1950s, the following statements were derived concerning the natural history of the disease (Carette *et al.* 1983):

- onset is insidious
- it progresses with a series of exacerbations and remissions
- limitation of spinal movements and spinal deformity increase with time
- if peripheral joints are involved, this usually occurs early
- iritis develops early and tends to re-occur
- functional disability is usually mild
- the course is more severe if onset is during childhood or adolescence
- the disease has a milder form in women than in men.

Back and/or thigh pain is the presenting feature in over 70%, with peripheral joint disease in about 20% (Wordsworth and Mowat 1986). Pain and stiffness
tends to be a series of exacerbations and remissions (Goodacre *et al.* 1991; Mau *et al.* 1988). Radiological verification of sacro-iliitis or spinal involvement may not be present for ten years (Mau *et al.* 1987, 1988).

Carette *et al.* (1983) reported a long-term study of fifty-one patients with ankylosing spondylitis with mean disease duration of thirty-eight years. The average age at onset was 24 years old. About a third of patients denied any pain, another third described it as mild, 26% as moderate and only 4% as severe. Pain was generally most severe in the first ten years and then gradually decreased. Over the forty years only five deteriorated, and fourteen improved. Nearly all were working or had been working and were now retired due to age rather than the disease. Spinal restriction was mild in 41%, moderate in 18% and severe in 41%; deformity was mild in 67%, moderate in 15% and severe in 18%. A quarter of those with moderate or severe loss of mobility had little or no deformity. Peripheral joint involvement was noted in 36% in order of frequency: shoulders, hips, knees, ankles and metatarsophalangeals. Peripheral joint involvement and iritis, present in 24% of this sample, were both associated with more severe disease. Most had sacro-iliitis and spondylitis according to radiography.

In summary, for most individuals with this disease it takes a benign course with minimal pain, loss of mobility or functional disability (Mau *et al.* 1987). Less than 20% of patients with adult onset ankylosing spondylitis progress to significant disability, with early peripheral involvement suggesting more severe disease. In most the pattern of disease is established in the first ten years.

Diagnostic criteria

Recognition of patients with more advanced disease may be possible on radiography. Early disease is less easily detected. Diagnostic criteria for ankylosing spondylitis were specified at the Rome conference in 1963 and modified in New York in 1966. These were a combination of clinical and radiological criteria. Further modifications have been proposed that merge the two sets of criteria (Table 12.6).

Table 12.6 Modified New York criteria for diagnosis of ankylosing spondylitis

A. DIAGNOSIS
   1. Clinical criteria:
      a) Low back pain and stiffness for more than three months, which improves with exercise, and is not relieved by rest.
      b) Limitation of motion of the lumbar spine in both sagittal and frontal planes.
      c) Limitation of chest expansion relative to normal values corrected for age and sex.
   2. Radiological criterion:
      a) Sacro-iliitis grade 2 or more bilaterally or grade 3 – 4 unilaterally.

B. GRADING
   1. Definite ankylosing spondylitis if the radiological criterion is associated with at least one clinical criterion.
   2. Probable ankylosing spondylitis if:
      a) Three clinical criteria present.
      b) The radiological criterion is present without any clinical criteria.

Source: van der Linden *et al.* 1984

Symptoms said to be suggestive of back pain of an inflammatory nature are: back pain at night enough to leave the bed, early morning stiffness for more than half an hour, pain and stiffness made worse by rest, improvement with exercise, association with other joint problems and an absence of nerve root signs (Calin and Fries 1975; Gran 1985). Many patients also have a positive family history. These

characteristic clinical features led to the proposal for solely clinical criteria as a screening test for ankylosing spondylitis (Calin *et al.* 1977). Five features were found to best discriminate back pain due to ankylosing spondylitis from back pain of other causes (Table 12.7). The authors stated 95% sensitivity and 85% specificity against the control group for four or more of these features. However, when the same criteria were applied to other samples, a sensitivity of only 23% or 38% was found (Gran 1985; van der Linden *et al.* 1984).

Table 12.7 The clinical history as a screening test for ankylosing spondylitis

- onset of back pain before the age of 40
- insidious onset
- persisting for at least three months
- associated with morning stiffness
- improved with exercise.

Source: Calin *et al.* 1977

Tests purporting to identify involvement of the sacro-iliac joint (SIJ) suffer from poor reliability and unproven validity (see section on SIJ, Chapter 13). SIJ tests have been examined in ankylosing spondylitis patients; one study found little correlation between different tests (Rantanen and Airaksinen 1989). Commonly used tests have been shown to be unhelpful in distinguishing ankylosing spondylitis patients from those with other sources of back pain (Russell *et al.* 1981; Gran 1985). However, Blower and Griffin (1984) found two tests significantly associated with patients with ankylosing spondylitis – pain on pressure over the anterior superior iliac spine and local sacral pressure. These tests were not positive in every patient, and they were not always both positive in the same patient. In clinical practice some patients can experience significant exacerbation of symptoms in response to Cyriax's (Cyriax 1982) three pain provocation tests, which can last several days. Many such patients have gone on to be proven to have AS.

The sensitivity and specificity of individual criterion is low, but items related to the history perform better than items of physical examination (van den Hoogen *et al.* 1995). The prevalence of diseases has a profound effect on the value of a test. The study by Calin *et al.* (1977) was performed in a hospital population, in which with higher prevalence rates the positive predictive value of a test will be greater.

In primary care, when screening for a rare disease such as ankylosing spondylitis, the positive predictive value of a positive test is extremely low, but the negative predictive value of a negative test is high (Streiner and Norman 1996).

In summary, specific inflammatory conditions such as AS, as well as non-specific spondylarthropathies, are diseases that run a chronic course. Earlier and milder forms may often be undiagnosed and may be more common than previously imagined. As in other conditions, a clinical reasoning process and combination of features is likely to be most helpful in identifying patients with presumed ankylosing spondylitis or a non-specific inflammatory joint condition who will need further investigation to confirm this diagnosis. Such patients also respond to a mechanical evaluation in an atypical way. Patients who are suspected to have this pathology should be referred to a rheumatologist.

Conclusions

This chapter has considered some of the most common specific and serious pathologies that may affect the lumbar spine. These conditions are rarely encountered in clinical practice, but occasionally patients with these problems may appear, despite being screened by GPs or physicians. It is thus vital, in terms of safe practice, that clinicians are aware of these entities and the 'red flags' that might indicate their presence, as well as the atypical responses to mechanical evaluation that may accompany them.

Some of these conditions are absolute contraindications to mechanical therapy – cauda equina syndrome, fractures, cord signs and spinal infection. *If it is suspected that patients have any of these pathologies, urgent referral is essential.* If suspicion is supported by several factors in the history and physical examination, it is always better to be safe than sorry – get the patient to a specialist as soon as possible. In the presence of ankylosing spondylitis, osteoporosis or even cancer, if a mechanical problem is also present, cautious and appropriate management can be offered. If these pathologies are suspected, but not diagnosed, then appropriate referral is necessary.

The detail provided in this chapter is summarised in the form of criteria and operational definitions contained in the Appendix – these are essential for identification of the different pathologies.

13: Other Diagnostic and Management Considerations

Introduction

The majority of patients with back pain will be included in the mechanical syndromes (see Chapter 8). From time to time consideration of other diagnoses may have to be made. In this chapter certain specific conditions are described, as well as certain non-specific entities whose existence is controversial.

Specific conditions, such as spinal stenosis, hip joint problems and spondylolisthesis are described in this chapter. These are differential diagnoses that will have to be considered on some occasions. Other management issues are considered here, such as back pain in pregnancy,
are also described in this chapter whose existence or clinical recognition is somewhat more contentious, such as zygapophyseal joint disorders and instability. Conditions are briefly described, and key features and suggested management approaches are mentioned.

A normal mechanical evaluation, as outlined in Chapters 14 and 15, is always conducted first. These conditions only need to be considered with a failure to identify a mechanical syndrome. As will be made clear, putative recognition of these problems is often difficult and can only be done once a thorough mechanical evaluation has excluded one of the more common mechanical syndromes. *Only after the completion of a thorough mechanical evaluation, possibly over several days and/or generation of an atypical response, should these differential diagnoses be considered.*

The following sections are presented in this chapter:

- spinal stenosis
- hip problems
- sacro-iliac joint problems
- low back pain in pregnancy
- zygapophyseal joint problems
- spondylolysis and spondylolisthesis
- post-surgical status

- chronic pain
- mechanically inconclusive
- surgery
- post-surgical status
- chronic pain
- Waddell's non-organic signs and symptoms
- treating chronic backs – the McKenzie Institute International Reabilitation Programme.

Source: McKenzie Institute International Rehabilitation Programme

The detail provided in this chapter is summarised in the form of criteria and operational definitions contained in the Appendix – these are essential for identification of the different syndromes.

Spinal stenosis

Patients who have spinal stenosis that has been confirmed objectively by imaging studies may benefit from mechanical evaluation or generalised physiotherapy advice. With an ageing population it is highly likely that patients with undiagnosed stenosis will be encountered in physiotherapy clinics. In hospital populations an annual incidence of fifty per million inhabitants has been estimated, but many patients with minor symptoms do not seek medical attention, so its prevalence in the general community is unknown (Johnsson 1995). Although spinal stenosis can frequently be suspected by clinical information, objective investigations are needed to make the diagnosis. Imaging studies are essential for the definitive diagnosis of lumbar spinal stenosis (Yoshizawa 1999).

Pathophysiology

Stenosis is a condition associated with extensive degenerative changes of the disc and zygapophyseal joints at multiple levels, which may include degenerative spondylolisthesis (Amundsen *et al.* 1995). However, stenosis has both a structural and a dynamic component. The postural nature of the patient's pain is partly related to the narrowing effect that extension has on the spinal canal and the intervertebral foramen. The more the canal is structurally narrowed by the degenerative process, the more easily slight extension motion causes compression of the nerves (Penning and Wilmink 1987;

Penning 1992; Willen *et al.* 1997). Extension also causes an increase in epidural pressure, which is raised anyway in individuals with stenosis (Takahashi *et al.* 1995a, 1995b). Flexed postures have the reverse effects, widening the canal and foramen and reducing the epidural pressure, which explains why temporary relief can be gained in sitting or leaning forward.

Clinical presentation

Two types of stenosis are described depending upon whether the degenerative changes affect the nerve roots in the spinal canal or in the intervertebral foramen (Porter 1993; Heggeness and Esses 1991; Getty 1990). Laterally the root may be entrapped by bony changes, giving unremitting radicular pain from which there is no relief even at night, and which is made worse on walking. With central stenosis there is little or no leg pain at rest. This is brought on in one or both legs with walking a limited distance, termed neurogenic claudication, and is relieved with flexed postures (Porter *et al.* 1984; Porter 1993). In practice, the distinction between the two types of stenosis may be less clear (Amundsen *et al.* 1995). To further confuse diagnosis, stenosis and disc herniation may occur together (Sanderson and Getty 1996). Central stenosis can also produce signs and symptoms of cauda equina syndrome, with considerable variability in the reported prevalence of this condition (Johnsson 1995; Oda *et al.* 1999).

There has often been a long history of back pain with subsequent development of leg pain, and the condition is rarely found in those under fifty (Getty 1990; Heggeness and Esses 1991). The distinguishing feature of the condition is the postural nature of the patient's pain, with aggravation of leg symptoms when standing, and especially when walking. Leg pain is likely to be worse than back pain. Conversely, patients report relief of symptoms when they adopt positions of flexion, such as sitting or leaning forward. Walking distances can be severely impaired because of neurogenic claudication. Extension is often very limited and may provoke leg symptoms if sustained, while flexion may be maintained. Signs and symptoms of motor, sensory and reflex deficit and root tension signs are less common than with disc herniations, occurring in about 50% of patients (Heggeness and Esses 1991; Amundsen *et al.* 1995; Getty 1990; Fritz *et al.* 1998; Jonsson *et al.* 1997a; Onel *et al.* 1993; Hall *et al.* 1985; Zanoli *et al.* 2001).

There are, however, no clear clinical presentations that distinguish the different nerve root compression syndromes of lateral and central stenosis and disc herniation (Jonsson and Stromqvist 1993). One study found that history findings most strongly associated with the diagnosis of spinal stenosis are greater age, severe lower limb pain and the absence of pain when sitting. Physical examination findings most strongly associated with the diagnosis were wide-based gait, abnormal Romberg test, thigh pain with thirty seconds of lumbar extension and neuromuscular deficits (Katz *et al.* 1995). Differential diagnosis between stenosis and derangement is considered in Table 13.2 (from original idea Young 1995).

As part of their previous study, Iversen and Katz (2001) examined forty-three patients with radiographically confirmed structural evidence of spinal stenosis. The correlation between radiological changes and severity of symptoms was poor. The mean age was 72, the mean duration of symptoms three years. The prevalence of certain findings is presented in Table 13.1. Walking and standing were the most common aggravating factors, but getting up from a chair made pain worse in 43%, and sitting and leaning forward in about 25%; bending forward only made 15% better. Reduced or absent lordosis and minimal extension were common features, and if extension was maintained pain tended to radiate further down the leg. About 60% of subjects reported numbness or tingling and weakness, and findings of sensory or muscle impairment were common.

Table 13.1 Features of history and examination in spinal stenosis

Clinical feature	*Prevalence rate in sample of 43*
History	
Severe difficulties with walking	63%
Worse walking uphill	78%
Worse walking on flat ground	72%
Worse standing for 5 minutes	65%
Better side lying	68%
Better / worse seated	52% / 24%
Physical examination	
Wide-based stance	43%
Romberg test positive	39%
Reduced lumbar lordosis	65%
Lumbar extension < 10 degrees	65%
Pain on flexion	79%

Continued next page

Clinical feature	*Prevalence rate in sample of 43*
Pain on 5 sec extension in back	67%
Pain on 30 sec extension in back / thigh / calf	77% / 51% / 28%
Absent or reduced pinprick	60 – 79%
Weakness extensor hallucis longus	79%

Source: Iversen and Katz 2001

Differential diagnosis – derangement or stenosis

Table 13.2 Distinguishing spinal stenosis from derangement with leg pain

Clinical presentation	*Derangement*	*Spinal stenosis*
Age	20 – 55	>> 50
History	Sudden / gradual onset Episodes	Long history LBP Gradual onset leg pain
Status	Improving Unchanging Spontaneous resolution more likely	Unchanging Worsening Spontaneous resolution unlikely
Symptom behaviour	Variable Centralisation / peripheralisation	Consistent pattern Walking distance limited
Aggravating factors	Variable Often flexion activities – bending, sitting, driving, etc. Sometimes flexion and extension activities	Consistent Always walking Sometimes standing Activities of extension
Relieving factors	Variable Often walking, moving about, lying	Consistent Always flexion activities Bending, sitting, stooping often relieves pain temporarily
Radiography	Variable Clinically insignificant	Extensive degenerative changes Degenerative spondylolisthesis
Mobility	Major losses flexion and extension common	Extension always limited or absent Flexion well maintained
Neurological presentation	Variable sensory and motor deficit Positive tension test	Sensory and motor deficit less common Negative tension test
Response to repeated movement testing	Better / worse Centralisation / peripheralisation Obstruction to curve reversal Variable mechanical presentation	Extension produces no worse Flexion reduces no better Consistent response Mechanical presentation unchanging

Source: adapted from Young 1995

Spencer (1990) discusses the essential difference between a disc herniation and spinal stenosis as relating to the mechanism of insult to the nerve root. The latter, being due to compression, occurs without nerve tension signs in the older patient, with spontaneous resolution less likely; there is pain during walking and relief with sitting. In contrast, symptoms from a disc herniation are due to tension or compression on the nerve root, the patient is younger, with nerve tension signs, is made worse by flexion and better with extension and has a good chance of spontaneous resolution. These two clinical presentations represent extremes at either end of a continuum; in clinical practice combinations of the different mechanisms of symptom production may be found.

Management

Computed tomography, myelography and magnetic resonance imaging (MRI) are the most important imaging studies for evaluating and quantifying the degree of forminal stenosis and making the diagnosis (Jenis and An 2000; Yoshizawa 1999). However, studies into these technologies lack methodological rigour and do not permit strong conclusions about the relative diagnostic accuracies of the different procedures (Kent *et al.* 1992). Furthermore, degenerative changes are not closely correlated with symptoms (Iversen and Katz 2001; Amundsen *et al.* 2000). Abnormal findings occur in the asymptomatic population; in those over 60 years of age, 21% had spinal stenosis (Boden *et al.* 1990).

In the US in the previous two decades, surgery for spinal stenosis has more than quadrupled (Taylor *et al.* 1994). However, the long-term effects of surgical intervention are uncertain and deteriorate with time, and over a third of patients have only fair to poor outcomes (Katz *et al.* 1991,1996; Jonsson *et al.* 1997b; Tuite *et al.* 1994; Turner *et al.* 1992). In one of the latest reviews on surgical interventions for back pain, the authors concluded that there is no acceptable evidence for the efficacy of any form of decompression for spinal stenosis or for any form of fusion (Gibson *et al.* 1999).

Even when the long-term result is more favourable compared to conservative treatment, failure to improve with surgery is still common. After four years, about 30% of one surgical cohort were the same or worse, compared to about 50% of those who had been treated conservatively (Atlas *et al.* 2000). In the most recent comparison of surgical and conservative management of stenosis, in

which a subgroup of patients was randomised to different treatment groups, the outcome was most favourable for surgically treated patients, especially those with very severe symptoms. However, many improved with conservative management also, especially those with milder symptoms, and those who had an unsatisfactory result treated later with surgery still had a good outcome. Results were entirely independent of the radiological degree of degeneration, which could not be used to predict the outcome of treatment (Amundsen *et al.* 2000).

Despite being a degenerative condition, the natural history of spinal stenosis is frequently non-progressive and conservative management is thus a valid alternative (Porter *et al.* 1984; Johnsson *et al.* 1991,1992; Atlas *et al.* 1996c). Patients followed up over five to ten years have reported an improvement in symptoms (15 – 20%) and symptoms unchanged (60 – 70%), as well as a worsening of symptoms (15 – 20%) (Johnsson *et al.* 1992; Oda *et al.* 1999).

Various conservative treatments have been proposed, usually involving multiple and vigorous therapies, although none have been adequately evaluated (Fritz *et al.* 1997,1998; Onel *et al.* 1993; Heggeness and Esses 1991; Oda *et al.* 1999; Simotas *et al.* 2000). Reviewing some of these programmes, which typically include exercises and drug therapy or epidural steroid injections, it is reported with follow-up between one and five years that 15% to 43% of patients will have continued improvement after conservative treatment (Simotas 2001). A mechanical evaluation is worth undertaking to see if any element of the condition is reversible. These patients may benefit from advice to avoid positions of extension and use of flexion exercises. Failure to change the level of symptoms and disability is likely to be common in this group.

Table 13.3 Significant history and examination findings in identification of spinal stenosis

- history of leg symptoms when walking
- may be eased when sitting or leaning forward
- absence of directional preference
- no lasting change in symptoms in response to therapeutic loading strategies
- loss of extension
- possible provocation of symptoms in sustained extension, with relief on flexion
- age greater than 50

- possible nerve root signs and symptoms
- extensive degenerative changes on x-ray
- confirmation by CT or MRI.

Hip problems

Although not a condition of the lumbar spine, hip problems should be considered in the differential diagnosis as the referral of pain pattern can be similar in both. The history in hip problems is generally distinctive, and the lumbar spine is often excluded from the outset. Generally the pain pattern and the aggravating and relieving factors sound like the hip, and this is confirmed by the finding of restricted movement and/or reproduction of pain with hip tests. If the hip proves to be negative, then look at the lumbar spine.

Wroblewski (1978) has described the location of pain in eighty-nine patients (102 hips) with primary osteoarthritis (OA) who were awaiting hip surgery (Table 13.4). None of the sites featured alone; all patients described pain in several locations, with the most frequent combination including the greater trochanter, anterior thigh and knee. In 108 patients with less severe OA, who had minimal limitation of activities, pain in the anterior thigh was experienced by over half, and smaller proportions had pain in the posterior and lateral aspect of the thigh and in the knee (Jorring 1980). These pain patterns are not unique to the hip joint.

Table 13.4 Pain sites in hip osteoarthritis

Site[1]	*Proportion of hips affected* *Wroblewski 1978*	*Jorring 1980*
Greater trochanter	70%	17%
Knee	69%	18%
Anterior thigh	62%	56%
Groin	46%	8%
Shin	39%	
Buttock	39%	

[1]More than one site affected in most individuals.

Source: Wroblewski 1978; Jorring 1980

The pain is usually associated with weight-bearing, especially early in the course of the disease, but may become more constant as it progresses. Often patients will report an easing of or no pain when

sitting, in contrast to many spinal problems. Morning stiffness, pain on first weight-bearing, pain on movement of the limb and during weight-bearing are common but not universal findings (Jorring 1980).

Symptomatic hip OA occurs in about 5% of adults, most commonly in those over fifty, while over 20% of those over 55 display radiographic changes of hip OA (Felson 1988; Lawrence *et al.* 1998). Younger individuals may show no radiographic signs of involvement of the hip, and non-specific conditions may cause the joint to be symptomatic.

In OA fibrosis, thickening and contracture of the capsule produces stiffness, reduced mobility and pain at end range of movements (McCarthy *et al.* 1994). Different patterns of radiological and pathological changes have been observed (Cameron and Macnab 1975). While in 60% of the patients studied capsular restrictions were minimal until there were gross degenerative changes, in 40% there were early and marked capsular restrictions without major radiological changes. Movements commonly implicated, and which need to be included in the physical examination of the hip, are flexion, medial rotation, abduction and extension (Dieppe 1995). The hip quadrant (a combination of flexion / adduction) is also a useful test movement (Maitland 1991). Resisted tests should also be conducted, a common cause of groin pain being adductor strains. When a symptomatic hip is present, some or all of these tests should provoke the patient's pain and may form a useful part of treatment. If these tests are negative, attention focuses on the lumbar spine.

Table 13.5 Significant history and examination findings in hip joint problems

- pain worsened by weight-bearing, eased by rest
- worse with first few steps after rest
- pain pattern – groin, anterior thigh, knee, anterior shin, lateral thigh, possibly buttock
- positive pain provocation tests (reproduction of patient's pain) using passive or resisted movements.

For management considerations of hip joint problems, see *The Human Extremities: Mechanical Diagnosis and Therapy (McKenzie and May 2000).*

Sacro-iliac joint problems

The role of the sacro-iliac joint (SIJ) in spinal problems is one of the more controversial issues in back pain. While some authorities claim

a predominance of SIJ disorders among back pain patients (Don Tigny 1990), others state it has a negligible role (Cyriax 1982). It is instructive to be aware that the issues of reliability and validity of 'SIJ tests'
this stage, is this a recognisable entity? This is a necessary, but as yet incomplete, preliminary process before it can be decided which is the best way to manage the problem.

Several studies using SIJ blocks have shown that the joint is a definite if minor source of back pain. Schwarzer *et al.* (1995) found that 13% of 100 consecutive chronic back pain patients had a positive response to a single SIJ intra-articular injection of anaesthetic. In a sample of eighty-five patients chosen with suspicion of SIJ involvement due to the area of pain, 53% were positive to a single joint block (Dreyfuss *et al.* 1996). However, a positive response to a single intra-articular injection cannot be seen as a 'gold standard' test. Zygapophyseal joint injections in the cervical and lumbar spine have revealed a placebo response to a single injection of 27% and 38% (Barnsley *et al.* 1993; Schwarzer *et al.* 1994a). Likewise, in the SIJ when double injections have been used, 53% demonstrate a placebo response; that is, relief on the first injection, but failure to gain relief on the second (Maigne *et al.* 1996). In a sample that was carefully selected as likely to have SIJ problems, 18% of fifty-four patients responded to double joint blocks.

Diagnosis

All these studies compared clinical features, pain patterns and responses to commonly used 'SIJ tests' in those who responded to the injections and those who did not (Schwarzer *et al.* 1995; Dreyfuss *et al.* 1996; Maigne *et al.* 1996). *No historical features nor physical examination procedures, nor constellation of such demonstrated worthwhile and consistent diagnostic value.*

Before consideration is given to the SIJ as a possible source of symptoms, it is essential first to exclude the lumbar spine and hip joints, otherwise tests for SIJ will generate many false-positive responses. In a population of 202 chronic back pain patients, 60% had at least one positive SIJ pain provocation test (Laslett 1997). However, once lumbar and hip joint pathology were excluded, only 17% were left with at least one SIJ positive test. When a criterion of at least three and preferably four positive tests was used to distinguish SIJ pathology, only 6.5% and 3.5% were truly

were detected using the following criteria: McKenzie mechanical evaluation to detect centralisation, pressure on lumbar spinous processes to provoke familiar pain and the presence of acute lateral shifts. Hip problems were excluded using pain provocation tests – passive medial rotation and abduction, and resisted lateral rotation.

The ability of non-SIJ problems to mimic true SIJ problems is further supported by another study (Slipman *et al.* 1998). Fifty patients were selected who had pain over the SIJ area and who were positive to three SIJ pain provocation tests. Only thirty patients had a positive response to a single intra-articular anaesthetic block (60%), which meant that at least 40% of those positive to pain provocation tests are false-positives. As only a single joint block was used, the proportion of those mimicking SIJ is likely to be considerably higher.

It is thus apparent that SIJ problems are not easy to differentiate. The most common site of pain is over the buttock and posterior thigh (Slipman *et al.* 2000), but the pain pattern has no clear distinguishing characteristics. Asymptomatic volunteers who allowed SIJ injections to provoke pain described an area of pain just inferior to the posterior inferior iliac spine, with some also describing referral into the lateral buttock and thigh (Fortin *et al.* 1994). Other studies have demonstrated referral down the full length of the limb, both anteriorly and posteriorly. Two studies (Schwarzer *et al.* 1995a; Dreyfuss *et al.* 1996) that attempted to differentiate subjects with SIJ pathology from those without it by using SIJ injections found that referral of pain below the knee was as common in both groups. Groin and anterior thigh and leg pain were more common, and pain above L5 was rare in the SIJ groups. These were not exclusive characteristics, and one study found lower lumbar pain to be common and pain patterns to be highly variable (Slipman *et al.* 2000). These studies show that SIJ pathology cannot be recognised by pain patterns alone.

One study has tried to compare findings from the history and mechanical assessment in a group of chronic patients who responded to SIJ, facet injections or discography (Young and Aprill 2000). Findings from the facet and SIJ groups were similar, both showing lack of obstruction or movement loss after repeated movements, lack of centralisation or peripheralisation, and sometimes abolition of distal symptoms without centralisation. The entire SIJ group had three or more SIJ pain provocation tests positive, compared to 25% or 30% in the other two groups, and all but one had no pain at or above L5.

Pain provoked on rising from sitting was present in most of both disc and SIJ groups.

There exist numerous test manoeuvres that are said to diagnose SIJ disorders. These have been widely investigated and found wanting on many counts. Two sorts of test exist, those that attempt to provoke the patient's pain by 'stressing' the SIJ mechanically and those that seek to implicate the SIJ by trying to observe or palpate a difference in mobility or alignment with the asymptomatic side. Generally pain provocation tests are much more reliable between testers than tests that are based upon palpation or observation, which are frequently unreliable (Potter and Rothstein 1985; Lindsay *et al.* 1995; Laslett and Williams 1994; Carmichael 1987; van Deursen *et al.* 1990). Although pain provocation tests have also been found not to be reliable (McCombe *et al.* 1989; Strender *et al.* 1997), these tests generally perform much better than tests based on palpation or observation.

A selection of these studies is presented in Table 13.6. Mostly trials have been included that reported the Kappa statistic (see Glossary). As in palpatory procedures for the lumbar spine, intra-tester comparisons are more reliable, with poor to moderate reliability, than inter-tester ones, with only poor reliability. Overall tests that use pain provocation (shown in bold) have considerably better reliability than tests based upon palpation (shown in ordinary text).

Table 13.6 Reliability of examination procedures of the sacro-iliac joint (SIJ)

Reference	*Assessment procedure*	*Intra-tester reliability (mean Kappa)*	*Inter-tester reliability (mean Kappa)*
Carmichael 1987	Gillet test	0.18	0.02
Meijne *et al.* 1999	Gillet test	0.055*	-0.025*
Van Deursen *et al.* 1990	6 palpatory tests		0.04*
Mior *et al.* 1990	Mobility testing • Students • Chiropractors	} 0.50*	0.09* 0.08*
Vincent-Smith and Gibbons 1999	Standing flexion test	0.46	0.05

Continued next page

Reference	*Assessment procedure*	*Intra-tester reliability (mean Kappa)*	*Inter-tester reliability (mean Kappa)*
O'Haire and Gibbons 2000	Palpation and observation of SIJ anatomy	0.26*	0.06*
Freburger and Riddle 1999	Instrumented SIJ alignment		0.18
Lindsay *et al.* 1995	Mobility and positional tests **Pain provocation** tests		0.16* 0.33*
Dreyfuss *et al.* 1996	Gillet test 4 **pain provocation** tests		0.22 0.54*
Laslett and Williams 1994	7 **pain provocation** tests		0.70*
Strender *et al.* 1997	SIJ compression – **pain provocation**		0.26
McCombe *et al.* 1989	3 **pain provocation** tests		0.23*
			Agreement
Potter and Rothstein 1985	11 palpatory tests 2 **pain provocation** tests		39% 85%
Mann *et al.* 1984	Iliac crest heights		Mean 6.6 out of 11

K = kappa
* = calculated from original data

A systematic review considered the reliability of clinical tests for the SIJ (van der Wurff *et al.* 2000a). They found no evidence of reliable outcomes for mobility tests, while some studies demonstrated reliability for some pain provocation tests.

Multiple tests perform better, and single positive tests should be viewed as irrelevant (Laslett 1997; Cibulka *et al.* 1988; Osterbauer *et al.* 1993; Cibulka and Koldehoff 1999; Broadhurst and Bond 1998). A multi-test regime using five pain provocation tests has been found to have good reliability, Kappa value 0.70 (Kokmeyer *et al.* 2002). The authors recommended three positive tests out of five be conducted.

Various palpation or mobility tests have been examined on 'normal' volunteers with 'positive'
et al. 1994; Egan *et al.* 1996; Levangie 1999a). These tests cannot be said to diagnose SIJ problems as the asymmetrical mobility that they rely on is found in the asymptomatic population. No substantive positive association between pelvic asymmetry and back pain was found in a study of over 100 patients and controls (Levangie 1999b).

Attempts to palpate movement abnormalities should be further cautioned against due to the minimal movement that occurs at the joint – a review of sixteen in vitro and in vivo studies found this to be less than four degrees of rotation and about 3mm of translatory motion (Walker 1992). Recent high-quality studies using implanted tantalum balls and radiography have found no significant difference in mobility between symptomatic and asymptomatic joints in patients with unilateral symptoms (Sturesson 1997). The amount of movement found was minimal, less than two degrees, and during the Gillet test is *"so minute that external detection by manual methods is virtually impossible"* (Sturesson *et al.* 2000a, 2000b).

Two studies (Maigne *et al.* 1996; Dreyfuss *et al.* 1996) have tested the diagnostic validity of twelve commonly used SIJ tests against the results of double or single anaesthetic blocks of the joint. Neither pain provocation nor palpatory tests were useful predictors of a positive response to injection. Thus none of these tests, either singly or in combination, demonstrated worthwhile diagnostic value when compared with SIJ pathology identified by intra-articular blocks. However, the results may have differed if pathology was related to para-articular structures, such as ligaments. A systematic review of the validity of clinical tests for the SIJ concluded that there is no evidence to support the diagnostic value of either mobility or pain provocation tests (van der Wurff *et al.* 2000b).

A recent review of the published evidence to guide examination of the SIJ reached the following conclusions (Freburger and Riddle 2001). A combination of positive pain provocation tests and pain pattern may be useful for considering a diagnosis of SIJ. The most useful tests appear to be Patrick's test, pressure over sacral sulcus, thigh thrust / posterior shear, resisted hip abduction, and iliac compression and gapping. The most useful indicators in the pain pattern are absence of pain in the lumbar area, pain below L5, around the posterior superior iliac spine and in the groin area. Movement and symmetry tests appear to be of little value.

Attempting to detect a SIJ problem is thus extremely problematical, and a staged differential diagnostic process should always be used (Table 13.7). Given the likelihood of false-positive test results, without care, it is very likely that SIJ problems are needlessly overdiagnosed.

Table 13.7 The staged differential diagnosis for SIJ problems

1. Exclusion of more common causes of buttock, thigh and groin pain, namely lumbar and hip problems. A normal mechanical evaluation should be conducted and the patient may be given a trial of exercises over a twenty-four-hour period to further test out responses. There is no value in conducting a barrage of SIJ tests on day one, as there will be a large number of false-positive responses. A relevant lateral shift is produced by lumbar problems, to which the treatment should be directed. Centralisation, reduction or abolition of pain with repeated lumbar movements confirms a mechanical syndrome and further testing becomes irrelevant.
2. Clinicians may be alerted by failure to respond, atypical responses to repeated movements and lack of directional preference.
3. The hip joint should first be discounted using pain provocation testing (see appropriate section)
4. Pain must be present over the buttock, but may radiate anteriorly and posteriorly.
5. Multiple pain provocation tests (Laslett and Williams 1994; Kokmeyer *et al.* 2002) should be undertaken and at least three and preferably four should provoke the patient's pain for a positive identification of a SIJ problem.
   a. Distraction*
   b. Compression*
   c. Posterior shear or thigh thrust or posterior pelvic pain provocation test* (see section below: Back pain in pregnancy)
   d. Pelvic torsion or Gaenslen's test* (both sides)
   e. Sacral thrust
   f. Cranial glide
   g. Patrick sign or Faber test*

*Five tests used by Kokmeyer *et al.* 2002

Using such a clinical reasoning process with a patient history, dynamic mechanical evaluation and pain provocation testing of first the hip and then the SIJ has been compared to double anaesthetic joint blocks (Young *et al.* 1998). Agreement between the physical examination and the injection was 91%, with a Kappa value of 0.82.

Table 13.8 Significant examination findings in identification of SIJ problems

- exclusion of lumbar spine by extended mechanical evaluation
- exclusion of hip joint by mechanical testing
- negative response to mobilisation of lumbar spine
- positive pain provocation tests (reproduction of patient's pain) – at least three tests.

Management

A wide range of interventions has been proposed for the treatment of SIJ syndrome. This includes exercise, manipulation, injections of corticosteroid and local anaesthetic, injections of sclerosing agents, and even surgical arthrodesis (Bernard 1997). Within the field of physical therapy, there are some ornate classification systems of SIJ syndromes based upon pathological models of innominate subluxations and fixations (such as Lee 1997; Don Tigny 1997). Palpation and mobility testing are used to discern these, but there is little evidence of reliability or validity, as already noted. With the problems involved in recognising true SIJ pathology and the difficulty of assembling such a cohort, scientifically testing out specific interventions has never been satisfactorily achieved. Thus no evidence exists as to the efficacy of any proposed interventions for the SIJ.

There is an incomplete understanding of the pathology of the SIJ and the reason for pain, although various theoretical models exist. It is not known if the source of symptoms is articular or para-articular – if it is the latter, then the injection studies mentioned earlier may not expose it. Pain may be due to a mechanical articular lesion and sometimes responds to repeated end-range exercises or clinician techniques. If pain appears to be mechanical – intermittent, twinges, unilateral, activity-related – it is worth exploring symptom response to repeated end-range anterior and posterior pelvic rotation. On the other hand, pain may be due to soft tissue insufficiency around the pelvis and require stabilising with a belt – see next section on back pain in pregnancy.

It has also been suggested that SIJ pathology is primarily inflammatory – in such a case pain would be a constant, dull aching, aggravated by mechanical therapy. Bone scanning with quantitative sacro-iliac scintigraphy has provided evidence of inflammation in women with chronic non-specific back pain (Davis and Lentle 1978; Rothwell *et al.* 1981). Inflammatory sacro-iliac disease, not related to ankylosing spondylitis, was diagnosed in this way in twenty-two of fifty patients, compared to two of sixty-six controls (Davis and Lentle 1978). If such pathology is the root of symptoms, mechanical therapy will be unhelpful.

If, having performed the staged differential diagnostic process outlined above, mechanical SIJ involvement is suspected, management will be determined by response to repeated movements. These movements need to focus on rotation of the pelvis. With the present uncertain

understanding of pathology and lack of clearly evaluated practice, a degree of experimentation may be warranted, but failure to respond is common. For SIJ problems related to pregnancy, see the following section.

Low back pain in pregnancy

Prevalence

Back pain is a common although not universal experience for many women during pregnancy. As in other types of back pain, there are still ambiguities inherent in the terminology, diagnosis and classification (Heiberg and Aarseth 1997). This section draws together some of the evidence relating prevalence and classification as well as making some suggestions concerning management.

Following large cohorts of women through pregnancy with repeated questionnaires and a good response rate (over 85%) is the best way of establishing incidence and prevalence. Those studies that have done this have found prevalence rates of between 47% and 76% (Mantle *et al.* 1977; Berg *et al.* 1988; Ostgaard *et al.* 1991, 1994a; Kristiansson 1996a; Sturesson *et al.* 1997). The mean rate across multiple studies thus gives a prevalence of back pain of just over 50% of pregnant women. This compares to a one-year prevalence rate in the general population of about 40% (see Chapter 1).

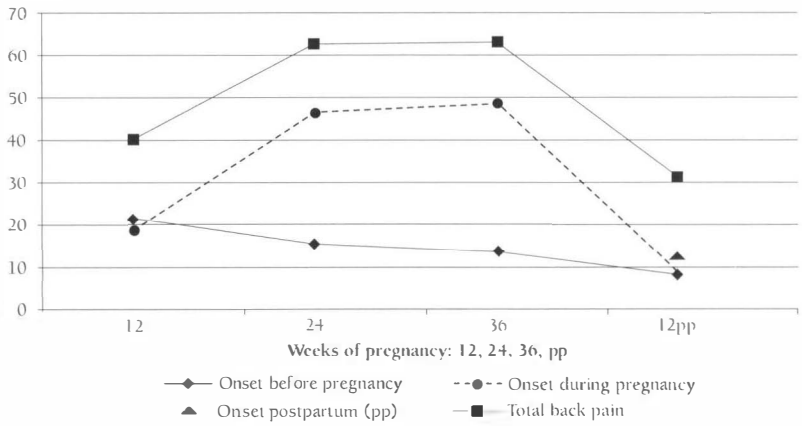
Natural history

Back pain during pregnancy is not a static entity, but changes during trimesters. Onset is most common during the third to seventh months of pregnancy (Fast *et al.* 1987; Mantle *et al.* 1977), and there is an increase of back pain as the pregnancy proceeds (Ostgaard *et al.* 1997a; Kristiansson *et al.* 1996a). In those who had back pain prior to pregnancy, there is in fact a decreased rate of back pain during the pregnancy, and following the birth there is a rapid decline in back pain. The incidence of back pain during pregnancy is considerably greater and accounts for the cumulative increase in total back pain (Figure 13.1). Ostgaard *et al.* (1997a), in 362 women, found 18% had back pain before pregnancy, 71% during and 16% six years later.

There is also a variability of impact and severity of back pain during pregnancy. Less than 20% appear to have constant pain, and intermittent symptoms are much more common (Berg *et al.* 1988; Fast *et al.* 1987). Ten to 15% of pregnant women suffer severe back

pain that interferes with daily activities, and may need time off work, while far more women suffer troublesome, but not severe pain (Mantle *et al.* 1977; Berg *et al.* 1988; Fast *et al.* 1990; Heiberg and Aarseth 1997). In general there is a tendency for increasing severity of pain as the pregnancy proceeds. On a 0 – 10 visual analogue scale, average pain intensity before the pregnancy and at weeks twelve and thirty was respectively 1.3, 3.9 and 4.5 (Ostgaard and Andersson 1991).

Figure 13.1 Back pain during pregnancy



Source: Kristiansson *et al.* 1996a

Multiple variables that may be risk factors for back pain in pregnancy have been investigated. The strongest and most consistent associations with back pain during pregnancy are a prior history of back pain, mechanical and psychosocial stresses at work, the hormonal effects of pregnancy and method of birth.

Classification of back pain in pregnancy

The site of symptoms has been used as a means of classifying back pain during pregnancy. Although these groups are distinguished by pain distribution and appear to behave differently, definitely established pathological models have not been proven. *"Pregnant women with 'back pain' can be separated into two groups with different pain patterns – one group with pain in the back and one group with pain in the posterior pelvis"* (Ostgaard *et al.* 1994a).

Posterior pelvis pain (PPP) is felt over the buttock and sacro-iliac area and back pain is felt in the lumbar region. PPP appears to be more common during pregnancy (range 24 – 48%) than low back pain (range 10 – 32%); combinations of the two types of pain are also

common (Ostgaard *et al.* 1991, 1994a, 1996; Kristiansson *et al.* 1996a; Noren *et al.* 1997; Sturesson *et al.* 1997).

During the pregnancy these different symptoms behave differently. Low back pain is more common both before and after pregnancy, but remains relatively stable or even declines in prevalence during the pregnancy. PPP, which increases dramatically during pregnancy, is probably the most common form of back pain (Ostgaard *et al.* 1991, 1994a, 1994b, 1996; Mens *et al.* 1996; Kristiansson *et al.* 1996a, Kristiansson and Svardsudd 1996). One study found the point prevalence of back pain to remain stable at about 7%, while PPP increased from 10 – 30% during the early part of the pregnancy (Ostgaard *et al.* 1994a). 'Normal' low back pain and PPP are differentiated by pain patterns and by certain other features of history and physical examination (Table 13.9).

Table 13.9 Distinguishing features of low back pain and posterior pelvic pain

Low back pain (LBP)	*Posterior pelvic pain (PPP)*
History of back pain prior to pregnancy	No previous history of back pain
Pain – lumbar region Nerve root pain unusual (1% all women)	Pain – buttock, SIJ area, radiation into thigh, also possibly pubic area, groin, coccyx and pelvis No nerve root pain
Lumbar flexion aggravates	Pain aggravated by weight-bearing
Loss lumbar range of movement	Lumbar range of movement normal
Pain on lumbar pressure	Pain-free intervals
Negative PPP provocation test	Positive PPP provocation test

PPP provocation test – patient lies supine with hip flexed to 90 degrees, clinician stabilises pelvis and pushes posteriorly through femur. Positive test reproduces concordant pain with gentle pressure (Ostgaard *et al.* 1994b). Also known as thigh thrust or posterior shear test.

Source: Ostgaard *et al.* 1991, 1996; Kristiansson and Svardsudd 1996; Mens *et al.* 1996

The thigh thrust or PPP provocation test was evaluated in a consecutive group of seventy-two pregnant women (Ostgaard *et al.* 1994b). One clinician took the history and one performed the test, blind to whether the women had pain or what type of pain. Twenty-seven women had PPP, twelve had LBP or thoracic pain and thirty-three had no pain. The sensitivity of the test in identifying PPP was 81%, its specificity in excluding those who did not have PPP was 80%, and positive and negative predictive values were 71% and 88% respectively.

It is postulated that low back pain is 'normal' back pain as experienced by the non-pregnant population, while PPP is specific to the pregnant condition (Ostgaard 1997b). It is suggested that the effect of hormones on the aetiology of PPP is significant, with serum relaxin – which is released during pregnancy – causing a softening of ligamentous restraint and producing ligamentous insufficiency or instability at the joints of the pelvis (Kristiansson 1997, 1998; MacLennan *et al.* 1986; Ostgaard 1997b). Significant correlations have been found between mean relaxin levels and back pain and, in those with pregnancy onset back pain, a positive PPP provocation test (Kristiansson *et al.* 1996b). Symptoms may derive from instability at one or both SIJ, the symphysis pubis, or all three articulations (Albert 1998).

Lordosis and pregnancy

Pregnancy produces altered mechanical stresses on the lumbar spine. Different studies suggest that biomechanical response to the pregnant state is different in different women, and at different times of the pregnancy. Ostgaard *et al.* (1993) found no change in lumbar lordosis between the twelfth and thirty-sixth week of pregnancy, but did find a significant correlation between a large lumbar lordosis and back pain. Bullock *et al.* (1987) found a significant increase in lordosis between about the eighteenth and thirty-eighth weeks. There was a mean increase of 7.2 degrees, but with considerable variety, with some women showing a marked increase – one woman's increased by 22.3 degrees. Increasing lordosis was associated with increased height and weight, but no correlation was found with back pain. Dumas *et al.* (1995) also found a significant increase in lordosis up to about thirty-two weeks. This increase continued in multigravidas after this point, but the lordosis decreased in primagravidas after thirty-two weeks.

Mechanical response to pregnancy may in fact be variable and individual. Of twenty-five women, about half showed a decrease in lordosis initially and about half stayed the same or increased (Moore *et al.* 1990). However, later in the pregnancy about half showed an increase in lordosis and about half stayed the same or showed a decrease in the lordosis. The tendency for the lordotic curve to increase with the progression of the pregnancy was associated with a greater likelihood of back pain.

Management of back pain during pregnancy

Evidence about management of back pain in pregnancy is rather thin; a Cochrane review only contained one trial that fulfilled their inclusion criteria (Young and Jewell 1999).

It is likely that women with PPP and ordinary lower back pain (LBP) will respond differently. Education and exercise programmes have produced better outcomes than control groups and have been found useful by the majority of women with LBP (Ostgaard *et al.* 1997a; Noren *et al.* 1997; Mantle *et al.* 1981). Women with PPP did not benefit from a programme of exercises and education, nor did they benefit from the protective effect of pre-pregnancy fitness, as did women with LBP (Ostgaard *et al.* 1994a). Women with PPP may worsen if treated with back strengthening exercises (Ostgaard 1997b). However, a study of women with persistent PPP after pregnancy showed no difference in outcome between groups randomised to education and refraining from exercise and those given exercises (Mens *et al.* 2000). Abdominal training was performed either focusing on diagonal or longitudinal trunk muscles, with the latter viewed as placebo. All groups could also use a pelvic belt. After eight weeks of intervention there was no significant difference between the groups, but 64% reported improvement.

Several investigators have found that women with PPP report a reduction of pain and disability, especially when walking, with the use of a non-elastic sacro-iliac or trochanteric belt (Ostgaard *et al.* 1994a; Berg *et al.* 1988; Mens *et al.* 1996).

Reports suggest variable mechanical responses to pregnancy, one of which is increased lordosis. These women may report themselves to be much worse when standing or walking, but better when sitting – such women may respond to the flexion principle (see Chapter 25 for details). Certain of the procedures may need to be adapted to cope with the pregnant abdomen, for instance by abduction at the hips. Alternatively, increased lordosis may cause postural strains and respond to postural correction in standing.

Other women may respond to the extension principle. Due to the pregnancy, certain procedures are ruled out. After a certain point in time it is not appropriate for women to lie prone; the exact time varies. When prone lying or extension in lying become impossible, extension in standing is usually still tolerated. From a four-point

kneeling position, a certain amount of extension can also be gained by dropping the abdomen to the floor. Inability to reach end-range extension may limit the effectiveness of these procedures and full reduction may not be obtained.

If PPP, as defined above, is present, women should be offered a firm belt and advised about restricting weight-bearing activities. Overdoing activities may aggravate pain the following day. Keeping generally fit with activities such as swimming may help. Although pain may not recede during the pregnancy, prognosis post-partum is good. Some women present with a mixture of LBP and PPP.

Table 13.10 General guidelines on management of women with back pain during pregnancy

- A distinction must be made between LBP and PPP. In terms of natural history and response to interventions, these appear to be different entities, and therefore management must distinguish between the two.
- Women with PPP benefit less from educational and exercise programmes, but frequently get some benefit from a firm support belt.
- Women with LBP may be classified according to one of the mechanical syndromes:
  - Derangements commonly respond to the flexion principle, some to the extension principle
  - Postural syndrome should also be considered
- An educational and exercise programme appears to be beneficial in some women, especially those with LBP. Programmes involve the following (Ostgaard 1994a, 1997a; Noren *et al.* 1997):
  - individualised according to the type of back pain
  - no passive treatment
  - lifting/working techniques and discussion of vocational ergonomics
  - muscle training and general exercise involving back extensors, abdominals and pelvic floor
  - relaxation
  - didactic educational component
  - home programme

Zygapophyseal joint problems

Diagnosis

Zygapophyseal or 'facet' joints have long been assumed to be a cause of back pain; however, its prevalence rate or means of recognition is unclear. The most effective way to establish that a zygapophyseal joint is the source of a person's back pain is to inject the joint with

anaesthetic. This should be done under fluoroscopic guidance to ensure that the injection is accurately located. Based on single diagnostic blocks of this type, the prevalence of zygapophyseal joint pain has been reported to range from 8 – 75% in sixteen different studies (Dreyer and Dreyfuss 1996).

Unfortunately, such intra-articular injections are associated with a high rate of false-positive findings. Substantial numbers have pain abolished by a placebo injection or respond to a first but not a second injection. Rates of such false-positive responses to single lumbar zygapophyseal joint blocks have been shown to occur in 32%, 38% and over 60% of individuals (Schwarzer *et al.* 1992, 1994a, 1994d). The positive predictive value of a single joint block has been rated at only 31% (Schwarzer *et al.* 1994a). Furthermore, the amount injected must respect the capsule of the joint, which will leak or tear if more than a few millilitres are injected, and thus may affect other structures (Raymond and Dumas 1984). These factors invalidate previous attempts to describe this entity using only single joint blocks, some with excessive quantities of contrast agent, saline or analgesic.

Prevalence

Using a rigorous research design involving two separate joint blocks, the prevalence of zygapophyseal joint pain has been estimated at 15% of 176 (Schwarzer *et al.* 1994b) and at 40% of sixty-three (Schwarzer *et al.* 1995b) patients with chronic back pain. In another study that used pain provocation and pain relief to make the diagnosis, 17% of fifty-four chronic back pain patients had the diagnosis confirmed (Moran *et al.* 1988). In another study involving ninety-two consecutive chronic back pain patients, both the zygapophyseal joints and the intervertebral disc were investigated as sources of pain (Schwarzer *et al.* 1994d). The latter were diagnosed by exact pain reproduction on discography, with abnormal image, provided no pain was reproduced at a control segmental level. Thirty-nine percent had positive discograms, while 9% were positive to double zygapophyseal joint blocks. Only 3% of the patients had a combination of zygapophyseal and discogenic pain.

Clinical features

Clinical features have not been found that could predict patients' response to such injections. Factors such as movement limitation, day or night pain, pain on certain movements, pain aggravated or relieved by certain activities, and area of pain referral could not

distinguish those patients who responded to zygapophyseal joint injections from those who did not (Schwarzer *et al.* 1994b, 1995b; Jackson *et al.* 1988). For instance, features such as aggravation of pain by rotation, or extension and rotation, or referral of pain were poor discriminators of zygapophyseal pain. Two earlier studies (Fairbank *et al.* 1981; Helbig and Lee 1988) suggested certain features that were present in patients who responded to zygapophyseal joint injections. However, these criteria were later found to be unreliable in distinguishing this from other sources of pain (Schwarzer 1994c).

Direct stimulation of facet joints has produced mostly local or buttock pain (Marks 1989). However, those responding to double joint blocks are as likely to have symptoms radiating into the thigh and lower leg as those who do not respond (Schwarzer *et al.* 1994b). The only pain pattern that appears to differentiate between responders and non-responders is central pain, which was never found in those responding to double joint blocks (Schwarzer *et al.* 1994b, 1994d). Computed tomography was unable to distinguish painful joints either (Schwarzer *et al.* 1995c).

One small study has demonstrated the accuracy of diagnosis by manual examination when compared to zygapophyseal joint blocks in the cervical spine (Jull *et al.* 1988). In twenty patients, manual therapy showed 100% sensitivity and specificity in diagnosing cervical zygapophyseal joint pain. Such a study has not been reproduced, nor has it been replicated in the lumbar spine.

Recently a new set of criteria to identify patients with painful zygapophyseal joints has been identified and proposed through studying responders and non-responders to joint injections (Revel *et al.* 1992, 1998). Pain should always be relieved by recumbency, and four of the following variables also had to be present:

- age greater than 65 years
- pain not exacerbated by coughing
- pain not worsened by hyperextension
- pain not worsened by flexion
- pain not worsened rising from flexion
- pain not worsened by rotation-extension.

If the patient has five of these seven criteria, has not had spinal surgery, does not have true sciatica, does not have upper lumbar or sacro-iliac joint pain, there is a greater than 90% chance that they will respond to an injection. These characteristics should not be considered diagnostic for zygapophyseal joint pain, but only indicative of a patient who will respond to a zygapophyseal joint injection.

One study has tried to compare findings from the history and physical examination in a group of chronic patients who responded to SIJ, facet injections or discography (Young and Aprill 2000). Findings from the facet and SIJ groups were similar, both showing lack of obstruction or movement loss after repeated movements, lack of centralisation or peripheralisation, and abolition of distal symptoms without centralisation.

Management

Not only is the identification of this group problematical, but no effective treatment has been identified. Open uncontrolled studies evaluating the value of intra-articular steroid injections report relief in 18 – 63% of subjects in ten studies; however, such study designs are inherently biased and are likely to report favourable outcomes (Dreyer and Dreyfuss 1996). Corticosteroid injections into zygapophyseal joints when evaluated under randomised, controlled study design are no more effective than injections of saline (Lilius *et al.* 1989; Carette *et al.* 1991).

Radiofrequency facet joint denervation is a recent treatment option that appeared to have positive short-term effects in two small studies (Gallagher *et al.* 1994; van Kleef *et al.* 1999). However, a larger, more recent study found the intervention to lack treatment effect at twelve weeks (Leclaire *et al.* 2001).

In summary, zygapophyseal joints can be a source of pain, but identification through a normal clinical examination appears to be unlikely. At this stage there is no clinical benefit in identifying them as a separate group. Such patients may respond mechanically.

Spondylolysis and spondylolisthesis

Definitions and classification

Spondylolysis is a defect in the pars interarticularis. Spondylolisthesis denotes a forward displacement of a vertebral body, which can occur if there are defects in both neural arches.

Spondylolysis and spondylolisthesis have been classified according to origin. The commonly accepted classification is as follows (Macnab and McCulloch 1990):

- dysplastic
- isthmic
- degenerative
- traumatic
- pathological.

This classification refers largely to onset – dysplastic being due to a congenital deficiency, isthmic occurring in childhood. In essence, the first two are 'developmental', the other categories being 'acquired' in later life (Smith and Hu 1999). Developmental defects and those that occur as a result of disc degeneration are the categories that are most likely to be seen clinically, and so are considered here. The incidence of spondylolisthesis due to trauma or bone disease is unknown, but clinically should be considered a 'red flag' condition unsuitable for mechanical therapy.

Isthmic spondylolisthesis is further categorised as follows (Stinson 1993):

- fatigue fracture of the pars interarticularis with slippage
- an intact, but elongated, pars interarticularis
- acute fracture.

The degree of the slip has been graded according to two methods. The Meyerding classification divides the top of the sacrum into four equal sections. A slip in the first quarter is grade I, a slip in the last quarter is grade IV. A more accurate measurement can be given in percentage terms (Hensinger 1989).

The majority of individuals with spondylolisthesis have low-grade slippages. In a population survey, which found sixty-nine cases in a sample of 1,147 subjects (6% prevalence), the degree of slip was grade I in 79%, grade II in 20% and grade III in 1% (Osterman *et al.* 1993). In over 300 patients, nearly 90% were classified as grade 0, I or II (Danielson *et al.* 1991).

Relevance to symptoms

Despite the alarming nature of the abnormality, spondylolisthesis is not inevitably a source of back pain. Van Tulder *et al.* (1997c) conducted a systematic review of radiographic findings and back pain. Six studies investigated spondylolysis or spondylolisthesis, of which five concluded there was no association between these findings and back pain. One study of middle-aged patients found the association between spondylolisthesis and back pain to be weak and only present in women (Virta and Ronnemaa 1993). *"Roughly half of patients with this finding do not have back pain, so finding may be unrelated"* to symptoms (Roland and van Tulder 1998).

The prevalence rate of isthmic spondylolysis in the general adult population and in the back pain population is generally about the same, around 6%. If such defects were a common source of back pain, these findings would be much more common in the latter (Porter and Hibbert 1984; Micheli and Yancey 1996; Macnab and McCulloch 1990).

The role of disc pathology as a confounding factor in the presence of spondylolisthesis has been demonstrated in several studies (Macnab and McCulloch 1990; Henson *et al.* 1987; Deutman *et al.* 1995). These are reminders that the finding of spondylolisthesis may be irrelevant to symptoms, and that a mechanical evaluation should always be attempted.

Prevalence

There is no evidence that the defect exists at birth; it most commonly appears between the ages of 5 and 7, with a subsequent increase during adolescence, after which prevalence rates remain relatively static during adulthood (Ciullo and Jackson 1985; Johnson 1993). Defects of the pars interarticularis are strongly associated with spina bifida occulta (Fredrickson *et al.* 1984). Isthmic spondylolysis and spondylolisthesis occur predominantly at L5 – S1.

In 500 school children, the incidence of spondylolysis was 1.8% and isthmic spondylolisthesis 2.6%; this increased to 2% and 4% respectively in young adulthood (Fredrickson *et al.* 1984). Osterman *et al.* (1993) reported an incidence of 6% of isthmic spondylolisthesis in a random population survey of adults. Macnab and McCulloch (1990) found the incidence of spondylolisthesis in nearly a thousand patients to be 7.6%, but in those under 25 it was 19%, in those between 26 and 39 it was 7.6% and in those over 40 it was 5.2%.

They concluded that in the younger patient it was more likely that the defect was the cause of their symptoms.

Numerous reports have suggested that the prevalence rate is greater, sometimes up to 50%, in the young athletic population (Jackson *et al.* 1976; Micheli and Wood 1995; Johnson 1993; Morita *et al.* 1995; Jackson 1979; Foster *et al.* 1989; Hardcastle *et al.* 1992; Hollenberg *et al.* 2002). Association suggests there may be a causal relationship between some sports and symptomatic spondylolysis. However, an awareness of risk in this group should be tempered by several factors: the high prevalence of back pain in all adolescents, the uncertain nature of sport as a risk factor for back pain and the biased study designs that have been used to look at this question.

Backache is commonly reported by school children and rises linearly during teenage years (Duggleby and Kumar 1997; Leboeuf-Yde and Kyvik 1998; Taimela *et al.* 1997; Burton *et al.* 1996). According to one study, the one-year period prevalence of back pain is about 10% in 12-year-olds, rising to over 40% in 20-year-olds (Leboeuf-Yde and Kyvik 1998). By early adulthood, the high prevalence rates of back symptoms are already well established, after which the steep increase flattens out (Leboeuf-Yde and Kyvik 1998; Burton *et al.* 1996).

In fact, both physical inactivity and sporting activity have been associated with adolescent back pain (Burton *et al.* 1996; Taimela *et al.* 1997; Prendeville and Dockrell 1998). Participation in sport is not clearly a risk factor for juvenile non-specific back pain, while hours of television watching has been significantly associated with back pain (Duggleby and Kumar 1997).

Most studies in sporting groups have been conducted in limited populations in which the diagnosis has been sought – such a study design may produce a biased sample. In a population study of over 3,000 élite, adult Spanish athletes, the general prevalence of spondylolysis was 8%, although certain sports demonstrated much higher rates (Soler and Calderon 2000). This would suggest that sport itself is not a risk factor in adults, but that *certain* sports may be more associated with the defect.

In adolescents, only infrequently is spondylolysis or spondylolisthesis the cause of back pain, but, especially in athletes, this diagnosis should be considered. Micheli and Wood (1995) compared the final diagnosis

after investigations in 100 randomly selected adolescent patients with back pain from a sports medicine clinic and 100 randomly selected adult patients with acute back pain. Average age in the two groups were 16 and 32. A stress fracture of the pars interarticularis was found in 47% of the adolescents, but only 5% of the adults. The authors recommend that the index of suspicion should be raised if there has been a history of repetitive hyperextension training, such as gymnastics, cricket or baseball, and pain is provoked on hyperextension.

Aetiology

Isthmic spondylolysis does not exist at birth. It is acquired during growth caused by a stress fracture of the pars interarticularis. Its acquisition is thought to be related to weight-bearing (Rosenberg *et al.* 1981).

Cadaveric experiments have induced fractures of the neural arch with repetitive cyclical loading, especially implicating extension forces (Cyron *et al.* 1976). However, mechanical fatigue of the pars is possible during any strenuous activity that generates sufficient force and number of cycles, especially in young people, since their intervertebral discs are more elastic and their neural arch may not be completely ossified (Cyron and Hutton 1978). It is thought that the defect is a fatigue fracture due to repeated minor trauma or stress rather than the result of one traumatic incident (Wiltse *et al.* 1975).

Besides mechanical factors, a familial tendency also exists for the development of pars interarticularis defects (Wiltse *et al.* 1975). Prevalence of 33% has been reported among those with a family history of spondylolysis (Johnson 1993).

Unlike other stress fractures, defects of the pars interarticularis frequently persist and fail to heal (Wiltse *et al.* 1975). A possible cause for this persistence is the formation of a pseudo-arthrosis at the site of the defect because of communication with adjacent zygapophyseal joints. Synovial cells and tissue and loose fibrous tissue similar to a joint capsule have been commonly found at these sites (Shipley and Beukes 1998). Furthermore, neural elements have been identified within the pars defect and in the 'ligament' associated with it, and thus it is a feasible source of back pain in some (Schneiderman *et al.* 1995; Eisenstein *et al.* 1994)

Progression and natural history

Not all spondylolysis progresses to spondylolisthesis. Progression of the slip occurs most commonly in a short period and during the adolescent growth spurt between eight and fourteen years of age, after which it tends to remain stable (Comstock *et al.* 1994; Lonstein 1999; Fredrickson *et al.* 1984). During the growth period, a stress fracture or slippage triggered by excessive exertion may become symptomatic (Hensinger 1989; Micheli and Yancey 1996). Progression is said to be rare once individuals reach adulthood (Danielson *et al.* 1991; Fredrickson *et al.* 1984), but may occur. This is more likely in the case of a spondylolisthesis than a spondylolysis (Ohmori *et al.* 1995). Progression of the slip is not prevented by surgical intervention (Seitsalo *et al.* 1991).

Progression of isthmic spondylolisthesis during adulthood has been reported and is said to be a possibility in about 20% of individuals with this finding (Floman 2000). Thus, an incidental and irrelevant finding can become a source of symptoms; the average age in a set of eighteen patients was forty-four. The individuals had incapacitating low back and leg pain, with most reporting radicular pain due to local spinal stenosis brought about by the narrowing and the increased slip (Floman 2000).

Degenerative spondylolisthesis occurs most frequently at L4 – L5, in those over fifty, and is more common in women, especially those who have had multiple pregnancies (Grobler *et al.* 1994; Herkowitz 1995; Sanderson and Fraser 1996). Vertebral displacement with an intact neural arch can critically narrow a small spinal canal (Porter 1993). Clinical findings are thus those of spinal stenosis (see section on spinal stenosis) from other degenerative causes – leg pain brought on by walking, relieved by flexion, low prevalence of neurological signs and symptoms and restricted range of extension (Herkowitz 1995). A long history of back pain is usual and radiographs should display considerable degenerative changes.

Clinical presentation

As has been stressed before, both pars fractures and spondylolisthesis can be asymptomatic and incidental findings in the normal population, or an individual can have these abnormalities as well as unrelated back pain (van Tulder *et al.* 1997c).

In patients with a finding of spondylolysis the main symptom is back pain, with or without radiation into the thigh (Porter and Hibbert 1984). The pain is localised around L5; patients are said to be able to point to the site of pain (Ciullo and Jackson 1985; Johnson 1993). In patients with a symptomatic spondylolisthesis, back and radicular pain may be present; neurological signs and symptoms are also found less commonly (Frennered *et al.* 1991; Seitsalo 1990; Seitsalo *et al.* 1990; Boxall *et al.* 1979; Kaneda *et al.* 1985).

The adolescent group should be assessed with a greater index of suspicion concerning this diagnosis, especially those involved in vigorous sport. It is suggested that a number of different sports are risk factors for developing spondylolysis (Duggleby and Kumar 1997). Those that involve repetitive hyperextension may involve the greatest risk, such as gymnastics, baseball and bowling in cricket. Trauma is not often involved and in many instances symptoms have an insidious onset, but may coincide with the adolescent growth spurt (Micheli and Yancey 1996).

In some individuals the degree and angle of slippage increases when they move from lying to standing (Boxall *et al.* 1979; Lowe *et al.* 1976), thus sustained weight-bearing is likely to be a cause of aggravation and recumbency a cause of relief. Prolonged standing, walking or sitting may bring on symptoms, which are relieved by lying. Symptoms may be initiated or aggravated by strenuous activity in the adolescent group, such as sporting participation, and decreased by rest.

Physical findings are likely to vary depending on the grade or stage of the defect. Very often there is full range of movement. Extension of the spine is often painful and exacerbates or produces the patient's symptoms (Balderston and Bradford 1985; Micheli and Yancey 1996; Hardcastle 1993; Hollenberg *et al.* 2002; Micheli and Wood 1995). This will be a consistent and unchanging response, which does not get easier, as might occur in derangement. Both repeated flexion and extension might worsen symptoms (Payne and Oglive 1996).

In more extreme cases, signs may be more pronounced. Distortion of the pelvis and trunk, tight hamstrings with a waddling gait, a prominent step-off at the level of the slippage, and folds and protrusion in the abdominal wall have been reported (Balderston and Bradford 1985; Hensinger 1989; Harris and Weinstein 1987).

McKenzie (1981) recommends a simple clinical test to help determine if a spondylolisthesis is responsible for the presenting symptoms, as it often reduces or abolishes pain in the presence of this condition. With the patient standing, place one hand across their sacrum and the other firmly against their abdomen. With further compression from both hands, pain arising from spondylolisthesis is markedly reduced or abolished. On sudden release of pressure, which must be maximal, there may be a sharp return of pain of short duration. The test should be repeated three times, and if pain is experienced on release of pressure each time, it is likely that pain is from the spondylolisthesis. Pain from derangement is usually worsened, and that from other mechanical syndromes unaffected. Another provocative manoeuvre is the one-leg lumbar hyperextension test, in which the patient stands on the ipsilateral leg and bends backwards in an attempt to reproduce their familiar symptoms (Ciullo and Jackson 1985). Neither test has been formally evaluated.

A comparison has been made of 111 adult patients with isthmic spondylolisthesis with at least one year of back pain and/or sciatica to thirty-nine chronic patients prior to surgery (Moller *et al.* 2000). Most of the slippages were grade I or II; symptoms were mostly constant, worsened by sitting and walking, woke patients at night and were associated with moderately restricted function. Sciatica was present in 70%, but positive signs were unusual, with tight hamstrings, positive straight leg raising and sensory disturbance present in 20% or less. The profile of functional disturbance, aggravating factors, and signs and symptoms were strikingly similar for both spondylolisthesis and non-specific chronic back pain groups. *This study shows that in adults at least there is no clear clinical presentation that distinguishes back pain patients with spondylolisthesis from those with non-specific back pain.*

Diagnosis

Ultimately, to make the diagnosis of spondylolysis or spondylolisthesis, imaging studies are required. Radiographs can be insensitive tools in the detection of the defect (Congeni *et al.* 1997). If the defect is large it may be visible on ordinary lumbar radiographs, while a spondylolysis or minimal slippage may only be revealed on oblique radiography (Hensinger 1989). Different radiographic views have different sensitivity to the lesion, with lateral and oblique views picking up over 75% and anterior-posterior views detecting 50% or less (Amato *et al.* 1984).

Various specialist imaging techniques are also used. Computed tomography scans with reverse gantry angle technique and scintigraphy or single photon emission computed tomography (SPECT) are more sensitive than radiographs (Saifuddin *et al.* 1998; Harvey *et al.* 1998; Bodner *et al.* 1988; Bellah *et al.* 1991; Collier *et al.* 1985). SPECT may be particularly useful in the identification of early lesions, when fractures are still metabolically active and x-rays may be normal (Lowe *et al.* 1984; Harvey *et al.* 1998). Later, when the lesion is well established, radiography is more specific (Papanicolaou *et al.* 1985).

Identification of a lyses defect by imaging, *let a*lone any attempt to establish a causal link with the patient's pain through such means, clearly requires sophisticated techniques in the hands of a specialist.

Management

The literature is dominated by surgical interventions. Comparisons between surgical and conservative treatment of spondylolisthesis are rare; a convincing case for the superiority of surgery, even in more severe slippages, has not been previously made (Seitsalo *et al.* 1991; Seitsalo 1990; Harris and Weinstein 1987). However, in the first randomised trial comparing conservative and surgical treatments ever to be done, and including a two-year follow-up, the superiority of the surgical treatment was clear (Moller and Hedlund 2000). While function improved by 19% and pain by 26% in the surgery group, the comparative changes in the conservative treatment group were 0% and 9%. The exercise programme consisted of back and abdominal strength training conducted over at least one year, two or three times a week.

Conservative treatment of symptomatic spondylolisthesis does not favour any particular approach; rather, the literature consists of a few contradictory interventions. In one trial involving patients with a radiographic diagnosis of spondylolysis or spondylolisthesis, mostly with a minimal or absent slip, normal management was compared to specific stabilising exercises. Only the intervention group showed a statistically significant reduction in pain and disability, which was maintained at thirty months (O'Sullivan *et al.* 1997).

Both flexion and extension exercises have been used in patients with spondylolisthesis, and both have been found superior. One trial compared the effect of abdominal or back strengthening exercises,

although details of the duration and number of sessions were not given. The overall recovery rate in the active extension group at three months was 6% and at three years was 0%, compared to 58% and 62% in the active flexion group (Sinaki *et al.* 1989). The authors state their belief that flexion exercises are preferred and that extension exercise should be avoided. This is based on the putative role of lumbar extension in causing fractures of the pars interarticularis.

Although this opinion is common, an extension programme has also been shown to be beneficial. A group of patients classified by their translational findings as spondylolisthesis, retrolisthesis or no defect were randomised to extension, flexion or control treatments. The exercise groups performed exercises and used a lumbar brace to maintain the appropriate posture. At one-month follow-up only the extension group patients showed a significant improvement across time, and this occurred in all translation subgroups (Spratt *et al.* 1993). The authors suggest that the favourable response to extension treatment, despite spondylolisthesis or retrolisthesis, may be because these findings are secondary to underlying disc pathology. The opinion that extension exercises should be contraindicated in the presence of a spondylolisthesis is not borne out by this study.

Most fatigue fractures mend with time, and spondylolysis are unusual in that this normal healing process does not always occur. However, healing can happen, and this is more likely when the fracture is still at a relatively acute stage (Hardcastle 1993). When 185 adolescents with spondylolysis were classified into early, progressive and late stage defects, according to computer tomography (CT) findings, their response to conservative management was significantly different. While 73% of those in the early stage achieved bony union according to radiography and/or CT three to six months later, only 38% of those in the progressive stage and 0% of those in the final stage did so (Morita *et al.* 1995). These findings make clear the importance of early detection of the fracture to ensure appropriate management, which in this case entailed absence from sport and use of a lumbar corset for three to six months.

In the young sporting population, reduction or cessation of the aggravating activities and stretching and strengthening programmes are recommended, with a gradual return to sport as symptoms allow (Johnson 1993). Some recommend the use of a brace to facilitate healing, although this is not universally required. While the results

of a series of sixty-seven patients were good or excellent in 78% fitted with an anti-lordotic Boston brace, the intrusiveness of the intervention was extreme. It was to be worn twenty-three hours out of twenty-four for six months, and then reduced over a further six months (Steiner and Micheli 1985).

Summary

The finding of spondylolysis or spondylolisthesis on a radiographic report may be quite unrelated to a patient's symptoms, with even quite severe slippages present in individuals without back pain. A full mechanical evaluation may be safely conducted, and many such patients respond in a normal mechanical fashion. Atypical responses may imply that the defect has significance. Furthermore, certain items during history-taking and physical examination may alert the clinician to the possibility of this diagnosis.

Symptomatic isthmic spondylolysis should be suspected in adolescent sporting participants with a gradual onset of low back pain that is sports-related. Those involved in repetitive flexion/extension and/or ipsilateral side bending or rotation movements may be at particular risk. Extension is likely to increase symptoms, although not necessarily worsen them, and tight hamstrings may be present. This is a stress fracture, and referral to a sports physician is most appropriate; relative rest is the best management and mechanical therapy is contraindicated. However, only a minority of back pain in adolescents is due to spondylolysis. Mostly they present with symptoms from either postural or derangement syndromes.

Instability

Lumbar segmental instabilities have been categorised by cause as being due to fractures, infections, neoplasms, spondylolisthesis or degeneration. Degenerative lumbar instabilities are either primary or secondary, with the latter resulting from surgical destruction of some kind (Bogduk 1997). Primary instabilities are defined by their direction; for instance, translational instability, characterised by excessive anterior translation of one vertebra on another during flexion.

Primary instability has been variously defined as loss of motion segment stiffness, an increase in mobility or an increase of segmental rotations or translations (Richardson *et al.* 1999). Definite instability is indicated by more than 4 – 5mm of translation on a flexion–extension

radiograph (Fordyce *et al.* 1995) and is traditionally associated with degenerative disc disease. More recently, clinical instability has been defined as *"a significant decrease in the capacity of the stabilizing system of the spine to maintain the intervertebral neutral zones within physiological limits which results in pain and disability"* (Panjabi, in Richardson *et al.* 1999, p. 13). The neutral zone is the area where movement of a motion segment occurs with minimal resistance from ligamentous structures, which offer restraint in the elastic zone to limit end-range movement. The stabilising system is comprised of three components: the passive system of the spinal column, the active system of the muscles and a neural control system. Back pain is said to occur when there is a deficit in any of the three components, resulting in abnormally large segmental motions that cause compression or stretch on pain-sensitive structures (Richardson *et al.* 1999).

Despite much discussion and considerable theoretical work that has elaborated the concept of primary instability, there are still numerous problems concerning definition, criteria, relationship to a pain state and clinical identification (Porter 1993; Spratt *et al.* 1993; Dupuis *et al.* 1985). Most definitions of instability involve increased or abnormal segmental motion. Some studies have shown large amounts of translation are more common in those with back pain compared to the general population (Spratt *et al.* 1993; Lehmann and Brand 1983; Sihvonen *et al.* 1997). However, 4 mm and more of anterior translation has also been found in 10% to 20% of asymptomatic populations (Woody *et al.* 1983; Hayes *et al.* 1989). Only one study has demonstrated a link between the amount of translation and the degree of symptoms (Friberg 1987). In fact, all these studies have involved individuals with a diagnosis of spondylolisthesis or retrolisthesis. Various methods have been used to try to expose abnormal segmental motion during dynamic radiographic studies: centrode patterns, dynamic traction-compression and flexion-extension radiography.

All these techniques have flaws. Centrode patterns, the locus of successive positions of instantaneous centres of rotation, have been studied in vitro and in vivo (Gertzbein *et al.* 1984, 1985; Pearcy and Bogduk 1988). The group that developed centrode patterns found them to be associated with a high degree of error and inaccuracy, and they subsequently abandoned the technique as a clinical investigative tool (Weiler *et al.* 1990). A study using dynamic traction–compression radiography found that the severity of symptoms related to the amount of translation at the level of the spondylolisthesis

(Friberg 1987). Mean movement in the asymptomatic, moderate and severe pain groups were 0.7mm, 5.2mm and 7.5mm respectively. However, the technique was found to have a poor correlation with the results of dynamic flexion-extension radiography, which is the traditional method of diagnosis. By traction–compression, 8% of a cohort of patients were diagnosed with instability, compared to 96% by flexion–extension radiography (Pitkanen *et al.* 1997).

Flexion–extension radiography was the original method used to reveal instability. The technique was unable to expose abnormal or erratic motion during movement, but only at end-range (Stokes and Frymoyer 1987). There can be inconsistencies and inaccuracies in flexion-extension radiography, errors in classification and lack of definition about what is normal and what is pathological (Shaffer *et al.* 1990; Spratt *et al.* 1993). As a consequence of these failings, there is no gold standard method of diagnosing or measuring instability, nor is it a morphological abnormality that is correlated with back pain.

"Difficulties lie, particularly in vivo, in gaining a definition of instability that would indicate a relationship to a pain state and that would generate a method of quantification to demonstrate its presence. As a consequence, there is currently neither a gold-standard definition of clinical instability nor a gold-standard measure" (Richardson *et al.* 1999, p. 12).

"Various clinical criteria have been proclaimed as indicative or diagnostic of lumbar instability. At best, these constitute fancy. To be valid, clinical signs have to be validated against a criterion standard. The only available criterion standard for instability is offered by radiographic signs, but the radiographic signs of instability are themselves beset with difficulties. Consequently, no studies have yet validated any of the proclaimed clinical signs of instability" (Bogduk 1997, p. 224).

Degenerated discs have been correlated with higher levels of instability factor, which is a combination of translation and angulation (Weiler *et al.* 1990), and with an increasing spread of axes of movement (Penning and Blickman 1980). It has been suggested that instability may need to be considered an irrelevant product of disc pathology rather than a distinct clinical syndrome (Spratt *et al.* 1993). This is supported by some studies that have found radiographic instability persisting after symptoms have resolved. Radiographic instability has been shown

both to improve spontaneously over time and to persist when symptoms have resolved (Sato and Kikuchi 1993; Lindgren *et al.* 1993).

The evidence does not prove that an excessive amount of translation at a lumbar segment is a source of symptoms, although it does suggest that there are serious difficulties in measuring this. If a gross abnormality such as a spondylolisthesis is not always directly related to symptoms, the role of lesser 'instabilities' in back pain awaits further elucidation. Furthermore, with the lack of 'gold standard' diagnosis, there are no clinical criteria that have been validated as being sensitive and specific in the recognition of this entity.

Mechanically inconclusive

There is a small group of patients whose symptoms are influenced by postures and movements, and yet who do not fit one of the three mechanical syndromes. Symptoms are affected by loading strategies, but in an unrecognisable or inconsistent pattern. This group does not display a mechanical presentation – range of movement is preserved, and there is no obstruction to movement. Pain may be constant or intermittent, and is frequently produced or increased at end-ranges. Repeated end-range movements in all planes may produce a worsening of symptoms, but no obstruction of extension or flexion by loading in the opposite direction. Thus, no directional preference is indicated.

There may be variations on a similar theme; for instance, catches of pain during movement, or initially there is a favourable response to repeated movement in one direction, which then becomes inconsistent or causes a worsening of symptoms if continued or if force progressions are included. The key to this mechanically inconclusive group, who nonetheless have symptoms that respond to loading strategies, is that a consistent directional preference cannot be found.

Criteria for mechanically inconclusive group are:

- symptoms affected by spinal movements
- no loading strategy consistently decreases, abolishes or centralises symptoms, nor increases or peripheralises symptoms
- inconsistent response to loading strategies.

This group sometimes responds to mid-range postures rather than end-range movements. Maintenance of posture correction, use of mid-range movements, especially slouch-overcorrect, avoidance of end-range postures and movements and interruption of painful positions may be helpful for this group.

Surgery

Lumbar disc herniation is one of the few clear occasions when surgery or other invasive treatment might be considered. Because many will improve if treated conservatively, early surgery should generally be avoided. The only specific indicators for early surgery are cauda equina syndrome and progressive or profound neurological deficit (Saal 1996). Otherwise if surgery is to be considered, certain strict criteria are necessary (see Table 5.1), as well as the failure of six weeks of attempted conservative therapy (see Table 5.4 for characteristic presentation of extrusions and sequestrations). Patients with these more severe disc herniations may do better with surgery than patients with protrusions (Hoffman *et al.* 1993).

Scheer *et al.* (1996) reviewed thirteen randomised controlled trials for sciatica and discogenic back pain, concentrating on the outcome of return to work. Chemonucleolysis, discectomy and epidural steroid injections were included in the review. For all interventions they found the evidence to be equivocal. In particular, they could not infer that surgery was better than conservative therapy in the long-term. Hoffman *et al.* (1993), in a literature synthesis, concluded that standard discectomy appears to offer better short-term outcomes than conservative treatment, but long-term outcomes are similar.

In a recent Cochrane review of surgery for lumbar disc prolapse, twenty-six randomised controlled trials were identified (Gibson *et al.* 1999). Meta-analyses showed that chemonucleolysis was clearly better than placebo, and discectomy was better than chemonucleolysis, and therefore discectomy is better than placebo. There was no difference in outcomes between microdiscectomy and standard discectomy, although both produced better results than percutaneous discectomy. Only one trial compared surgical with conservative treatment (Weber 1983). There were significant differences in favour of surgery at one year, but not at four or ten years. These reviewers concluded that there was considerable evidence for the clinical

effectiveness of discectomy for *carefully selected* patients with sciatica who fail to improve with conservative care. All reviews comment on the poor quality of design methodology and reporting.

The trial by Weber (1983) is a randomised comparison between conservative and surgical treatment of disc herniations; such comparisons are rare, and so it is given considerable importance. In fact, it suffers from certain design faults that limit its implications. Critical defects include the large number of crossovers, the inadequate sample size and insensitive outcome measures (Bessette *et al.* 1996). It was a prospective study in which eighty-seven patients with mild symptoms were treated conservatively, sixty-seven patients with severe symptoms underwent surgery and 126 patients with uncertain indications for surgery were randomised. All but five of the latter group were followed up at one, four and ten years. At one year 92% of the surgery group were satisfied, compared to 79% in the conservatively treated group. Seventeen patients allocated to conservative treatment were operated on, and one patient allocated to surgery refused the operation. At four and ten years in those patients who were located, satisfaction in those allocated and treated surgically was 86% and 93%; and in those allocated and treated conservatively, 90% and 92%. Only at one year were there significant differences favouring the surgical group.

In a non-randomised study with over 500 patients treated either surgically or conservatively, follow-up was performed at one year (Atlas *et al.* 1996b). Surgical patients tended to have more severe symptoms and few patients with severe symptoms were treated conservatively, but about half of each treatment group had symptoms that were categorised as moderate. For the predominant symptom, 71% of the surgery group and 43% of the non-surgery group reported definite improvement. Those undergoing surgery saw quicker and more dramatic improvement in symptoms.

Although it seems fairly clear that appropriately selected patients will make quicker improvements with surgery, many patients will have satisfactory outcomes with conservative treatment, especially those with mild or moderate symptoms. Some of the drawbacks of surgery should also be remembered. The long-term follow-up of some surgical series shows high levels of persisting or recurring symptoms, unsatisfactory outcomes, further operations and a deterioration of results over time (Loupasis *et al.* 1999). Four to seventeen years after

operation in a partial follow-up of over 500 patients, 70% complained of back pain, 45% of sciatica, 35% were still receiving some kind of treatment and 17% had undergone repeat operations (Dvorak *et al.* 1988). In another study with a minimum of ten years follow-up, 75% reported back pain and 56% leg pain (Yorimitsu *et al.* 2001). Hoffman *et al.* (1993) estimated that 5 – 15% of all operations lead to poor outcomes and further surgery.

Although certain clinical and morphological factors are significant in outcomes from lumbar discectomy, psychosocial and work-related factors can be as significant or more so (Schade *et al.* 1999). In this prospective study of forty-six patients, the size of herniation, nerve root compression, depression, occupational mental stress and support from the spouse were associated with post-surgical pain relief. However, only psychosocial factors were associated with return to work. Careful patient selection for surgery is clearly crucial.

Epidural steroid injection for sciatica

A less invasive medical intervention sometimes considered for sciatica is epidural steroid injection. Although there is limited evidence that this intervention may offer short-term pain relief, convincing proof of its therapeutic value is missing. In 1995 two systematic reviews of this intervention were published (Watts and Silagy 1995; Koes *et al.* 1995). Rather alarmingly, they came to different conclusions despite reviewing mostly the same studies. Ten papers were common to both, one extra paper was exclusive to one review and two additional papers were exclusive to the other review. According to Watts and Silagy (1995), epidural corticosteroid is effective in the management of lumbosacral radicular pain. However, the conclusion of Koes *et al.* (1995) was that the best studies showed inconsistent results, and the efficacy of steroid injections is as yet unproven. Given that such reviews are supposed to be based on a rigorous and objective analysis of the evidence, their conflicting conclusions attest to the qualitative judgements that may occur in this process (Hopayian and Mugford 1999).

Since then a further systematic review into injection therapy in general has been published (Nelemans *et al.* 2001). This included twenty-one papers, including one (Carette *et al.* 1997) published since the previous reviews. They considered studies as being either explanatory or pragmatic, where comparison with a placebo injection was termed an explanatory trial. They located four explanatory trials into the efficacy of epidural injections for sciatica. Although all four reported greater

pain relief short-term in the experimental group, this was not statistically significant. More than six weeks after the intervention, there was no difference. Six pragmatic trials looked at the effects of epidural injections in a range of conditions, including sciatica. Four showed a non-significant positive effect short-term, and neither of the two that reported on long-term pain relief found any significant difference. Their overall conclusion was that convincing evidence about the efficacy of injection therapy is lacking (Nelemans *et al.* 2001).

Recent studies have tended to confirm the lack of efficacy of epidural corticosteroid injections. The placebo controlled study by Carette *et al.* (1997) is a recent high-quality paper examining the effects of methylprednisolone acetate compared to saline in 158 patients with sciatica due to a disc herniation. Improvements in function were better, but not significantly in the active treatment group at three weeks, and just significantly better regarding leg pain at six weeks. At three months there were no differences between groups, and at one year the incidence of surgery was the same in both groups. They conclude epidural injection may provide short-term pain relief only.

This same short-term result was produced in a similar recent study (Karppinen *et al.* 2001a) in which leg pain was significantly better in the active treatment group at two weeks. However, there later appeared to be a 'rebound' effect, with back pain less in the placebo group at three months and leg pain less at six months. Use of steroid did not obviate the need for surgery, rates being similar in both groups. However, sub-group analysis suggested that for contained herniations, the steroid injection produced significantly better results than for extrusions (Karppinen *et al.* 2001b). Buchner *et al.* (2000) found no significant difference in pain or function in conservatively treated groups, one of which received steroid injections, at two or six weeks and at six months.

Only one recent study, which used fluoroscopic imaging to ensure the steroid injection was delivered precisely to its target site, has shown results that clearly favour this intervention (Vad *et al.* 2002). However, in the study patients were not blinded to the intervention and a true placebo comparison was not used.

Post-surgical status

Those with symptoms may be those who have had successful surgery, but in whom pain has re-occurred, or else those who are surgical failures in whom the original symptoms may be reduced, but still remaining. Reoccurrence of symptoms may be due to a second disc herniation or perineural fibrosis (Spitzer *et al.* 1987).

Mechanical evaluation should always be offered to post-surgical patients. If symptoms have re-occurred, it is important to distinguish whether the cause is derangement or post-surgical adhesions – the latter presents as a flexion dysfunction or an adherent nerve root. These presentations should be treated in the normal manner described in the relevant chapters.

Early active rehabilitation has an important role post-surgery. The evidence suggests better outcomes can be gained if patients are put through a dynamic exercise programme after surgery than with surgery alone. Early active training involving extension, flexion and active straight leg raising instigated immediately post-surgery resulted in significantly less leg pain for at least three months compared to a less active control group, although at one year results were about the same (Kjellby-Wendt and Styf 1998). Dynamic exercise programmes have also been instigated at about one month following surgery, again producing better outcomes than a lighter exercise comparison treatment, especially at six months (Manniche *et al.* 1993a; Danielsen *et al.* 2000). In another study, six weeks after microdiscectomy patients were entered into an exercise or control group and followed up at one year (Dolan *et al.* 2000). The exercises consisted of a four-week programme of general mobility and strengthening exercises. The exercise group showed further improvements in pain and function that were maintained at one year, whereas the control group made no further improvements except those made by surgery. The post-surgical programme is clearly important in an early restoration of confidence and function.

One aspect of post-surgical rehabilitation that has been shown *not* to be beneficial is neural mobilisation, using initially passive and then active movements, such as straight leg raise and neck flexion (Scrimshaw and Maher 2001). The neural mobilisation group had worse outcomes, although the differences were not statistically significant. Both groups performed active strengthening exercises. "*This randomized controlled*

trial demonstrates not only that neural mobilization after spinal surgery is of no benefit to patients but it suggests that this physical regimen may in fact be harmful" (Fraser 2001).

The value of an active exercise approach for those more than six months after surgery is also apparent (Manniche *et al.* 1993b; Timm 1994). Following a twelve-week course of dynamic extension exercises, there was a significant improvement of pain in 70% of those who completed the programme (Manniche *et al.* 1993). Timm (1994) compared passive modalities, manipulative therapy and low- and high-tech exercises for chronic back pain following an L5 laminectomy, with the low-tech group using extension and stabilisation exercises. Both exercise groups had significant and lasting improvements in mobility and function, and reduced disability. The passive treatment group was no better than a no-treatment control group, and the manipulative therapy group also produced minimal changes.

The above studies make clear that outcomes from surgical procedures can be significantly improved with the application of a dynamic exercise programme during the rehabilitation period.

Chronic pain

Chronic pain has traditionally been defined by pain duration; for instance, symptoms that have persisted for more than three to six months. However, timescale alone is now generally considered to be an inadequate definition for chronic pain. Other factors are considered important in the chronic pain experience. Psychosocial and behavioural factors complicate the clinical problem, and pain is disassociated from tissue damage. Patients may experience widespread pains, and the problem is more likely to prove difficult to treat (Spitzer *et al.* 1987; Adams 1997).

From the review of the epidemiology of back pain in Chapter 1, it is apparent that many individuals have persistent symptoms, but that in this group severity and disability are often minimal. Waddell (1998) estimates that while 6 – 10% of all adults may have persistent or recurrent back pain, most lead relatively normal lives, are working, do not seek health care and have little disability. Categorisation of chronic patients should not be determined simply by pain duration. Of those who have persistent symptoms, many demonstrate mechanical responses, although sometimes response may be slower.

The length of time that symptoms have been present should never be seen as a deciding factor in the application of therapy. Many of those with chronic symptoms can benefit from a mechanical assessment. *Patients who have long-standing low back pain should not be denied a mechanical assessment*. Many patients with long-term problems display directional preference for certain repeated movements (Donelson *et al.* 1990, 1991, 1997; Long 1995; Rath and Rath 1996). Not all will resolve their problems, but many patients with chronic symptoms improve their ability to manage their condition. Because of the length of time the problem has been present, a slower and more ambivalent response may occur. However, also within this group it should be recognised that alternative approaches may be appropriate.

Within the group with chronic pain are also found those who demonstrate multiple 'yellow flags', inappropriate pain behaviours, widespread pain and aggravation of symptoms with all activity. Just 1 – 2% of the adult population has chronic, intractable pain with major disability. They have been off work for months or years, and they absorb considerable health care resources (Waddell 1998). Those most severely disabled by pain are likely to exhibit some or all of the features listed (Table 13.11); those who are moderately distressed may only show one or two features.

Table 13.11 Possible characteristics of patients with chronic intractable pain

- persistent pain
- interruption of work, social and other activities of daily living
- depressed
- distressed
- unhelpful beliefs
- multiple health care interventions
- multiple treatment failures
- anger.

Source: Waddell 1998

Symptoms may become complicated and persist due to non-mechanical problems. These are considered in more detail in Chapter 3, but in brief these consist of psychosocial or neurophysiological factors that act as barriers to resolution and obscure a mechanical problem. Psychosocial and cognitive factors are closely related to the

development of chronic back disability. Depression, anxiety, passive coping and attitudes about pain are associated with chronic pain and disability. Catastrophising, hyper-vigilance about symptoms and fear–avoidance behaviour are some of the attitudes and beliefs that have been highlighted as being significant in this context (Linton 2000).

The timescale when these factors may become active modulators of patients' pain experience may be in the first few weeks (Philips and Grant 1991; Burton *et al.* 1995; Fritz *et al.* 2001). This further discounts the significance of pain duration for categorisation. It is also highlights the prominence of psychosocial factors at an early time in the natural history of back pain. It suggests that at no time, whether the patient is in the acute or chronic stage, can we afford to ignore these potential modulators of the pain experience.

Furthermore, persistent peripheral nociceptive input can induce changes in the central nervous system (Woolf 1991; Melzack and Wall 1988). This may lead to the sensitisation of neurones in the dorsal horn – a state characterised by reduced thresholds and increased responses to afferent input, such that normal mechanical stimuli is interpreted as pain. In this situation pain, aching and tenderness are likely to be widespread, and most normal activity is perceived as painful.

Thus a chronic pain state is not simply related to the time that symptoms have been present. These are patients in whom a mechanical response to loading strategies is obscured by non-mechanical factors, which may be psychosocial or neurophysiological in origin. Symptoms are likely to have been present for a prolonged period, but this may not always be so. Interruption of their normal lifestyle has usually occurred. Multiple or widespread pain sites are common. All activity increases symptoms, at least initially. There is no obvious directional preference, nor clear mechanical response; again, at least not initially. Often these patients display exaggerated pain behaviours and vocalisation. They nearly always hold mistaken beliefs and attitudes about pain and movement, and in particular are fearful of movement. Depression, anxiety and distress are all commonly found. They may display multiple Waddell's non-organic signs and symptoms, but other features may be more revealing.

Table 13.12 Key factors in identification of chronic pain patients

- no lasting change in pain location or pain intensity in response to therapeutic loading strategies
- persistent widespread symptoms
- all activity increases symptoms
- exaggerated pain behaviour
- mistaken beliefs and attitudes about pain and movement.

Waddell's non-organic signs and symptoms

For a further review of this topic, see Scalzitti (1997). Waddell *et al.* (1980) developed a collection of eight signs that are said to be indicative of non-organic pathology. Individual signs are not considered significant, and a cut-off point of three or more is recommended. In the original study, three positive signs were found in 33% and 50% of chronic problem backs, 12% of acute backs and 0% of normal subjects.

Table 13.13 Inappropriate signs

- superficial tenderness
- non-anatomical tenderness
- back pain on axial loading
- back pain on simulated rotation
- distraction test, such as straight leg raise
- regional, non-dermatomal weakness
- regional, non-dermatomal sensory disturbance
- over-reaction to examination / overt pain behaviour.

Waddell *et al.* (1984) have also described a series of seven inappropriate symptoms, in which patients offer descriptions that do not fit with normal clinical experience, again with the inference that they are related to psychological rather than physical features. Isolated symptoms are not relevant, and as some can occur in serious spinal pathology, they are only appropriate to non-specific back pain in which specific pathology has been excluded. Such symptoms were reported by an average of 36% of problem patients, 18% of referrals from primary care and 7% of normal subjects.

Table 13.14 Inappropriate symptoms

- tailbone pain
- whole leg pain
- whole leg numbness
- whole leg giving way
- no pain-free spells
- intolerance of treatments
- self-admission to hospital emergency department with back pain.

The aim of these behavioural signs and symptoms is to try to distinguish between physical and non-organic complaints, to assist in the identification of patients in whom there was a behavioural component to disability, and to prevent the administration of inappropriate treatment. Their presence does not indicate faking or simulated incapacity; rather, the authors see them as a form of communication between the patient and the clinician indicating distress and the need for more detailed psychological assessment (Main and Waddell 1998).

In the original study, agreement over the detection of non-organic signs was high (86%) between two examiners (Waddell *et al.* 1980). In a later report, Kappa values were given for inappropriate symptoms and signs of between 0.55 and 0.71 (Waddell *et al.* 1982). McCombe *et al.* (1989) found poor reliability in detecting individual signs, with a mean Kappa score of 0.15. This finding should further warn against the importance of individual non-organic signs.

Furthermore, as indicators of distress the signs may not be stable over time, but reflect the patient's attitude towards their back problem and their treatment. Clinically it has been found that whereas on initial assessment signs may be positive, several days later they no longer are. This may be a display of patient's initial distress, which is reduced a few days later when they have gained confidence in the clinician and the way that they are being managed. Werneke *et al.* (1993) found the presence of the signs reflected the success or failure of a rehabilitation programme to return patients to work. There was a significant reduction in their presence in those who returned to work, but no change in those who did not.

There is conflicting evidence about the clinical utility of non-organic signs to predict outcomes. Studies have found them useful in predicting poorer results in lumbar spinal surgery (Dzioba and Doxey

1984; Waddell *et al.* 1986) and correlated signs with poorer treatment outcomes in conservative management (Lehmann *et al.* 1983; Karas *et al.* 1997). In acute back pain patients, the presence of signs has been associated with poorer return to work, more treatment and the use of more imaging technology (Gaines and Hegmann 1999). Other studies have found no correlation between signs and return to work, health care use and later outcomes in acute and chronic patients (Fritz *et al.* 2000b; Polatin *et al.* 1997; Bradish *et al.* 1988; Werneke *et al.* 1993).

These signs and symptoms clearly need to be used with a certain amount of caution, and used in the context of the whole clinical picture, but may be useful on occasions when mechanical response is unclear. Other 'yellow flag' indicators are likely to be present; for instance, the patient displaying exaggerated pain behaviour and mistaken beliefs and attitudes about pain, activity and/or work. To be of significance, at least three signs should be present when tested, with the presence of multiple signs and symptoms being more compelling evidence of inappropriate behaviour. This does not indicate that the patient is malingering or in some way 'faking it'; rather, they have an inappropriate behavioural response to back pain, as well as possibly as a physical problem, and may need further psychological assessment. Such signs may vanish if the patient's anxieties and distress is moderated and their back pain is managed in a way that is satisfactory to them. However, attempting to treat their physical problem may not be successful if the behavioural problem is not also addressed. A multi-disciplinary pain management or cognitive behavioural functional rehabilitation programme may be more appropriate in some patients.

Management of chronic pain patients

So-called 'yellow flags' are not, however, a diagnostic category, but rather they are a confounding factor that may be a barrier to recovery. If these psychosocial concerns can be dealt with, then treatment may proceed straightforwardly. If they are not addressed, then these factors often prevent successful management.

This may be a difficult group to treat, but it is apparent that the emphasis should be on improved function, coping and self-management rather than resolution of pain. Foremost in the clinician's mind when assessing the patient should be the importance of focusing on functional changes rather than highlighting the effects of repeated

movements on pain. The confounding effect that non-mechanical factors can have on the efficacy of purely mechanical interventions should be recognised.

For chronic musculoskeletal problems, it is recommended that a cognitive-behavioural framework be used for interaction with the patient (Turner 1996). This requires:

- awareness of and enquiries into psychological 'yellow flags' that suggest inappropriate pain behaviours and beliefs about pain and can be risk factors for the development of persistent pain
- appropriate information provision – the importance of the self-management principle for ongoing health problems, activity for musculoskeletal conditions and reassurance that pain on movement does not mean an exacerbation of the problem
- encouragement of a graduated, systematic resumption of activities.

Gifford (1998b) offers a useful approach to this small, but difficult patient group. *"On-going pain states are best explained to patients in terms of an altered sensitivity state as a result of altered information processing throughout the system, and not solely a result of damaged and degenerating tissues. This helps patients accept the notion that hurt does not necessarily equate with harm – which leads on to the positive message that carefully graded increases in physical activity mean stronger and healthier tissues. By contrast, continued focus on a tissue as the pain source reinforces fear of movement and activity, the need to be constantly vigilant for pain and the desire for increasingly expensive passive therapeutic interventions that are yet to demonstrate convincing efficacy"* (p. 33).

Failure to improve after a time-limited period of individual therapy should lead to recommendation for a chronic pain management, general exercise, functional restoration programme or behavioural therapy approach (Flor *et al.* 1992; Cutler *et al.* 1994; van Tulder *et al.* 1997b, Bendix *et al.* 1998; van Tulder *et al.* 2000c). Within the framework of the biopsychosocial model of pain is the proposal for active, behavioural therapy and exercise-based management (Wheeler and Hanley 1995; Rose *et al.* 1997; Frost *et al.* 1995, 1998).

Common features of successful programmes for chronic back problems have been identified (Linton 1998):

- use a multidimensional view of the problem, including psychosocial aspects
- conduct a thorough 'low-tech' examination
- communicate the findings of examination to the patient and an explanation of why it hurts and how to best manage it
- emphasise self-care, and explain that the way the patient behaves is integral to the recovery process
- reduce any unfounded fears or anxiety about the pain and movement ('hurt does not mean harm')
- make clear recommendations about starting normal activities and a graded approach to exercises
- do not medicalise the problem: avoid 'high-tech' investigations, long-term sick leave and advising the patient to 'take it easy'.

Treating chronic backs – the McKenzie Institute International Rehabilitation Programme

In New Zealand, the Accident Rehabilitation and Compensation Insurance Corporation (ACC) evaluated the effectiveness of four treatment programmes for chronic compensated back pain patients (Borrows and Herbison 1995b). All programmes used different exercise and rehabilitation regimes, one of which was a McKenzie regime. Nearly 800 patients, with an average of twenty months on compensation, were allocated, not randomised, to the different programmes.

The outcomes from the McKenzie programme are summarised in Chapter 11. In summary, the results show that not all functional rehabilitation programmes are the same. While two programmes produced significant improvements in a range of outcomes, the other two programmes hardly had any impact at all. 'Fitness to work' was the primary goal; this improved by 35% in the McKenzie programme, 20% in the next best intervention and by less than 4% in the other two. Functional disability and depression also improved markedly in the two best programmes, but minimally in the least effective two (Borrows and Herbison 1995b).

The timescale for providing these outcomes was very different. The average duration of the three other programmes was from 103 to 127 days, with all exceeding their initial estimated duration by nearly

50%. In comparison, although the McKenzie programme was residential, it had a finite duration of only fourteen days. The cost implications of this are not calculated in the original report, but could be considerable.

The authors of the report comment on the characteristics of the successful programmes (Borrows and Herbison 1995b):

- passing the responsibility for improvement to the patient
- ignoring or downplaying the significance of pain
- individual biomechanical assessment
- individual exercise programme
- pleasurable recreational activities.

The background of the ACC

In the 1980s the ACC was the sole provider of insurance cover for injuries arising from an accident at work, irrespective of fault. Part of their responsibility was to provide treatment and physical rehabilitation to restore 'injured' workers to 'workability'. Ultimately the scheme was discontinued as financially insolvent, as it was unable to cope with the escalating costs of providing compensation and rehabilitation. In an attempt to reduce costs, the organisation cut funding for rehabilitation and simply paid long-term earnings-related compensation. The situation arose in 1990 that almost 14,000 individuals with work-related 'injuries' were receiving earnings-related compensation. They had been off work for up to two years and no attempt was being made to rehabilitate them back to work.

In order to try to reinvigorate the rehabilitation process that had been put in abeyance, the McKenzie Institute International approached the government agency responsible for the ACC, who eventually were instructed to fund the trial mentioned above.

All participants had been on earnings-related compensation for at least three months and were willing to participate. The McKenzie programme excluded patients in whom specific pathology was diagnosed, scored high on psychometric questionnaires, if they refused to be compliant with the programme or if no movement reduced, abolished or centralised symptoms. Of those included some responded to extension, a few to flexion and some just responded to movement and reactivation in general. After two days' testing, 219

patients were accepted onto the programme from 252 referred by the ACC (87%). On the other programmes acceptance levels were 70%, 74% and 89%.

Patients were assessed independently by an ACC representative. This was done according to a standardised protocol, and a battery of functional and psychometric tests were applied that allowed independent verification of the outcome data. This was done prior to a full history-taking and mechanical evaluation, as well as further questionnaires, by programme personnel. Following this, patients were classified according to their mechanical syndrome.

The details of the McKenzie ACC programme

Many patients with a directional preference for extension were then assigned to repeated sessions on the REPEX machine. The Repeated End Range Passive Exercise (REPEX) machine causes repeated movements to end-range that are done while the patient lies on the equipment. These sessions were repeated for a maximum of ten minutes each hour on the first day, but according to the patient's tolerance. The machine allowed them to experience the sensation of movement without exertion, which was generally well tolerated. Following a session on the REPEX, the patient repeated extension in lying actively. REPEX was employed for up to the first seven days. The machine was important in extension responders, as most had very little range of movement and REPEX sped up the process of regaining this movement. It helped those who could not tolerate active extension exercises every hour to participate in regular movement. Many patients with gross losses of movement improved range dramatically, demonstrating what appeared to be the reduction of very stubborn lumbar derangements.

If patients demonstrated a preference for extension, as well as the hourly sessions on the REPEX, they were also given lumbar rolls, education on posture and introduced to the gym. There they undertook upper and lower body strengthening exercises and back extension exercises. They were also encouraged to take a short walk on an hourly basis.

Patients with a preference for flexion performed flexion in lying and in the gym performed exercises that promoted flexion. REPEX was generally unnecessary in this group as they were achieving end-range flexion effectively in their exercise programme.

If patients had no clear directional preference, they were assigned to the flexion group as a provocative regime. Unless patients developed a clear derangement, they were kept on unidirectional exercises for the first few days, with frequency and load gradually increased. Some patients responded positively to general mobilisation and reactivation.

In this deconditioned population, active participation, especially in the case of extension in lying, sometimes led to significant fatigue in the shoulders and arms. If this occurred, patients were advised to perform active sessions a little less frequently. To allow recovery from what for many was an excessive amount of exertion, they had twice daily sessions in the swimming pool. There the patients repeated their assigned exercises in a weight-relieving aquatic environment and also participated in water polo games, hydro-aerobics and unstructured fun activity. About an hour was allocated for lunch. The afternoon session repeated what had occurred in the morning, and the last two hours of each day were spent in 'play hardening' as opposed to 'work hardening' activity. This consisted of tennis, volleyball, table tennis, net ball, ten-pin bowling, horse riding, snooker, golf, jogging or brisk walking.

Day two largely repeated the format of the first day with REPEX, active exercise, gym, pool sessions and sporting participation. The number of sessions remained the same, but the passive and active repetitions were increased and the amount of participation in pool and sporting activity was increased where indicated.

By the second or third day, use of the REPEX was discontinued in many cases and active participation in gym activities was substituted. If it brought definite benefit, REPEX was used for longer periods. In the gym over the whole programme, the time spent on equipment, the number of equipment units utilised and the loading was progressively increased. Likewise, levels of self-applied end-range motion were progressed, with most attaining end-range by the fourth or fifth day. By day four or five the opposite movement was introduced – so, for instance, if a patient had been put on an extension regime, flexion and rotation exercises were started.

The remainder of the programme consisted of the same activities. Gym work on equipment and aerobics, active end-range movements, educational sessions, activities in the pool and 'play hardening'. Patients were individually assessed on a daily basis and their regime

progressed accordingly. Group work could be somewhat challenging, depending on how the negative or positive attitude of the dominant personality affected group dynamics. The frequency of the pleasurable recreational activities was increased as it became apparent that patients would often forget their anxiety and fear about movement and their disability as they threw themselves vigorously into the spirit of the game. Games such as tennis and volleyball, which involved considerable flexion to retrieve the ball, were especially good at producing a return to normal function and overcoming individuals' fear of movement.

Patients' response to the programme varied. Some, with considerable disability or fearful behaviour, required more individualised education and instruction. In some their fear of activity was considerably worse than the effect of the activity itself. Once they had experienced that movement and activity could be tolerated, and especially when their enthusiasm for the recreational activities was stimulated, these fears were overcome.

Day four of the programme was often difficult, as the majority experienced an increased level of pain at about this point. Encouragement to persevere and focus on improving function, rather than pain, was especially important at this phase. A patient's belief systems were often a major part of the problem and had to be fully explored and dealt with. Mistaken beliefs and attitudes about pain and activity were often the result of iatrogenic advice. Patients had been told, for instance, 'don't move if it hurts', 'rest or you will do damage', or 'if you are in pain, take medication'. The programme appeared to alter the moods and attitudes of the patients. As they increased their activity, this was reflected in increased confidence and reduced disability and impairment. At the same time their depression and anxiety noticeably lessened.

While most of those who reported high rates of disability were consistent in their reporting and in their activities, a small number appeared to deliberately exaggerate their disability. These patients demonstrated multiple Waddell's non-organic signs and symptoms. They displayed exaggerated vocalisation and body language on testing and movement, but were able to play tennis, volleyball, snooker and other games with ease.

Those patients who reported no improvement on completion of the programme, those with the most intractable disability or psychological distress, may have received benefit from a multi-disciplinary pain management programme.

Conclusions

This chapter considers other categories of back pain besides the mechanical syndromes. If after a detailed and thorough mechanical evaluation conducted over a few sessions there has been a failure to classify the patient into one of the mechanical syndromes, only then should other diagnoses be considered. This occurs in only a minority of patients. Among those with specific pathology the group that is most important to recognise are those with serious spinal lesions. Recognition of these pathologies is gained largely from the history and is detailed in the previous chapter.

Appendix

Classification and operational definitions

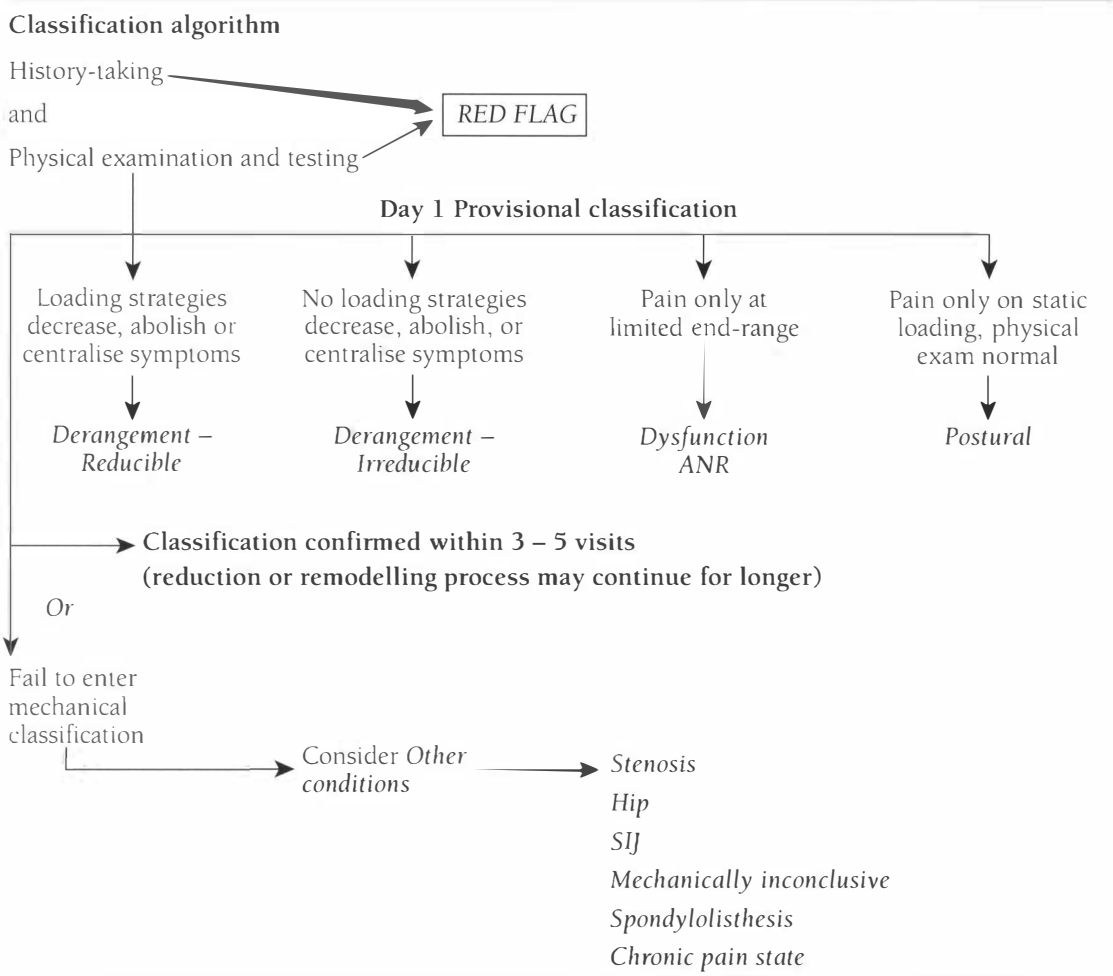
Category *Mechanical syndrome*	Definition	Criteria** *Symptom response*
Reducible derangement	Internal disc displacement with competent annulus	Centralisation Abolition Decrease
Irreducible derangement	Disc displacement with incompetent or ruptured annular wall	Peripheralisation Increase in peripheral pain No centralisation, reduction or abolition
Dysfunction	Soft tissue structural impairment	Intermittent pain when loading restricted end-range
Adherent nerve root	Adhesions producing functional impairment of nerve root or dura	Intermittent pain at limited end-range flexion in standing and long sitting
Postural syndrome	Prolonged mechanical deformation of normal soft tissues	Pain only with prolonged loading Physical examination normal
OTHER	*Exclusion of above*	*Lack of above responses, plus the following*
Spinal stenosis	Bony or soft tissue narrowing of spinal or foraminal canal causing neurogenic claudication May be associated *with* degenerative spondylolisthesis	History – leg symptoms when walking, eased in flexion Minimal extension Sustained extension may provoke leg symptoms
Isthmic spondylolisthesis	Slippage of vertebral body	Sports-related injury in adolescence Worse with static loading
Hip	Pain-generating mechanism due to mechanical, inflammatory or degenerative changes in or around hip joint	History – pain on walking, eased on sitting Specific pain pattern Positive 'hip' tests
SIJ	Pain-generating mechanism due to mechanical, inflammatory or degenerative changes in or around SIJ	Three or more positive SIJ pain provocation tests
Mechanically inconclusive	Unknown intervertebral joint pathology	Inconsistent response to loading strategies No obstruction to movement

Continued next page

Category *Mechanical syndrome*	Definition	Criteria** *Symptom response*
Chronic pain	Pain-generating mechanism influenced by psychosocial factors or neurophysiological changes peripherally or centrally	Persistent widespread pain Aggravation with all activity Exaggerated pain behaviour Inappropriate beliefs and attitudes about pain

Serious spinal pathology – suspected	Definition	Criteria
Cauda equina	Compression of sacral nerves by disc herniation or tumour	Bladder / bowel involvement Especially urinary retention Saddle anaesthesia Sciatica
Cancer	Growth of malignant tumour in or near vertebrae	Age > 55 History of cancer Unexplained weight loss Constant, progressive pain unrelated to loading strategy, not relieved by rest
Fracture	Bony damage to vertebrae caused by trauma or weakness due to metabolic bone disease	Significant trauma Trivial trauma in individual with osteopenia
Spinal infection	Infection affecting vertebrae or disc	Systemically unwell Febrile episode Constant severe back pain unrelated to loading strategy
Ankylosing spondylitis	One of the systemic inflammatory arthropathies affecting spinal and other structures	Exacerbations and remissions Marked morning stiffness Persisting limitation all movements No directional preference, but better with exercise, not relieved by rest Systemic involvement Raised ESR, + HLA B27

** The operational definitions provided below present the criteria in more detail. These give the symptom responses and timescale by which classification should be recognised.



Operational definitions

The operational definitions describe the symptom and mechanical behaviours and the timescale needed to document each category.

Reducible Derangement

Centralisation: in response to therapeutic loading strategies, pain is progressively abolished in a distal to proximal direction, *and*

- each progressive abolition is retained over time until all symptoms are abolished, *and*
- if back pain only is present this moves from a widespread to a more central location and then is abolished *or*
- pain is decreased and then abolished during the application of therapeutic loading strategies
- the change in pain location, or decrease or abolition of pain, remain better, *and*

- should be accompanied or preceded by improvements in the mechanical presentation (range of movement and/or deformity).

Timescale

A derangement responder can be identified on day one, *or*

- a derangement responder will be suspected on day one and a provisional diagnosis made. This will be confirmed by a lasting change in symptoms after evaluating the response to a full mechanical evaluation within five visits
- decrease, abolition or centralisation of symptoms is occurring but the episode may not have completely resolved within five visits
- aggravating factors may precipitate a deterioration in symptoms and a longer recovery process.

Irreducible Derangement

Peripheralisation of symptoms: increase or worsening of distal symptoms in response to therapeutic loading strategies, *and/or*

- no decrease, abolition, or centralisation of pain.

Timescale

An irreducible derangement patient will be suspected on day one and a provisional diagnosis made; this will be confirmed after evaluating the response to a full mechanical evaluation within five visits.

Dysfunction

Spinal pain only, *and*

- intermittent pain, *and*
- at least one movement is restricted, and the restricted movement consistently produces concordant pain at end-range, *and*
- there is no rapid reduction or abolition of symptoms, *and*
- no lasting production and no peripheralisation of symptoms.

ANR

History of sciatica or surgery in the last few months that has improved, but is now unchanging, *and*

- symptoms are intermittent, *and*
- symptoms in the thigh and/or calf, including 'tightness', *and*
- flexion in standing, long sitting, and straight leg raise are clearly restricted and consistently produce concordant pain or tightness at end-range, *and*
- there is no rapid reduction or abolition of symptoms and no lasting production of distal symptoms.

Timescale

- a dysfunction/ANR category patient will be suspected on day one and a provisional diagnosis made; this will be confirmed after evaluating the response to a mechanical evaluation within five visits
- if the patient fails to fit all criteria another category must be considered
- rapid change will not occur in this syndrome, and symptoms will gradually reduce over many weeks, as range of movement gradually improves.

Postural

Spinal pain only, *and*

- concordant pain only with static loading, *and*
- abolition of pain with postural correction, *and*
- no pain with repeated movements, *and*
- no loss of range of movement, *and*
- no pain during movement.

Timescale

- a posture category patient will be suspected on day one and a provisional diagnosis made. This will be confirmed after evaluating the response to a mechanical evaluation within two to three visits
- if the patient fails to fit all criteria, another category must be considered.

'Other' categories are only considered on failure to enter a mechanical diagnosis within five treatment sessions. To be designated into 'Other' category, patients will fulfil:

- 'other' criteria, *and*
- criteria for specific other category as listed below.

'Other'

- no centralisation, peripheralisation, or abolition of symptoms, *or*
- does not fit derangement, dysfunction or posture criteria
- no lasting change in pain location or pain intensity in response to therapeutic loading strategies, *and*
- fulfils relevant criteria in suspected 'other' pathology listed below.

Indicators for possible 'Red Flags'

Cauda equina

- bladder dysfunction (urinary retention or overflow incontinence)
- loss of anal sphincter tone or faecal incontinence
- saddle anaesthesia about the anus, perineum or genitals
- global or progressive motor weakness in the lower limbs.

Possible cancer

- age greater than 55
- history of cancer
- unexplained weight loss
- constant, progressive pain not affected by loading strategies, worse at rest.

Other possible serious spinal pathology

One of the following:

- systemically unwell
- widespread neurology
- history of significant trauma enough to cause fracture or dislocation (x-rays will not always detect fractures)
- history of trivial trauma and severe pain in potential osteoporotic individual
- sudden and persistent extremes of pain causing patient to 'freeze'.

Possible inflammatory disorders

- gradual onset, *and*
- marked morning stiffness, *and*
- persisting limitation of movements in all directions
- peripheral joint involvement
- iritis, psoriasis, colitis, uretheral discharge
- family history.

Stenosis

- history of leg symptoms when walking upright
- may be eased when sitting or leaning forward
- loss of extension
- possible provocation of symptoms in sustained extension, with relief on flexion
- age greater than 50
- possible nerve root signs and symptoms
- extensive degenerative changes on x-ray
- diagnosis confirmed by CT or MRI.

Hip

- exclusion of lumbar spine by mechanical evaluation, *and*
- pain worsened by weight bearing, eased by rest or worse first few steps after rest, *and*
- pain pattern – groin, anterior thigh, knee, anterior shin, lateral thigh, possibly buttock, *and*
- positive hip pain provocation test(s) – (concordant pain).

Symptomatic SIJ

- exclusion of lumbar spine by extended mechanical evaluation, *and*
- exclusion of hip joint by mechanical testing, *and*
- positive pain provocation tests (concordant pain) – at least three tests.

Mechanically inconclusive

- symptoms affected by spinal movements
- no loading strategy consistently decreases, abolishes or centralises symptoms, nor increases or peripheralises symptoms
- inconsistent response to loading strategies.

Symptomatic spondylolisthesis

- suspect in young athletic person with back pain related to vigorous sporting activity
- worse with static loading.

Chronic pain state

- persistent widespread symptoms
- all activity increases symptoms
- exaggerated pain behaviour
- mistaken beliefs and attitudes about pain and movement.

Other definitions

Definition of centralisation

- in response to therapeutic loading strategies pain is progressively abolished in a distal to proximal direction with each progressive abolition being retained over time until all symptoms are abolished
- if back pain only is present, this is reduced and then abolished.

Criteria for a relevant lateral shift

- upper body is visibly and unmistakably shifted to one side
- onset of shift occurred with back pain
- patient is unable to correct shift voluntarily
- if patient is able to correct shift, they cannot maintain correction
- correction affects intensity of symptoms
- correction causes centralisation or worsening of peripheral symptoms.

Right and left lateral shift

- a right lateral shift exists when the vertebra above has laterally flexed to the right in relation to the vertebra below, carrying the trunk with it; the upper trunk and shoulders are displaced to the right
- a left lateral shift exists when the vertebra above has laterally flexed to the left in relation to the vertebra below, carrying the trunk with it; the upper trunk and shoulders are displaced to the left.

Contralateral and ipsilateral shift

- contralateral shift exists when the patient's symptoms are on one side and the shift is in the opposite direction; for instance, right back pain, with / without thigh / leg pain, and upper trunk and shoulders displaced to the left
- ipsilateral shift exists when the patient's symptoms are on one side and the shift is to the same side; for instance right back pain, with / without thigh / leg pain, with upper trunk and shoulders displaced to the right.

Criteria for a relevant lateral component

- acute lateral shift deformity OR loss of frontal plane movements *and / or*
- unilateral / asymmetrical symptoms affected by frontal plane movements
- symptoms fail to improve with sagittal plane forces *or*
- symptoms worsen with sagittal plane forces *and*
- symptoms improve with frontal plane forces.

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Glossary of Terms

Anterior compartment

The compartment of the intervertebral segment that is compressed with flexion forces.

Centralisation

The phenomenon by which distal limb pain emanating from although not necessarily felt in the spine is immediately or eventually abolished in response to the deliberate application of loading strategies. Such loading causes an abolition of peripheral pain that appears to progressively retreat in a proximal direction. As this occurs there may be a simultaneous development or increase in proximal pain. The phenomenon only occurs in the derangement syndrome.

Curve reversal/obstruction to curve reversal

In an asymptomatic state, individuals can move from an extreme position of flexion to an extreme position of extension without impediment; in derangement this can become difficult or impossible. Following a period of loading or repeated movements in one direction the opposite movement may become obstructed, and recovery is slow, gradual and/or painful. Thus, after spending a period of time in flexion, as in bending or sitting, or after repeated flexion, the patient is unable to regain the upright position immediately or without pain. They are forced to gradually and painfully resume the erect posture or movements into extension. In severe derangements patients may have difficulty straightening after one flexion movement.

Deformity

The patient experiences a sudden onset of pain and immediately or subsequently develops a loss of movement and a deformity so severe that they are unable to move out of the abnormal posture. The patient is fixed in kyphosis, lateral shift or lordosis and is unable to self-correct this very visible anatomical misalignment. If they are able to correct the deformity, they cannot maintain the correction. This phenomenon only occurs in derangement and must be immediately recognised as it determines treatment.

- *Kyphotic deformity* – the patient is fixed in flexion and is unable to extend.

- *Lateral shift* – the patient is fixed in (for instance) right lateral shift and is unable to bring his hips back to the mid-line or assume a position of left lateral shift. In the case of a 'hard' deformity, the patient will need clinician assistance to correct it, while in the case of a 'soft' deformity, the patient may be able to self-correct with repeated movements.
- *Lordotic deformity* – the patient is fixed in extension and is unable to flex.

Derangement syndrome

Rapid and lasting changes, sometimes over a few minutes or a few days, in pain intensity and location. Mechanical presentation can occur in this syndrome with the performance of movements or the adoption of sustained postures. Loading strategies produce a decrease, abolition or centralisation of symptoms. Opposite loading strategies may cause production, worsening or peripheralisation of symptoms if prolonged over a sufficient time. A distinguishing set of characteristics will be found during the history-taking and physical examination. The conceptual model involves internal articular displacement that causes a disturbance in the joint, which produces pain and impairment.

Deviation

There are two types of deviation: a) postural b) on movement.

a) Postural deviations – patients may prefer to hold themselves shifted to one side or in a degree of flexion because this brings temporary easing of their condition. However, they are capable of straightening, which distinguishes this group from those with a deformity. Both occur only in derangement.
b) Deviation on movement – for instance, as the patient flexes, they deviate away from the pure sagittal plane to left or right. This is indicative of either an adherent nerve root or a derangement.

Directional preference

The phenomenon of preference for postures or movement in one direction that is a characteristic of the derangement syndrome. It describes the situation when postures or movements in one direction decrease, abolish or centralise symptoms and often increase a limitation of movement. Postures or movements in the opposite direction often cause these symptoms and signs to worsen. This does not always occur, and may be a product of the length of exposure to provocative loading.

Distal symptoms

The symptoms located furthest down the leg; these may be radicular or somatic referred pain, or paraesthesia. During the evaluation of symptomatic responses to mechanical loading, the most distal symptoms are closely monitored. Movements that decrease or abolish these symptoms are prescribed, while movements that increase or produce them are avoided.

Dysfunction syndrome

Pain from the dysfunction syndrome is caused by mechanical deformation of structurally impaired soft tissues. This abnormal tissue may be the product of previous trauma or degenerative processes and the development of imperfect repair. Contraction, scarring, adherence, adaptive shortening or imperfect repair tissue become the source of symptoms and functional impairment. Pain is felt when the abnormal tissue is loaded. A distinguishing set of characteristics will be found during the history-taking and physical examination. In spinal dysfunction pain, is consistently produced at restricted end-range, and abates once the loading is released. Dysfunction may affect contractile, peri-articular or neural structures, with the latter two occurring in the spine.

Extension principle

This principle of treatment encompasses procedures, both patient- and therapist-generated, that produce extension of the lumbar spine. In a posterior derangement these will be used to abolish, decrease or centralise symptoms. In an extension dysfunction, the extension principle is used for remodelling.

Flexion principle

This principle of treatment encompasses procedures, both patient- and therapist-generated, that produce flexion of the lumbar spine. In an anterior derangement these will be used to abolish, decrease or centralise symptoms. In a flexion or ANR dysfunction, the flexion principle is used for remodelling.

Force alternatives

A change in the manner in which a force may be applied during the exploration of loading strategies to reduce derangements. For instance, alternative start positions (standing or lying), force directions (sagittal or lateral), dynamic (repeated movements) or static forces (sustained positions).

Force progressions

Within each principle of treatment direction (extension, flexion, lateral), there is a range of loading strategies available. These involve greater or more specific forces, but are still in the same plane of movement. For instance, sustained mid-range positions, end-range patient-generated movement, patient-generated force with clinician overpressure, clinician-generated force, or repeated movements over several days. Force progressions are used to determine the correct directional preference and when lesser forces are not able to maintain improvements.

Kappa

The Kappa coefficient is commonly used in studies to address the reliability of two testers to come to the same conclusion about a test. It takes account of the fact that there is a 50% probability of chance agreement even if random judgements are made. It reports a numerical value, with 1.00 being perfect agreement and 0.00 for agreement no better than chance. Negative values imply that agreement is worse than what would be expected by chance alone.

Guide to Kappa values

Kappa value	*Strength of agreement*
<0.20	Poor
0.21-0.40	Fair
0.41-0.60	Moderate
0.61-0.80	Good
0.81-1.00	Very good

Source: Altman 1991

Lateral compartment

The compartment of the intervertebral segment that is compressed with lateral forces. The lateral compartment becomes relevant if lateral forces influence the patient's symptoms.

Relevant lateral component

This refers to patients with derangement who have unilateral or asymmetrical symptoms that do not improve with sagittal plane forces. When the lateral component is relevant, asymmetrical forces are necessary to achieve centralisation or decrease of symptoms.

Lateral principle

This principle of treatment encompasses procedures, both patient- and therapist-generated, that produce an asymmetrical force on the lumbar spine. In postero-lateral or antero-lateral derangement these will be used to abolish, decrease or centralise symptoms.

Loading strategies

Describes the applied movements, positions or loads required to stress particular structures, and may be dynamic or static – dynamic would be a repeated movement; static, a sustained posture. The significant loading strategies, postures and repeated movements are those that alter symptoms.

Mechanical presentation

The outward manifestations of a musculoskeletal problem such as deformity, loss of movement range, velocity of movement or movement deviations. Very important in re-assessment of treatment efficacy.

Mechanical response

Change in mechanical presentation, for instance an increase or decrease in range of movement in response to a particular loading strategy.

Mechanical syndromes

Refers to the three mechanical syndromes as described by McKenzie – derangement, dysfunction and posture, which describe the majority of non-specific spinal problems.

Non-mechanical factors

Factors that are non-mechanical in nature that may influence a patient's experience of pain. For instance, in the acute phase of a problem, the pain-generating mechanism may be primarily inflammatory. In the chronic stage, various non-mechanical factors, such as central or peripheral sensitisation or psychosocial factors, may influence pain modulation.

Pain

Acute pain

Pain of recent onset of less than seven days. This includes some with pain of an inflammatory nature, but many will experience pain of a mechanical nature due to derangement.

Sub-acute pain
Pain that has lasted between seven days and seven weeks. In some this may represent an interface between inflammatory and mechanical pain, but again, mechanical factors are likely to predominate.

Chronic pain
Pain that has lasted for longer than seven weeks. In the majority this will be mechanical in nature, and non-mechanical in a minority.

Chronic pain states
Pain of long duration in which non-mechanical factors are important in pain maintenance. These factors may relate to peripheral or central sensitisation or psychosocial factors, such as fear–avoidance, etc. Symptoms are often widespread and aggravated by all activity, and patients display exaggerated pain behaviour and mistaken beliefs about movement and pain.

Chemical or inflammatory pain
Pain mediated by the inflammatory chemicals released following tissue damage, or due to systemic pathology, such as ankylosing spondylitis.

Mechanical pain
Pain that results from mechanical deformation of tissues. This occurs with abnormal stresses on normal tissues, as in the postural syndrome, and normal stresses on abnormal tissues, such as in derangement and dysfunction.

Constant pain
Constant pain describes symptoms that are present throughout the patient's waking day, without any respite, even though it may vary in intensity. This may be chemical or mechanical in origin, and may also exist in chronic pain states.

Intermittent pain
This describes pain that comes and goes during the course of the day. Commonly this relates to intermittent mechanical deformation that results in pain. Pain may be momentary or appear and linger for varying amounts of time, but does at some point during the day completely stop.

Site and spread of pain
The area in which pain is perceived in terms of the extent of referral into the limb. The most distal site of pain is important to monitor regarding centralisation and peripheralisation. This information provides important information during assessment and re-assessment of the symptomatic presentation.

Severity of pain
This provides important information during assessment and re-assessment of the symptomatic presentation. Either the patient is asked on a one-to-ten scale about the intensity of the pain on different occasions, or in retrospect is asked to compare present pain to when they first attended.

Peripheralisation
Peripheralisation describes the phenomenon when pain emanating from the spine, although not necessarily felt in it, *spreads* distally into, or further down, the limb. This is the reverse of centralisation. In response to repeated movements or a sustained posture, if pain is produced and remains in the limb, spreads distally or increases distally, that loading strategy should be avoided. The phenomenon only occurs in the derangement syndrome. The temporary production of distal pain with end-range movement, which does not worsen, is not peripheralisation, as this response may occur with an adherent nerve root.

Posterior compartment
Describes the compartment of the intervertebral segment that is compressed with extension forces.

Postural syndrome
Mechanical deformation of normal soft tissues arising from prolonged postural stresses, affecting any articular structures and resulting in pain. A distinguishing set of characteristics is found during the history-taking and physical examination. If prolonged sitting produces pain, it will be abolished by posture correction. Range will be full and pain-free, and repeated movements have no effect.

Red flags
This refers to features of the history-taking that may indicate serious spinal pathology, such as cancer, cauda equina syndrome or fracture. If possible 'red flag' pathology is suspected, further mechanical therapy is contraindicated and the patient should be referred to a specialist.

Reliability
This is the characteristic of a test or measuring tool to give the same answer in different situations. Inter-tester reliability examines the degree of agreement between different clinicians on the same occasion; intra-tester reliability examines the degree of reliability of a single

tester on different occasions. Results are presented in several ways: as a percentage agreement, correlation coefficients, or Kappa values.

Sensitivity

This is a characteristic of a clinical test used to diagnose a problem. The sensitivity is the ability of the test to be positive in all who have the problem. When a test is 100% sensitive, it is able to detect all who have the condition of interest. The sensitivity is the true positive rate. When sensitivity is extremely high (>0.95 or 95%), a negative test response rules out that disease. Poor sensitivity indicates a test that fails to identify many of those with the disease of interest.

Specificity

This is a characteristic of a clinical test used to diagnose a problem. The specificity is the ability of a test not to be positive in those who do not have the problem; it is thus the true negative rate. When a test is 100% specific it is able to identify all those who do not have the condition of interest. When specificity is extremely high (>0.95 or 95%) a positive test result gives a definite positive diagnosis. Poor specificity indicates a test that fails to exclude many individuals without the disease of interest.

Stage of condition

All musculoskeletal conditions can be anywhere on the continuum from acute to sub-acute to chronic. These stages are often of more significance to management than a structural diagnosis.

Standardised terms

These are used to make consistent descriptions of symptomatic responses to different loading strategies to judge their value for self-treatment. The description of symptoms during and *after* loading is significant in determining the management strategy to be applied. These are the words used to describe symptom response during the physical examination.

During loading:

Increase	Symptoms already present are increased in intensity.
Decrease	Symptoms already present are decreased in intensity.
Produce	Movement or loading creates symptoms that were not present prior to the test.
Abolish	Movement or loading abolishes symptoms that were present prior to the test.

Better Symptoms produced on movement, decrease on repetition.

Centralises Movement or loading abolishes the most distal symptoms.

Peripheralises Movement or loading produces more distal symptoms.

No effectMovement or loading has no effect on symptoms during testing.

End-range pain Pain that only appears at end-range of movement disappears once end-range is released, and in which the range does not rapidly change. In end-range pain due to derangement, increased force reduces symptoms, while with end-range pain due to dysfunction, increased force will increase symptoms.

Pain during movement

Pain produced during the range of movement, but then subsides or remains when the individual moves further into the range of movement. In the three mechanical syndromes in the spine, this only occurs in derangements.

After loading

Worse Symptoms produced or increased with movement or loading remain aggravated following the test.

Not worse Symptoms produced or increased with movement or loading return to baseline following the test.

Better Symptoms decreased or abolished with movement or loading remain improved after testing.

Not better Symptoms decreased or abolished with movement or loading return to baseline after testing.

Centralised Distal symptoms abolished by movement or loading remain abolished after testing.

Peripheralised Distal symptoms produced during movement or loading remain after testing.

No effect Movement or loading has no effect on symptoms during or after testing.

State of tissues

This describes the different conditions that tissues could be in. They may be normal or abnormal. Abnormal tissues may be injured, healing, scarred or contracted, with healing suspended, hypersensitive to normal loading due to changes in the nervous system, degenerated or painful due to derangements.

Status of condition

This describes the direction of the condition relative to recovery. It may either be improving, worsening or unchanging. Its status is significant in decisions concerning management.

Symptomatic presentation

This describes the details of the patient's complaints and can be assessed and re-assessed regarding site, intermittency/constancy, diurnal variation, severity, consequent analgesic/NSAID consumption and self-reported functional disability. This is very important in re-assessment of treatment efficacy.

Symptomatic response

The behaviour of pain in response to a particular loading strategy, for instance centralisation, peripheralisation, worse or better.

Traffic light guide

Identification of patient's responses to loading strategies, using standardised terminology, determines the appropriateness of a management direction. If the patient remains worse afterwards, this is a 'red light' to that procedure; if the patient remains better, this is a 'green light' for that exercise; if there is no change, an 'amber light', a force progression or force alternative *may* be required. An 'amber' response is also a 'green light' in the presence of a dysfunction.

Treatment principle

The treatment principle defines the force direction used in management; they are termed extension, flexion or lateral. Each principle of treatment contains patient- and clinician-generated force progressions. In a derangement, the treatment principle is determined by the direction that causes a decrease, abolition or centralisation of pain. In a dysfunction, the treatment principle is determined by the direction that reproduces the relevant symptom.

Validity

This is the ability of a test to diagnose or measure what it is intended to diagnose or measure. There are various dimensions of validity, but criterion validity is critical to the accuracy of a diagnosis. This is the ability of a test to determine the presence or absence of a particular pathology. The value of a test is judged by its ability to diagnose the pathology compared to a 'gold standard'. The validity of the 'gold standard' is meant to be about 100%. Validity is measured by sensitivity and specificity.

Yellow flags

Term used to describe psychosocial risk factors for developing or perpetuating long-term disability or sick leave as a consequence of musculoskeletal symptoms. They include factors such as the attitudes and beliefs of the patient about their problem, their behavioural responses to it, compensation issues, inappropriate health care advice, information or treatment, emotions such as depression, anxiety and fear of movement, and relations with family and work.

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THE LUMBAR SPINE MECHANICAL DIAGNOSIS & THERAPY

VOLUME TWO

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The Lumbar Spine
Mechanical Diagnosis & Therapy

Volume Two

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14: The History

Introduction

"Every patient contains a truth.... The (clinician) must adopt a conscious humility, not towards the patient, but towards the truth concealed within the patient" (Cyriax 1982, p. 45). The patient knows the details of the history, onset, symptom pattern and behaviour since onset, and aggravating and relieving factors. This information is vital to gain knowledge of the disorder. In order to access this truth the clinician must approach the patient in a respectful and friendly way, they must have a logical format for collecting information, and, most importantly, they must actively listen to the patient's responses. Only from the patient is it possible to gain insights into various aspects of the clinical presentation, which are essential to inform issues such as the stage and nature of the disorder, the prognosis and the management. Very often the history-taking provides information that is at least as important as that gained from the physical examination, if not more so.

The interview requires the clinician to have skills in questioning accurately and appropriately and of listening. It is important that we make the patient as comfortable and as relaxed as possible. Avoid the use of medical words or phrases that may be foreign to the patient. Conversation should be conducted using terms and phrases with which the patient feels comfortable. See the chapter on patient management for a longer discussion about the importance of good communication.

In mechanical diagnosis and therapy, we wish to understand the effect that different movements and positions have on symptoms and use this understanding to shape an appropriate management strategy. This understanding comes through analysis of the history and physical examination. The theoretical basis for interpreting the effect of posture and movement is described in Chapter 6.

Using a structured but flexible interview format so that all pertinent factors from the history and behaviour of the condition are collected facilitates a good understanding of the patient's problem. A thorough 'low-tech' assessment is an essential prerequisite for the provision of information and reassurance concerning their problem.

The standardised assessment form includes the most important aspects of the history that need gathering; mostly it will be unnecessary to add to this information. Be wary of gathering excessive amounts of information – always consider, 'how will the answer help in managing this patient?' If, however, it is deemed essential to gather further details about a particular aspect of the clinical presentation that causes concern, the form should not prevent further specific questioning. It is best to gather the information using open-ended questions first, so that patients may volunteer their own answers, rather than using leading questions. Focused questions may be used to pursue particular aspects that require more detailed information. Thus management decisions can be grounded in the particular patient's problem and their response to it.

Sections in this chapter are as follows:

- aims of history-taking
- interview
- patient
- symptoms
- previous history
- specific questions.

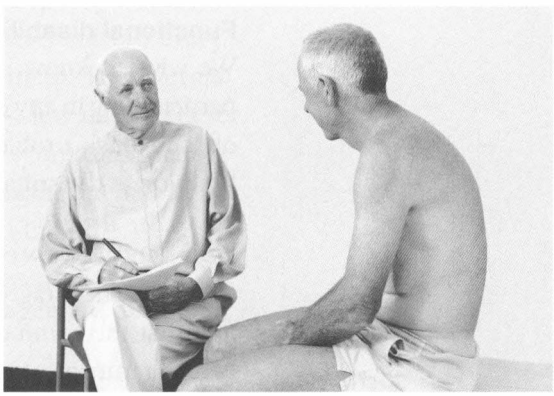
Aims of history-taking

By using the form and the appropriate questioning technique, ideally at the end of the history-taking the following will have been obtained:

- an overall impression of the clinical presentation
- site of the back pain: central / symmetrical, or unilateral / asymmetrical; if unilateral is the pain in the back or thigh, or referred below the knee
- the stage of the disorder – acute / sub-acute / chronic
- the status of the condition – improving / unchanging / worsening
- identification of 'red flags' or contraindications
- baseline measurements of the symptomatic (and mechanical presentations) against which improvements can be judged

- factors that aggravate and relieve the problem and the role of posture, which may help guide future management
- the severity of the problem, which may guide the vigour of the physical examination
- the functional limitations that the condition has caused on the patient's quality of life
- an impression about the way the patient is responding to their condition, and how much encouragement, information, reassurance or convincing they may need to be active participants in their own management
- a hypothetical diagnosis by syndrome.

Photo 1: Taking the history.
Allow the patient to relax unsupported to expose the true nature of their sitting posture.



1

Interview

During the history-taking, seat the patient on the assessment plinth or backless chair so that they reveal their true relaxed sitting posture.

Patient

Age

Patients are more susceptible to certain problems at different times of life. Postural syndrome is more likely to be present in the young, while young to old adults have derangements and dysfunctions. Spondylolysis is more relevant in the young, while osteoporosis is generally only relevant in the elderly, especially postmenopausal women, although there are exceptions. The general state of spinal tissues varies according to the age of the individual. With increasing age spinal degeneration is more likely to be present: the intervertebral discs become more dehydrated and fibrosed, and osteophytes and other bony changes can occur around the zygapophyseal joints and vertebral bodies (Bogduk 1997). Such changes may predispose to spinal stenosis. Malignancies are also more common in the older age group.

The age of the patient may also be significant in their response to the problem. Increasing years not only raise the susceptibility to disease and injury, but also reduce the body's ability to recover from the effects of musculoskeletal disease and injury (Buchwalter 1993). A patient's age may thus be important in their prognosis.

Occupation / leisure activities

It is important to know the individual's occupation and the kind of postural stresses it entails. Do they spend their day mostly sitting, driving or bending forwards; are they constantly changing activity; are they on their feet most of the time? Knowing the predominant activities of their working hours means that detrimental daily loading factors can be eliminated or lessened.

We also wish to know their usual sporting or recreational activities outside of work. Do they exercise regularly, or do they lead a largely sedentary life? It is also useful to know if there has been a recent change in occupation, from a sedentary to a more manual job, or vice versa. Either change may be a trigger to potential overloading.

Functional disability

We wish to know if the patient is off work at present, and/or not participating in any of their usual sporting or leisure activities because of their back problem. The earliest possible return to full normal function is the suitable goal for management.

A substantial review of back pain and work recommended that the worker should be encouraged to remain at work or to return as soon as possible, and that the common misconception that they should be pain-free before returning to work should be addressed. (Carter and Birrell 2000). Returning to work should be a primary outcome of treatment.

We should be aware of any normal sporting or recreational activities that they have stopped because of the back pain. Returning to such activities as soon as possible, in a gradual way, should be encouraged. General fitness has a therapeutic as well as protective effect for back pain, and management of the patient should address these issues.

A knowledge of the activity limitations that back pain has caused in the patient's normal lifestyle provides some understanding of their response to the problem in terms of their fear and anxiety. A brief and temporary interruption of normal activity may be necessary in some episodes; a long-term abstention from normal activity is generally unnecessary and disproportionate. Persistent avoidance of daily routines often indicates an exaggerated and inappropriate response to pain. Such patients need specific encouragement to return to normal activities and care must be made not to further exaggerate such inappropriate fear–avoidance behaviour.

Symptoms

Symptoms this episode

Relevant symptoms

We need to know all the areas where the symptoms have been felt during this present episode – this involves pain and paraesthesia. We wish to know if the present pain is central / symmetrical or unilateral / asymmetrical. If symptoms are unilateral or asymmetrical, is the pain felt in the back and thigh, or is it referred below the knee? We wish to know the most distal extent of any pain. If the patient reports pain in the leg, they should be asked if 'pins and needles', tingling or numbness are present at any time, and exactly where.

All the symptoms that have occurred during the present episode should be accurately marked on the body chart. To ensure accuracy, this can be shown to the patient and checked by them. The ***relevant symptoms*** are those that have been felt in the last few days – these are noted on the line below, so on this line those symptoms that are still a problem are described. In later chapters management will be described relative to different symptom patterns (Table 14.1).

Table 14.1 Symptom patterns relevant to management decisions

- central / symmetrical
- unilateral / asymmetrical +/- pain to knee
- unilateral / asymmetrical pain below knee.

The site of pain provides various useful pieces of information. Central or bilateral symptoms invariably need sagittal plane procedures. Patients with unilateral symptoms commonly require lateral forces in their management, although their response to sagittal plane forces is usually tested first.

The extent and degree of referred or radiating pain and other symptoms gives some indication of the severity of the problem. Greater referral of symptoms tends to indicate a more severe problem, and paraesthesia and weakness may accompany pain below the knee.

If the pain has changed since onset, this may provide a clue to the status of the condition. Pain that was felt in the leg and is now felt only in the back is describing an improving situation. Conversely, pain that began in the back and is gradually spreading down the leg is describing a worsening situation.

Baseline symptoms that are still troubling the patient must be recorded in full so that any changes in pain pattern over time can be appreciated.

The location of pain gives some insight into mechanical syndrome classification. Pain of postural or dysfunctional origin is almost always felt locally, with no radiation of pain. An exception to this is referred pain caused by an adherent nerve root, which is described later. If pain radiates into the buttock, thigh or calf, a derangement is likely.

Nerve root involvement is possible if pain or paraesthesia is described in the typical pattern of a dermatome, especially when other neurological signs are present – paraesthesia of diagnostic significance occupies the distal end of the dermatome, and may take the form of tingling or numbness (Smyth and Wright 1958; Nitta *et al.* 1993). However, leg pain per se is *not* an indicator of nerve root involvement; somatic structures, such as discs, zygapophyseal and sacro-iliac joints are all capable of causing leg pain (Schwarzer *et al.* 1994b, 1995a, 1995b; Dreyfuss *et al.* 1996). These studies failed to find any pain pattern characteristics that clearly distinguished these conditions. Paraesthesia felt around the perineum is a possible red flag (see ***Red Flags***).

The diagnostic utility of pain patterns should not be taken too far. One study found considerable overlap in pain drawings from patients with and without nerve root compression as diagnosed by magnetic resonance imaging. The drawings, which included pins and needles and numbness as well as pain and aching, allowed correct classification of only 58% of those with nerve involvement (Rankine *et al.* 1998). There is considerable overlap in pain patterns between L4 – L5 and L5 – S1 disc herniations when the L5 and S1 nerve roots are affected (Vucetic *et al.* 1995; Smyth and Wright 1958).

Pain that is due to degenerative symptoms from the hip joint is most commonly felt over the greater trochanter, anterior thigh, and knee, but also in the buttock, groin and shin (Wroblewski 1978; Jorring 1980). Patients usually describe pain in several locations. It should be remembered that these pain patterns are not unique to the hip joint. The pain is usually associated with weight-bearing, especially early in the course of the disease, but may become more constant as it progresses. Often patients report an easing of pain when sitting, in contrast to many spinal problems. Morning stiffness, pain on first weight-bearing, pain on movement of the limb and during walking are common but not universal findings with hip pathology (Jorring 1980).

Duration

This question is to determine when this *particular* episode started. If the patient has suffered recurrent problems, at this stage we are only interested in the present attack. Very often the patient is aware of the time an episode started. If pain has been present for a long time, an acute exacerbation of a chronic problem may have caused them to seek help. In this case, the episode has lasted since the original onset.

It is helpful to know if we are dealing with an acute, sub-acute or chronic problem. In this text these will be defined in line with the Quebec Task Force definitions (Spitzer *et al.* 1987), which correspond with the known healing process reviewed in Chapter 3.

Table 14.2 Definitions of acute, sub-acute and chronic

- acute – less than seven days
- sub-acute – between seven days and seven weeks' duration
- chronic – more than seven weeks' duration.

The length of time that the condition has been present may give some indication of the stability of the problem. Acute problems can easily be worsened as well as improved, so care with movement testing may be necessary. Acute and sub-acute problems are most probably due to derangement, while any three of the mechanical syndromes could be the cause of chronic problems.

Knowing the length of time that the problem has been present allows us to determine the state of the tissues. Days after onset tissues may be damaged and inflamed, whereas a few weeks later tissues may be healing. If the symptoms have been present for a couple of months, adaptive changes may have occurred in the collagenous repair tissue, indicating that dysfunction may be the cause of persisting symptoms. If the problem has been present for many months, although a straightforward mechanical condition may be present, the chance that the tissues are hypersensitive and deconditioned should be borne in mind. Chronic pain syndromes often complicate the management of persistent pain and may, though not always, make treatment less effective. Pain that has been present for many months, as well as having a poorer prognosis, may respond more slowly when it does respond. Many patients have a long or recurrent history of their problem, and therefore the educational component of management is particularly important to improve their future self-care. The duration of the episode thus provides diagnostic and prognostic information.

The length of time that the patient has had symptoms can also guide us in deciding how vigorous we can be with mechanical assessment procedures. If a patient has had symptoms for several months and has been able to work or remain active during this time, he or she will probably have placed more stress on the structures at fault than we are likely to apply during our assessment process. This allows us to be fairly vigorous with the overall mechanical assessment. However, someone who presents with a very recent onset needs to be examined with more care, at least initially.

Status

It is important to know if the patient thinks their problem is improving, worsening or unchanging. Judgements about the status of a condition may be based on five criteria (Table 14.3).

Table 14.3 Criteria for defining status of condition

Criteria	*Dimensions*
Time	Constant / intermittent Frequency increase / decrease
Intensity	Increase / decrease
Referral of pain	Centralising / peripheralising
Mechanical presentation	Movement increase or decrease
Activity limitation	Increase / decrease

When the patient reports that their condition is improving, a review of the problem and its prognosis is often all that is required. Avoid the inclination to embark on a programme of passive therapies. If history and evaluation of repeated movements confirm the process of recovery is under way, continuing at a steady rate and accompanied by improvement in function, there is no justification for any intervention other than education and assurance, unless or until progress comes to a halt. Provide guidelines for the progression of activity and exercise, and give advice on posture where necessary, but such patients do not require attending a clinic for regular 'treatment'.

If the pain is unchanging, a routine approach to the assessment can proceed. Stable and persistent symptoms generally permit a reasonably vigorous approach to assessment and management. Derangement or dysfunction may cause pain and functional impairment that may continue unabated for weeks or months, and may only be exposed using vigorous procedures.

If the patient reports pain that has persisted for many months, which may be constant or intermittent, and classification according to one of the mechanical syndromes is unclear, then a chronic pain syndrome may be suspected. Getting such patients started on regular, graduated exercise programmes frequently leads to an improvement in symptoms, function, and patient's perceived self-efficacy. Initially they may experience an exaggeration in symptoms due to the nature of chronicity, which is likely to involve sensitisation of certain tissues. They should be encouraged to pace their activities, not do too much too soon, and alternate activity with rest. Unless findings emerge from the assessment process that suggest further tests or more caution is required, *education and instruction* in a vigorous self-treatment programme are indicated. Clinician intervention at this point is unnecessary, but may follow at a later date should self-treatment and guidance fail to provide improvement.

In the event that the patient describes that his or her symptoms are worsening since onset, it is necessary to investigate the cause of deterioration. A rather gentle approach to the mechanical evaluation is always required if the patient describes that their pain is progressively increasing, and symptom response must be very carefully monitored. Under these circumstances, a purely educational approach may be indicated, certainly for the first twenty-four to forty-eight hours. Sustained positions may be of more use than repeated movements in attempting to improve symptoms. Escalating pain intensity could indicate more serious pathology, but certainly indicates an unstable condition in which greater care should be taken.

Patients whose symptoms are worsening should be seen on a regular basis until stability or improvement occurs, or until it becomes obvious that referral for further investigation is necessary. If the patient describes any of the 'red flag' indicators of serious pathology or if the reactions to mechanical evaluation are atypical or if they fail to affect the symptoms, referral for further investigation should be considered. For instance, an insidiously worsening condition in an older patient who looks unwell should be the cause of some concern. Appropriate blood tests or radiological assessment may shed light on the origin of the symptoms in such cases.

Onset

From this question we wish to know what the patient was doing when the pain started. In most instances there is no apparent reason (McKenzie 1981; Kramer 1990; Hall *et al.* 1998).

What the patient was doing immediately before the onset of pain may be important. On further questioning they may reveal that the ache started during an activity, for instance an evening spent sitting on the couch watching television. Other patients report the onset of pain following a particularly long drive, a bout of coughing and sneezing, or as a consequence of digging in the garden. These activities all involve flexion; temporary avoidance of these postures will probably be significant in management.

Often it is the case that patients are reluctant to accept that there is no obvious reason for the onset of symptoms and ascribe a causative role to some recent event. When questioned more closely, it is apparent that the event – taking some unusual exercise or a fall – is a week removed from when the problem actually started. It is especially important to disabuse patients of misconceived causality when it is apportioned to an attempt at exercise.

Sometimes back pain commences with an obvious incident. Significant accidents can cause multiple and specific injuries, should be treated with caution, and may require further investigation in their management (see ***Red Flags*** section below). Particular caution should be exercised with older women developing severe lumbar or thoracic pain from relatively trivial injuries – possibly suspect osteoporosis. In adolescents involved in sport who relate sport-associated onset back pain, possibly suspect spondylolisthesis.

Where symptoms have commenced for no apparent reason and are progressively worsening, it is always possible that some more sinister cause may be present. The likelihood of the presence of serious pathologies should be determined from further 'red flag' questioning.

Symptoms at onset

This question is to determine if, since onset, the area where the pain is felt has changed. Specifically we wish to know if pain and other symptoms are peripheralising into the leg – a worsening scenario – or if there has been a resolving of leg pain that was originally present – an improving scenario. Patients describing symptoms that change location always indicates derangement.

Constant or intermittent

This is one of the most important questions we must ask all patients with musculoskeletal disorders. Be sure that by 'constant pain' the patient does actually mean that their pain is there '100% of their waking day', from the moment they get up to the moment they fall asleep. Because some patients can always produce their pain with certain movements, they interpret this as constancy. Pain must be classified as intermittent even if there is only half an hour during the day when they are pain-free.

Constant pain is caused by inflammatory diseases and is present also where patients have suffered recent trauma causing an inflammatory response. Constant pain can also be the result of constant mechanical deformation, which is only present in derangement syndrome.

If the cause of the constant ache or pain is chemical, the symptoms will not be reduced or abolished by mechanical assessment procedures. Normally innocuous mechanical stresses can become painful under these circumstances. Movements may enhance existing chemical pain, but they never reduce or abolish it (McKenzie 1981, 1990). In general, the inflammatory period following trauma does not exceed five days (Evans 1980; Hardy 1989). If an inflammatory disease or arthropathy is present, chemical pain persists for longer periods.

If the cause is mechanical in origin, movements and positions can usually be found that reduce or stop the aching or pain. Constant mechanical pain is the result of internal derangement. The derangement syndrome can be associated with a constant ache, whereas patients with the postural and dysfunction syndromes do not experience constant pain. Constant pain is more diffi
than intermittent pain because the patient is usually unable to identify a directional or postural preference.

Chronic pain states usually present with constant pain, which is likely to worsen initially on mechanical assessment. The history will reveal the length of time symptoms have been present and also may relate previous failed treatments.

Unremitting, constant pain that does not abate even with recumbency, which is worsening, may be caused by serious non-musculoskeletal pathology (see *Red Flags* section below).

A description of intermittent pain generally excludes the possibility that the pain is inflammatory. Should the pain cease at rest or when the patient is still, the pain is not a result of an inflammatory response and must therefore be mechanical in origin. It could be postural, or result from dysfunction or derangement. Likewise, pain that changes location is not the product of an inflammatory process. However, inflammatory pain may spread as it worsens.

Intermittent pain is relatively easy to treat because if there is one hour in the day when no mechanical deformation is present, it is possible to gradually to extend that pain-free period by duplicating the favourable circumstances. In addition, the patient is usually already aware of certain movements or positions that bring relief, thus indicating the likelihood of the presence of a directional or postural preference.

Both a change in the frequency of the pain from constant to intermittent or reduction of intermittent pain from one that is present most of the time to one that is present only sometimes represents improvements in symptomatic presentation. Such improvements should help in the overall assessment of management strategies. It is therefore useful to ask about the proportion of the day that pain is present. This could be expressed as a percentage, for instance, 'on average, are your symptoms there for 80% of the day, 50% of the day, 20% or how much of the time?' Alternatively, patients may report that their pain occurs intermittently, and increasing or decreasing frequency of pain occurrence can be used to evaluate management efficacy. The different ways to measure symptomatic and mechanical improvements are considered in detail in Chapter 16.

What makes the pain worse / better?

These questions allow the patient to provide us with the information that is likely to lead to the appropriate management. They are designed to determine what movements or positions produce or abolish, or increase or decrease, mechanical loading and/or deformation of the affected structures. It is important to record those movements, positions or activities that specifically reduce or relieve the pain as this information will be utilised in our management protocols. It is also important to record which movements or activities aggravate the symptoms. It is often helpful at this point in the recording of the history to ask, "Of all the things that cause you pain, what bothers

you most of all?" This provides a yardstick by which to measure improvement at a later date.

It is also useful to know which other type of loading strategies, static or dynamic, affects the symptoms most. The patient may reveal that sustaining a position reduces their symptoms, and so static forces would be explored in the physical examination, or they may reveal that a prolonged position produces their pain after a certain time, and so interruption of aggravating postures before pain is created is important in management. As part of the educational strategy, it will be necessary temporarily to avoid such causative factors, or alter the way an activity is performed so that stresses are lessened.

Specifically, we must ask about the effects of sitting, standing, walking, lying and bending activities on the patient's symptoms. Everyone is subjected to these forces every day, so questioning can generally be confined to what are universal daily activities. Furthermore, in these positions the anatomical alignment of lumbar joints is relatively well understood (see Chapter 6). In general, sitting, driving and bending are activities of flexion, standing and walking activities of extension. This allows us to determine which situations increase and which situations decrease mechanical deformation (McKenzie 1981, 1990). This information is vital for optimal management. In lying, the posture of the spine varies according to whether the individual adopts a supine, prone or side lying position.

When asking about the effect of rising from a sitting or forward flexed position, we wish to know if they have difficulty assuming the upright posture after a period of prolonged flexion. This usually indicates the presence of a posterior derangement. In this situation, deformation appears to occur following prolonged flexion that inhibits or prevents immediate curve reversal of the spine into extension, or else the act of leaning forward in order to stand may cause a momentary increase in pain prior to standing erect.

We also wish to know if the symptoms are better when the patient is still or better when on the move. Very often patients feel less pain when they are moving regularly and worse when still. These patients often respond well to an exercise programme. Some patients with more severe conditions only gain some relief when they are still.

If symptoms are intermittent, it is important to know the positions, movements or activities that produce the pain. We also wish to know if these activities consistently produce the pain, and what happens when the activity is stopped. If pain is always brought on by the same activity and ceases shortly after cessation of that activity, we may begin to suspect dysfunction. Conversely, if activities are *sometimes* painful and *sometimes not,* derangement is usually implicated.

It is helpful to know if the movement that relieves or aggravates the pain brings about a lasting improvement or worsening. A lasting improvement following a particular loading strategy gives a useful idea about self-treatment procedures. If pain is produced by certain activities, but quickly abates once the movement ceases, the disorder is at a reasonably stable stage. If pain remains worse for hours after a relatively trivial movement or sustained posture, the disorder is likely to be an unstable derangement. Thus, knowing if the pain remains worse or better afterwards has important implications for self-treatment procedures.

If uncertainty exists as to whether a condition is spinal or peripheral, the activities that produce or aggravate symptoms may also be helpful in determining the source of the problem.

Some patients who have had pain for a long time can have difficulty in determining what makes their symptoms better or worse. Further detailed questioning may provide the relevant information. However, at times patients may have become incapable of analysing their pain, or movements and positions may have no consistent affect symptoms, in which case the information obtained from the history is insufficient, and during the physical examination it is essential to produce a change of symptoms by utilising end-range repeated movements and sustained positions. An indeterminate effect of loading strategies on symptoms is common in chronic pain states, when mechanical factors have become less relevant to pain persistence.

Diurnal pattern

We wish to know if there is any consistent pattern to the symptoms during the day. Are they better or worse first thing in the morning? Do they get better or worse as the day goes on? What happens to the symptoms in the evening? Does the pain wake them at night? Their answers provide some clues as to the effect of different activities over time and the effect of general activity compared to rest.

The diurnal pattern of symptoms can give an indication of overall prognosis. Patients who report they are better as the day progresses frequently do well, while those who report they are worse as the day progresses may have a poor prognosis. This depends, however, on the postures adopted during the day. If they are worse as a result of sitting all day, posture correction and appropriate exercise is usually effective. If the patient reports that they are worse in the morning, it can be useful to know if this is on waking, or on rising, when they are first bearing weight. If they are worse on waking, performance of extension in lying should be investigated before getting out of bed.

Some patients report that their worst symptoms are always in the night and the first few hours of the morning, but then once they are moving symptoms are much better for the rest of the day. Sometimes this is accompanied by a painful limitation to extension. Such scenarios frequently respond well to an extension principle exercise programme.

Patients with back pain are often woken by pain at night, but generally fall back to sleep quite quickly with a change of position (Boissonnault and Di Fabio 1996). Night pain per se is not an indication of serious pathology. If night pain is a problem, the sleeping position or surface of the bed may need to be considered. Night pain that drives the individual from the bed and prevents further rest may be an indicator of serious pathology.

When marking the form, circle an activity if it always makes the pain worse or better, underline it if it sometimes makes the pain worse or better, and cross it through if it has no effect.

Previous history

Back pain / treatment

At this point we wish to know their history of back pain. Is this their first episode? Have they had previous episodes? Just a few episodes, or many? How frequent have they been? We wish to know if previous episodes have been the same as this one or if there is a gradual worsening of symptoms with repeated episodes. Is the site of pain the same, or has it spread? How long have previous episodes lasted? Is this one about the same?

We also wish to know in brief what, if any treatment, they have received before. Particularly we are interested in anything that the

patient thinks was effective. Also, if they have been given exercises before, what were they, what effect did they have, are they still doing them, and with what effect now?

X-ray / imaging

Has the patient received radiography or another form of spinal imaging? The results are not particularly important to us as the abnormal morphology portrayed may have little bearing on the presenting symptoms, but in certain clinical presentations imaging may be necessary to exclude fractures or other specific pathology – for instance, following trauma, adolescent back pain related to sports, or degenerative or osteoporotic changes in older people.

Specific questions

Tingling / numbness / weakness / disturbed gait

The aim of these questions is to help identify certain specific pathologies. The presence of paraesthesia and/or numbness should be enquired about if the patient reports leg pain. This may be an indication of nerve root involvement, although this is not always so (Rankine *et al.* 1998). Nerve root involvement may also be suspected if the patient reports foot drop or weakness when pushing off during walking; therefore the patient is questioned about whether their gait is normal or has changed with the onset of their back problem.

Red Flags

The aim of these questions is to help exclude certain specific pathologies that contraindicate mechanical therapy and may need urgent onward referral. In particular we are concerned about cancer, systemic disease, fractures, cauda equina syndrome and cord signs – for further description of these pathologies, see Chapter Twelve. The questions do not allow diagnosis of these problems, but may lead to suspicion of them, especially when response to mechanical evaluation is atypical. Sometimes it may be clear just from the history alone that sinister pathology must be considered in the differential diagnosis, in which case no further testing should be done. At any point if any of these specific pathologies are suspected then specialist referral is indicated, and in the case of suspected cauda equina syndrome this is urgent.

Serious spinal pathology accounts for less than 1% of all back pain (Waddell 1998; CSAG 1994). Inflammatory arthropathy also accounts

for less than 1% of all back pain. These specific conditions are rare, but they will be missed if they are not at least considered. This is the basis of the triage system – to filter out those few individuals who need further investigation and referral. At this point exact diagnosis is not necessary, but suspicion of serious spinal pathology demands action. A simple screening process can be conducted if a few factors are considered.

Deyo *et al.* (1992) and Waddell (1998) identified a few key features that distinguished patients with serious spinal pathology. These came to be termed 'red flags' (*AHCPR* 1994; CSAG 1994). The physical examination is less useful in identifying serious spinal pathology than certain features to be discovered in the history-taking. Most simple backache affects the lumbar region, varies with time and activity, especially affects the middle-aged and does not impact upon general health. Serious spinal pathology has the opposite features and offers warning signs in the history (Waddell 1998).

Table 14.4 Features of history ('red flags') that may indicate serious spinal pathology

- Age. Those under 20 and those over 55 with a new onset of back pain should be considered carefully. Structural problems such as spondylolisthesis should be considered in the young group; tumours or osteoporosis are more likely in the older group. Approximately 80% of patients with cancer are over 50 years of age (Deyo *et al.* 1992).
- Non-mechanical back pain. Normally back pain varies over time and with different activities. Some movements make the pain worse and some make it better. In contrast, non-mechanical back pain is often unrelated to activity, often gets gradually worse, rest or exercises do not relieve it and pain may be worse at night. Most patients with cancer-related back pain report that it is unrelieved with recumbency (sensitivity >0.90), but the finding is non-specific (Deyo *et al.* 1992)
- Thoracic pain may be a warning sign.
- Violent trauma. Considerable force may cause a fracture. Postmenopausal women or patients on systemic steroids may incur fractured vertebra with relatively trivial injuries.
- Long-term use of steroids can cause bone weakening, and immune suppression drugs can predispose to infection.
- Previous medical history. A previous history of cancer has such a high specificity (0.98) that such patients should be considered to have cancer-related back pain until proven otherwise (Deyo *et al.* 1992). However, only about one-third of patients with cancer have had cancer previously (sensitivity 0.31).
- Systemic symptoms. However severe the back pain, a patient with ordinary backache is well. If the patient is generally unwell, suffering from malaise, fever or unintentional weight loss, suspicion should be raised.

- Persistent severe restriction of lumbar flexion. Most features of the physical examination are unhelpful in determining the presence of serious spinal pathology. Severe and persistent restriction of flexion may be a warning sign; however, it is also common in acute disc prolapse.
- A normal x-ray and physical examination do not exclude serious spinal pathology.
- Bladder function. Abnormal bladder function (retention, incontinence or difficulty imitating micturation) is the most consistent feature of cauda equina syndrome (Deyo *et al.* 1992).
- 'Saddle area' anaesthesia. This is another variable feature of cauda equina syndrome. Additionally, unilateral or bilateral sciatica, motor deficit, root tension signs and absent reflexes may be noted.

Suspicion of serious pathology must be made from clinical reasoning based on a thorough history-taking. Van den Hoogen *et al.* (1995) reviewed several articles that evaluated the accuracy of different items of the clinical presentation in diagnosing vertebral cancer. Individual items of history-taking and physical examination in general have low sensitivity or specificity. Combinations of factors are more significant. For instance, in an individual with several of the following features, suspicions should be raised immediately: history of progressive, insidious back pain, unrelieved by recumbency, in an individual over 50, with a previous history of cancer, with unexplained weight loss and failure to improve with conservative therapy. In a series of nearly 2,000 back pain patients, no cancer was diagnosed in any patient who was under 50, without a history of cancer, without unexplained weight loss or who improved with conservative therapy. Conversely, these four features identified all cancer-related back pain with a combined sensitivity of 100% (Deyo *et al.* 1992).

Some of these issues have been covered in previous questions. Additional questions can be introduced if aspects of the presentation demand further enquiry, but the following should always be included:

Is their general health good, fair or poor?

Is there any systemic ill health or malaise?

Is there any history of serious disease? (specifically cancer)

Has there been any major surgery?

Have there been any serious accidents recently?

Is their bladder function normal for them?

What medications is the patient taking for this or any other conditions?

Has there been any unexplained weight loss?

Conclusions

At the end of the first page of the assessment sheet, certain aspects of the patient's presentation have been clearly identified. The clinician must feel happy to continue to explore mechanical therapy and that 'red flag' pathology has been discounted. A hypothetical mechanical diagnosis should be under consideration, or possibly a non-mechanical syndrome. It is important at this point to summarise briefly to the patient the main points in the history from your understanding; this ensures that your impression of their problem accords with their knowledge.

Various issues of prognostic significance will have been explored. This includes the stage and status of the condition, its apparent mechanical sensitivity, and any 'yellow flag' factors concerning the patient's response to the problem. The mechanical sensitivity, as in the aggravating and relieving factors, provides clues for management strategies.

A range of baseline measurements will have been taken against which to judge the effects of subsequent mechanical therapy. These include the site, spread, temporal component of the pain and functional limitations.

Information gained during the history-taking should provide insights into what needs to be explored in the physical examination and how vigorous it can be. For instance, should a neurological examination be conducted? Is there a strong suggestion of a directional preference, or is a protracted period of exploring loading strategies more likely?

15: Physical Examination

Introduction

The mechanical assessment relates closely to the findings from the initial interview. From the history provided by the patient, the clinician should already have gathered sufficient information to be making tentative conclusions about certain aspects of the case. Very often it is clear from the history to which mechanical syndrome the patient belongs, whether the patient has simple back pain or with nerve root involvement and whether there exists a directional preference. Details may also have been gathered that suggest serious spinal pathology, which needs further investigation.

The clinical examination is designed to confirm the initial findings and fully expose the mechanical nature and extent of the problem. The two parts of the first day's assessment should thus produce a good general picture of the patient's symptomatic and mechanical presentations. From these findings will come the optimal management of the condition. The whole assessment provides baseline measures of pain, movement and function against which to judge the worth of any subsequent intervention. The information also gives prognostic indicators, derived from such items as the duration of the problem, previous history, the age of the patient or the constancy of the pain.

The physical examination involves various observations and movements about which the clinician must make judgements. It should be remembered that such perceptual tests, in which a human being is the measuring device, are bedevilled by variability of results. Intraobserver and interobserver variability is seen as the inevitable consequence of such perceptual tests (Gray 1997). Although we cannot totally prevent this phenomenon, we can limit its impact by conducting the examination in the same way each time it is done. Clinicians need to perform tests consistently on each occasion, and patients must always start from the same position – in this way we can be more certain that different test results reflect changes in the mechanical presentation rather than being the fault of inconsistent examination technique.

Given that a high level of observer error is present during the taking of the history and the performance of the physical examination, it

has been proposed that if a lot of detailed information is sought much of it will be unreliable (Nelson *et al.* 1979). If the examination is limited, the reliability of the information gathered is better. It is especially important not to complicate the physical examination with an excessive number of tests and movements. Multiple tests have a high probability of generating unreliable information, and may only serve to confuse rather than enlighten the examining clinician.

Sections in this chapter are as follows:

- aims of physical examination
- sitting posture and its effects on pain
- standing posture
- neurological tests
- movement loss
- repeated movements
- examination of repeated movements
- examination of sustained postures
- testing inconclusive
- other examination procedures.

Aims of physical examination

During the physical examination the following points should be exposed:

- usual posture
- symptomatic response to posture correction
- any obvious deformities or asymmetries that are related to this episode
- baseline measures of mechanical presentation
- neurological examination
- symptomatic and mechanical response to repeated movements.

The following conclusions should be made:

- syndrome classification

- appropriate therapeutic loading strategy, *or*
- appropriate testing for loading strategy.

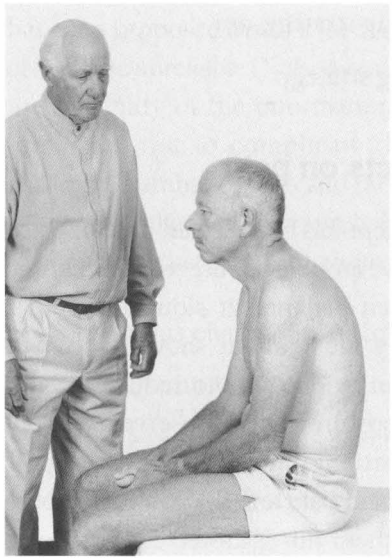
Sitting posture and its effects on pain

If during the history-taking the patient has been seated unsupported on a plinth or examination couch, we are able to observe their natural unsupported seating posture. Often patients sit slouched or in a posture of flexion; sometimes they move about showing obvious discomfort in that position, or lean to one side to reduce weight-bearing on the painful side. The posture can be observed without the patient's awareness. Some patients are more aware of the relationship between their posture and pain and make an attempt to sit upright as experience has told them this is easier. Such patients are few.

An important component and often helpful indication of the nature of the patient's problem can be obtained immediately following the interview process. At the conclusion of the interview, they have been sitting in a relaxed posture for fifteen to twenty minutes. Very often this means they have been in a posture of sustained flexion for that length of time. It is valuable at this point to test the effect of posture correction on their symptoms. As when evaluating any mechanical loading strategy on the symptomatic presentation, the present level of symptoms should first be noted. 'While you are sitting here now, do you have back pain?' If symptoms are present, we also wish to know where they are located, as well as the most distal extent of any referred pain. Enquiry may reveal a gradual worsening of symptoms while they have been sitting.

Posture correction is then performed by the clinician, which ensures the lumbar spine is pulled into extension (see procedure 4, Chapter 17). Ask the patient, 'What effect does that have on your pain? Does it make it better, worse or have no effect?' If referred symptoms are present, we wish to know the effect of posture correction on the most distal area of pain. Symptom response will vary. Often patients report an easing or centralising of back or referred symptoms. This should confirm a directional preference that has already been exposed during the history-taking and is also is a useful teaching tool for the patient. Sometimes posture correction aggravates symptoms, and less often has no effect. Each of these responses often parallels information

Photos 2, 3, 4:
At the conclusion of the history and while the patient is still slouched, ask, 'In this position, what pain do you have?' Record the location (2). Then press the patient's upper sternum, at the same time pulling the lower back into lordosis, thus guiding the patient into a lordotic sitting posture (3). Having corrected the patient's flexed posture, ask, 'What do you feel in this position?' Record the effects of posture correction (4).



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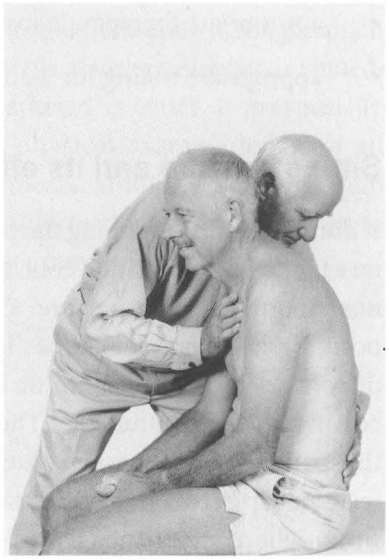

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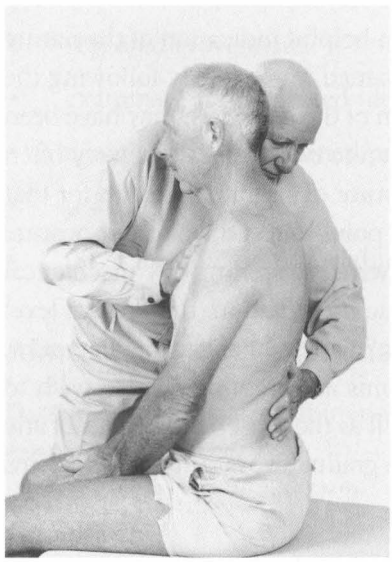

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that the patient has already provided about aggravating and relieving factors. Whatever the response, knowledge is provided about their response to loading strategies that helps to provide the appropriate management.

After prolonged sitting, the effect of restoring the lordosis may be to centralise, reduce or increase the pain. In more severe, acute cases, posture correction may aggravate symptoms, as the weight-bearing nature and speed of application of extension is a problem rather than the direction itself. On other occasions it could be an early indication of the need to explore frontal plane movements. The increase of pain can be due to the speed of adopting the extended posture – a useful check is to compare the response to posture correction after fifteen minutes of sitting to the response after a series of extension exercises. It will

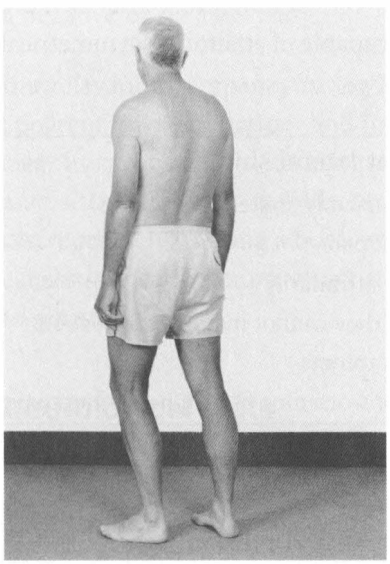
The variability of symptom response only occurs in derangement syndrome when posture correction may reduce, abolish, centralise,

increase, produce or peripheralise symptoms. In posture syndrome, posture correction abolishes symptoms immediately or within a minute or two, which will not return during the rest of the physical examination. In extension, dysfunction syndrome posture correction may produce symptoms, and in flexion dysfunction posture correction may abolish symptoms.

Standing posture

The patient is then asked to remove their shoes, expose their lower back and stand facing away from the examining clinician. They may display problems with curve reversal upon moving from the flexed sitting posture to the erect standing position. A consistent position will be obtained if they are asked to stand with their legs apart and their arms relaxed by their side. Observation, along with palpation, is one of the least reliable aspects of examination (Kilby *et al.* 1990; Donahue *et al.* 1996); therefore any observed minor abnormality should always be used as part of the overall examination findings and never taken on its own to justify management. The more extreme cases of lateral shift or lumbar kyphosis, which *will* determine treatment direction, are apparent and cannot be missed. The following features are observed.

Photo 5: Standing posture. Observe standing posture: check lordosis, check for presence of lateral shift, ignore minor asymmetry.



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Lordosis

The most common deviation from normal is the flattened lumbar spine or reduced lordosis (McKenzie 1981). Much less commonly, the lumbar lordosis is accentuated. In especially severe and acute cases, sometimes the patient has been forced into a posture of kyphosis by the problem, and they are unable to tolerate standing upright at all.

Lateral shift

A departure from the midline, causing a lateral shift, may be detected. This can be noted from the sagittal alignment of the

lumbar spine, and, sometimes more easily, by the distance between the trunk and the arms, which will be symmetrical.

When the upper part of the body is shifted to the right relative to the lower part, that is, the vertebra above has shifted right carrying the rest of the body with it, this is described as a right lateral shift (McKenzie 1981). The converse situation is a left lateral shift.

A lateral shift may be present for several reasons. It could have always been present and not be related at all to the present episode of pain – a congenital anomaly may be the cause, or there may be some remote mechanical asymmetry, such as leg length inequality or pelvic tilt. Those who have a congenital scoliosis are at no greater risk of developing back pain than those without; asymmetry is not a cause of back pain (Dieck *et al.* 1985).

It is important to determine if it has always been present or if the shift came on at the same time as the back pain. Derangement of intervertebral discs is believed to be the cause of shifts related to back problems (O'Connell 1943, 1951; Spurling and Grantham 1940; Falconer *et al.* 1948). However, it should also be noted that very rare causes of non-mechanical back pain, such as osteoid-osteoma and discitis, are also associated with rapid onset scoliosis (Keim and Reina 1975; Greene 2001). If the shift came on at the same time as the back pain, and therefore is related to it, we need to know if it is voluntarily maintained or if the patient is unable to correct it. Sometimes patients stand with their weight shifted to one side because this is more comfortable, but they are perfectly capable of attaining a symmetrical posture. This is not a lateral shift.

Table 15.1 Criteria for a relevant lateral shift

- upper body is visibly and unmistakably shifted to one side
- onset of shift occurred with back pain
- patient is unable to correct shift voluntarily
- if patient is able to correct shift, they cannot maintain correction
- correction affects intensity of symptoms
- correction causes centralisation or worsening of peripheral symptoms.

Leg length inequality

There are various manual methods of measuring leg length, such as palpating or comparing levels of the iliac crests and skin creases at the buttocks and knees, or measuring and comparing the length from

anterior superior iliac spine to medial malleolus with the legs in neutral (Corrigan and Maitland 1983). Such methods are unreliable and have been associated with errors of up to 1cm (Friberg 1983). The only reliable way of assessing leg length inequality is radiography of the pelvis.

In a thousand asymptomatic subjects 7% were found to have a discrepancy of half an inch or more, when assessed clinically, while the prevalence in a smaller number of back pain patients was 22% (Nichols 1960). Friberg (1983) found discrepancies of over 5mm, measured radiographically, in 75% of patients and 43% of controls. However, other studies have refuted the relevance of differences in leg length to back pain. No significant differences have been found in the proportion of those with leg length inequalities in groups with and without back pain (Soukka *et al.* 1991; Pope *et al.* 1985). Differences greater than 10mm were equally distributed in those with disabling back pain as well as those without a history of back pain. In a case-control study with 140 subjects, lower limb and pelvic asymmetries were as common in the control group as in the back pain group (Grundy and Roberts 1984).

There is thus a lack of evidence to substantiate a definite role for leg length inequality as a cause of back pain. In a review of individual risk factors, Nachemson and Vingard (2000) list eight studies that have found a positive association between a leg length discrepancy of less than 2.5cm and back pain, and nine studies that have found a negative or no association.

If a relevant discrepancy in leg length is encountered it should be apparent on examination, and its relevance to symptoms should be clear from the history. Standing and walking enhances the back pain and sitting relieves it. Where it is relevant, adjustment of the discrepancy by placing a book under the foot for five to ten minutes causes immediate improvement, and removing it causes symptoms to return.

Neurological tests

If a neurological examination is deemed necessary, this is a suitable time to perform it. It is important that a neurological examination be conducted before the instigation of an exercise programme so that

the effect of exercises on nerve root signs and symptoms can be monitored. One should be conducted if nerve root involvement is suspected.

Table 15.2 Criteria for conducting a neurological examination

- paraesthesia in the leg
- weakness in the leg
- thigh or leg symptoms, especially in a radicular pattern.

Neurological examination may involve four components:

- sensation
- muscle power
- reflexes
- nerve tension tests.

Comparing the perception of light touch with the opposite limb tests sensation. More sophisticated testing can be done using sharp or blunt instruments in the affected dermatome. The distal end of the dermatome is the area that should be tested as this is the area most likely to be affected.

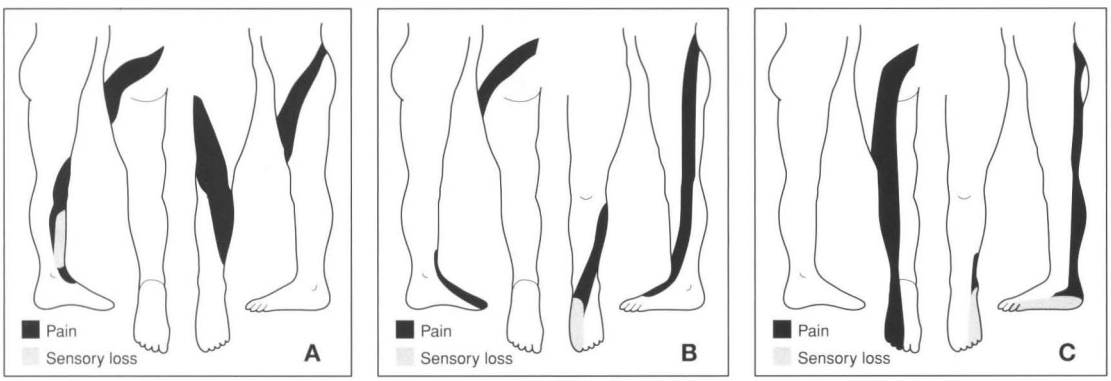
While the patient is standing, they can be asked to walk on their toes (S1) and on their heels (L4). Sitting or standing, extensor hallucis longus (L5) and dorsiflexion (L4) can be tested, and the sensitivity to touch of the medial aspect of the leg (L4), the big toe (L5) and the outer border of the foot (S1) on both legs can be compared. Details of the most important neurological signs and symptoms to be considered are given in Table 15.3 and Figure 15.1. Different authorities give slightly different descriptions of the same segmental level. This is a reflection of the normal variability between individuals and anomalous patterns of innervation (Kadish and Simmons 1984; McCulloch and Waddell 1980). This causes the overlap between signs and symptoms at different segmental levels that is commonly recorded, especially L5 and S1 (Kortelainen *et al.* 1985; Aronson and Dunmore 1963; Falconer *et al.* 1947).

Table 15.3 Typical signs and symptoms associated with L4 – S1 nerve roots

	L4	*L5*	*S1*
Distribution of pain and *sensory loss*	(Anterior thigh) Anterior / *medial leg* (Great toe)	(Lateral thigh) Lateral leg Dorsum of foot Great toe	Posterior thigh Posterior leg *Lateral border of foot* Sole
Motor weakness	Quadriceps Dorsiflexion	Big toe extension Extension of the toes	Plantarflexion Eversion
Reflex	(Knee)		Ankle

Source: Waddell 1998; Nitta *et al.* 1993; Smyth and Wright 1958; Butler 1991; Kramer 1990

Figure 15.1 Typical areas of pain and sensory loss L4, L5, S1



Over 95% of disc herniations occur at the L4 – L5 and L5 – S1 levels, thus the nerves most commonly affected are L5 and S1 (Andersson and Deyo 1996). Upper lumbar disc herniations are rare compared to herniations in the low lumbar spine, but they do occur. Of seventy-three patients with upper lumbar disc herniations, 70% had involvement of L3, 25% involvement of L2 and 5% involvement of L1 (Aronson and Dunsmore 1963). Radiation of pain was primarily over the lateral and anterior aspect of the thigh, and some cutaneous sensory loss was present in about 50% of patients in the same area. Muscle weakness mostly affected quadriceps or psoas, but extensor hallucis longus was occasionally affected. The knee jerk was reduced or absent in 50% of patients.

Movement loss

Initially we are interested in assessing the quality of movement and pain response. Gathering a picture of their mechanical presentation provides a baseline against which to judge response to management at a later date. The range of movement, movement pathway, pain response, confidence and willingness to move, and curve reversal are all considered.

If pain is present with the movement, we may also wish to know if the pain is present *during* the movement or at *end-range*. Ask the patient, 'Do you have pain as you move, or at the end of movement?' If movement is less than expected, we also wish to know if it is limited by pain, or by stiffness – the patient is simply unable to move further, but it is not pain that prevents the movement.

Single movements provide an inadequate mechanical evaluation by themselves and rarely provide enough information to decide on the correct loading strategy. Most importantly, in terms of management we wish to know the effects of repeated movements and any lasting changes that result in the patient's symptoms. Only when movements are repeated do symptom responses become clear. Repeated movement testing is discussed below.

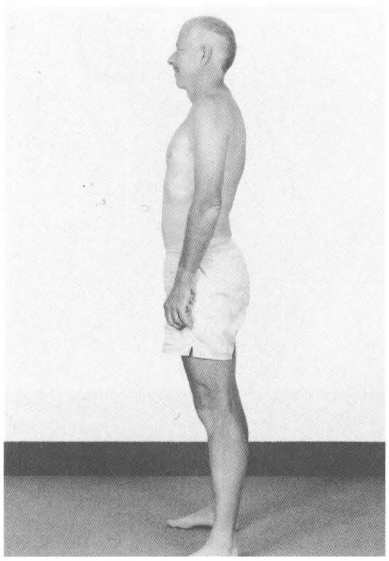
The patient stands with feet apart so that they have a good base of support, and one movement is performed in each direction. It is best to give verbal instruction and to demonstrate to the patient what is required in each case. The following movements are examined

Flexion

The patient, standing with feet shoulder-width apart, places their hands on the front of their thighs. Then, maintaining knee extension, they run their hands down the front of their legs. They are encouraged to go as far as possible – 'further, further, further'. They then immediately return to neutral standing. The distance that the hands reach down the legs gives an accurate baseline measure of functional flexion and should be noted. We ask if the movement is painful, and if so, 'Is it painful as you move or only at the end of movement?' We also ask, 'Is it pain that stops you, or you just can't go further?' If it is painful, we need to know where the pain is felt.

Loss of flexion should be recorded as major, moderate or minimal. Gauging the degree of loss is dependent partly on the patient's normal

Photos 6, 7: Range of movement in standing - flexion.
Assess flexion before extension. Ensure patient achieves maximum flexion. 'Further, further, further'



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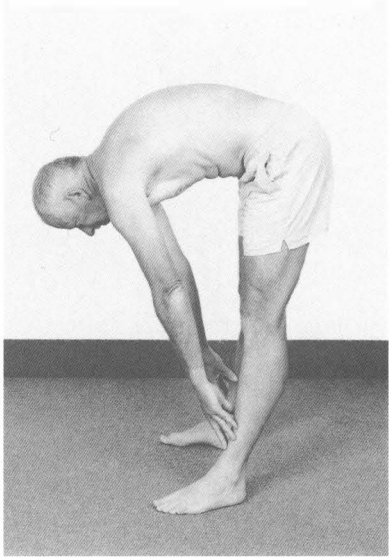

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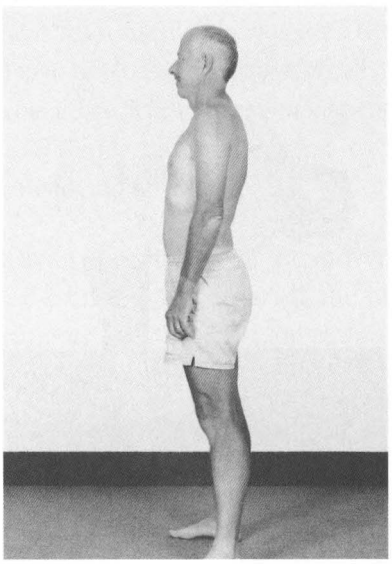
range of movement, which should be enquired about. Not everyone can touch his or her toes. An approximate level of impairment is as follows: a major loss means they are unable to reach to their knees, a moderate loss they reach to about their upper shin and a minimal loss they reach to their lower shin. Flexion range also depends on the time of day, as range increases from morning to afternoon by over ten degrees, with the biggest change occurring in the first part of the day – in comparison, extension range is independent of time of day (Ensink *et al.* 1996).

The other way that a loss of flexion manifests itself is a deviation from the normal sagittal pathway as flexion is performed. Any asymmetrical impediment to flexion may cause the spine to take the path of least resistance, resulting in a detour to the right or left. This may occur in an arc-type movement, the flexion commencing and ending in midline; or, once movement is commenced, it may divert from the midline and increase its departure as long as flexion is continued. Very often if the deviation is prevented and the patient held in the sagittal plane, a considerably greater loss of flexion is observed with a concomitant increase in pain. Deviation and limited range may co-exist.

Photos 8, 9: Range of movement in standing – extension.
Assess extension after flexion. Patient should achieve maximum extension. 'Further, further, further'. Ensure patient returns to upright position immediately.



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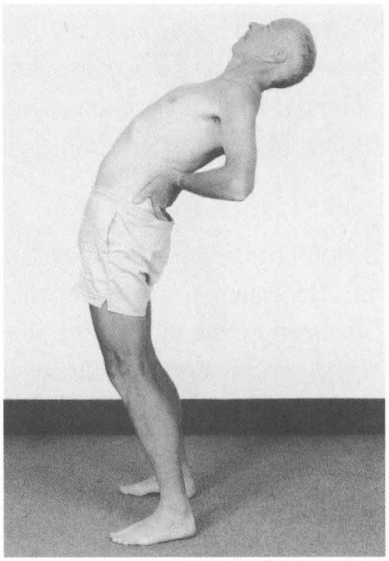

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Deviation in flexion

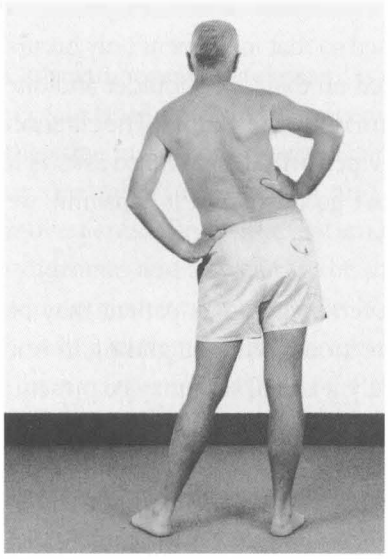
McKenzie (1981) identified three causes for deviation in flexion. As the mechanism is different in each case, the treatment must vary accordingly.

- Derangement. In the absence of nerve root irritation, deviation in this instance generally occurs away from the painful side. However, in some patients deviation may vary – one day to the right, one day to the left.
- Dysfunction. This develops following repair after derangement and limitation of flexion. Scarring prevents flexion in the sagittal plane, whereas deviation occurs either towards or away from the painful side, but never varies.
- Adherent nerve root. In this situation, following the resolution of sciatica, adhesions or scarring about the nerve root prevents flexion from occurring in the sagittal plane. Adherence of the nerve root now acts as an anchor and pulls the patient towards the side of the adherence; deviation and limitation of flexion may be very severe and always occurs towards the painful side.

Extension

With feet about shoulder-width apart to provide a good base of support, the patient is asked to place his hands in the small of his

Photos 10, 11: Side gliding in standing. Check side gliding movement in both directions.



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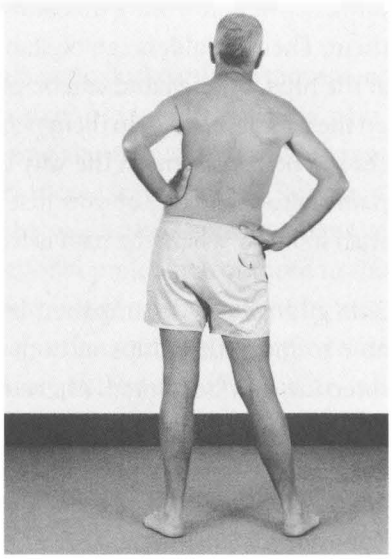

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back and bend backwards as far as possible, letting the head arch backwards at the same time. They are encouraged to go as far as possible – 'further, further, further'. They then immediately return to neutral standing. We ask, 'Is it pain that stops you, or you just can't go further?' If it is painful, we need to know where the pain is felt.

Impairment of extension is harder to quantify than flexion, but gross movement loss is reasonably obvious. Again, loss should be recorded as major, moderate or minimal. Deviation may occur on extension, but this is less common than with flexion. This is generally the result of derangement.

Side gliding

Movement in the frontal plane is assessed best by examining lateral gliding of the hips (McKenzie 1981). During this manoeuvre, upper lumbar movement is restricted and the focus is on lower lumbar movement, whereas during side bending or lateral flexion most of the movement occurs at the upper lumbar segments (Mulvein and Jull 1995). When the hips are taken to the left, right side gliding or lateral translation occurs; when the hips are taken to the right, left side gliding or lateral translation occurs.

On completion of ten to fifteen movements the patient is told to relax, and after a minute or two they are questioned again about their symptom status. At this point we wish to know if the area or the severity of symptoms has changed or if pain that was present before has been abolished, or if pain that was not present before has been produced. Afterwards the patient is either 'better', 'no better', 'worse', or 'no worse'. If a patient reports no pain prior to testing, but an improvement in mechanical or symptomatic response occurs during repeated movements, this should also be recorded as 'better' afterwards, and the details noted.

Repeated movements in derangement syndrome

In derangement syndrome, repeated movements in the direction that produces greater deformation of spinal structures will produce, increase or peripheralise the symptoms, and frequently may additionally cause an obstruction to movement. The performance of movements in the opposite direction reduce deformation of those structures, cause reduction in the derangement and bring about an abolition, decrease or centralisation of symptoms. In this syndrome movement is usually impaired, but performance of the appropriate repeated movement brings about a recovery of all movements, not just the one being repeated. Thus, repeated movements are diagnostic of the derangement syndrome as well as confirming the directional preference of the management strategy, to which clues will have been provided in the interview. Once a repeated movement has been found that decreases, abolishes or centralises symptoms, and/or improves the mechanical presentation, no further testing is necessary and that movement is used in the management strategy.

Very often a definite symptomatic and mechanical response is apparent on the occasion of the first assessment, and the history-taking and physical examination produce a consistent picture. At times the initial response to repeated movements is more equivocal, and after several batches of repeated movement there is no change. In such instances repeated movements should be explored vigorously over the subsequent day(s), and other force progressions considered, until elucidation of directional preference is produced.

Repeated movements in dysfunction syndrome

In articular dysfunction syndrome, repeated movements in the direction that puts tension on adaptively shortened structures produces end-range pain on every occasion it is performed.

Alternatively, repeated compression of structurally impaired tissue could consistently reproduce the patient's symptoms at end-range. Repeated movements will not make the patient progressively worse. When they return to the neutral position
will pain be peripheralised. On every subsequent occasion the same exercise will generate the same response. There will be no rapid change in range of movement. Thus, repeated movements are diagnostic of dysfunction syndrome and also reveal the movement that requires repetition to remodel adaptively shortened tissues.

Repeated movements in postural syndrome

Patients with posture syndrome experience no pain on any test movements or their repetition, and they display no loss of normal range of movement. Only with sustained positioning do these patients experience their pain.

Selecting repeated movements

It is assumed that the movements having the greatest influence on levels of pain will also have the greatest influence on the pathology and pain generator and can be used to improve or worsen the condition. The movements that usually have the greatest effect on pain are flexion and extension, although sometimes it is side gliding. For this reason the repeated movement testing initially only uses sagittal plane movements. Except in the case of an obvious lateral shift, where frontal plane movements are necessary and sagittal plane movements are initially undesirable, sagittal plane movements are always explored first. Frontal plane movements are introduced if sagittal plane movements worsen or peripheralise symptoms. Lateral forces may also be introduced if sagittal plane forces do not improve the symptomatic or mechanical presentation. Different effects are produced when the test movements are performed in standing compared with lying positions.

Once a repeated movement decreases, abolishes or centralises pain, and thus it is apparent that the directional preference has been located, further testing is unnecessary.

Repeated movements – flexion in standing compared to flexion in lying

Flexion is examined in standing and in lying. There are several differences between these two manoeuvres: first the effect of gravity is far greater in standing, which can make that movement much more difficult in more severe cases. In flexion in standing the

movement occurs from the top down (Harada *et al.* 2000), whereas in flexion in lying it occurs from below upwards.

Flexion in lying exerts a stronger stretch, especially at L5 – S1, with the passive overpressure provided by the arms. Patients with a flexion dysfunction describe a stretch pain in lying that they may not experience with flexion in standing. As tension on the lower lumbar segments occurs much more quickly with flexion in lying, pain is produced rapidly and then increase as more pressure is applied. In flexion in standing, the same segments do not come under tension until near the end of physiological flexion, and pain is experienced at the end of movement.

The effects on the sciatic nerve roots are also different between the two manoeuvres. In flexion in standing, as long as knee extension is maintained, the sciatic nerve comes to be placed on full stretch, producing effects identical to those obtained in a straight leg raising test (McKenzie 1981). Flexion in lying, performed with hip and knee flexion as described, has the opposite effect on root tension – in this position nerve roots are relaxed and a problem with root adherence or root tension will not be identified.

In flexion in standing, enhancement of pain may be caused by a derangement or an adherent nerve root. However, aggravation of pain in flexion in lying can only be caused by a derangement. This simple test can be used to differentiate whether sciatic pain results from an adherent nerve root or from a derangement (McKenzie 1981).

In the presence of increasing peripheral pain or peripheralisation, continuing repetitions of flexion should not be performed as a worsening of symptoms may ensue. Very often the patient's history makes it clear that sustained flexion, such as sitting or driving, makes their symptoms worse. In such instances a protracted testing of flexion is unnecessary.

Repeated movements – extension in standing compared to extension in lying

Extension is also examined in standing and lying. The different manoeuvres, as with flexion, can produce markedly different symptomatic responses. The gravitational forces are different in the two positions. In extension in lying the force is almost perpendicular to the plane of the motion segments and, coupled with the weight of the pelvis and abdomen, a maximal mechanical effect is produced.

The greatest extension stretch that a person can apply to his or her own back is achieved in this position. In extension in standing, the gravitational force acts on the segments at an angle of forty-five degrees from the perpendicular and therefore is less efficient. The extension range and stretch are never as complete in this position.

Sometimes in the presence of the compressive force that occurs with extension in standing, range can be limited and symptoms can be aggravated. However, reduction of the same derangement becomes possible in extension in lying with the compressive force removed, when a gradual accomplishment of full range is achieved.

Because of these and possibly other unknown variables, the differences in symptomatic response between a manoeuvre performed in standing and lying can be marked. A patient can be made worse by repeated extension in standing, but better when extension is performed in lying. Flexion in standing can worsen a patient, while repeated flexion in lying has no effect.

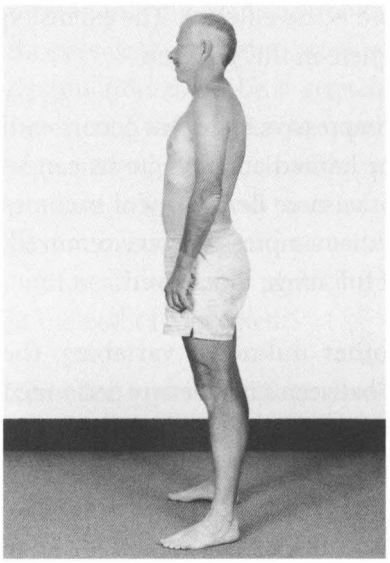
Examination of repeated movements

In general, all patients should perform the sagittal plane test movements. If it is clear from the history that flexion activities produce or worsen the patient's symptoms, it is unnecessary to test repeated flexion extensively, or even at all. In the case of patients with major acute derangements and severe symptoms, prolonged testing should be avoided. Apart from the exception of lateral shift already noted, the sagittal plane movements should be performed in the following order.

Flexion in standing

The pain status of the patient in neutral standing is recorded prior to the performance of the repeated movements. The patient, standing with feet shoulder-width apart and maintaining knee extension, is asked to run his or her hands down the front of the legs as far as pain allows, and then immediately return to neutral standing. The effects on the pain of one movement of flexion are recorded, and the movement is then repeated up to ten times, with the maximum possible stretch being obtained during the last few movements. The effect on pain while the movements are being performed is noted, and whether pain is felt during the movement or at end-range, as well as if there is improvement in the mechanical or symptomatic response during the repeated movements. Most importantly, the pain

Photos 12, 13: Effects of repeated movements: flexion in standing. After determining pre-test level and location of most distal symptom, test effects of repeated movement in standing, flexion first. 'Further, further, further.' Record effect on level and location of pain.



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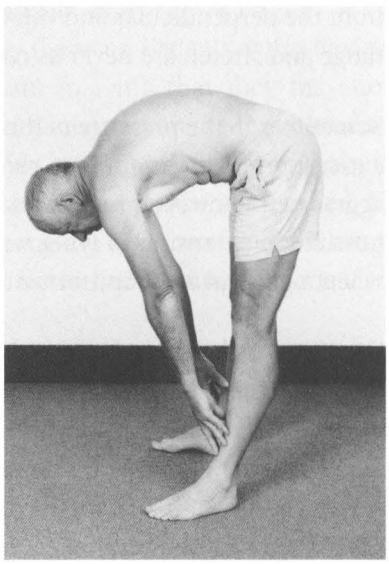

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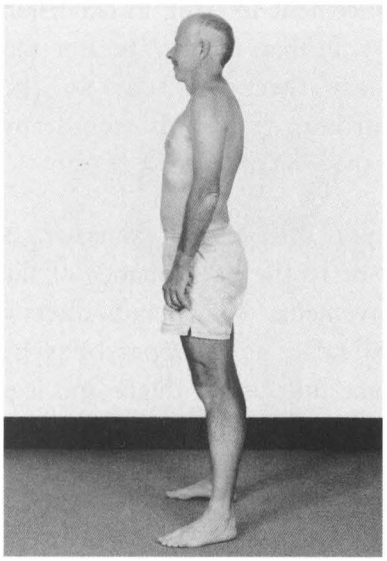
status is then recorded a minute or two *after* the completion of the repeated movements. Note is made of any changes in range that accompany the repeated movements.

Extension in standing

The pain status of the patient in neutral standing is recorded prior to the performance of the repeated movements. The patient stands with feet shoulder-width apart for a good base of support, with hands in the small of the back to act as a fulcrum. He or she then bends backwards as far as possible, letting the head drop backwards also, and then returns to neutral standing. The test movement is performed once and its effect on pain is recorded. The patient is then asked to repeat the movement up to ten times, ensuring that the maximum possible stretch is obtained during the last few movements. The effect on pain while the movements are being performed is noted, and whether pain is felt during the movement or at end-range, as well as if there is improvement in the mechanical or symptomatic response during the repeated movements. Most importantly, the pain status is then recorded a minute or two *after* the completion of the repeated movements. Note is made of any changes in range that accompany the repeated movements.

Photos 14, 15: Effects of repeated movements: extension in standing. After determining pre-test level and location of most distal symptom, test effects of repeating movement in standing, extension second. 'Further, further, further.' Afterwards record effect on level and location of pain.



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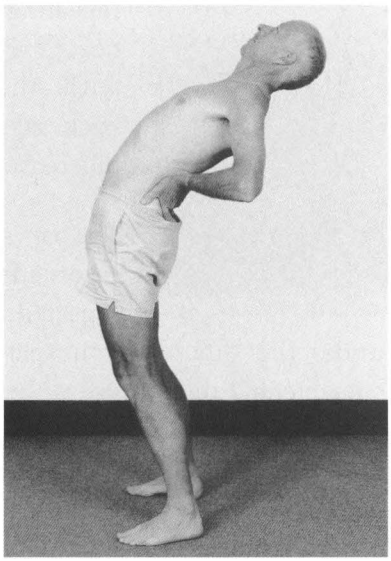

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Flexion in lying

The pain status of the patient in crook lying is recorded prior to the performance of the repeated movements. From supine with hips and knees bent so the feet rest flat on the plinth, the patient brings the knees up to the chest. The knees are clasped with the hands and a firm overpressure is applied to produce maximum possible lumbar flexion. The legs are then returned to the starting position and the effect of the movement on the pain is recorded. The patient is then asked to repeat the movement up to ten times, ensuring that the maximum possible stretch is obtained during the last few movements. The effect on pain while the movements are being performed is noted, and whether pain is felt during the movement or at end-range, as well as if there is improvement in the mechanical or symptomatic response during the repeated movements. Most importantly, the pain status is then recorded a minute or two *after* the completion of the repeated movements. Note is made of any changes in range that accompany the repeated movements.

Photo 16: Effects of repeated movements: flexion in lying. After determining pre-test level and location of most distal symptom, test effects of repeating movement in lying, flexion first. 'Further, further, further.' Afterwards record effect on level and location of pain.

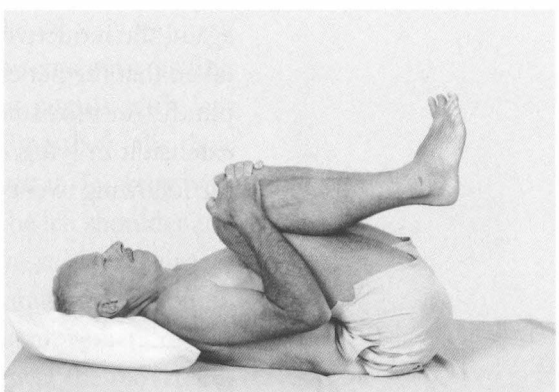

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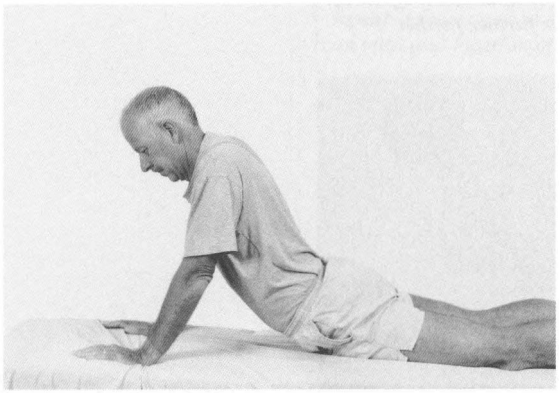
Photo 17: Effects of repeated movements: extension in lying.



17

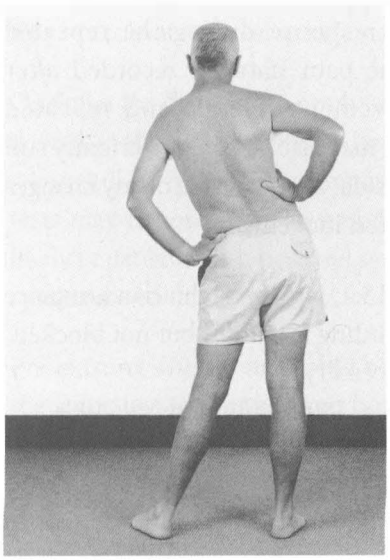
Extension in lying

After determining pre-test level and location of most distal symptom, test effects of repeating movement in lying in extension next. 'Further, further, further.' Breathe out with the last three of ten, so the back sags further. Afterwards record any difference in the level or location of pain.

The pain status of the patient in prone lying is recorded prior to the performance of the repeated movements. With hands directly under the shoulders, the patient raises their upper body by straightening their arms, at the same time as the thighs and legs remain on the plinth. The patient is encouraged to concentrate the bending movement at the low back. They then return to the starting position and the effects of the movement on the pain are recorded. The patient is then asked to repeat the movement up to ten times, ensuring that the maximum possible stretch is obtained during the last three or four movements. To do this the patient is encouraged to attain full elbow extension as the arms are straightened, and at the same time allow the body to sag at the lower back. The effect on pain while the movements are being performed is noted, and whether pain is felt during the movement or at end-range, as well as if there is improvement in the mechanical or symptomatic response during the repeated movements. Most importantly, the pain status is then recorded a minute or two *after* the completion of the repeated movements. Note is made of any changes in range that accompany the repeated movements.

If symptoms that were abolished recur after the patient has stood up again, the reductive process should be repeated and care should be taken that the patient maintains the lordosis as they get up from the plinth. An assessment should be made of the effects of repeating extension in lying, or whatever was the reductive movement, over the following twenty-four hours with attention to posture.

Photos 18, 19: Effects of repeated movements: side gliding in standing.
After determining pre-test level and location of most distal symptom, test effects of repeated side gliding if sagittal testing is inconclusive.
First hips away from the painful side. Afterwards record effect on level and location of pain (18). Then, if necessary, towards the painful side (19).



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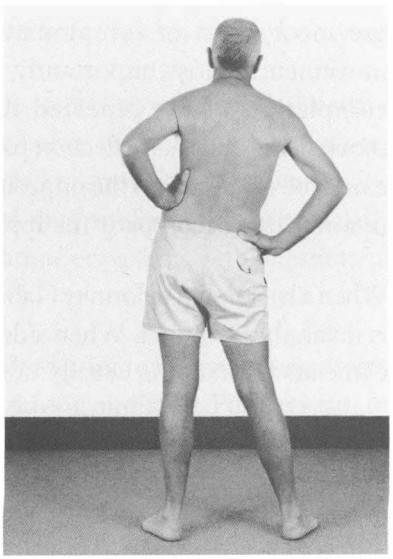

19

Side glide in standing

This manoeuvre will not be routinely done as a repeated movement, but always as a single test movement to gain a baseline impression of frontal plane movements. Frontal plane repeated movements *may* be required on the first day's assessment if there is lack of response to pure sagittal plane movements. They *are* required on the first day if there is a clear indication of a relevant lateral component (Table 15.4). Either a clear lateral shift might be present (see Table 15.1) or it rapidly becomes apparent that sagittal loading strategies cause symptoms to worsen or peripheralise.

The clinician should demonstrate to the patient what is required and assist them in the manoeuvre if necessary as some patients find it difficult to perform this action. The patient stands with feet shoulder-width apart. To perform right side gliding they take their hips to their left while their trunk remains in neutral. To assist with this movement, the clinician places one hand on the left shoulder and the opposite hand on the right iliac crest, and presses both towards the midline. The shoulders should always remain parallel to the ground. One movement is performed and the effect of the movement on the pain is recorded. The patient is then asked to repeat the movement up to ten times, ensuring that the maximum possible stretch is

obtained during the last few movements. To do this, overpressure can be added to the patient's opposite shoulder and iliac crest to generate a 'squeezing' effect. The effect on pain while the movements are being performed is noted, as well as if there is improvement in the mechanical or symptomatic response during the repeated movements. Most importantly, the pain status is recorded *after* completion of the repeated movements. Single and repeated movements and their effect on pain may also, where uncertainty still exists, be examined to the opposite side. Note is made of any changes in range that accompany the repeated movements.

When a lateral shift deformity (Table 15.1) is present, clinician assistance is invariably required. When side gliding is painful but not blocked, clinician assistance is usually unnecessary.

Symptoms may initially require sagittal plane forces, and then when improvements plateau, lateral forces. Equally the opposite may occur, that frontal plane forces are first required to centralise or reduce symptoms, but when improvements cease, sagittal plane forces must be introduced.

Table 15.4 Criteria for a relevant lateral component

- acute lateral shift deformity or loss of frontal plane movements *and/or*
- unilateral / asymmetrical symptoms
- symptoms fail to improve with sagittal plane forces *or*
- symptoms worsen with sagittal plane forces *and*
- symptoms improve with frontal plane forces.

Examination of sustained postures

Besides [illegible] of directional preference is elusive, static tests or sustained postures also need to be explored. The effect of prolonged loading at end-range while sitting and subsequent correction of posture should be routinely done and is discussed above. Other static tests are used as appropriate from the patient's history and physical examination. Long sitting, for example, sometimes provokes sciatic pain where that symptom is felt intermittently. Sustained end-range extension is a provocative test procedure used in patients who are suspected of having an anterior derangement.

Some derangements respond better to sustained rather than repeated movements, usually when time is an important factor. Static positions can be very important procedures in derangements that require a sustained time element in their reduction. For instance, some patients may not tolerate extension in lying, but improve with static prone lying.

One clue for the need to apply static tests is revealed when a patient describes their symptoms as being better when moving and worse when still. If repeated movements fail to provoke symptoms, static tests may expose the true nature of the problem. This is especially likely in patients with postural syndrome or patients with intermittent symptoms in derangement.

Pre-existing symptoms should be noted prior to the patient assuming the static test and monitored both during and after. Postures can be sustained for up to five minutes. Static mechanical evaluation can be conducted in the following positions:

- sitting slouched
- long sitting
- sitting erect
- standing slouched
- standing erect
- lying prone in extension.

Testing inconclusive

If testing has not produced a clear symptom response, so the patient is no better or unaffected by extension in lying, certain procedures may help to clarify a directional preference on day one. Overpressure may be applied in extension to see if this generates a favourable response. The patient lies prone and relaxed, the therapist hand placement is as for extension mobilisation (procedure 7), and a gradual slow increase of extension pressure by weight transference is made until the patient is able to identify a response. 'When I apply more pressure, do you feel more pain, less pain or is there no effect?' A favourable response indicates extension, otherwise the lateral component should be explored.

Photo 20: Extension overpressure.
If testing extension is inconclusive, determine if more pressure causes more or less pain. Apply an extension overpressure. If more pressure causes less pain, progressions are indicated. If more pressure causes more pain, other levels may be tried or force alternatives may be required.

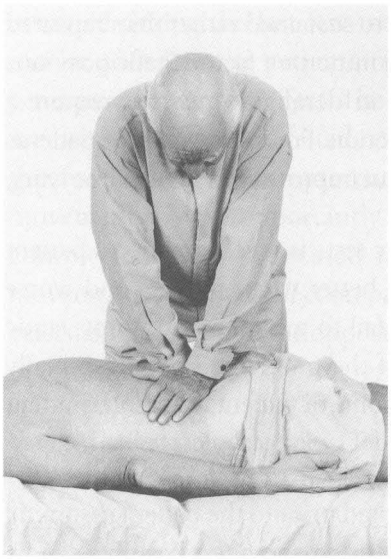

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Photo 21: Extension in lying with hips off centre.
If testing is inconclusive and pain unilateral asymmetrical, place hips off centre, away from the side of pain and repeat extension in lying.

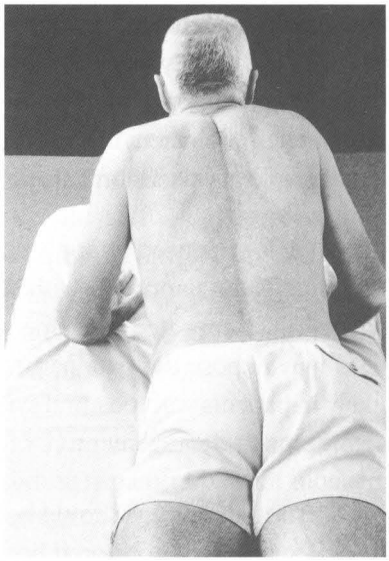

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Photo 22: Extension in lying with hips off centre with lateral overpressure.
If necessary, the clinician can apply overpressure to retain the position of the hips during the extension procedure.

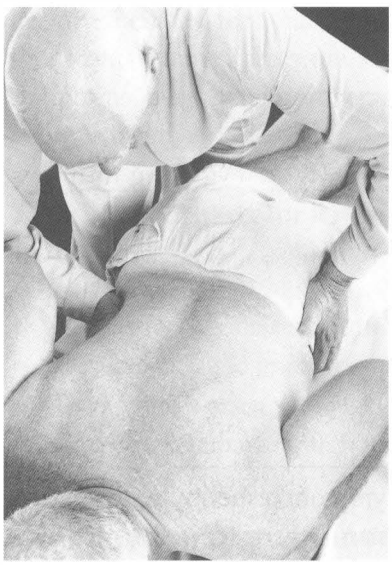

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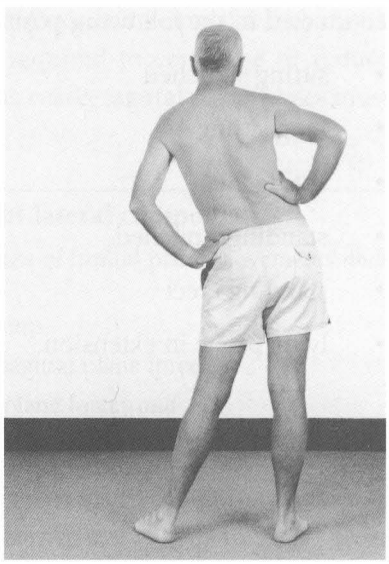
Photo 23: Side gliding.
A lateral force may be applied in standing if this appears inadequate in lying. Repeat side gliding in standing, move hips away from the side of pain.



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If patients have unilateral or asymmetrical symptoms, the lateral component can be explored and again overpressure can be applied.

If a definite symptomatic or mechanical response has not been elicited during the physical examination despite application of the above strategies, then the mechanical evaluation may be continued with the patient performing a specific loading strategy at home. Patients should be told about the expected symptom response and warned about peripheralisation. If the patient's condition is severe or worsening,

they should be reviewed within twenty-four hours. Frequently when patients have repeated an exercise regularly over a day or two a more distinct response occurs, and this becomes clear at review. The presence of a mechanical syndrome should be determined within five treatment sessions; often it will be much sooner.

Other examination procedures

At this stage further examination procedures should not be used until a thorough mechanical evaluation, which includes a trial of therapeutic exercises and use of force progressions, has been conducted over several days. *If multiple other tests are carried out on day one, numerous false-positive results can be generated.* If mechanical evaluation has allowed the elucidation of a management programme, further testing is irrelevant anyway.

Palpation adds very little to a mechanical evaluation and is rarely needed. Palpation is consistently of poor reliability between clinicians and thus is not a firm ground to base management strategies upon. Multiple studies have found poor reliability of palpation, especially when compared to pain response. The relevant literature is summarised in Chapter 11. Furthermore, the early forces used are patient-generated forces, which are not segmentally specific, and therefore palpation is not relevant or useful.

If non-lumbar sources of pain, such as the hip or sacro-iliac joints (SIJ) are thought to be the cause of symptoms, it is essential that the lumbar spine be categorically ruled out before further testing is pursued. In the event of a negative mechanical evaluation of the lumbar spine and appropriate findings from the history, then the physical examination should include the hip joint and/or SIJ. These conditions are discussed in the appropriate sections in Chapter 13, where the recommended mechanical stress tests are also mentioned. For more detail on the process of analysing symptomatic and mechanical presentations see Chapter 16, and for the review structure see Chapter 19.

Conclusions

Having listened to the information provided by the patient's history and conducted an appropriate and thorough physical examination, including the relevant repeated movements, certain conclusions can now be drawn.

At this point serious spinal pathology should be excluded from the equation. The patients should have simple mechanical back pain with or without nerve root symptoms. Mechanically we wish to know if the patient has a derangement, a dysfunction or a postural problem. The majority of patients will have derangements, a minority dysfunctions, and a few postural syndrome. In the case of some patients mechanical testing may be inconclusive, and may need to be continued over a few days to reach a definitive conclusion or to determine a non-mechanical source of symptoms. If it is determined that a mechanical syndrome is present, then one of the principles of mechanical therapy is selected for the management strategy.

Mechanical syndromes

A brief summary is presented; see Chapter 5 for more detail on definition, algorithm at the end of this chapter (and in Appendix) and appropriate chapters in section 7 for clinical presentations.

Postural syndrome

Pain from the postural syndrome in the spine is caused by mechanical deformation of normal soft tissues arising from prolonged end-range loading affecting the articular structures.

Dysfunction syndrome

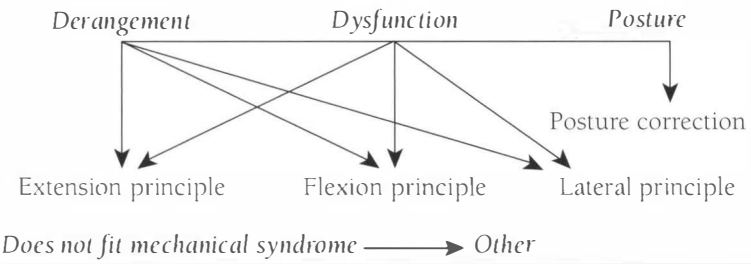
Pain from the dysfunction syndrome is caused by mechanical deformation of structurally impaired tissues. Pain is felt when the abnormal tissue is loaded. In the spine the syndrome presents as articular dysfunction, with pain at limited end-range.

Derangement syndrome

Internal derangement causes a disturbance in the normal resting position of the affected joint surfaces. Internal displacement of articular tissue obstructs movement. Derangement syndrome is characterised by a varied clinical presentation and typical responses to loading strategies. This includes worsening or peripheralisation of symptoms in response to certain postures and movements. It also includes the decrease, abolition or centralisation of symptoms, and

the restoration of normal movement in response to therapeutic loading strategies.

Figure 15.2 Principles of management in mechanical therapy



Derangement

This will be the conclusion in the majority of all patients. Once it is determined that a derangement is present, the key management decision concerns the directional preference. The movement that centralised, decreased or abolished the symptoms during the examination will be the one chosen for the patient to perform. The movements that the patient reported to aggravate or produce their symptoms are those that need to be *temporarily* avoided. The management strategy should evolve out of the findings of the two parts of the assessment. *In the treatment of derangement we must choose the movement that relieves the pain, as this movement decreases the mechanical deformation by reducing the derangement.* Very often the appropriate movement is painful to perform, but the patient feels better afterwards.

The majority of patients with derangement require the *extension principle*. This is applied when extension movements centralise, decrease or abolish symptoms, and the opposite movement, flexion, causes a worsening or peripheralising of symptoms. The extension principle includes a variety of procedures, including extension in lying, extension in standing, prone lying and extension mobilisation.

A smaller group of patients with derangement require the *flexion principle*. This is applied when flexion movements centralise, decrease or abolish symptoms. The flexion principle involves all manoeuvres that utilise this directional tendency, including flexion in lying, sitting and standing, and rotation mobilisation in flexion.

Some patients with derangement require the *lateral principle* or a combination of forces. These are applied when a relevant lateral component is present, when movements in the frontal plane, or with a frontal plane element, centralise, decrease or abolish symptoms. An acute lateral shift deformity may be present, which indicates an immediate need to address the lateral component. Or, in a patient with unilateral or asymmetrical symptoms, pure sagittal plane movements are found to worsen or peripheralise the pain. Sometimes a relevant lateral component is indicated by a failure of sagittal plane movements to bring about any lasting change after several days, rather than an actual worsening of symptoms. In these instances some combination of extension and lateral movements may be required.

Procedures are described in Chapter 17, with combined techniques listed under the appropriate extension or flexion principle. Patients who commence management with the lateral principle should, as for all patients, be constantly monitored to ensure continuing suitability for this principle. Sometimes after an initial period of lateral movements a change to the extension principle, for instance, may be required.

The response of some derangements to *all* mechanical testing will be an increase or worsening of symptoms. The working hypothesis in such a case is an irreducible derangement. Before this poor prognosis is conveyed to the patient, a period of mechanical testing and re-evaluation is worthwhile. However, once it is clear that there are no mechanical loading strategies that decrease, abolish or centralise symptoms, the patient should be advised of the situation. Further investigation may be necessary; the focus of therapy should change to functional rehabilitation, but certainly a lengthy period of ineffective treatment should not be undertaken.

Dysfunction

A much smaller number of patients will be placed in the dysfunction category. Again, once it is decided that the patient has a dysfunction, the appropriate direction of movement must be selected. *In dysfunction syndrome the movement chosen is the one that consistently produces the patient's pain, as this movement gradually remodels the structural impairment.* The movement chosen reproduces the symptoms on each occasion, but these abate shortly after the movement ceases.

The majority of patients with dysfunction need the *extension principle* or the *flexion principle* and make use of the manoeuvres mentioned above. A few may need to use the *lateral principle*.

Postural

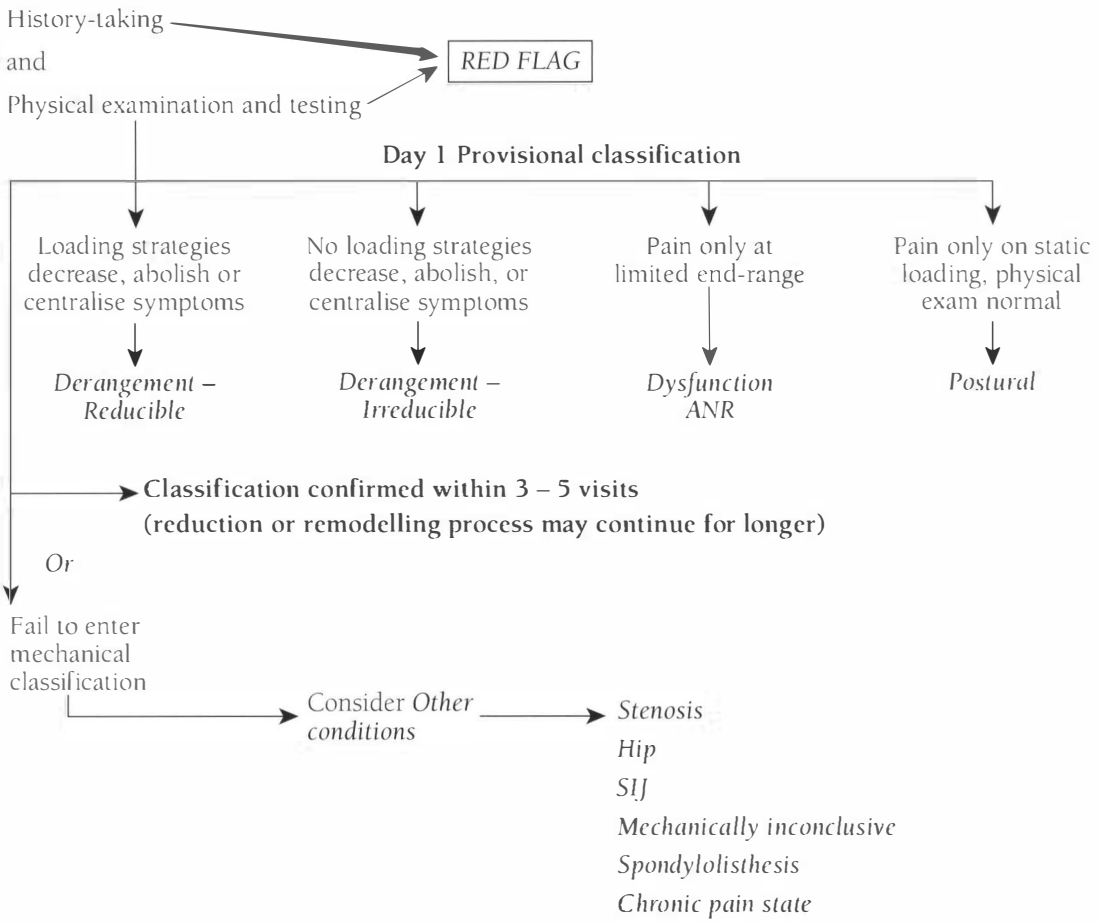
Very rarely are patients with pain of purely postural origin found. These patients simply require postural correction and education. However, poor posture is a perpetuating factor in all three syndromes, and most patients have pain to some extent from this cause.

Inconclusive

It may be necessary, in order to identify the causative syndrome, to deliberately provoke the patient's symptoms. However, not every patient displays an obvious mechanical response to provocative loading strategies on the first assessment. Especially if symptoms have been present for some time, a more extended period of mechanical testing may be necessary. As long as baseline measures have been gathered against which to judge change, if the initial response is equivocal it is very often worthwhile to encourage the patients to test out a particular provocative or reductive loading strategy over the next day(s). Multiple sessions of repeated movements performed over several days often provide more definitive information than one or two sessions in the clinic. Other ways of making force progressions in order to facilitate the diagnostic process are described in the chapter on symptomatic and mechanical presentations (Chapter 16). Within the history and mechanical presentation, there are often clues as to directional preference that become clearer once explored more fully over time.

Even if a non-mechanical syndrome is suspected, it is generally unwise to embark on additional pain provocation testing on the initial visit as this often produces false-positive responses. Testing of the sacro-iliac or hip joints, or consideration of the 'other' category should generally wait until a thorough evaluation of the response to a regular loading strategy has been applied over twenty-four to forty-eight hours, and the response is atypical or negative. The full diagnostic process should be completed within five clinic sessions, but very often is completed much more quickly than this.

Figure 15.3 Classification algorithm (Should be read with operational definitions in Appendix)



16: Evaluation of Clinical Presentations

Introduction

During the first assessment, data is gathered about the patient's pain and the impact that this is having on their function and normal activity. Thus, during the history-taking and physical examination, baseline measures are collected on the symptomatic and mechanical presentations. On all subsequent occasions, clinicians must evaluate the effect of the management strategies being used against these baseline measures. This evaluation needs to address both the pain, which is frequently the patient's main complaint, and the impaired function. These two elements should be assessed on each occasion and will generally improve or worsen in parallel – in other words, as the pain eases, the function also returns to normal. Depending on the effect of the management strategies on the symptomatic and mechanical presentations, these should be continued, abandoned or supplemented with force progressions, as appropriate.

This chapter considers some of the aspects that are relevant to assessment of symptomatic and mechanical presentations, which are the factors that are involved in evaluation and re-evaluation of clinical presentations. The specific way to conduct a review evaluation is detailed in Chapter 19.

Sections in this chapter are as follows:

- symptomatic presentation
- assessment of symptomatic response
- use of symptom response to guide loading strategy
- mechanical presentation
- assessment of mechanical presentation
- use of mechanical response to guide loading strategy
- symptomatic and mechanical presentations to identify mechanical syndromes
- chronic pain – interpretation of symptom responses.

Symptomatic presentation

Pain is usually the main complaint of patients with musculoskeletal problems, although paraesthesia, numbness or weakness may also be relevant. Pain as an outcome measure is criticised as 'soft' data, which lacks objectivity. However, while pain is by its very nature a subjective experience, it can be recorded and assessed in a reliable way, especially when using serial measurements of pain taken from a single individual (Sim and Waterfield 1997). Numerous articles and books present ways of assessing pain, and some of the problems of so doing (for instance, Jadad and McQuay 1993; Sim and Waterfield 1997; Adams 1997). The most common tools are rating scales of pain intensity or pain relief, visual analogue scales, analgesic consumption, pain frequency and pain questionnaires, which investigate multidimensional aspects of pain (Jadad and McQuay 1993). For any therapeutic intervention whose goal is the reduction of pain, the assessment of pain must rank as one of the most important and relevant measures of improvement (Holmes and Rudland 1991).

The symptomatic presentation has various dimensions by which changes can be assessed.

Table 16.1 Dimensions of symptomatic presentation to monitor progress

- site of pain
- constant or intermittent
- severity
- paraesthesia
- number of analgesics and non-steroidal anti-inflammatory drugs (NSAIDs)
- pain on movement.

Site of pain. Pain of spinal origin may centralise or peripheralise (McKenzie 1981, 1990; Donelson *et al.* 1990,1991), and this phenomenon is discussed at length elsewhere. In essence, the further the pain spreads into the limb, the worse the presentation. If the area of symptoms can be reduced or moved further up the leg, this is an improvement. Change in pain site is one of the most important factors used in determining directional preference, and thus the management strategy to be implemented. Centralisation has been the subject of numerous studies (see Chapter 9) and has been shown to be reliable and to predict a good prognosis. Just as centralisation offers a good

prognosis and is positively sought after, its opposite, peripheralisation, should be avoided. It is apparent that not only peripheralisation, but also non-centralisation – that is, failure to alter the site of the symptoms – is also associated with a poor prognosis (Werneke *et al.* 1999).

- Frequency of symptoms. A patient may complain of constant pain. If this subsequently becomes intermittent, an improvement has been made. If an intermittent pain, which is present for most of the day, is reduced to being present only 20% of the day, this also is an improvement.
- Severity. Intensity of pain can be assessed in various ways. This can be done formally using a Visual Analogue Scale, with the patient marking the pain intensity on a line between 'No pain' and 'Pain as bad as it could be' (Huskisson 1974). They can be asked the same question at a later date for comparison. They can be asked whether their symptoms are severe, moderate or mild. Alternatively, at re-assessment patients can be asked how their present symptoms compare to when they first attended. 'If you had 100 units of pain when we commenced treatment, how many do you have now?' Some patients even volunteer that they are '80% better'.

When centralisation of symptoms occurs or lumbar exercises are initiated, this can sometimes result in a temporary and localised increase in symptoms. As long as this *is* temporary, is *not* excessive, is located in the *back*, and especially when associated with centralisation, this should be seen as normal, and not a contraindication for that exercise. Peripheral pain that is being produced or made more severe by a movement is a contraindication for that procedure.

- Paraesthesia. Patients with sciatica may also present with a sensation of tingling, pins and needles or numbness. The presence of these symptoms should always be enquired into if the patient presents with pain into the calf or thigh. In such patients a full neurological examination should be conducted involving appropriate dermatomal, myotomal and reflex testing.

Trauma to the nerve root-dorsal ganglion-spinal nerve complex can cause demyelination or axonal degeneration, leading to changes in nerve function. This can be experienced as muscle weakness, sensory deficit or hyperexcitability of the nerve tissue causing pain (Rydevik *et al.* 1984). Radicular pain is the most common symptom of nerve

root interference; sensory deficit and muscle weakness are variable findings. Vague feelings of tingling or sensory impairment can sometimes be reported anywhere in the leg, but marked nerve root interference is denoted by a sensory loss in the distal part of the dermatome. The most common sites are the medial side of the lower leg (L4), the big toe (L5) and the lateral border of the foot (S1) (Nitta *et al.* 1993).

Such symptoms do not always respond so directly to repeated movements as does pain. There is no 'centralisation' of paraesthesia. Although radicular pain may be centralising or resolving, improvements in sensory deficit usually take place less rapidly, and sometimes not at all. Improvement may occur in one of the ways listed below.

Table 16.2 Criteria by which paraesthesia may be improving

- numbness may become more a 'tingling' feeling
- the severity of the numbness may lessen
- the constancy of the paraesthesia may lessen
- the area of paraesthesia may diminish.

It is important to ensure that there is no worsening of such symptoms with mechanical therapy, which may be judged by the opposite of the above criteria or the onset of sensory deficit.

At times pain will have more or less resolved and the individual can still be left with an area of reduced sensation. If this starts to improve, either lessening in severity, constancy, or size, continuing improvements are likely. However, when there is no early easing of these symptoms, recovery is less likely; some individuals are left with a patch of numbness that never improves. They should be reassured that this does occur, but is nothing to worry about. After surgery, which is likely to involve patients with a more significant neurological deficit, normalisation of sensory changes occurred in only 35% of about ninety patients at four months, increasing to no more than 40% by two years (Jonsson and Stromqvist 1996).

- Consumption of analgesics and NSAIDs. The number of tablets being taken daily at the beginning of the episode should be recorded and compared with consumption later.

- Pain on movement. Even if movement appears to be full-range, pain may be felt during the movement or at end-range. An improvement is made if the patient reports that over time the difficulty of performing the movement and the degree of discomfort it engenders has lessened or if there has been an increase in the range of pain-free movement.

When other responses are equivocal and a management strategy is unclear, a painful movement is sometimes the only variable that can be assessed to determine directional preference. For instance, in a case where right side gliding is painful and repetition of side gliding causes no change, the patient should perform extension in lying. Although this apparently has no effect, it may be the appropriate exercise. This can be confirmed by again checking the response to side gliding. If the pain is improved, extension is in the appropriate direction. Conversely, flexion may worsen the effects of side gliding, thus also indicating extension.

Although often pain responses are a useful determinant of appropriate mechanical therapy, it should also be borne in mind that excessive attention on the pain can heighten the pain response (Arntz *et al.* 1991). By contrast, use of distraction techniques can minimise the pain (Klaber Moffett and Richardson 1995). With some patients the focus should be on function rather than pain; this is especially relevant in patients with chronic pain states.

Assessment of symptomatic response

Standardised terms (McKenzie 1981; Van Wijmen 1994) are used to evaluate the patient's pain responses during mechanical testing (see Glossary for summary of terms). The pain status is established before, during and after test procedures. If we wish to compare the effect of movements on the symptoms, *it is vital we know the pain status prior to testing*. It is too late to establish this once the patient has started to do the movements. We are most interested in the effect of the movements a minute or so after testing.

At baseline the patient is either with or without pain. During the test movements this can be *increased*, *decreased*, *abolished*, *produced* or the movements have *no effect*. Also during the test movements symptoms may show signs of *centralising* or *peripheralising*. The

significant response, which helps determine the treatment strategy, is not, however, due to a single movement. Response to repeated movements often reveals the paradoxical nature of pain and movement. While a single movement may produce or increase pain, the accumulated and lasting effect of repeating that movement may be to reduce the overall pain. Thus the key symptomatic response is based on the effects of *repeated* movements and not on the effects of a single movement. The choice of terms to describe responses must await the completion of perhaps two or three series of ten movements. The final decision should be made after the patient has had time to assess the effects. Sometimes it is best to get the patient to walk around for a few minutes and then ask, 'Do you have more or less pain, or is it unchanged?'

Another favourable symptom response that can occur is production of pain on the first movement, which decreases on repetition. With each repeated movement pain is still felt, but this gets less and less with each repetition, so that by the last movement pain production is minimal or absent. After the test movements, the patient returns to being pain-free. Although strictly speaking this 'before and after' response should be recorded as *produced, no worse*, the symptom modification in response to loading strategy is clearly favourable and worth noting. When this response is observed to repeated movements, this should also be recorded afterwards as *better*.

Different terms are used to describe any changes that persist after the completion of one or more series of repeated movements. At this point pain, which was increased or produced by the movements, can either remain *worse* or be *no worse* if it returns to its former state when the movement has ceased. Pain, which was decreased or abolished during the movements, can either remain *better*, or be *no better* when the movement has ceased. If during and after the movements the symptoms remain completely unchanged, they are said to have *no effect*. These terms are listed and defined in the Glossary. At first glance they appear very obvious to apply; however, their careful use requires experience and a thorough understanding of these terms is a prerequisite of mechanical diagnosis.

If following test movement the site of pain has changed, symptoms are then reported to be *centralised* or *peripheralised*. If the patient has reported referred pain into the limb, then we must know the

extent of referral prior to testing. *We should enquire about the most distal point that pain is felt in the leg at that moment*. The movement of pain proximally or distally is a key determinant of directional preference. If after a series of test movements pain that was felt as far as the calf is now felt only in the thigh, then symptoms are in the process of centralising. If pain that was initially felt all across the back comes to be felt solely in the centre of the back, again the centralisation process is occurring. However, if pain is made to appear more distally, and further repeated movements increase the distal symptoms, peripheralisation is occurring.

Use of symptom response to guide loading strategy

Using these standardised terms to define patient's responses to repeated movements allows us to determine the appropriateness of those particular movements. A system termed the 'traffic lights guide to the progression of forces', initially devised by W. and J. Rath (Robinson 1994), addresses the therapeutic implications of the pain response after the test movements. Table 16.3 is an adaptation and development of their earlier work.

Table 16.3 Traffic Light Guide to symptom response before, during and after repeated movement testing

Pain status before test	*Pain response during test*	*Pain response after test*	*Implications (Traffic Light Guide)*
Pain	Increase	Worse	Red (if distal pain)
		Not worse	Amber
	Decrease	Better	Green
	Abolish	Not Better	Amber
No Pain	Produce	Worse	Red
		Not worse	Amber/Green
	Produce, better with repetition	No pain	Green (dysfunction)
Proximal pain	Peripheral pain produced	Worse	Red
		Not Worse	Amber
Distal pain	Abolish	Better	Green
	Decrease	Not Better	Amber
	Increase	Worse	Red

The traffic light guide allows a logical formulation of appropriate mechanical loading strategies based on the patient's symptom responses. If repeated movements abolish the most distal symptoms in the patient's leg, or if symptoms give the appearance of moving proximally up the leg, the process of centralisation is occurring and the correct strategy has been selected. Equally, if the patient's symptoms are abolished or reduced after the test movements, the correct movement has been selected and treatment should be continued unaltered. These are both examples of a 'green light' to more of the same procedure. ***If the patient is showing improvements with the prescribed management, there is no justification for changing or supplementing it in any way***. In the case of dysfunction, if pain produced by end-range movement ceases afterwards and this response is consistently produced, this too is a 'green light' for more of the same.

If peripheral pain is produced by the repeated movements and remains worse afterwards, then the wrong procedure has been applied. If the pain remains worse after test movements, then either the direction, speed of movement or starting position is wrong, and that particular exercise should be modified and, if indicated, stopped. Movements in the opposite direction should be tested or lateral movements need to be fully explored. If movements still aggravate symptoms, non-mechanical problems or an irreducible derangement might be suspected. Movement may have commenced too early during healing and the inflammatory process is being prolonged, the wrong starting position may be being used, or the procedure may be too vigorous for the stage of the disorder. Whatever the specific cause, these are indications of a 'red light' to that particular procedure. If everything appears to aggravate the patient's symptoms, they should be spared further testing and reviewed in a week's time.

In the case of the 'amber light', essentially nothing is changed by the test movements. Although they may produce, increase, abolish or decrease symptoms during repeated movements, afterwards the patient reports that they are just the same as they were before the test procedures. In this instance the application of more force is justified to see if the traffic light changes to give a clearer indication of the appropriate direction of therapeutic exercise. Only with an 'amber light' response is it justifiable to use force progressions. More force may be applied by the patient exercising more regularly, or with patient overpressure, or by the patient testing the movement

over a twenty-four- to forty-eight-hour period. If this does not clarify the response, clinician force becomes necessary.

The 'amber light' is the expected response in patients with dysfunction syndrome, and in this instance this response is a 'green light' for that particular exercise. In articular dysfunction if pain is produced on end-range stretch or end-range compression, which ceases on release, that loading strategy should be continued unaltered.

Mechanical presentation

The mechanical presentation refers to the outward manifestations of the problem that may limit or alter normal movement, posture and function. The mechanical presentation has various dimensions by which it can be assessed. Broadly, these address impairment and disability. Impairment refers to an anatomical or physiological abnormality leading to loss of normal bodily ability, such as loss of movement. Disability is the diminished capacity for everyday activities and normal occupation, which is the degree to which the individual is affected by that impairment (Beattie and Maher 1997; Waddell 1998). In a small group of acute back pain patients, it has been demonstrated that symptoms, function and range of movement were closely correlated, and all showed a similar pattern of recovery over a twelve-week period (Ferguson *et al.* 2000); thus function and mobility can be valid ways of assessing change over time.

Although back pain, impairment and disability are related to each other, there is not always a close correlation between these different aspects of a clinical presentation (Waddell 1998). In a lower limb amputee impairment is relatively straightforward to assess, although disability varies considerably depending on age, functional goals, psychological drives and so on. In back pain we cannot measure impairment or disability directly. Instead we get proxy measures, by seeing what functional limitations patients report and show when we examine them, which they ascribe to their back pain. Current functional limitations associated with pain may be the result of an anatomical impairment. It should also be remembered that these findings measure performance and depend upon the patient's effort and willingness to move. Reduced ability to perform a movement or task may relate to fear of further 'injury' or fear of pain, rather than pain itself, and thus result from a patient's beliefs rather than an anatomical impairment.

Assessment of mechanical presentation

Patients, especially if acute, often present with significant losses of normal bodily ability and loss of normal function. Losses of range of motion, deviations and asymmetries from normal movement, deformity, absence from work, or inability to enjoy usual pastimes are all commonly observed in back pain patients. Changes in these aspects of the clinical presentation can be used to monitor progress.

Table 16.4 Dimensions of mechanical presentation by which to assess change

- range of movement
- deformity
- deviation on movement
- quality of movement
- curve reversal
- loss of normal function.

There is considerable variation in spinal mobility in the general population (Twomey and Taylor 1994b), and the reliability of simple ways of measuring movement has been questioned (Gill *et al.* 1988). Several tools are now available to make measurement of spinal mobility more 'objective'. Clinical utility demands that methods are simple, inexpensive and easy to use, as well as accurate, reliable and sensitive to change. Some sort of trade-off between accuracy and simplicity may be necessary, but should not compromise clinical integrity. Changes in movement patterns that occur over an episode of care are often substantial, especially when patients are seen from the acute stage. Minor alterations that need to be measured in centimetres are generally not relevant. Gross examples of loss of mobility can be determined by 'eyeballing' and do not need objective tools. At the outset we wish to know from the patient if their present range of movement is abnormal for them.

It is vitally important that the examination process is standardised; tests should always be done from a consistent position to optimise reliability and sensitivity to change. *Range-of-movement tests should always be conducted from the same starting position and in consistent ways so that the results can be compared on different occasions.* Increased range of movement is the main improvement to watch for, therefore an accurate baseline measurement is important. It is essential that to determine a baseline measurement the patient is encouraged

to move as far as possible, for instance by saying, 'further, further, further'. Only then is a reliable start position established. The occurrence and severity of pain during movement and the quality of movement are other ways that the mechanical presentation can change.

Measurement of flexion by distance of fingers-to-floor has been shown to be reliable (Newton and Waddell 1991; Gauvin *et al.* 1990). Although fingers-to-floor is a composite movement of the spine, hips and hamstring flexibility (Waddell *et al.* 1982), true lumbar flexion and total lumbosacral/hip flexion are closely correlated and are equally relevant as a measurement of impairment (Rainville *et al.* 1994). Total flexion is also found to closely reflect self-reported disability in activities of daily living (Rainville *et al.* 1994; Waddell *et al.* 1992; Michel *et al.* 1997). Flexion measured by fingers-to-floor is a quick, easy, reliable and relevant measure of improvement in function. It is suggested it may be the single most valid measure of lumbar impairment (Waddell 1998).

The measurement of extension is more difficult to do accurately and has been shown to be less reliable than flexion (Reynolds 1975; Lovell *et al.* 1989; Beattie *et al.* 1987). Reliable measures of total extension can be obtained using an inclinometer, though separation into the pelvic and lumbar components of the movement is difficult (Newton and Waddell 1991; Waddell *et al.* 1982, 1992). Marked changes in range in extension are quite noticeable, and in lying patients ability to extend their elbows fully can give a simple proxy measure of lumbar extension.

The measurement of side glide movements is also more difficult to do accurately, but again, gross losses or asymmetries are easy to detect.

Time of day affects available range of motion. Sagittal range in patients has been found to increase as the day progresses, with the significant change occurring in flexion, while extension remained relatively stable throughout the day. From morning to evening there is an average gain of eleven degrees of flexion, but only three degrees of extension (Ensink *et al.* 1996).

Loss of movement occurs most dramatically in the derangement syndrome, when, with the onset of pain, all movements can be dramatically reduced. This is most noticeable in sagittal plane movements, but occurs also to a lesser extent in frontal plane movements. Equally with derangements there can be rapid

improvements in range of movement. In the dysfunction syndrome the affected movement displays a marked loss of mobility. This will have been present for some time and will only gradually improve. In the postural syndrome there will be no loss of movement.

- Deformity. In some patients the onset of pain is accompanied by a sudden loss of movement that is so severe that they are unable to move out of the abnormal posture. The patient is locked in kyphosis, lateral shift or lordosis and is unable to self-correct this very visible anatomical misalignment, or if able to correct, cannot maintain the correction. This phenomenon only occurs in derangement and must be immediately recognised as it determines treatment.
- Kyphotic deformity – the patient is locked in flexion and is unable to extend.
- Lateral shift – the patient is locked in (for instance) right lateral shift and is unable to correct shift or maintain shift correction.
- Lordotic deformity – the patient is locked in extension and is unable to flex.

Recognition of deformity is straightforward. It will have come on dramatically with the pain, and active attempts to regain a normal upright posture provoke intense pain and prove impossible. The patient generally needs clinician assistance to correct the deformity; this is explained in the chapter on procedures. If accurate measurement of a lateral shift is required, a simple plumb line is reliable and simple to use (McLean *et al.* 1996). When patients respond to the appropriate treatment, there is rapid improvement in the deformity and more normal active movement begins to return.

Patients with deformity are instantly recognised. They have severe symptoms and a marked postural misalignment that is of recent origin. Patients who present with minor deviation from normal alignment, those who have asymmetrical movement losses and patients who are able to correct a pain relieving posture do not have deformity. In other words, if the presence of deformity needs to be considered and is not obvious, they do not have one.

- Curve reversal. One of the ways in which movement can be affected is a problem with curve reversal (McKenzie 1981). In an asymptomatic individual, movement from full flexion to full

extension is performed without hesitation or interruption. In patients with derangement, problems can occur after sustained positions or repeated movements. Following a period of sitting or bending, they report having to ease themselves gradually into an upright position and that initially they cannot attain full extension because of pain. They may report that initially they walk the first few steps bent forward, gradually attaining an upright posture. Problems with curve reversal are either due to pain or an obstruction to movement.

Provoking problems with curve reversal can be a way of determining directional preference when this is unclear (Van Wijmen 1994). For instance, if a limited number of repeated movements have failed to clarify the appropriate management strategy, it may be necessary to do many repetitions of, for instance, flexion, and then re-check the patient's movement into extension. If, following fifty repetitions of flexion, there is now pain on or an obstruction of extension that was not present earlier, the symptom response has clarified management. Flexion must be avoided and extension, in this case, is the direction of preference. Conversely, sustained end-range extension may significantly diminish flexion in those with an anterior derangement.

- **Deviation on movement** One of the ways that movement can be altered in derangement or dysfunction is deviation from the normal sagittal plane. As the patient bends forwards or backwards, they deviate to the right or left. This is most noticeable and common in flexion. Usually this is masking a restriction of movement. If the clinician prevents the patient from deviating by holding them in the sagittal plane, very often there is considerably greater movement loss. See Chapter 11 for more detail.
- **Quality of movement** Pain makes people move more cautiously (McGregor *et al.* 1995; Paquet *et al.* 1994). One study that used a computerised spinal motion analyser to generate a curve of motion during flexion–extension movements graphically showed the difference. The normal subject demonstrated a smooth 's-shaped' curve as they moved from neutral to flexion to extension in just over five seconds. The curve from a patient with acute back pain was much flatter, demonstrating losses of flexion and extension, and took nearly eight seconds to complete, with the movement being more jerky and laboured. This loss of velocity

has also been shown to improve following a rehabilitation programme (Magnusson *et al.* 1998a).

- **Functional disability** It is always important to ask which of their normal activities the patient is unable to do because of their back pain. Often work, recreational and domestic responsibilities and activities may be curtailed, and the goal of treatment is always a return to their normal level of function. More formal ways of assessing patient's function should be done using established disability questionnaires. These can be completed within five minutes by the patients themselves and are a good measure of how much they feel their normal lifestyle is affected by the back problem. When used at the beginning and end of an episode of treatment, these offer validated, reliable and sensitive research tools that are easily applied in the clinical environment. Examples of some of the most commonly used questionnaires are given below, although more are available. These three functional status questionnaires are all presented and reviewed by Beattie and Maher (1997).

Table 16.5 Some commonly used back disability questionnaires

- Roland and Morris Disability Questionnaire – Roland and Morris 1983.
- The Oswestry Low Back Pain Disability Questionnaire – Fairbank *et al.* 1980.
- Quebec Back Pain Disability Scale – Kopec *et al.* 1995.

Use of mechanical response to guide loading strategy

Just as symptom response can be used to guide loading strategy, so also can mechanical responses (Table 16.7). If there is an increase in range with repeated movements or sustained positions, this is interpreted as *better*. If with repeated movements or sustained positions there is a decrease in range, this is interpreted as *worse*. The Traffic Light Guide provides the same therapeutic implications as above – *better* is a green light to more of the same loading strategy, *worse* is a red light requiring that loading strategy be stopped or amended. Often these responses occur with the symptomatic changes outlined above. Sometimes initially only a symptomatic change occurs, or less commonly only a mechanical response occurs. In all three instances the mechanical response has the same therapeutic implications.

Table 16.6 Mechanical responses to loading strategy

Range of movement change	*Terminology*	*Implications*
Increase	Better	Green
Decrease	Worse	Red
No change		Amber

Symptomatic and mechanical presentations to identify mechanical syndromes

The different mechanical syndromes present with characteristic patterns of symptoms and movement abnormalities. Recognition of these assist in the identification of the different mechanical syndromes. Equally, their absence helps in the discovery of atypical and non-responders, who may require further testing or investigation to elucidate their problem.

The table below indicates the characteristics that may be present in each syndrome. These criteria either *must* be present for that mechanical classification to be indicated (shown as +) or are *variably* present in that mechanical classification, that is they *may* be present (indicated as (+)). If a particular feature is *never* found in that syndrome, this is indicated by **N**. For instance, if there are referred or constant symptoms, the patient cannot have postural syndrome. The table makes clear that the presentation of derangement is much more varied than the other two syndromes. Presentations in dysfunction and postural syndromes are reasonably consistent. In derangement many different signs and symptoms can be present, but not all need be present to classify as derangement.

Table 16.7 Characteristic symptomatic and mechanical presentations of the mechanical syndromes

Symptomatic and mechanical features	*Postural syndrome*	*Dysfunction syndrome*	*Adherent nerve root*	*Derangement syndrome*
Symptoms				
Back	+	+	(+)	(+)
Pain to knee	N	(+)	+ and	(+)
Pain to calf	N	N	/ or +	(+)
Pain to calf and neuro	N	N	(+)	(+)

Continued next page

Symptomatic and mechanical features	*Postural syndrome*	*Dysfunction syndrome*	*Adherent nerve root*	*Derangement syndrome*
Constant	N	N	N	(+)
Intermittent	+	+	+	(+)
Centralisation/ Peripheralisation	N	N	N	(+)
Pain during movement	N	N	N	(+)
End-range pain	N	+	+	(+)
Sustained loading produces pain	+	(+)	+	
Inconsistent pain response to loading	N	N	N	(+)
Painful arc, no movement loss	N	N	N	(+)
Mechanical features				
Movement loss	N	+	+	+
Acute deformity	N	N	N	(+)
Postural deviations	N	N	N	(+)
Deviation on movement	N	N	(+)	(+)
Problems with curve reversal	N	N	N	(+)

Key:
+ = must be present
(+) = may be present
N = never present

Chronic pain – interpretation of symptomatic responses

In the situation of chronic pain, when peripheral tissue and central nervous system elements may be sensitised and deconditioned to normal movement, the criteria of symptom response needs to be different. Under these circumstances, normal mechanical stimuli can produce pain, repeated movements may have a 'wind-up' effect on pain production, there may be a spread of painful areas and there may be ectopic nociceptive signals (Dubner 1991; Johnson 1997). These changes make the interpretation of mechanically produced symptom responses difficult and invalidate diagnostic labels applied to particular responses (Zusman 1992,1994). Psychosocial factors

that have been identified as factors in chronic pain and disability are passive coping strategies, fear–avoidance behaviour, lack of self-efficacy and depression (Linton 2000b). These characteristics may make patients overly anxious and fearful about pain responses, which they consequently exaggerate. These examples suggest that we should interpret the behaviour of chronic pain to repeated movements somewhat less rigidly. Non-mechanical factors may have become significant factors in perpetuation of pain.

The above effects are unlikely to be present to the same degree, or even in all patients with chronic pain; many such patients respond relatively straightforwardly to mechanical therapy. Some patients with chronic pain can have ongoing tissue damage or a degenerating disease (Johnson 1997). Most patients with chronic musculoskeletal pain have only mild or moderate symptoms and do not suffer major functional impairment (Magni *et al.* 1990; Von Korff *et al.* 1990). A proportion leave the pool of persistent pain sufferers if followed over a few years (Crook *et al.* 1989). Only a small proportion of patients with persistent pain (2%) are at the severe, disabled end of the spectrum; many respond normally to a mechanical evaluation. If the response is equivocal, test the patient's response over twenty-four hours or use one of the other suggestions above for facilitating the diagnostic process when it is unclear.

However, in the instance of chronic pain patients, it is sometimes permissible to allow a slight worsening of symptoms initially. Sometimes the response to mechanical therapy takes a while to elucidate, thus it is valuable to follow the approach for a few sessions rather than abandoning it as soon as there is slight worsening of symptoms. Sometimes the sensitisation induced by chronic pain states needs to be desensitised by encouraging gentle regular movement prior to establishing a more mechanical pattern of response. With chronic pain patients it is necessary to concentrate more on trying to improve coping strategies and function rather than focus on pain. Often improvement in general function and the psychological effect of doing something active about their problem can produce a reduction in pain. Patients with severe levels of dysfunctional behaviour due to persistent pain problems are best treated in a multi-disciplinary pain programme or a functional rehabilitation approach rather than on a one-to-one basis.

Conclusions

This chapter has discussed the means by which patients are assessed on their progress or lack of it. This involves regular interpretation of their pain response and examination of their mobility and function. The way to review and interpret the symptomatic and mechanical presentations in detail has been presented. Using these criteria, it can be gauged whether the patient is improving, worsening or unchanging. If they are getting better, nothing should be changed. If they are worse, further assessment is conducted and a change in management strategy is necessary. If they are the same, then a progression of forces should be considered and a re-analysis is conducted. Review should ideally be conducted on a daily basis until there is a definite improvement and confirmation of the management strategy. The specific way a review is conducted is presented in Chapter 19.

17: Procedures of Mechanical Therapy for the Lumbar Spine

Introduction

This chapter contains general descriptions of the procedures that may be needed in mechanical therapy of the lumbar spine, as well as indications for their application. The procedures described here include both patient and clinician techniques.

In most situations patient techniques are used first, and are frequently effective in resolving the problem without the need for more interventions. Provided there is adequate instruction and careful explanation regarding management of the problem, the self-treatment concept can be successfully applied to most back pain patients. Patients with postural syndrome can *only* resolve their problem with self-management strategies. Clinician interventions are ineffective without the patient being educated regarding the role of posture as a cause of their pain. In the dysfunction syndrome *only* the patient is able to provide the appropriate loading strategies with sufficient regularity to enable a remodelling of the structural impairment. Clinician techniques may aid this process, but by themselves are generally inadequate to resolve the tissue abnormality. In the derangement syndrome the majority of patients can successfully manage their own problem, while about 30% of patients will not recover with exercises alone and need clinician techniques in addition (McKenzie 1981).

Thus, in general, patient techniques are always used first and these are only supplemented by clinician techniques when this becomes necessary because of a failure to improve. While the patient is improving with self-management strategies, there is absolutely no need to supplement treatment with additional interventions that encourage patient dependency. In certain instances, most notably the acute lateral shift deformity, clinician techniques may be needed to bring about a situation that the patient can begin to manage alone.

It is one of the main tenets of the approach propounded in this book that many patients can be taught to treat, manage and control their own back pain (McKenzie 1981). In order to achieve this, it is necessary to depart from the traditional methods of treatment in

which the clinician does something to patients to bring about change. In that case the patient attributes recovery, rightly or wrongly, to what was done to them, and the patient is educated into dependency upon the clinician. By avoiding the use of clinician techniques, unless absolutely necessary, and using primarily patient or self-generated techniques, the patient will recognise that they are capable of managing their own problem both now and in the future.

However, there are instances where force progressions involving clinician techniques are needed. The role of force progressions and force alternatives in the elucidation of management strategies will be discussed. The therapeutic loading strategies that are used involve repeated movements and/or sustained postures, as well as posture correction.

Sections in this chapter are as follows:

- force progression
- force alternatives
- repeated movements or sustained postures
- procedures.

Force progression

This approach to musculoskeletal problems involves a progression of forces, initially starting with patient-generated forces, and only involving clinician-generated forces when needed (see Table 17.1). This has several advantages (McKenzie 1989). The patient can regularly apply the procedures throughout the day with far more frequency than would be possible if the patient was only treated in the clinic. If the patient is educated adequately and effectively in self-management, then the responsibility for their condition lies with the individual; the solution to their problem lies in their own hands. They are able to become independent of the clinician and are given the opportunity to manage the problem themselves should it reoccur in the future. Furthermore, should it be necessary to progress forces and include a manipulative procedure, the hundreds of repeated movements that will have preceded this intervention provide a built-in safeguard. The integrity of the structure will have been fully tested and any likelihood of exacerbating fragile pathology will have been exposed.

Contemporary guidelines about spinal care stress the importance of patient responsibility for management (CSAG 1994; AHCPR 1994). This responsibility can be encouraged if the patient is offered an approach that is based on self-management techniques. In contrast, the use of passive therapies, including clinician-generated mobilisation and manipulation, engender patient dependency. Their use implies that the patient is incapable of effecting their own cure, which depends entirely on the attentions of the clinician.

It is not always necessary to start with the earliest forces; the patient can enter at the stage that generates a positive effect. This is different for different patients; those with more severe and acute problems will likely need the earlier stages first – static patient-generated forces. Those with milder symptoms can usually start at dynamic patient-generated forces. Clinician-generated forces should never be used before patient-generated forces have been tried. Progression of forces (Table 17.1) is only introduced as needed, and is not an inevitable part of management.

Force progression is considered when the previously employed technique increases or decreases symptoms during the procedure, but afterwards they are no worse or no better. If a procedure results in the reduction, abolition or centralisation of symptoms, it does not need to be progressed or supplemented in any way, provided there is a continued increase of movement to end-range. If a procedure results in the worsening or peripheralisation of symptoms, it should be stopped and force alternatives considered. Only when symptoms remain unchanged following a procedure should force progressions be considered. Force progression could also include increasing the frequency of exercises and prolonging the period over which exercises are tested out. For instance, a twenty-four-hour test period may provide a more definite response than one gained during a short clinic visit. The progression for the application of forces is listed below (McKenzie 1989).

The progressions are given in the order that most frequently generate a favourable clinical response. However, in determining the appropriateness of loading strategies, some flexibility in the application of force progressions and force alternatives may be required. Application of force progressions and force alternatives should always be conducted with due consideration given to clinical reasoning and attentive interpretation of symptomatic and mechanical responses.

Table 17.1 Force progression

Static patient-generated forces:

- positioning in mid-range
- positioning at end-range

Dynamic patient-generated forces:

- patient motion in mid-range
- patient motion to end-range
- patient motion to end-range with patient overpressure

Clinician-generated forces:

- patient motion in mid-range with clinician overpressure
- patient motion to end-range with clinician overpressure
- clinician mobilisation
- clinician manipulation.

Force alternatives

At times, rather than a force progression, an alternative force is needed. For instance, the response to extension in standing may be equivocal, or even cause a worsening of symptoms; however, in the same individual, extension performed in lying may reduce symptoms. If at any point during exploration of sagittal plane movements these are all found to worsen symptoms, then lateral forces need to be considered. In patients with an acute kyphotic deformity, any attempt to force extension will result in a severe exacerbation of their problem. A gradual recovery of extension over time is the appropriate management.

Table 17.2 Force alternatives

- starting position, example: loaded or unloaded
- direction of loading strategy, example: sagittal or frontal plane movements, or combination
- sagittal direction: flexion or extension
- time factor, example: sustained positioning or repeated movements
- frontal plane angle during combined procedures, example: degree to which hips are shifted during EIL with hips off centre, or hip flexion angle during rotation mobilisation in flexion.

Repeated movements or sustained postures

Procedures are either static or dynamic – that is, either sustained positions or repeated movements. Repeated movements are used most

commonly. The optimum number of movements is about ten to fifteen repetitions in one 'set'. In certain instances, several 'sets' of exercises may be done in succession. The number of times in a day that the series of exercises should be done will vary according to the mechanical syndrome, the severity of the problem and the capabilities of the patient. In most instances, a minimum of four or five sets a day is necessary to produce a change.

It is essential that movements be repeated in order to gain a true understanding of their mechanical effect. Single movements rarely provide a thorough understanding of the effect of that movement. Sometimes the effect of repeated movements is rapidly apparent, while at other times repeated movements over a period of a day or two are necessary to produce a clear symptomatic and/or mechanical change.

Exercises or mobilisations are generally performed in a rhythmical pattern – the procedure should be followed by a brief moment of relaxation. With each subsequent movement, the range or pressure exerted should be increased, as long as the symptomatic response is favourable.

On occasions, static rather than dynamic procedures should be used. These should be considered,
when there is a poor response to repeated movements or when a time factor is important.

In assessing the patient's response to any technique, the symptomatic and mechanical response must be considered. In terms of the symptomatic response, the site, the severity and the frequency of the pain may alter. In terms of the mechanical presentation, the range of movement and the functional level may alter. *A thorough understanding of the appropriate way to interpret symptomatic and mechanical response is essential in order to safely and effectively manage the patient.* A careful monitoring of the patient's response to different procedures is vital. These issues are considered in depth in Chapter 16, and the structure of the review process is detailed in Chapter 19.

Procedures

The procedures are listed according to the treatment principle and according to whether the procedure is static or dynamic. Extension procedures are used most commonly; sometimes these require a

lateral component for a brief period. The use of extension/lateral forces for some of the treatment period and in combination with purely sagittal plane forces is not uncommon. Few patients require a lateral component for long. This may be required only during the first treatment session, and usually for no more than a few days. A minority require pure lateral forces only. Flexion and extension procedures are grouped as 'pure' movements or as those involving a lateral component.

Table 17.3 Treatment principles

- extension principle forces (Procedures 1 – 10)
- extension principle with lateral component (Procedures 11 – 17)
- lateral principle forces (Procedures 18,19)
- flexion principle forces (Procedures 20 – 22)
- flexion principle with lateral component (Procedures 23 – 26).

Table 17.4 Procedures (not all in order of force progression)

Extension principle – static:

- 1: lying prone
- 2: lying prone in extension
- 3: sustained extension
- 4: posture correction

Extension principle – dynamic:

- 5: extension in lying – EIL
- 5a: extension in lying (with patient overpressure) – EIL
- 6a: extension in lying (with clinician overpressure)
- 6b: extension in lying (with belt fixation)
- 7: extension mobilisation (in neutral or in extension)
- 8: extension manipulation
- 9: extension in standing – EIS
- 10: slouch – overcorrect/correct sitting posture

Extension principle with lateral component – dynamic:

- 11: extension in lying with hips off centre
- 12: extension in lying with hips off centre with clinician overpressure (a: sagittal; b: lateral)
- 13: extension mobilisation with hips off centre
- 14: rotation mobilisation in extension
- 15: rotation manipulation in extension

Continued next page

Lateral principle:

- 16: self-correction of lateral shift or side gliding
- 17: manual correction of lateral shift

Flexion principle:

- 18: flexion in lying – FIL
- 19: flexion in sitting
- 20: flexion in standing – FIS
- 21: flexion in lying (with clinician overpressure)

Flexion principle with lateral component:

- 22: flexion in step standing – FISS
- 23: rotation in flexion
- 24: rotation mobilisation in flexion
- 25: rotation manipulation in flexion

Extension principle – static

Procedure 1 – Lying prone

The patient lies on their front with arms alongside the trunk and the head turned to one side, and in this position relaxes. The position is sustained. In prone lying the lumbar spine falls automatically into some degree of extension.

Application

A basic requirement for the self-treatment of posterior derangement is that the patient can attain and maintain the prone lying position. As it is the starting point for other self-mobilisation procedures of the extension principle, it is essential that the patient can achieve this position.

In posterior derangements, lying prone is used as a sustained position. Initially this may increase symptoms in some patients, but as long as the increase of pain is felt centrally this is acceptable. When the position is maintained for five to ten minutes, there should be a reduction of symptoms. Further procedures of the extension principle can be considered once the symptoms have stabilised. If pain is increased or produced peripherally, the prone position should not be maintained and force alternatives must be considered.

Following a decrease of symptoms in the prone lying position, care should be taken in resuming the upright posture. Every effort should be made to maintain the restored lordosis while moving from lying to standing in order to maintain any reduction.

Photo 24: Lying prone.

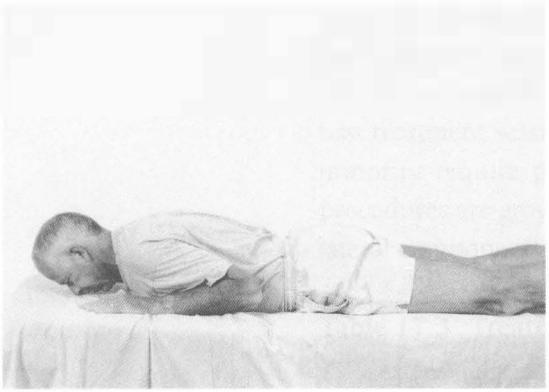


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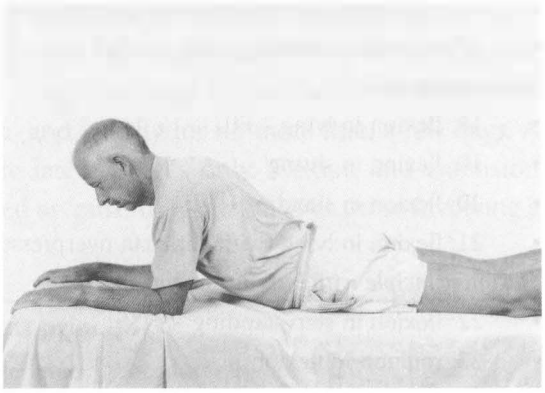
Photo 25: Lying prone in extension.



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In a major derangement, such as those presenting with an acute lumbar kyphosis, the degree of extension necessary to achieve prone lying may be unobtainable, in which case a gradual recovery over time is necessary. To do this, one or two pillows can be placed under the abdomen or, if on an appropriately hinged treatment couch, the wings of the couch can be lowered so that the patient lies initially in slight flexion, accommodating the deformity. After a period of five to ten minutes, during which time symptoms should ease, a pillow can be removed or the wings of the couch raised so that a prone position is gradually attained.

In extension dysfunction, there is always some loss of lordosis. In some patients the loss may be enough to prevent them lying prone without pain. For such people, lying on their front in bed or while sunbathing will have become impossible. The prone lying procedure by itself will not resolve the extension dysfunction. However, when adopted several times daily for five to ten minutes and in conjunction with other extension principle procedures, prone lying should become painless as remodelling of tissues occurs.

Procedure 2 – Lying prone in extension

After lying prone (Procedure 1), the patient places the elbows under the shoulders and raises the top half of his body so that it is supported by their elbows and forearms while the hips or pelvis remain on the couch. The patient relaxes in this position, allowing the low back to sag into more extension. The position is sustained for five minutes or more; however, in severe and acute cases this may need to be interrupted by a return to lying prone at regular intervals. In lying

prone in extension, the lumbar lordosis is automatically increased, but this is a position of mid-range extension.

Application

This procedure is a progression of Procedure 1 and enhances its effect by increasing extension and by being sustained. In derangements the time factor is often critical, and the position should be sustained for five minutes or more. During this period there may be an initial increase in symptoms followed by an eventual decrease, which remains better. If the patient finds it difficult to tolerate the position, a return to prone lying is indicated at regular intervals. Further procedures of the extension principle can be considered once the symptoms have stabilised. If pain is produced peripherally, the prone position should not be maintained and force alternatives must be considered. This includes a more gradual attempt at achieving extension.

Procedures 1 and 2 are important when a time factor exists in the restoration of extension. These procedures are particularly important where time is a factor in the production of symptoms; so time is also a factor in the resolution of symptoms.

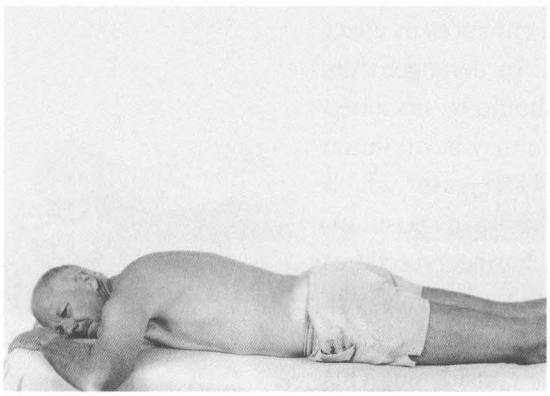
In more acute patients with derangements with an obstruction to extension, sustained extension may not be well tolerated due to pain. Such patients may initially require the application of mid-range extension on an intermittent basis. They hold the procedure for as long as they can tolerate, then relax briefly in prone lying before attempting a brief period of prone lying in extension again.

The procedure may also be useful in elderly patients who physically find it difficult to do an extension in lying and is also a starting point for marked extension dysfunction. It is, however, only a mid-range position of extension and needs progression as soon as possible.

Procedure 3 – Sustained extension

To apply a gradual and sustained extension stress to the lumbar spine, it is best to have an adjustable treatment table with one end that can be raised. If an adjustable plinth is not available or the procedure is to be done at home, then pillows or blankets can be used to adjust the angle of extension. The patient lies prone and their upper body is lifted gradually into extension a little at a time over an extended period, which may be half an hour or more. Once the maximum degree of extension is achieved, this position is held for a few minutes

Photos 26, 27, 28: Sustained extension.
Allow several minutes to pass before making the next progression (26). Raise the end of the couch in small increments. Allow the symptoms to guide progression (27). Having regained this amount of extension, do not progress further (28). This is sufficient to enable the patient to self-treat.



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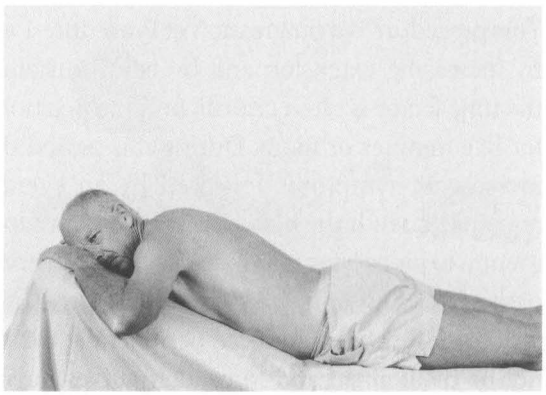

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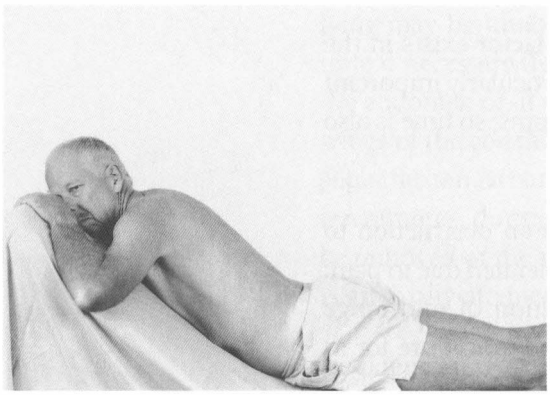

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according to the patient's tolerance. When returning the patient to the starting position, this also should be done gradually, over two or three minutes, otherwise the patient may experience severe back pain.

If the patient is not initially able to tolerate the prone lying position, as with an acute kyphotic deformity, the starting position has to be in slight flexion. If the patient is unable to attain the position, a gradual recovery of it is necessary. To do this, one or two pillows can be placed under the abdomen or, if on an appropriately hinged treatment couch, the ends of the couch can be lowered so that the patient lies initially in slight flexion, accommodating the deformity. After a period of five to ten minutes, during which time symptoms should ease, a pillow can be removed or the wings of the couch raised so that a prone position is attained in a gradual way.

Too rapid an increase in the extension range will cause severe pain and possibly a worsening of the condition. Progressions must be small, with the treatment couch being lifted only four or five centimetres at a time. With each progression the symptoms will increase in intensity, but after a few minutes this should start to settle. There may be an increase in pain centrally and a reduction of pain distally. Only once the pain has subsided to its former intensity should a further progression be made.

Should the pain fail to subside to its former level, the plinth must be lowered to the horizontal position and the patient allowed to rest in this position for a few minutes. Once the patient is recovered, another attempt at a gradual recovery of extension is made. This is often easier the second time. With each progressive increase in range, expect an initial increase in pain followed by centralisation or reduction.

If the recovery process is lengthy, it is necessary to allow the patient to rest for a few minutes every so often in the prone lying position. This allows recovery from the sustained exposure to extension, which can be difficult to tolerate. Following a recovery period it should be possible to raise the patient to the former extended position more rapidly. The blockage to extension will gradually diminish.

The aim is to regain as much extension as possible on the first treatment session, after which the patient should able to perform prone lying, prone lying in extension and extension in lying (Procedures 1, 2, 5) and continue with these procedures at home. However, in case of reversal of improvements or in case complete recovery is not achieved on the first occasion, patients must be instructed in home procedures. They should be told to lie over pillows on the floor or bed and gradually lower themselves into the prone position by removing the pillows one at a time.

Once the prone position is tolerated Procedures 1, 2 and 5 are implemented.

Application

This procedure is not a progression from previous procedures, but rather a different way of achieving extension in which time is more effective than repetition. It is only used when the patient is unable to tolerate previous procedures. The most likely need for its use is with patients who have a kyphotic deformity.

This procedure is only used in the reduction of major derangements. There is likely to be a major loss of extension movement; indeed, the suitable patient is normally stuck in flexion and unable to extend at all. In some patients a gradual and sustained extension stress has a better symptomatic response than intermittent stress, as in extension in lying.

During the application of this procedure, symptom response must be carefully monitored. Any suggestion of peripheralisation or worsening peripheral symptoms must lead to an immediate, but *gradual* lowering of the couch. An increase in central low back pain as the couch is lowered nearly always indicates a good response to treatment, whereas when this does not occur there tends to be little or no improvement following this procedure.

Once the patient is able to perform extension in lying (Procedure 5) exercises themselves, even if not to full range, this is implemented as part of the management strategy. If the application of this procedure is successful, then the use of Procedures 1, 2 and 5 becomes appropriate.

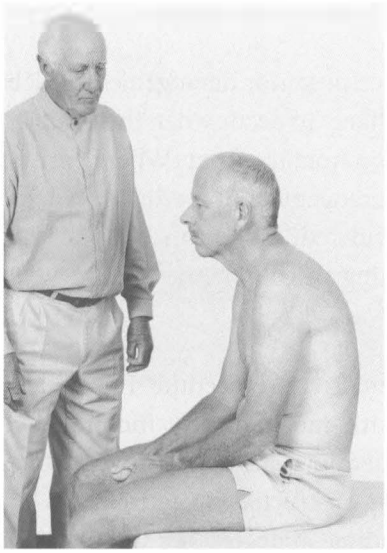
It is important to note that sustained forces may be more effective in the reduction of some derangements than repeated movements. These sustained forces can also be progressed by working in different parts of the range; for instance, starting in prone lying (Procedure 1), prone lying in extension (Procedure 2) and then sustained extension.

Note:
Sustained extension can also be used as a provocative test if anterior derangement is suspected, but unclear. With the end of the plinth raised, the patient is placed in sustained extension for up to five or six minutes. Their symptomatic and mechanical response is then evaluated. If pain becomes worse during this period or when the patient is upright again, an anterior derangement is likely. This can be confirmed by reviewing the mechanical response to flexion in standing. If the result of sustained extension is the production of a major loss of flexion, an anterior derangement is confirmed.

Procedure 4 – Posture correction
The patient is guided from a kyphotic sitting position to an upright sitting posture by anteriorly rotating the pelvis and accentuating the lordosis. This can be done by gentle pressure on the sternum and lumbar spine with the clinician to the side of the patient. Or, if the patient is more resistant to posture correction, from the front the clinician can adjust the pelvis with hands around the patient's waist.

As well as attaining a lordotic sitting posture, the patient should be shown how to maintain this position using a lumbar roll.

Photos 29, 30, 31: Posture correction.



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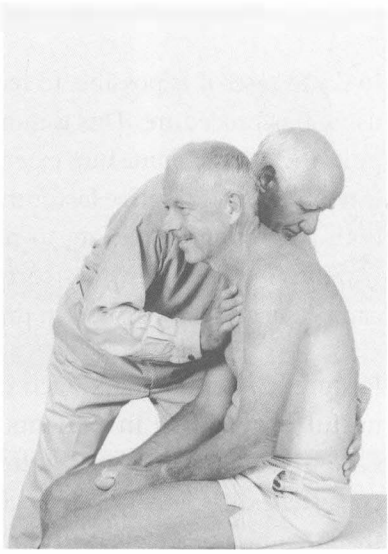

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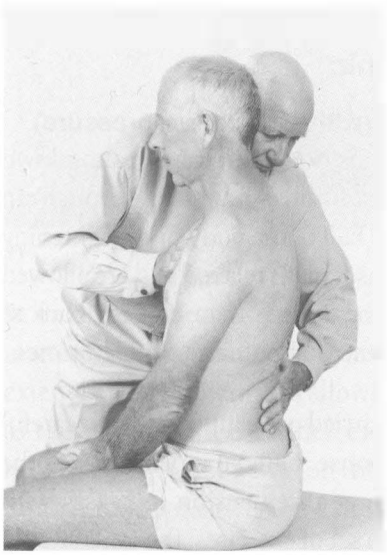

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Application

Posture correction is the main intervention for pain in postural syndrome when the aggravating factor is sitting. The procedure is discussed more in the appropriate chapter. Posture correction is also very important in management of derangement.

The importance of the postural component in a patient's symptoms can, in part, be determined by their response to posture correction after relaxed sitting during the interview. This can be an important learning experience for the patient. As with other procedures, symptom behaviour should be monitored as it is applied and afterwards.

In posterior derangement, it is very common that sitting is an aggravating factor. Management requires the absence or reduction of aggravating factors, such as sitting, as well as the introduction of therapeutic loading strategies. It is not uncommon for the patient to be able to reduce the derangement using repeated movements, but

symptoms return on sustained sitting in a poor posture. Derangement requires both reduction and maintenance of reduction.

In some cases it is possible to reduce posterior derangements solely using this procedure. This is more likely to occur when the clinician pulls the lumbar spine into extension from the front. Where posture is the obvious causative factor in derangement, it is educational, for both patient and clinician, to provide education in sitting posture without any exercises. It is surprising the number of patients who can be successfully treated in this way.

Posture correction and slouch overcorrect (Procedure 10) are also useful procedures in patients with mechanically inconclusive symptoms. These patients, infrequently seen, do not demonstrate consistent directional preference or obstruction to movement, but their symptoms are affected by loading strategies (see Chapter 13). They can benefit from mid-range work using these procedures.

Extension principle – dynamic

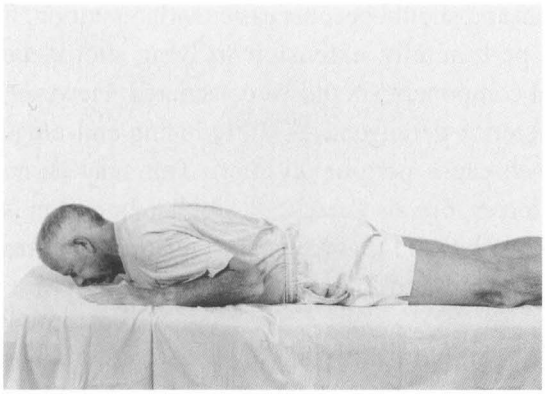
Procedure 5 – Extension in lying (with patient overpressure)

The patient starts in the prone lying position, with hands palms down under the shoulders, as for the traditional press-up or push-up exercise. They now raise only the top half of the body by straightening their arms, while the pelvis and thighs remain relaxed and are allowed to sag with gravity. The top half of the body is then lowered back to the plinth. The exercise is then repeated about ten to fifteen times.

The first few movements should be carried out cautiously, with careful monitoring of the symptomatic response. Often the patient finds the exercise becomes easier and the range of extension increases with repetition. As the patient completes a set of exercises, the arms should be fully straightened to achieve the maximum possible extension range.

As long as there is no lasting aggravation or peripheralisation of symptoms, a second set may be attempted. More vigour can be applied and the last three or four movements should be sustained for a second or two in a fully relaxed and sagged position. Allowing the abdomen to sag to the couch produces maximal possible self-generated extension. Further sets of ten can be repeated as continuing improvement indicates (or as indicated by continued improvement).

Photos 32, 33, 34: Extension in lying. Photo 35: Extension in lying with patient overpressure.



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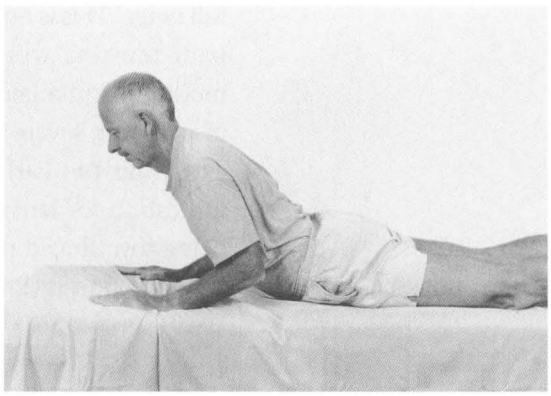

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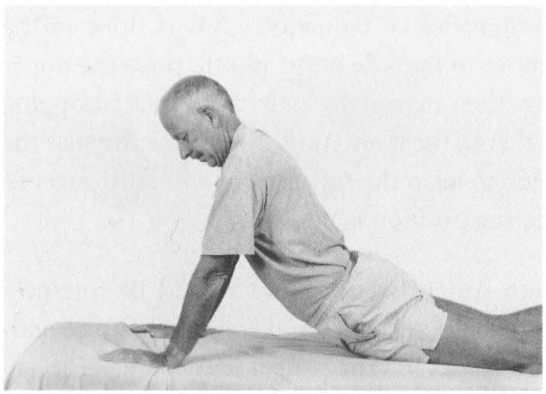

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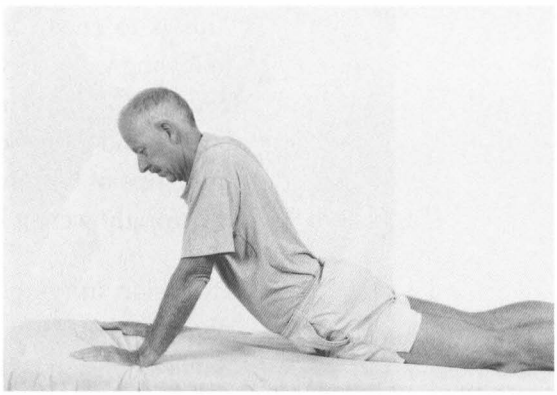

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In order to achieve extension in lying with overpressure, it does not matter if the hips and pelvis come off the plinth slightly as the patient extends their elbows. This allows space for the effect of the relaxation as they sag, which is to increase extension. If the patient is told to keep their hips and pelvis on the plinth, the effect of exhaling on the lumbar range of extension is less.

Application

This procedure is a progression from Procedures 1 and 2. Rather than a sustained extension force, an intermittent extension force is being applied, with greater amplitude. This procedure is the most important and effective exercise in the treatment of posterior derangement and extension dysfunction. Almost the maximum possible extension without external assistance is achieved with this manoeuvre.

In derangement, an increase in central low back pain may occur at full range. This is normal and should become easier with repetition. If pain remains worse peripherally, extension in lying should be modified and a lateral component should be considered. However, in acute or severe posterior derangements, introducing end-range extension too early can cause peripheralisation. This may be an indication for lateral forces, but also may indicate that extension is being introduced too quickly and that earlier sustained procedures should be used (Procedures 1 and 2).

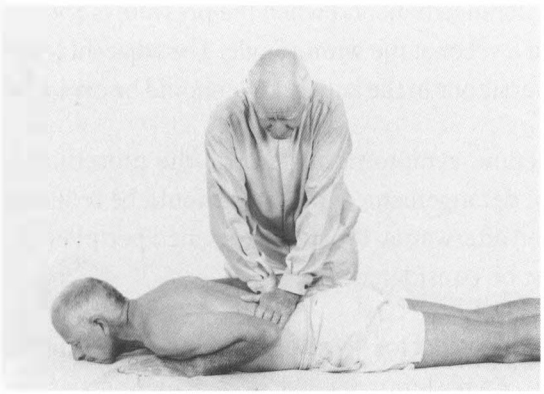
Following a decrease of symptoms with the exercise, care should be taken in resuming the upright posture. Every effort should be made to maintain the restored lordosis while moving from lying to standing. This is to ensure maintenance of reduction. This is done in the following way. They move to the side of the plinth, press the upper body up using the arm, then maximally extend the back, dropping one leg and then the other to the floor. At the same time they use the fingertips of both hands to keep the trunk more and more erect as the upright weight bearing position is regained.

Extension in lying with patient overpressure should be routinely performed as early as possible, usually on day one, to ensure end-range extension is being achieved. The patient is encouraged 'to sag the last two or three in each set of ten'. If this is not possible on day one, then this is the first force progression used at the next assessment.

Procedure 6a – Extension in lying (with clinician overpressure)
The patient's starting position and performance of extension in lying is as described in Procedure 5. External overpressure is applied at a segment so that the extension force is accentuated at that level. The treatment table is at the height that allows the clinician to be over the patient sufficiently for their line of force to be perpendicular to the spine. The patient lies close to the side of the table on which the clinician is standing. The level at which the overpressure should be applied is determined from the best symptomatic response.

The clinician stands to one side of the patient, crosses their arms and places the hypothenar eminencies on the transverse processes of the same segment. One hand is parallel to the spinous process and the other perpendicular to it, so that hands are at 90 degrees to each other. The clinician's chest is over their hands.

Photos 36, 37, 38, 39: Extension in lying with clinician overpressure.



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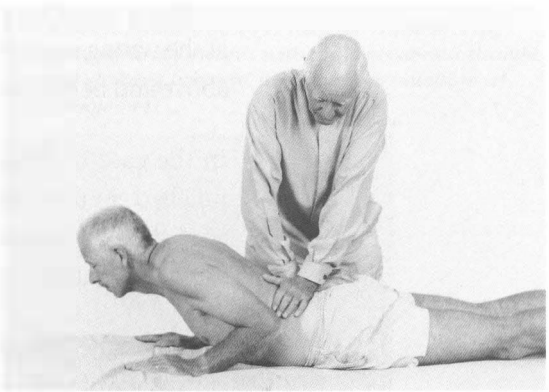

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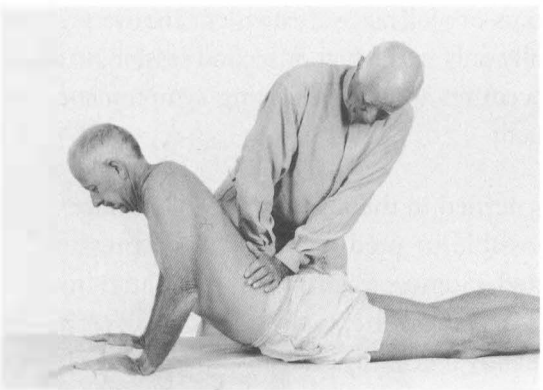

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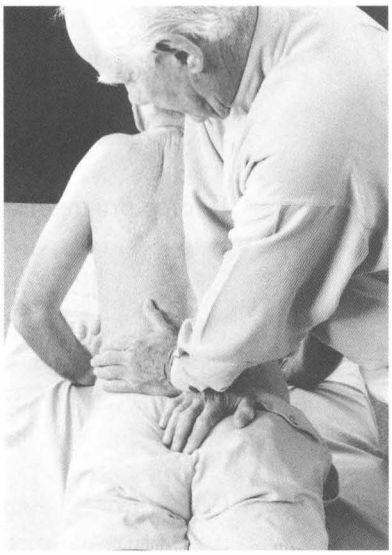

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Once in position, a gentle, symmetrical pressure is applied by leaning forward and transferring body weight through the arms. This is sustained as the patient performs extension in lying, but allows the movement to occur. As the patient extends, it is necessary to change the angle of the overpressure from directly downward to about forty-five degrees, so that the force remains parallel to the motion segment. This change in angle necessitates that the clinician alters their body weight onto their back leg. The level can be changed depending on the response of the symptoms, and the pressure can be increased if the response indicates.

Application

This procedure produces a greater and more localised passive extension stress than all previous procedures. This is used for two purposes: diagnostic and therapeutic. If more pressure produces more pain, a resistant derangement or dysfunction can be suspected. If more pressure produces less pain, a derangement is confirmed, as well as the correct

segmental level. More pressure causing more pain occurs in the presence of a resistant derangement and when the pressure is applied at the wrong segmental level or at the wrong angle. The adjacent levels above and below and variations in the lateral angle should be explored.

In the case of dysfunction, symptoms abate once the procedure is finished. In the case of derangement, symptoms should be reduced, abolished or centralised afterwards. If pain is worsened peripherally, force alternatives must be considered.

Overpressure may also be needed for therapeutic purposes during the early stage of reduction of a posterior derangement. Patient-generated procedures alone may not achieve a lasting reduction, abolition or centralisation of symptoms, or a full range of extension. The overpressure is applied briefly, usually only in the first or second session, to allow patient-generated procedures to achieve lasting symptomatic and mechanical improvement.

If force progression is needed in the management of derangement, it is generally not possible to predict whether overpressure or mobilisation is needed. Some patients respond better to this procedure, while others respond better to the procedure of extension mobilisation. Consequently it is always valuable to try both, if force progressions are required. Overpressure can be applied to the sacrum or the lumbar segments.

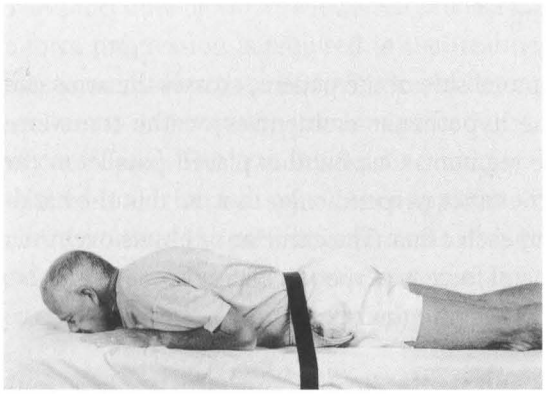
Procedure 6b – Extension in lying (with belt fixation)

An alternative method of achieving overpressure is provided by a safety or fixation belt, which can be applied around the plinth and over the pelvis. This prevents the pelvis from moving and accentuates the extension force at the lumbar segments. A belt can be loaned to the patient if they have something it can be attached around, or a member of the family can assist. Either they can sit on the pelvis or a towel can be laid across the pelvis and the assistant stand on either end of the towel, straddling the patient.

Application

The are two chief uses for extension in lying with belt fixation; first, in derangement as a home treatment for those who respond well to extension in lying with clinician overpressure (Procedure 6a); secondly, in extension dysfunction, which the previous procedure will have helped to confirm. This is only used if previous procedures prove inadequate, and is designed for long-term home use.

Photos 40, 41, 42: Extension in lying with belt fixation.



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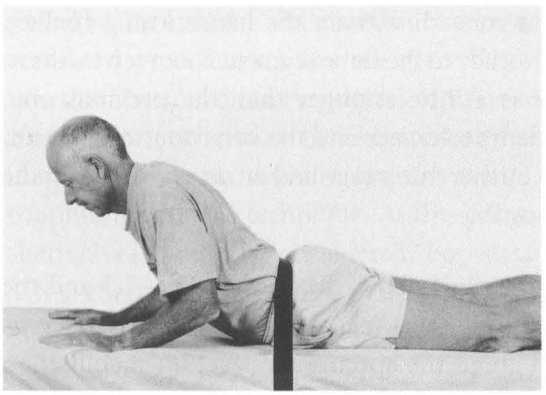

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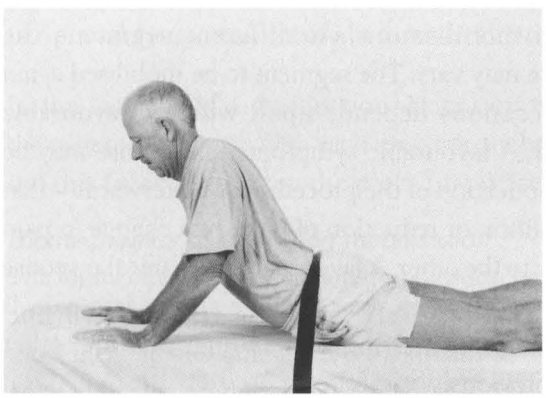

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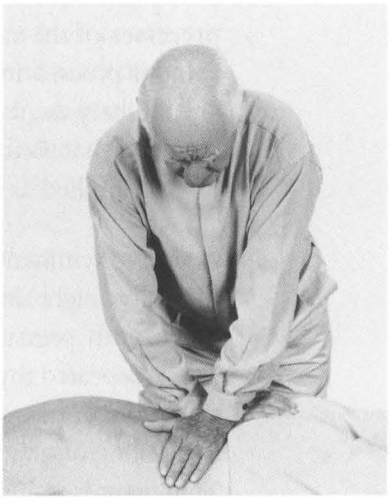
Photos 43, 44: Extension mobilisation. Stand well over the patient, lean forward, pressure on; lean back, pressure off (43). Keep the trunk moving backwards and forwards with the arms almost straight. Avoid applying pressure, using flexion/extension at the elbow (44).



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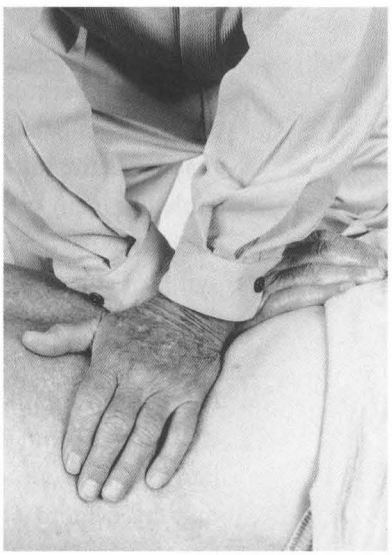

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Procedure 7 – Extension mobilisation

An adjustable-height plinth is preferred to perform this procedure most effectively. This should be at a level that allows the clinician to be over the patient sufficiently for their line of force to be perpendicular to the spine. The patient lies prone with their arms by

the side, as in Procedure 1, and near the edge of the plinth by which the clinician is standing.

The clinician stands to one side of the patient, crosses the arms and places the heel of the hypothenar eminencies on the transverse processes of the same segment. One hand is placed parallel to the spinous process and the other perpendicular to it, so that the hands are at ninety degrees to each other. The clinician's chest is over their hands so that their line of force is perpendicular to the segment. No force is applied as they position their hands.

A gentle, symmetrical and perpendicular pressure is applied using the bodyweight through the hands so that a rhythmical increase and decrease in pressure is created, without the hands losing contact. This is repeated rhythmically to the same segment about ten to fifteen times. Each pressure is a little stronger than the previous one, depending on the patient's tolerance and the behaviour of the pain. The intent is to move further into range and attain end-range in the last few pressures if possible.

The procedure is first applied about the level of L4 – L5 and the response determined, and then to adjacent segments above as required. Information about the appropriate level for mobilisation may have already been obtained during testing of extension in lying with clinician overpressure (Procedure 6a), which has probably preceded extension mobilisation. At different segments the symptomatic response may vary. The segment to be mobilised again or on subsequent occasions depends upon where a favourable response is generated. A favourable symptomatic response may be evident during the application of the procedure, or afterwards – that is, centralisation, abolition or reduction of pain, or a change in pain location from one side to the other. A favourable mechanical response may be evident afterwards when greater range and/or less pain is experienced with extension in lying or standing, or some other movement that was previously painful.

The procedure can be performed in varying degrees of extension if a plinth with an adjustable end is being used. The starting position would always be neutral, but if a progression is required, the mobilisation could be performed with the patient placed in a position of extension.

Application

This procedure produces a localised extension force and is used when a force progression is required in the treatment of derangement or extension dysfunction. For instance, in derangement, when there is a failure to improve with previous procedures, this technique should be considered. This is the clinician technique that is most commonly applied to the lumbar spine. After application of the procedure, the effectiveness of the patient-generated techniques are re-evaluated; if this now generates a favourable response, it is no longer appropriate to continue with manual techniques.

If clinician techniques are applied, these should always be accompanied by continuing with patient-generated techniques as well. The intent of the clinician's intervention is to make the patient-generated procedures more effective in resolving the condition. Whether the added intervention is of value can generally be judged after one or at the most two sessions of mobilisation. If after that there has been no definite improvement, there is no value in continuing with the technique. At this point manipulation or force alternatives should be considered. For instance, if this procedure fails to effect an improvement, performing the same procedure further into the physiological range of extension can sometimes be helpful.

As with every procedure, symptoms are carefully monitored during and after the mobilisation. If the manoeuvre results in worsened peripheral symptoms, force alternatives should be considered.

In the instance of a dysfunction, extension mobilisation produces the patient's local pain with each pressure on the appropriate segment, but this fades away when the procedure ceases.

Biomechanics of extension mobilisation

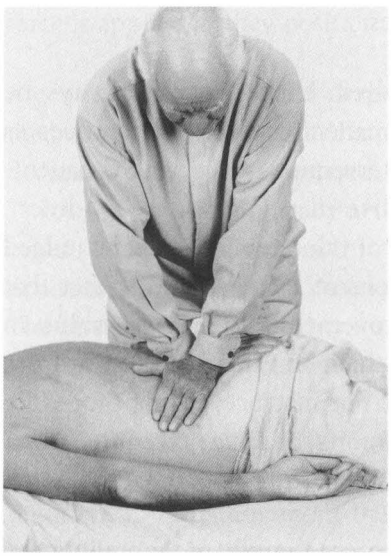
The biomechanics of extension mobilisation have been evaluated (Lee and Evans 1994). The procedure produces extension bending movement at all segments, but maximally at the point where force is applied. It also produces anterior shear forces in the segments below, and posterior shear forces on the segments above. The amount of intervertebral motion is very small, millimetres of translation and a few degrees of rotation. It is highly unlikely that this amount of movement can be palpated; what is felt is the movement of the whole lumbar spine as it sags under pressure between the two supporting ends, the pelvis and the thoracic rib cage. It is speculated that the

intervertebral disc is the major anatomical structure resisting the applied load.

Procedure 8 – Extension manipulation

The starting position for both patient and clinician is the same as extension mobilisation (Procedure 7). An adjustable-height plinth is preferred to perform this procedure most effectively. This should be at a level that allows the clinician to be over the patient sufficiently for their line of force to be perpendicular to the spine. The patient lies prone with their arms by the side, as in Procedure 1, and near the edge of the plinth by which the clinician is standing.

Photo 45: Extension manipulation. High velocity, small amplitude thrust.



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The clinician stands to one side of the patient, crosses the arms and places the heel of the hypothenar eminencies on the transverse processes of the same segment. One hand is placed parallel to the spinous process and the other perpendicular to it, so that the hands are at ninety degrees to each other. The clinician's chest is over their hands so that their line of force is perpendicular to the segment. No force is applied as they position their hands.

An extension force (Procedure 7) is then applied and sustained for five to ten seconds to ensure the patient can accurately assess the effects. The symptom response to this pre-manipulative testing procedure must be centralisation, reduction or abolition of symptoms during maintenance of the procedure, but which return soon after. If following this manipulation is indicated, the clinician leans over the patient with the arms perpendicular to the spine and applies pressure slowly until the spine feels taut. Then a high velocity thrust of very short amplitude is applied and immediately released.

The segmental level at which the manipulation is performed is decided by symptom response during the application of extension mobilisation, which will always precede this procedure. The

appropriate level is the one at which symptoms are centralised, reduced or abolished, but returns when the mobilisation pressure is released. Only one manipulative thrust should be performed at any one treatment session.

Application

This procedure produces the greatest localised extension force available. Manipulation is indicated in derangement when the desired result is not produced by extension mobilisation. The pre-manipulative testing with extension mobilisation, which centralises, reduces or abolishes symptoms temporarily, is essential. After application of the procedure, the effectiveness of the patient-generated techniques are re-evaluated, and if this now generates a favourable response, it is no longer appropriate to continue with manual techniques.

If clinician techniques are applied, these should always be accompanied by continuing with patient-generated techniques as well. Whether the added intervention is of value can generally be judged after one or at the most two sessions of manipulation. If after that there has been no definite improvement, there is no value in continuing with the technique.

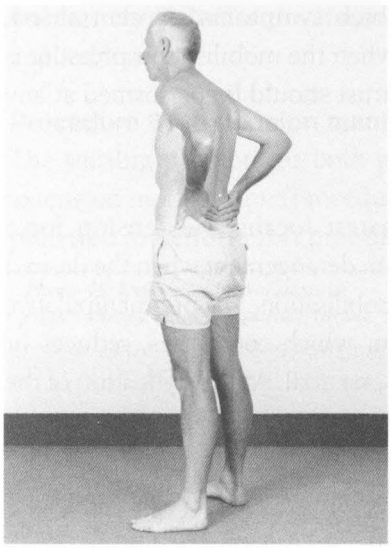
Procedure 9 – Extension in standing

The patient stands with the feet shoulder-width apart and knees straight so that they have a good base of support and feel stable when they perform the procedure. The hands are placed in the small of the back across the belt line with the fingers pointing downwards. The patient then leans backwards as far as possible, letting the head tip back also (care must be used if the patient has a cervical spine problem also), and uses the hands as a fulcrum by pressing them into the spine. They are encouraged to go 'further, further, further'. After arching backwards, the patient returns to neutral standing and the exercise is repeated about ten to fifteen times. As with extension in lying, full range is desirable for the best results. Initially this may not be possible; the patient may find the exercise gets easier on repetition and a fuller range of extension can be obtained.

Application

Extension in standing may be used in posterior derangement. It has little impact in the treatment of dysfunction. In major derangements the procedure may initially cause a worsening of symptoms, but the response improves after extension in lying is performed. Less movement is obtained in extension in standing than in lying, and

Photos 46, 47, 48: Extension in standing.



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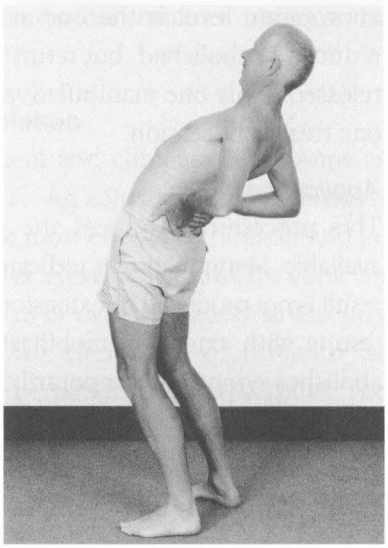

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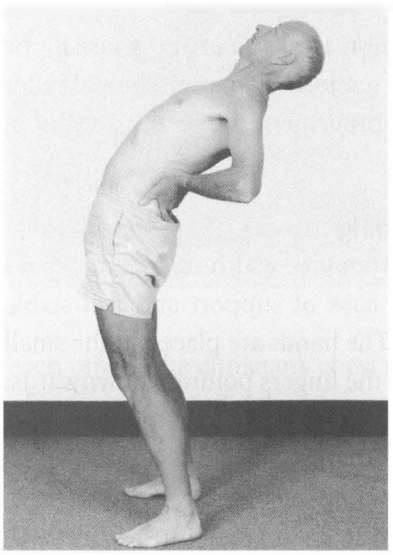

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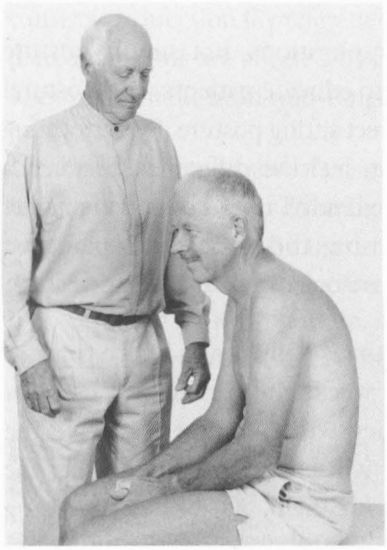
the two procedures also exert different forces on the spine, one being loaded and the other unloaded; for these reasons the standing manoeuvre is often less effective. This procedure is less likely to be appropriate if symptoms are severe or acute.

Derangements will rarely be completely reduced by extension in standing, although sometimes this occurs. However, the procedure is useful as a supplement to extension in lying, as long as the symptom response is satisfactory, especially when circumstances do not allow the performance of extension in lying. For patients who are still at work, or for some other reason struggle to perform extension in lying, the addition of extension in standing can be useful.

The manoeuvre is also very important in the prevention of the onset of back pain during or after prolonged sitting or bending, and is very effective when performed proactively before pain is actually felt. The procedure thus becomes very useful as symptoms of derangement are resolving and intermittent, and for prophylactic purposes.



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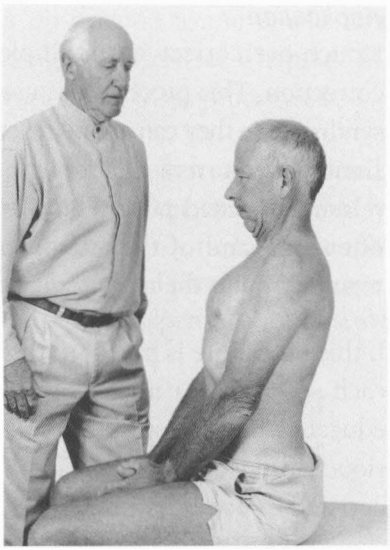

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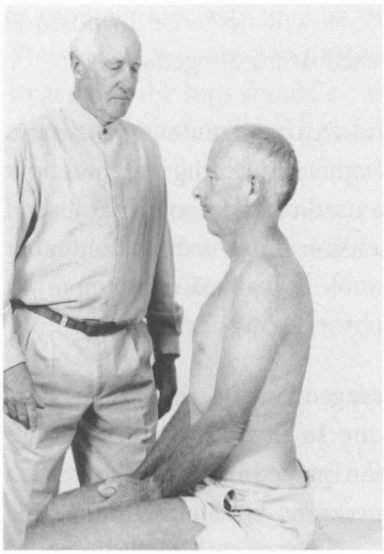
Photos 49, 50, 51: Posture correction.
Extreme of bad position (49).
Extreme of good position (50).
Extreme of good position less the strain (51).



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Procedure 10 – Slouch-overcorrect / Correct sitting posture

First the *slouch-overcorrect* procedure is performed. Sitting on a stool, the patient relaxes into the fully flexed slouched posture, with the head and chin protruding. The patient then smoothly moves into the erect sitting posture, with the lumbar spine positioned in maximal lordosis and the head retracted and chin pulled in. The sequence should be repeated several times in a flowing manner, so that the patient moves from an extreme flexed posture to an extreme of extended posture.

Once the patient has an understanding of good and bad sitting postures, they can then be taught the *correct sitting posture*. The overcorrected sitting posture is at an extreme of extension and becomes painful if maintained for long. The patient is thus instructed to move into an extreme of lordosis, but then release the last 10% of the movement. In this way they can attain a lordotic, but not fully extended, lumbar posture.

Application

Slouch-overcorrect has multiple applications, not simply posture correction. This procedure is used to educate patients with postural syndrome so they can attain the correct sitting posture. By performing slouch-overcorrect, the patient can feel the difference between a relaxed slouched posture and an extended one. These patients are often unaware of their body posture; this procedure, practised regularly, helps them to become conscious of a better sitting posture.

If this procedure is practised three times daily, ten to fifteen times at each session, in a matter of a few weeks the patient will have re-educated their postural habit. They will no longer perceive the slouched posture as 'normal'; they will have come to find that the corrected posture is now 'normal' for them. As well as practising slouch-overcorrect in order to retrain their postural 'habit' and to train their muscles to hold their trunk upright, the procedure should be done regularly if pain arises. Pain will not arise in postural syndrome if the patient avoids slouched sitting altogether.

The procedure may also be useful in derangement on certain occasions. Again, it can be a helpful way of educating patients about posture correction, but also can be used as a method, in a loaded posture, of regaining flexion or extension if this is difficult in other positions. The procedure may have a role in the flexion principle – it is often sufficient to unblock an anterior derangement.

This procedure is also useful in derangements in which directional preference alternates from extension to flexion. In these rather uncommon derangements, extension procedures initially cause a symptomatic and mechanical improvement, but after a brief period this ceases. Extension then causes an obstruction to flexion, and flexion procedures are required to improve symptoms and movement. However, in its turn, prolonged flexion also causes deterioration. Essentially, end-range persistent loading into flexion or extension eventually worsens the patient. Lasting improvement comes with a gradual resumption of both flexion and extension using the slouch-overcorrect procedure, followed for a few days by avoidance of end-range in either direction.

Posture correction (Procedure 4) and slouch overcorrect are also useful procedures in patients with mechanically inconclusive symptoms. These patients, infrequently seen, do not demonstrate

consistent directional preference or obstruction to movement, but their symptoms are affected by loading strategies (see Chapter 13). They can benefit from mid-range work using these procedures.

The importance and value of posture correction is often underestimated, and the procedure is often underused. We hold the view that if only one procedure was to be used, the most good for the most people would be achieved with the correct application of posture correction. It is certainly valid in certain instances to dispense only posture correction as your first intervention.

Extension principle with lateral component – dynamic

Procedure 11 – Extension in lying with hips off centre

This procedure is as Procedure 5, but the patient's starting position is asymmetrical, with the hips off centre in the prone lying position. Thus this is an extension procedure with an additional lateral force. In general, the hips should be shifted away from the painful side. If the patient has left-sided pain they are positioned with their hips off centre to the right, by moving the pelvis to the right away from the side with pain. They now raise the top half of the body by straightening their arms, while the pelvis and thighs remain relaxed on the plinth. The top half of the body is then lowered back to the plinth. The exercise is repeated about ten to fifteen times.

If moving the hips away from the side of pain worsens the symptoms or has no effect, the hips should be shifted the opposite way and the symptom response explored. As in all procedures, performance is dictated by favourable symptomatic and/or mechanical response.

Application

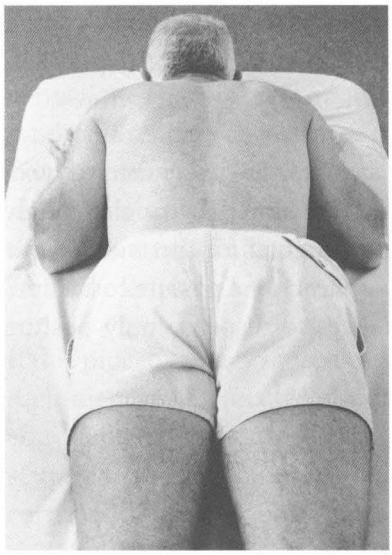
This procedure has a minimal lateral component and a strong extension component. It is used in derangements that have unilateral or asymmetrical symptoms and that have been worsened by, or not responded to, purely sagittal plane movements. It is the first procedure to be used in such a situation.

Procedure 12 – Extension in lying with hips off centre with clinician overpressure (sagittal or lateral)

The patient lies close to the side of the table where the clinician is standing. The level at which the overpressure should be applied is determined from the best symptomatic and/or mechanical response.

Photos 52, 53, 54: Extension in lying with hips off centre.

Photo 55: Extension in lying hips off centre with sagittal overpressure.



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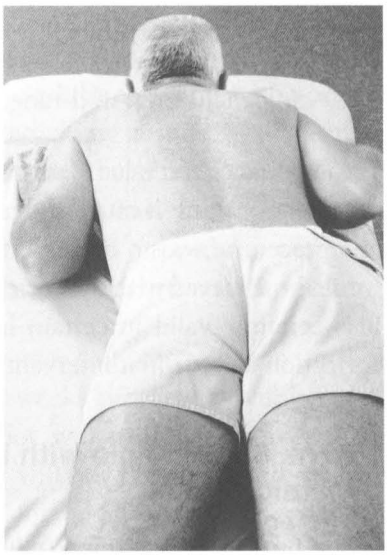

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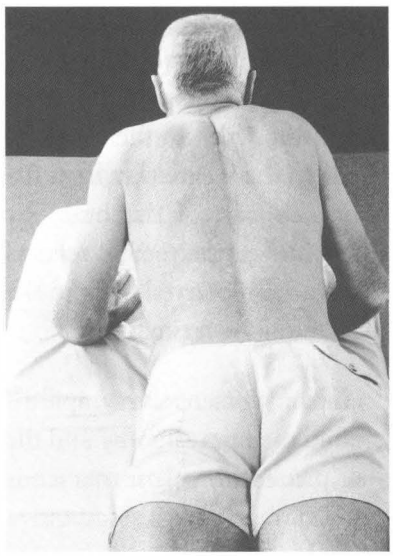

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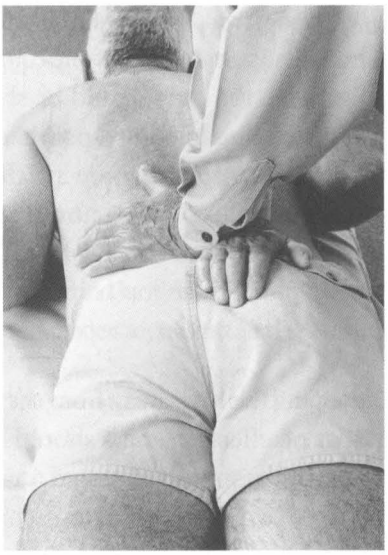

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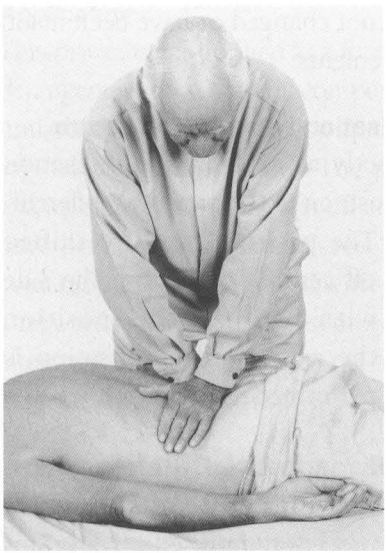
This procedure can be conducted in several ways depending on whether the sagittal or frontal plane component needs to be emphasised. The patient lies prone, prepared to perform repeated extension in lying (Procedure 5), but with hips off center, usually shifted away from the side with pain.

12a – Sagittal overpressure

If the sagittal plane is to be emphasised, the clinician stands to one side of the patient, crosses hands, and places the heel of the hypothenar eminencies on the transverse process of the same lumbar



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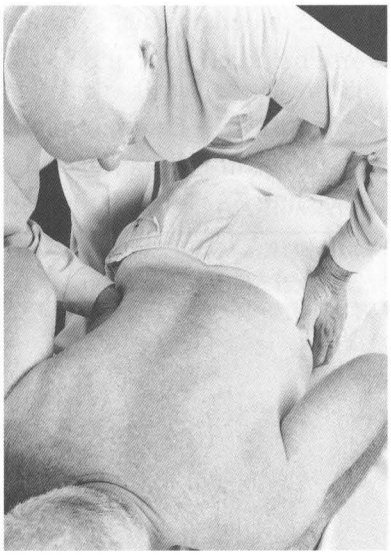

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Photo 56: Frontal plane overpressure.

Photo 57: Extension in lying hips off centre with lateral overpressure.

segment. The clinician's chest is over their hands. Once in position, a gentle, symmetrical pressure is applied using body weight through their arms. This is sustained as the patient performs extension in lying, but allows the movement to occur. This procedure is as extension in lying with clinician overpressure (Procedure 6a), with the addition of the hips off centre position.

If the frontal plane is to be emphasised, overpressure is applied at the iliac crest and at the lower ribs to emphasise the side gliding position. The clinician stands on the left side of the plinth if the patient had right-sided back pain, then lifts or slides the patient's pelvis to the left so that the patient is positioned in prone lying with hips shifted to the left. The clinician leans over the patient hands positioned as above, thus maintaining firm lateral overpressure as the patient performs extension in lying with hips off centre.

Application

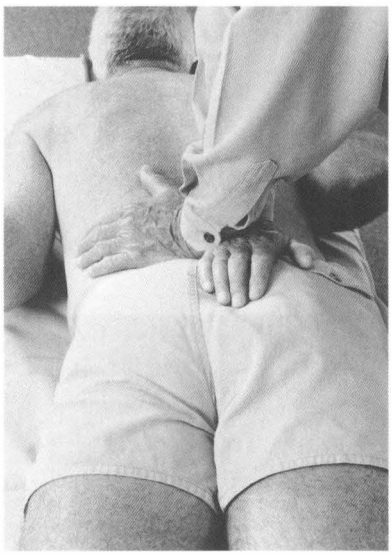
Overpressure is only applied when the response to the previous manoeuvre (11) is equivocal or does not last. If extension in lying with hips off centre decreases symptoms, which are no better afterwards, then the procedure with overpressure should be used. Overpressure is also applied if the response to the previous procedure (11) has been unclear. Overpressure is applied to emphasise the sagittal or lateral component of the procedure as indicated by symptom response. It is used in derangement and is discontinued if it causes a worsening of local or peripheral symptoms. It is only

applied in derangements that have not changed or have been made worse by purely sagittal plane movements.

Procedure 13 – Extension mobilisation with hips off centre

This procedure is performed exactly as extension mobilisation (Procedure 7); however, the start position of the patient is different. The patient's hips are shifted off centre, away from the side with pain. In this position the extension mobilisation is performed as outlined above.

Photo 58: Extension mobilisation with hips off centre.



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An adjustable-height plinth is preferred to perform this procedure most effectively. This should be at a level that allows the clinician to be over the patient sufficiently for their line of force to be perpendicular to the spine. The patient lies prone with their arms by the side, as in Procedure 1, and near the edge of the plinth where the clinician is standing.

The clinician stands to one side of the patient, crosses the arms and places the heel of the hypothenar eminencies on the transverse processes of the same segment. One hand is placed parallel to the spinous process and the other perpendicular to it, so that the hands are at ninety degrees to each other. The clinician's chest is over their hands so that their line of force is perpendicular to the segment. No force is applied as they position their hands.

A gentle, symmetrical and perpendicular pressure is applied using the bodyweight through the hands so that a rhythmical increase and decrease in pressure is created, without the hands losing contact. This is repeated rhythmically to the same segment about ten to fifteen times. Each pressure is a little stronger than the previous one, depending on the patient's tolerance and the behaviour of the pain. The intent is to move further into range and attain end-range in the last few pressures if possible.

Application

This procedure is applied as a force progression during treatment of a derangement with a lateral component, which will be in a patient with unilateral or asymmetrical symptoms who has not responded to pure sagittal plane movements. The patient will have been performing extension in lying with hips off centre and overpressure will already have been applied (Procedures 11 and 12). Symptoms may have been reduced or centralised with these procedures, but are no better afterwards. Alternatively, symptoms are increased, but no worse afterwards – in this instance the force progression is applied to help determine the appropriate loading strategy.

Procedure 14 – Rotation mobilisation in extension

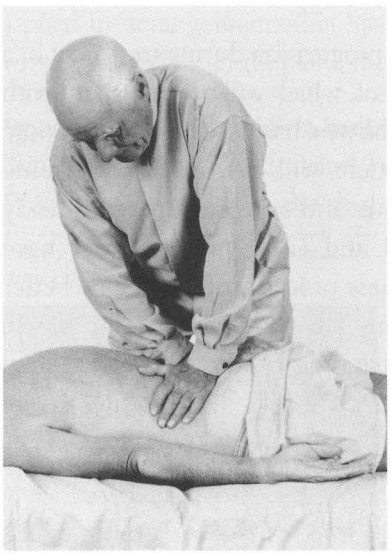
An adjustable-height plinth is recommended to perform this procedure most effectively. This should be at a level that allows the clinician to be over the patient sufficiently for their line of force to be perpendicular to the spine. The starting position for patient and clinician is as for Procedure 7, extension mobilisation. The patient lies relaxed, prone with their arms by their side, close to the side of the table where the clinician stands.

The clinician stands to one side of the patient, hands crossed, and places the heel of the hypothenar eminencies on the transverse processes of the same lumbar segment. One hand is parallel to the spinous process and the other is perpendicular to it, so that they are ninety degrees to each other.

A gentle pressure is applied and released first to one side, then to the other. By repeating the pressure on alternate sides a rhythmical rocking effect is obtained. As pressure is applied on one side a simultaneous reduction in pressure must occur on the other; thus, some extension force is always present, as well as an alternating lateral force. Pressure is achieved by applying the weight of the body through the arms, and the pressure is gradually increased with the intent of moving further into range. The technique is repeated about ten times on one segment and/or repeated on adjacent segments as necessary.

If pressure on one side is producing a favourable response, then mobilisation is restricted to that side only. The clinician stands on the opposite side from the side to be mobilised. Place one hand on top of other hand to perform the mobilisation on one side; force is directed anterior and slightly medially.

Photos 59, 60, 61: Rotation mobilisation in extension. Apply pressure evenly to both sides at first, then apply rhythmically to alternate sides (59). Apply pressure first to the transverse region on one side, and as you release slowly on that side, transfer pressure to the other side (60). Unilateral rotation mobilisation, once appropriate side established (61).



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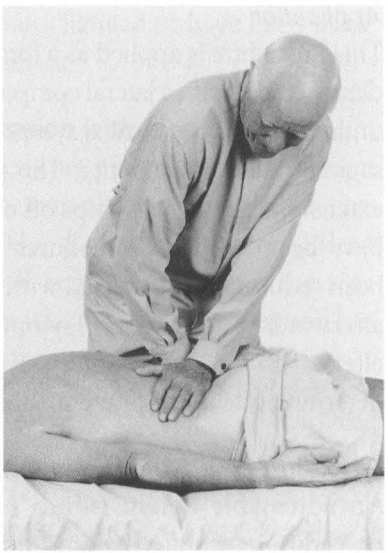

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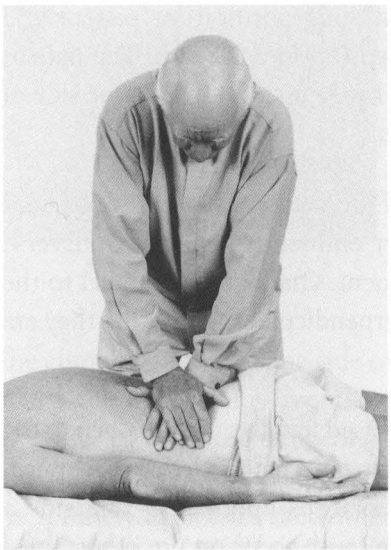

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Application

This procedure involves primarily extension, but with a lateral component. It is primarily used in derangement syndrome to reduce unilateral or asymmetrical symptoms that have remained unchanged following a thorough exploration of all previous procedures.

It may be used as a progression from extension in lying with hips off centre and extension in lying with hips off centre with clinician overpressure (Procedures 11 and 12) if response to these have been equivocal, that is, symptoms are no better or no worse afterwards.

Procedure 15 – Rotation manipulation in extension

An adjustable-height plinth is recommended to perform this procedure most effectively. This should be at a level that allows the clinician to be over the patient sufficiently for their line of force to be perpendicular to the spine. The starting position for patient and clinician is as for Procedure 7, extension mobilisation. The patient lies relaxed prone with their arms by their side, close to the side of the table where the clinician stands.

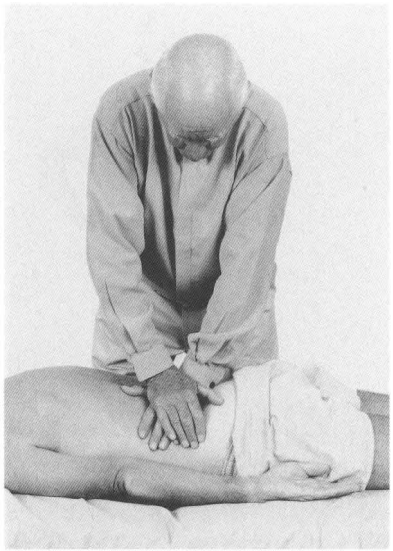

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Photo 62: Rotation manipulation in extension. With both hands on the appropriate side, apply high-velocity short-amplitude thrust.

A favourable response will have been generated previously with unilateral rotation mobilisation in extension, but symptoms have returned. The clinician stands on the opposite side from the side to be mobilised. Place one hand on top of other hand to perform the mobilisation on one side; force is directed anterior and slightly medially. The pre-manipulative test mobilisation should reduce symptoms that return afterwards; the manipulation is then indicated. The clinician leans over the patient with the arms perpendicular to the spine and applies unilateral pressure slowly until the spine feels taut. Then a high-velocity thrust of very short amplitude is applied and immediately released.

The segmental level at which the manipulation is performed is decided by symptom response during the application of extension mobilisation, which will always precede this procedure. The appropriate level is the one at which symptoms are centralised, reduced or abolished, but return when the mobilisation pressure is released. Only one manipulative thrust should be performed at any one treatment session.

Application

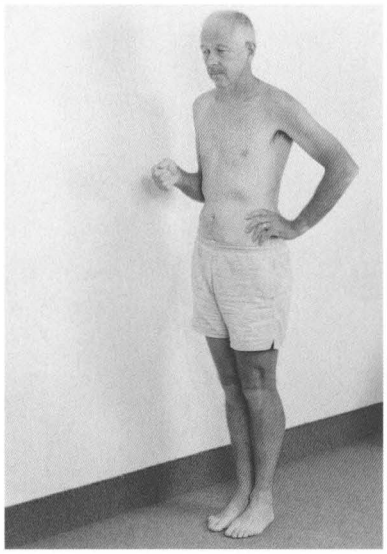
The application of this manoeuvre is as for the previous procedure. When the desired result is not obtained with the rotation mobilisation in extension, progression to a manipulation should be considered. Response to the pre-manipulative testing must be favourable before proceeding further.

Lateral principle – description of lateral procedures

Procedure 16 – Self-correction of lateral shift or side gliding

The direction of side gliding is nominated by the direction the shoulders move, rather than the hips. Thus, when the shoulders move to the right and the hips to the left, this is right side gliding. The side gliding procedure for self-correction of lateral shift can be performed

Photos 63, 64, 65, 66: Self-correction of lateral shift/side gliding – against a wall. Patient stands with feet together, 30 centimetres (twelve inches) out from the wall. The upper arm, on the side to which they are shifted, is held against the side of the ribcage, with the elbow at right angles. In the case of a right lateral shift, the patient stands with the wall at their right side (63). The patient leans against the wall and places the outer hand against the lateral crest of the ilium (64). The patient pushes the pelvis towards the wall, so that in this case the right hip approximates the wall. Keeping the upper trunk against the wall, the pelvis is then shifted away from the wall and the sequence repeated ten to twelve times (65). Patient positioning from behind, in this case demonstrating self-correction of a left lateral shift (66).



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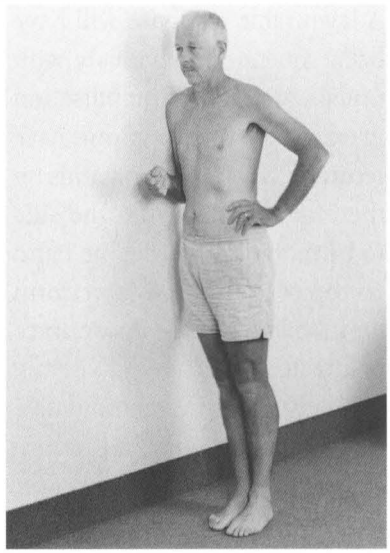

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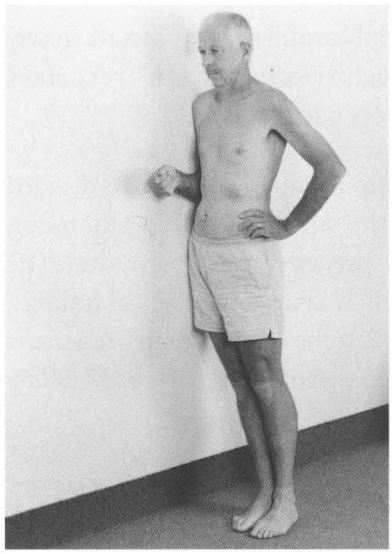

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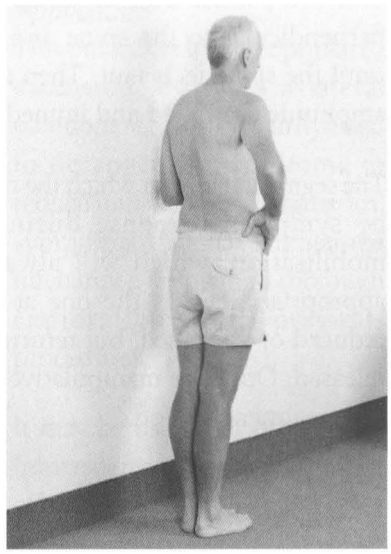

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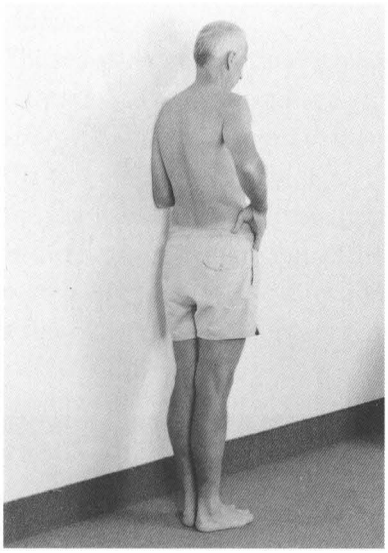
in several different ways. The different methods allow different amounts of pressure to be applied and also differentiate between sustained and repeated pressures. The procedure can be performed against a wall, in a doorway or freestanding. In all instances it is important to ensure that weight becomes evenly distributed on both legs.



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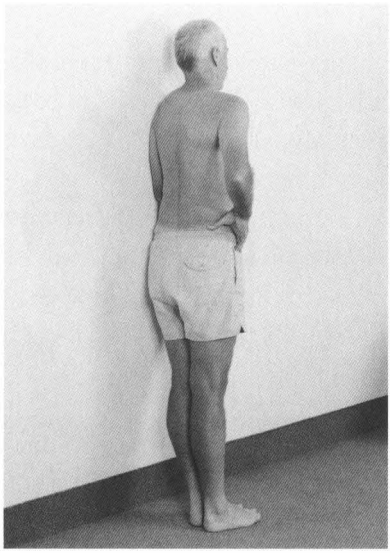

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Photos 67, 68: Patient positioning from behind, in this case demonstrating self-correction of a left lateral shift (67/68).

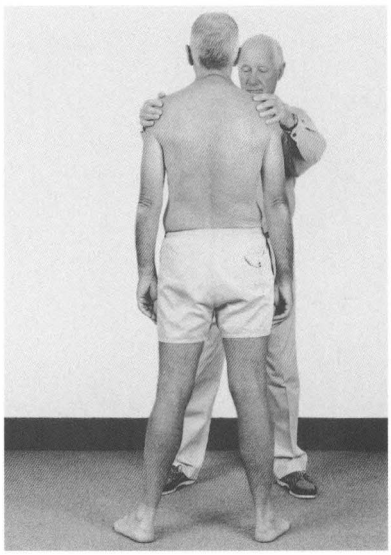
16a. Self-correction of lateral shift/side gliding – against a wall

Applying the procedure against a wall is the first choice and is most effective if the patient has lost some of the shift correction when they return home. They stand with feet together, one foot near to the wall, with the side to which they are shifted next to the wall. Thus for a right lateral shift, the right side is near the wall. The bent elbow is placed against the side of the patient as they lean against the wall. The hip is pushed towards the wall with the outer hand on the iliac crest. Overpressure is easily achieved with this method, and the movement can be repeated or sustained depending on the best response. Greater amounts of side gliding are achieved by starting with the outer leg further away from the wall. Once the movements or sustained position is finished, the patient should step the inner leg back towards the wall and return to neutral standing.

16b. Self-correction of lateral shift/side gliding – in a doorway

Alternatively, the procedure can be performed in a doorway of a suitable width. The patient stabilises their upper trunk by placing their forearms against the doorframe. Maintaining this position, they are instructed to move their hips sideways towards the doorframe. The movement is repeated up to ten times with each movement a little stronger and the final movements briefly held in the overcorrected position. The doorframe provides stability of the upper trunk and thus allows stronger pressures to be applied than the freestanding method.

Photos 69, 70: Self-correction of lateral shift/side gliding – freestanding. Clinician holds patient's shoulders to stabilise upper body and (69)... ...patient moves hips away from the side of pain (occasionally a favourable response is gained moving the hips towards the side of pain) (70).



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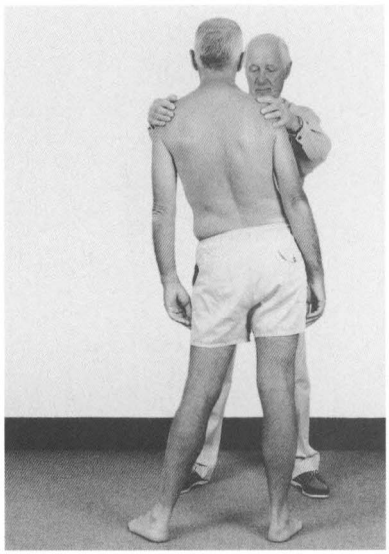

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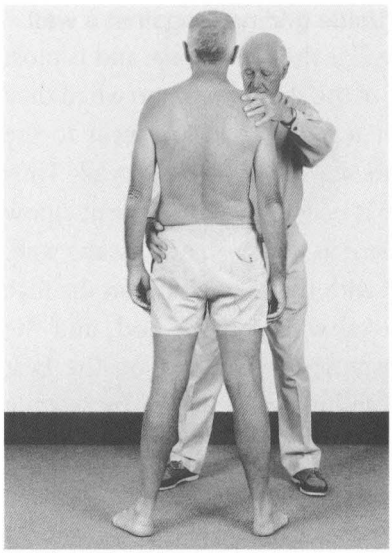
Photos 71, 72: Self-correction of lateral shift/side gliding – clinician overpressure. Clinician places hand on patient's shoulder on the side to which the patient is shifted. The other hand is placed on the opposite side on the patient's iliac crest. The patient is then instructed/guided to correct the shift in response to pressure provided by the clinician (71). Repeatedly applying and releasing pressure, the patient can quickly learn the appropriate manoeuvre (72).



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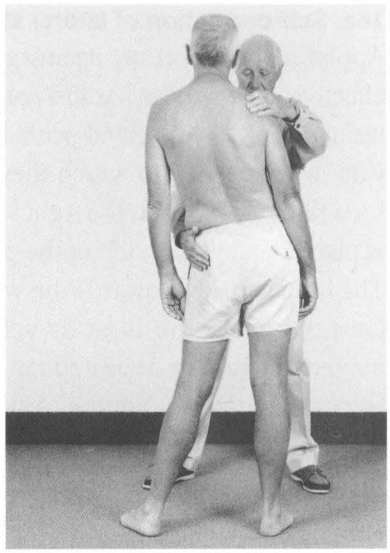

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16c. Self-correction of lateral shift/side gliding – freestanding

To perform the movement freestanding, the patient is instructed to glide their hips laterally while keeping their shoulders level with the floor. If patients are having difficulty performing the movement, they should be guided by the clinician applying pressure with one hand on the shoulder and one hand on the opposite iliac crest. The patient can then be taught how to apply overpressure in a similar way. Alternatively, the clinician can hold both shoulders and have the patient side glide the hips to the right or left below the fixed upper trunk. They repeat the movement up to ten times, briefly holding the last few movements in the overcorrected position.

Application

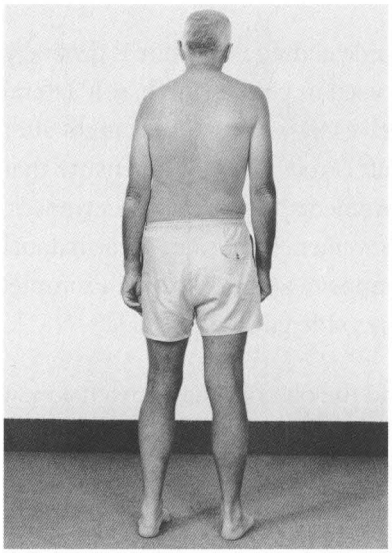
The self-correction of lateral shift or side gliding procedure is generally only applied to derangements. It is used in patients with 'soft' lateral shifts that are able to correct themselves. It is also always taught after the manual correction of lateral shift (Procedure 17) to ensure that the patient is able to maintain improvements and prevent recurrences. In this situation the direction of the movement is the same as the manual correction, which is the direction opposite to the shift. For example, a right lateral shift deformity requires side gliding to the left.

Having corrected the lateral shift and the obstruction to extension, it is essential to teach the patient to perform self-correction by side gliding in standing followed by extension in standing. This must be done on the very first day, so that the patient is equipped with a means of reducing the derangement himself at first sign of regression. Failure to teach self-correction leads to recurrence within hours, ruining the initial reduction, and the patient will return the next day with the same deformity as on the first visit.

The side gliding movement may also be applied to 'soft' lateral shifts. Such patients present with a lateral shift deformity, which initially appears to be resistant to self-correction. However, repetition of the procedure begins a gradual process of correcting the deformity and regaining the lost movement.

The side gliding movement is also applied to postero-lateral derangements that do not present with a lateral shift deformity, but that are either unchanged or worsened by extension principle procedures and respond to lateral procedures – in this case a loaded lateral procedure in a degree of extension. In this situation, the hip movement is generally applied away from the pain. For example, if the patient had right-sided pain they would perform right side gliding – shoulders are moved to the right and hips to the left. As in all procedures, performance is dictated by favourable symptomatic response; should it prove appropriate, although uncommon, movements are applied towards the symptomatic side. The procedure can also be applied with clinician overpressure that may help confirm an appropriate loading strategy.

Photos 73, 74, 75, 76: Manual correction of lateral shift. Clinician stands on the side to which the patient is shifted, in this case to the right (73). Patient places upper arm against rib cage with the elbow at ninety degrees (74). Clinician's arms encircle the patient, clasping hands with thumbs at the iliac crest (75). Clinician pulls patient's pelvis towards him/herself, while at the same time pushing against patient's upper arm and trunk. Repeatedly apply and release the pressure rhythmically and progressively the shift is gradually reversed (76).



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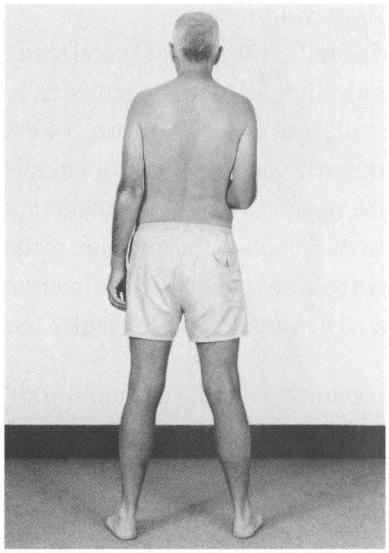

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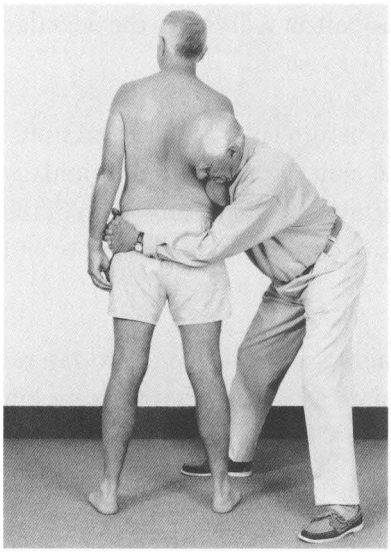

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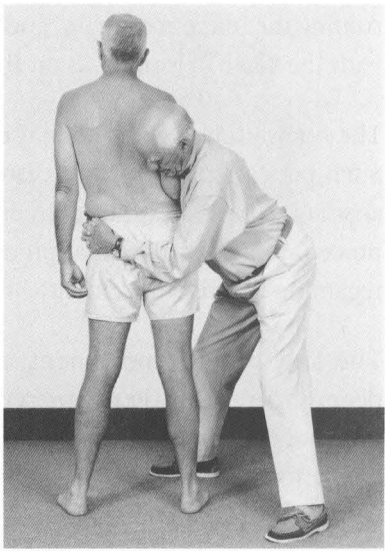

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Procedure 17 – Manual correction of lateral shift

The direction of lateral shift is nominated by the direction the shoulders are displaced, rather than the hips. Thus, when the patient stands with their upper body shifted to the right and hips to the left, this is a right lateral shift. This procedure has two parts: first the deformity of lateral shift is corrected; then, if present, the deformity of kyphosis is reduced and full extension is restored. It is very important to monitor symptom response at all times during this procedure. An increase of peripheral pain indicates a modification is required – for instance, altering the angle of flexion/extension. If no

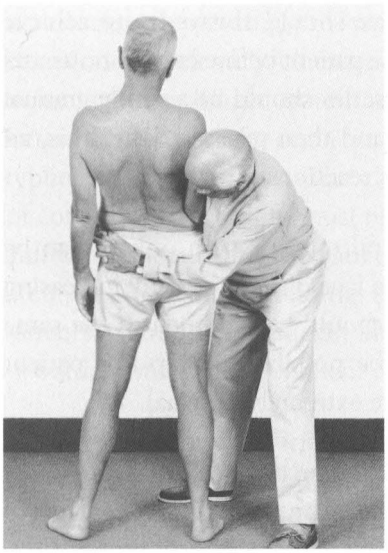

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Photo 77: Once a degree of over-correction is achieved, the patient is held over-corrected and extension recovery movements are added (77).

modification can be found to reduce, abolish or centralise symptoms, the manoeuvre should be abandoned.

The patient stands with the feet shoulder-width apart, attempting to weight bear evenly. As the deformity is corrected in the following way, the clinician ensures that the patient comes to stand with an even weight distribution, if this is not the case already. The elbow on the side they are shifted towards is bent at ninety degrees, just above the ilium.

The clinician stands on the side to which the patient is shifted and places his or her anterior shoulder against the patient's arm just above the elbow. They stand with their feet wide apart, their forward leg in front of the patient, and maintain a good back position with the knees bent. The clinician's arms encircle the patient's trunk, clasping the hands around the iliac crest with interlocked fingers, and with their head behind the patient.

The clinician applies a series of slow and small amplitude pressures with his or her shoulder against the patient's elbow, pushing the patient's trunk away while at the same time drawing the patient's pelvis t uards the clinician. The two movements are conducted simultaneously, thus producing a slide gliding movement, at the same time as trying to equalise the patient's weight. The movement is slow, rhythmical and of small amplitude. Each pressure should be held for three to five seconds prior to partial release, not back to the start position. Gradually, in this manner the lateral shift is reduced and overcorrected. If no reduction of symptoms occurs, a sustained, longer pressure may be necessary to attain end-range side glide movement.

Initially there will be significant mechanical resistance to the procedure, which may also cause an increase in pain. It is quite safe to continue with correction as long as centralisation of pain takes place, and therefore the patient must be questioned continually about the behaviour of their pain. Relaxation of the patient during the

procedure is very important and we should always try to achieve this. Explaining the procedure to the patient before correction assists this goal. The first pressure in the series should be a gentle gradual squeeze that is held momentarily and then released. After this, an accurate assessment of the patient's reactions must be made.

If, unusually, the patient has central midline pain only, it can be dangerous to force correction in the face of progressively increasing central pain. The angle of flexion should be modified. If the same response still occurs, it may not be possible to help this patient immediately. Such presentations are extremely unusual.

The application of too much pressure or too rapid a correction should be avoided; this could sometimes result in fainting and collapse of the patient. If well tolerated the pressure is applied a little further each time, again being momentarily held and then released, although not back to the beginning of the range. As correction progresses over ten to fifteen rhythmically applied pressures, the patient usually describes that the pain moves from a unilateral to a central position. By the time overcorrection is achieved, there will be a significant reduction in pain intensity or the pain may have moved slightly to the opposite side. If after a few rhythmical pressures no progress is made in the correction, it may be necessary to apply a longer and more sustained pressure.

Response to manual correction of the lateral shift can be extremely varied. Sometimes reduction may be felt clearly by the clinician and the patient's trunk is felt to move slowly but surely from its previous position. In lightly built or tall and slender patients shift correction may occur quite easily, and only a few minutes of ten to fifteen pressures are required to begin to reduce the derangement. On the other hand, some acute lateral shifts are extremely difficult to reduce and clinicians may have to perform five or six series of corrective pressures. Patients who present with lateral shifts and have pain only above the knee mostly respond. In those with pain below the knee, especially those with neurological signs and symptoms, lack of response is not uncommon. Such derangements often prove to be irreducible.

Once correction of the lateral shift deformity or centralisation of pain has been achieved, we must now proceed with restoring the lumbar lordosis. In some centralisation of pain occurs, but the shift is still apparent although less than it was; extension at this point can be

introduced, depending on symptom response. Introduction of extension is preferably commenced in the standing position. The patient may no longer exhibit a lateral shift but may still have a flattened lumbar spine, or, in a minority of patients, some degree of kyphosis may be evident. The clinician, still holding the patient as for correction of the shift, must maintain slight overcorrection of the shift while encouraging the patient to arch backwards. As the patient attempts extension in standing, the clinician's head can support the patient's trunk. The clinician should ensure the pelvis translates forward in order for the patient to remain balanced and relaxed.

A few movements will indicate the ease, or not, with which the lordosis will be restored. If the extension range improves rapidly, it is usually better to recover as much extension as possible in the standing position. If extension does not increase rapidly, then it is better to change to extension in lying with hips straight or off centre, depending on what is needed for symptom control. The clinician should hold the shift overcorrected as the patient performs extension in lying (Procedure 12b). This procedure should produce a steady and continuing reduction of central pain, and it should automatically follow for all patients once the lateral shift has been corrected and the symptoms have centralised.

Application

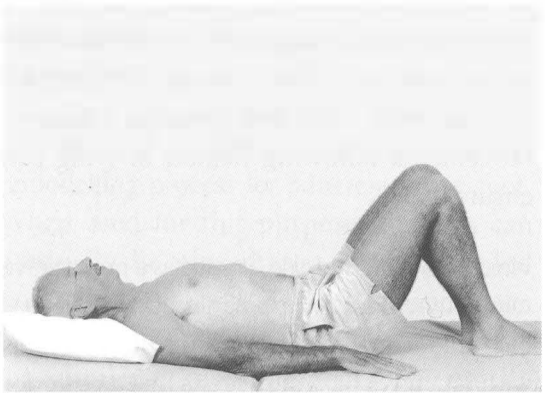
This procedure is only used for a particular sub-group of derangement that require the lateral principle, namely those with an acute lateral shift deformity. The patient will be fixed, for instance, in a right lateral shift, and is unable, without clinician assistance, to maintain correction of the deformity. In this instance, where patient-generated forces (Procedure 16) alone are unable to alter the mechanical or symptomatic presentations, then clinician-generated forces must be used to bring about a situation that the patient is able to self-manage. Some patients with a 'soft' shift are able to achieve shift correction independently, but those with a 'hard' shift will need clinician assistance. Those with more severe shifts are more likely to need clinician assistance. Following manual correction of a lateral shift, it is essential that patients be taught self-correction of a lateral shift (Procedure 16) so they are able to maintain improvements and prevent recurrences. Often the lateral principle of treatment is only required temporarily, in some cases just for the initial shift correction, after which they require the extension principle.

The lateral shift correction must not be hurried, but must proceed with care. In some difficult patients it may take up to forty-five minutes. Frequent repetition of the corrective procedure may be necessary to reduce peripheral symptoms. It is essential during the corrective procedure that continual reference is made to the patient's symptoms, especially those felt most distally. If there is a production or worsening of peripheral symptoms, the position that shift correction is being performed in should be considered. At times a change in the angle of flexion or extension of the lumbar spine may produce a better response. Occasionally, correction of the shift can be achieved with the patient sitting on a raised plinth in some degree of flexion.

Once the lordosis has been restored, this must be maintained from the first day in order to maintain reduction of the derangement. The most common cause of recurrence of symptoms shortly after correction of a lateral shift is poor sitting posture. For example, after a successful reduction, a patient may drive home for twenty minutes or more, and on leaving the car the derangement has recurred. Such situations must be anticipated; patients must be told about the importance of maintaining the lordosis in order to maintain the shift correction. They should be provided with a lumbar support on leaving the clinic. In an emergency, a rolled-up towel will provide temporary support, but long-term use causes more discomfort than it prevents.

Following reduction of acute cases the patient should go home and immediately check in a mirror to see if the shift is beginning to reoccur. If this is so, they should perform the self-correction of lateral shift (Procedure 16) before it becomes too difficult to correct the shift without external help. If the shift is corrected and pain centralised, they should then lie prone for a few minutes, and then perform a series of extension in lying with hips off centre (Procedure 11). This pattern should be repeated hourly, or as frequently as possible during that day, and sitting should be avoided as much as possible. If forced to sit, the patient must maintain the lumbar lordosis. They should lie with the spine overcorrected in extension for thirty minutes before sleeping. The next morning, although on first waking the pain may be quite noticeable, after performing the correction procedure and extension in lying a few times, there is usually a marked improvement in symptoms and deformity.

Photos 78, 79: Flexion in lying.
From start position raise knees to chest (78). Apply overpressure by pulling knees with both hands as far as possible – 'further, further, further' (79).



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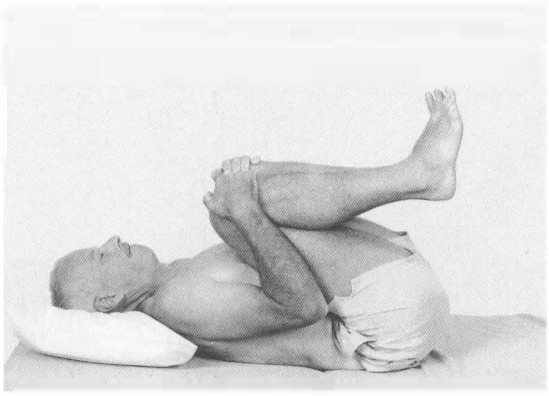

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Flexion principle

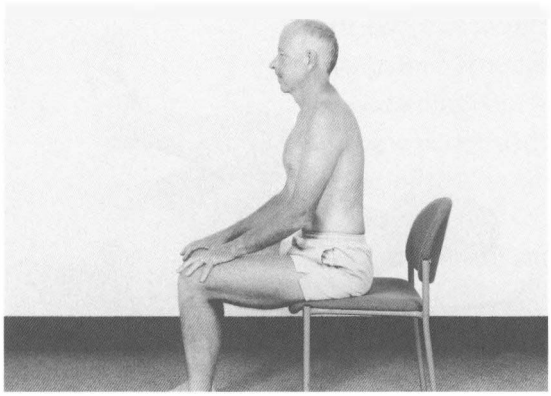
Procedure 18 – Flexion in lying

The patient lies supine with knees and hips flexed at about forty-five degrees and the feet flat on the plinth. The patient is then instructed to bring their knees up towards their chest, applying overpressure with hands around the knees to achieve maximum possible flexion. The knees are then released and the feet are placed back on the plinth in the starting position. The exercise is repeated about ten times. The first few flexion stresses can be applied cautiously; as long as the symptom response is satisfactory, overpressure may be applied more strongly with each movement, and maximally on the last repetitions.

Application

Flexion in lying is used in several mechanical syndromes. It is the first procedure in the reduction of anterior derangements, which improve with the flexion principle of treatment. It is also used in the remodelling of flexion dysfunction. It is important to test out flexion in lying following the reduction of posterior derangements, which improve with the extension principle of treatment. During the reduction of such derangements, flexion will have been avoided; in the restoration of function, it is essential that full-range pain-free flexion be attained and that reduction is stable. Flexion in lying is used to test the stability of reduction of a derangement prior to commencing more vigorous flexion procedures, such as flexion in standing, used to prevent the development of nerve root adherence. If the nerve root has already become adherent, there is little likelihood of recurrence of derangement, presumably as scarring has also stabilised the disc.

Photos 80, 81, 82: Flexion in sitting.
Sit upright with knees at right angles (80). Bend forwards, if possible touching the floor with fingertips (81). Progression can be made by clasping hands around the ankles and pulling down (82).



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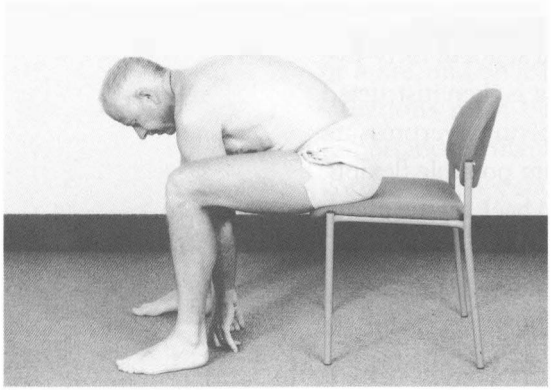

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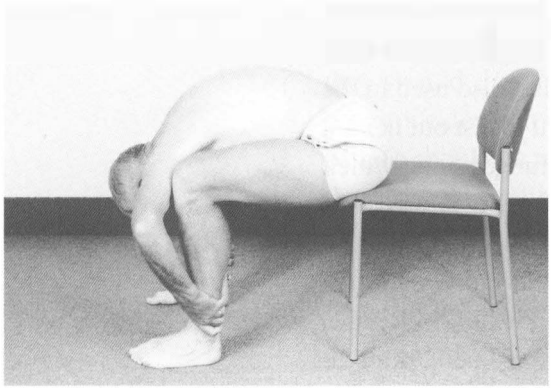

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When flexion procedures are used in the restoration of function or to test the stability of reduction following derangement, it is always important to ensure that this does not disturb the reduction or create the potential for recurrence. Full and pain-free extension movements following flexion in lying can ensure this.

Flexion in lying can also be used as a provocative manoeuvre if earlier mechanical evaluation has been inadequate. A worsening of symptoms with repetitive flexion suggests that a posterior derangement is present and that the extension principle should be applied.

As with every procedure, symptoms are carefully monitored during and after the exercise. If symptoms are worsened or peripheralised, the procedure is being applied too early or inappropriately.

Procedure 19 – Flexion in sitting

Sitting in a straight-back chair with knees and hips at ninety degrees, the patient puts his head between his knees, and then returns to an upright posture. The movement is repeated about ten times; with each repetition the patient tries to reach with the hands further under the chair. More pressure can be applied if the ankles are clasped and used to pull further into flexion. They return to an upright or slumped posture depending on the purpose of the applied procedure. Depending on the purpose of the procedure, knees can be extended.

Application

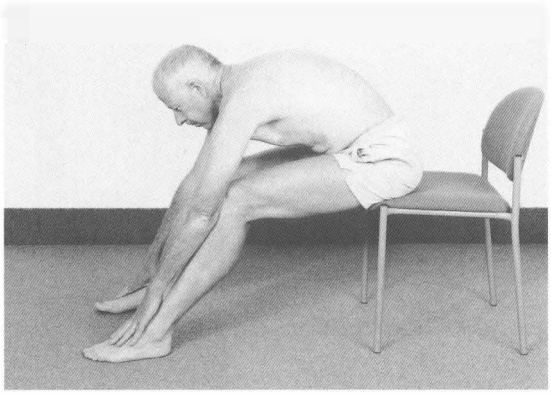
This procedure is used in the reduction of anterior derangements, those responding to the flexion principle of treatment. The procedure is a progression from flexion in lying. As these individuals generally sit with the lordosis fixed, their normal sitting posture should be avoided and a slumped sitting

position encouraged following completion of these movements. A brief period in extension is rarely provocative, but the patient should not be permitted to sit upright for more than a few minutes.

This procedure may also be used in the remodelling process for adherent nerve root. When used for this purpose, the legs can gradually be placed in a more extended position, which has the effect of enhancing the stress upon the affected tissue. Extending the legs until the knees are straight gradually increases the flexion force on the adherent nerve root. It is important for the patient to return to the fully upright position in this instance. Failure to ensure this is done causes flexion to be sustained throughout the procedure instead of intermittently applied. Remodelling or return to function without aggravation of symptoms is best achieved with intermittent loading when a posterior derangement has been present or is suspected.

This procedure is also used as the next progression after flexion in lying (Procedure 18) for recovery of function after a posterior derangement.

Photos 83, 84: A progressive increase in loading is achieved by partially extending the knees. This is also appropriate loading for adherent nerve root (83). A further progression, straightening both knees and reaching as far as possible towards the ankles (84).



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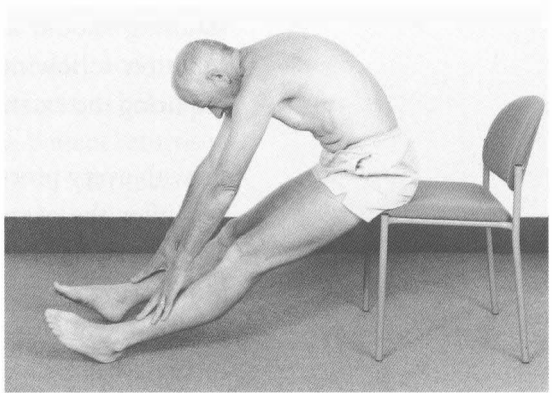

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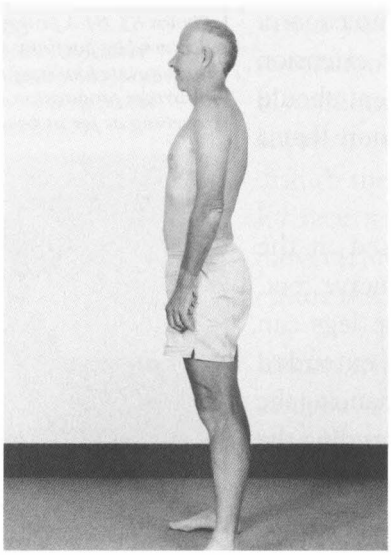
Procedure 20 – Flexion in standing

The patient stands with feet shoulder-width apart for a good base of support. They are instructed to place their hands on the front of their thighs, and then run them down the front of their legs, all the time maintaining straight knees. They are told to go as far as possible and then return to neutral standing. The exercise is repeated about ten times, initially with some caution, and latterly encouraging more vigour.

Application

Flexion in standing has several applications. It may be used as a progression from previous flexion procedures in the reduction of anterior derangements, those requiring the flexion principle of treatment. It is also the necessary loading strategy for management of adherent nerve root, a specific tissue dysfunction. Flexion in

Photos 85, 86: Flexion in standing.
Patient stands upright (85).
Patient bends forward as far as possible – 'further, further, further' (86).



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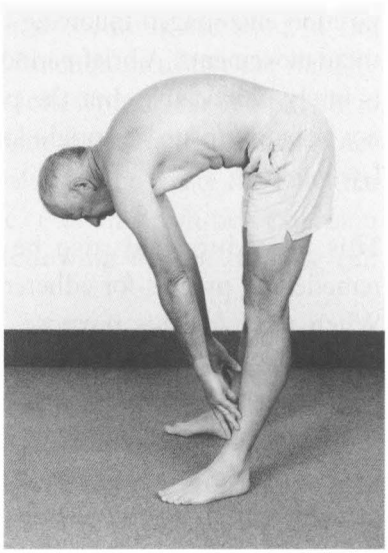

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standing should also be tested in the later stages of recovery of function following reduction of posterior derangements, those requiring the extension principle.

As with every procedure, symptoms are carefully monitored during and after the exercise. If it results in increased or peripheralised symptoms, the procedure is being applied too early or inappropriately. Monitoring of symptoms is especially important in recovery of function; while such patients may easily tolerate flexion in lying, the increased gravitational stresses of flexion in standing may easily aggravate symptoms.

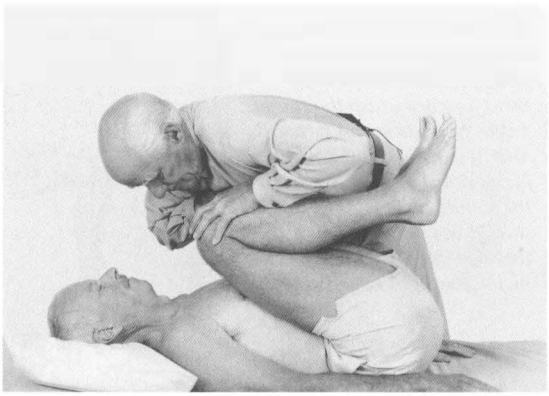
This procedure is also useful in chronic patients who have developed fear–avoidance towards activity. Overcoming a fear of flexion can be done by regular application of this procedure – that is, through exposure to the 'feared' activity. Once their anxiety about the consequence of flexion in standing is overcome, they find it easier to embark on other activities.

Procedure 21 – Flexion in lying (with clinician overpressure)
The patient lies supine with knees and hips flexed about forty-five degrees and the feet flat on the plinth. The patient brings their knees up towards their chest, and the therapist applies overpressure, pushing the patient's knees and legs downwards.

Application

This procedure is rarely used other than in the reduction of anterior derangements that have not fully responded to the previous flexion procedures (Procedures 18 – 20).

Photo 87: Flexion in lying with clinician overpressure. Apply the pressure slowly but steadily to maximum end-range.



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Flexion principle with lateral component

Procedure 22 – Flexion in step standing

The patient stands with one leg on the floor and one leg resting on a stool with the knee and hip flexed at about ninety degrees. The leg on the floor remains fully extended at the knee throughout the procedure. The patient is instructed to bend forward, keeping the trunk inside the raised leg so that the shoulder approximates the raised knee. The patient may apply more pressure by grasping the ankle of the raised leg and pulling themselves further into flexion so that the shoulder passes below the raised knee. The pressure is then released and the patient returns to the upright position. The sequence is repeated up to ten times. It is important that the upright position is regained between each movement and the lordosis restored.

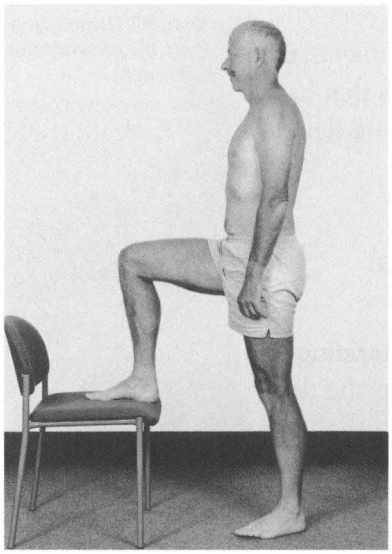
Application

This procedure causes an asymmetrical flexion stress and is applied when there is a deviation in flexion, which may be present in derangement or in dysfunction. In both syndromes the leg to be raised is that opposite to the side to which deviation in flexion occurs – for example, for deviation in flexion to the left, the right leg is raised.

As in all other procedures, response will be characteristic of derangement or dysfunction when the manoeuvre is applied in the different circumstances. In derangement the procedure should abolish, decrease or centralise symptoms; in dysfunction it should produce symptoms at end-range on each occasion, which will abate as soon as the exercise ceases.

In some cases of derangement, if the procedure is repeated excessively, a deviation to the other side results when the patient attempts sagittal flexion. When this occurs, pain can change sides also.

Photos 88, 89, 90, 91: Flexion in step standing.
Stand upright, place the foot (opposite to the side to which the deviation occurs) on a raised platform, such as the seat of a stable chair (88). Bend forward, clasp the raised ankle, pull the trunk forward and down (89). Pull down as far as possible (90). Note that the shoulder, where possible, should be inside the knee. In this case the right shoulder is inside the right knee (91).



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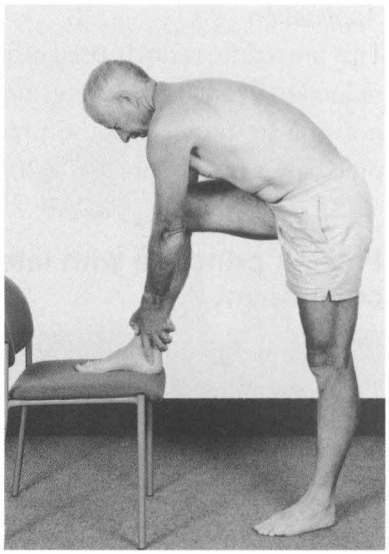

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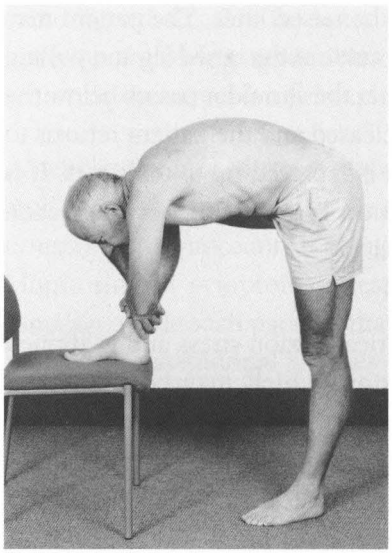

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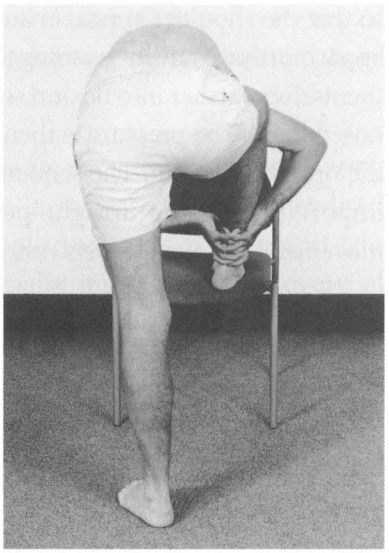

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Procedure 23 – Rotation in flexion

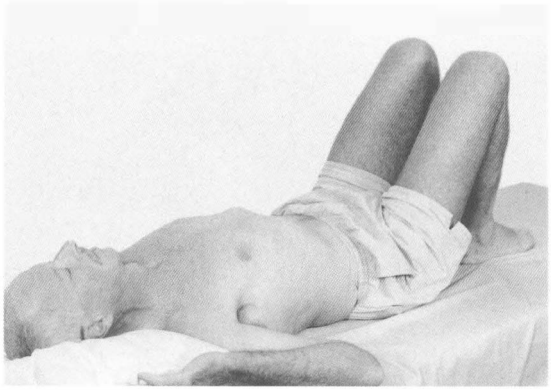
Due to the complex nature of this procedure, it is best that the patient practices it several times before leaving the clinic. At home it is most easily performed on a wide bed.

For the starting position, the patient lies supine with knees bent and feet flat on the bed. They lift their pelvis off the bed and place it off centre, away from the painful side. With right-sided pain the pelvis is shifted to the left and legs are rotated to the right. Knees are then raised until they are over the hips, and the trunk and lower limbs are at about ninety degrees to each other. The exact angle may need adjusting for different individuals. Keeping the appropriate hip angle, the knees are lowered to the bed. If overpressure is needed, the upper leg can be straightened. As long as the position is comfortable, and pain is reduced or abolished during the procedure, the position should be sustained for two to three minutes. They then return to the neutral position, but it may be repeated if desired. On completion of the procedure, the patient resumes the upright position by first getting into side lying.

Application

This procedure is used in the management of derangements that have not improved or have worsened with sagittal plane manoeuvres. These derangements require the lateral principle with a degree of adjunct flexion. In these circumstances the patient is instructed in this procedure and advised to repeat regularly as long as a favourable symptomatic response is still experienced.

Photos 92, 93, 94: Rotation in flexion. Patient lies supine with hips and knees flexed (92), then, shifts hips away from the side of pain (93). Then rolls knees towards the side of pain as far as possible (94), then returns to start position.



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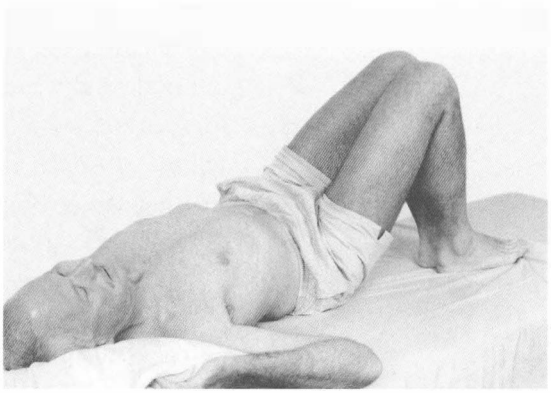

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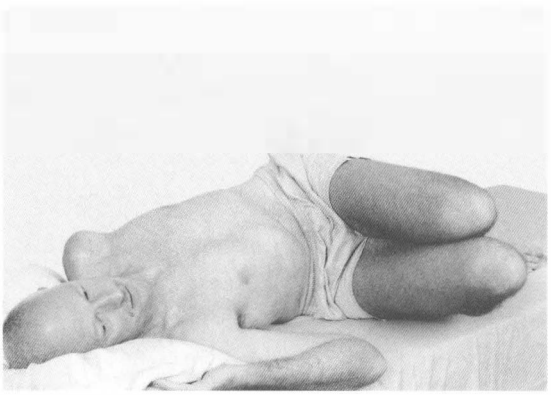

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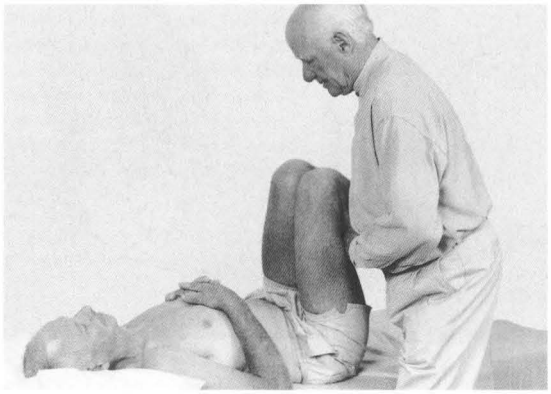
Photos 95, 96, 97: Rotation mobilisation in flexion (rhythmical).
Place hips at right angles with knees bent (95). Stabilise the shoulder, rotate the lumbar spine and repeatedly increase and decrease the degree of lumbar rotation (96). After rotating to end-range (97), return to neutral.



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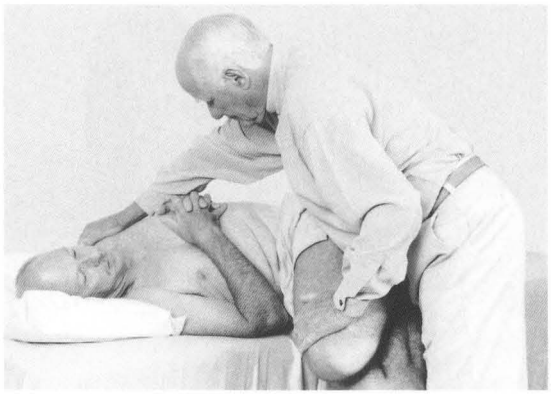

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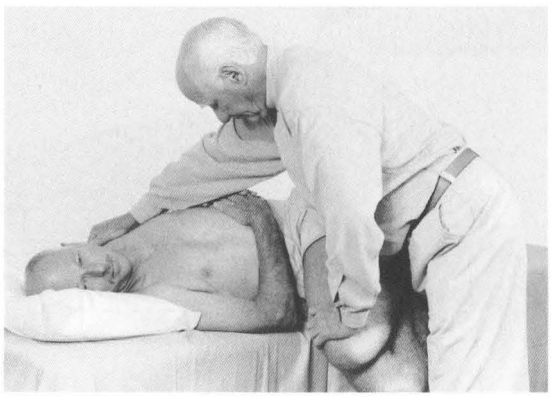

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Procedure 24 – Rotation mobilisation in flexion

The procedure is similar to the patient-generated procedure above (Procedure 23), but in this instance the clinician performs the manoeuvre. An adjustable-height plinth is preferable to perform this procedure most effectively. This should be lowered to a suitable height to permit full control of the patient and the technique. The suitable level is usually just above the knees. The patient lies supine, with knees flexed and feet flat on the plinth, with their body by the side of the plinth to which the legs will be rotated. The clinician also stands on that side, facing the patient. The patient must relax completely during this procedure.

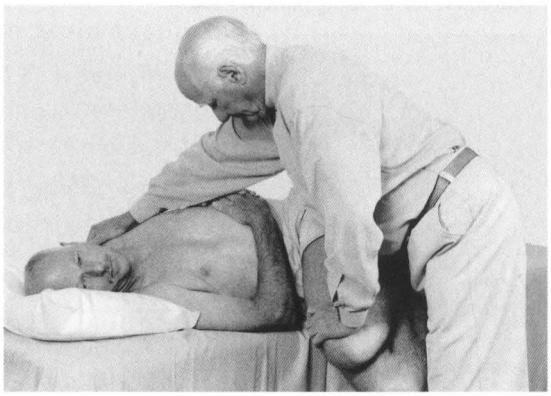
The clinician then flexes the hips and knees until the hips are at a right angle with the trunk. The knees are then lowered over the side of the plinth until the patient's lower leg rests on the clinician's thigh. Ensure the pelvis is at the edge of the plinth. The knees are allowed to sink as far as possible towards the floor. The clinician stabilises the patient's opposite shoulder, or lower ribs if the shoulder cannot be reached, and the position is sustained and symptom response is noted.

If this is favourable, overpressure is applied by pushing the knees further to the floor; moving the knees further to the floor alters the angle of rotation. The procedure can be done either as a rhythmical or sustained mobilisation. Flexing the knees further towards the chest alters the angle of flexion at the lumbar spine.

The angle of flexion at the hips can sometimes be critical in this procedure, with certain angles aggravating symptoms and other angles providing relief. Ninety degrees is the angle to

try first, but a certain amount of experimentation is sometimes necessary to find the angle that generates the best symptomatic and/or mechanical response. The angle of flexion at the hips affects the rotation of the pelvis and consequently alters the sagittal position of the lumbar spine. With increasing hip flexion there is increasing lumbar flexion, and as hip flexion is decreased the lumbar spine moves towards neutral.

Photo 98: Rotation mobilisation in flexion (sustained). Hold at end-range and monitor symptoms.



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Smaller clinicians with bigger patients may find it easier to get the patient to shift their hips away prior to the mobilisation, and may find it easier to stabilise the upper trunk with a belt.

Application

This procedure is used in derangements that have been unchanged or worsened by extension principle procedures and that respond to lateral principle procedures. This is a lateral manoeuvre with considerable adjunct flexion. It is a progression from the similar patient-performed manoeuvre (Procedure 23) and is used if this procedure causes no improvement or a reduction or centralisation of symptom that does not last.

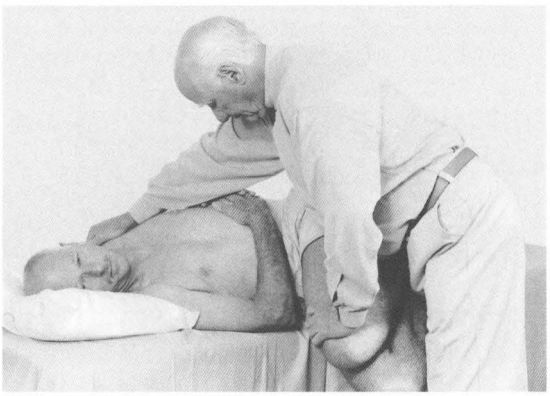
Usually the lower half of the body is rotated in the same direction as the painful side. If the patient has right-sided pain, their legs are moved towards the right. However, should this cause a worsening of symptoms, the opposite side rotation should be explored – as in every procedure, symptomatic response guides treatment direction. This procedure can be applied as a mobilisation, with repeated movements, or as a sustained procedure, but usually the latter generates the best response.

If the manoeuvre causes a reduction or abolition of distal pain, then the patient is advised to continue with a similar procedure at home, as a sustained position, on a regular basis (Procedure 23).

Procedure 25 – Rotation manipulation in flexion

An adjustable-height plinth is necessary to perform this procedure most effectively. This should be lowered to a suitable height to permit full control of the patient and the technique. The suitable level is

Photo 99: Rotation manipulation in flexion. Hold at end-range and apply high-velocity, short-amplitude thrust using patient's thighs.



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usually just above the knees. The patient and clinician starting positions are as for Procedure 24, which is always conducted as an essential pre-manipulative test procedure. The response to the rotation mobilisation in flexion will have been favourable, but symptoms have returned.

The pre-manipulative mobilisation (Procedure 24) is conducted first. The clinician flexes the hips and knees until the hips are at a right angle with the trunk. The knees are then lowered over the side of the plinth until the patient's lower leg rests on the clinician's thigh. Ensure the pelvis is at the edge of the plinth. The knees are allowed to sink as far as possible towards the floor. The clinician stabilises the patient's opposite shoulder, or lower ribs if the shoulder cannot be reached, and the position is sustained and symptom response is noted. If this is favourable, overpressure can be applied by pushing the knees further to the floor. The patient must relax completely for this to be successful. If symptoms return on reversing the mobilisation procedure, a manipulation should be considered.

The angle of flexion at the hips can sometimes be critical in this procedure, with certain angles aggravating symptoms and other angles providing relief. Ninety degrees is the angle to try first, but a certain amount of experimentation is sometimes necessary to find the angle that generates the best symptomatic and/or mechanical response.

For the manipulation itself, the same procedure is gone through as for rotation mobilisation in flexion. The patient is positioned in extreme end-range rotation, with the opposite shoulder or the trunk stabilised. This is either done with a hand on the opposite shoulder, or ribs, or if the patient is large and the clinician small, a fixation belt can be used. If the manipulation is indicated a high-velocity, short-amplitude thrust is delivered, pushing the knees further to the floor while simultaneously stabilising the shoulder/trunk.

Application

This manipulation is generally applied to derangements that have failed to improve or worsened with sagittal plane movements. They will have shown some improvement with rotation mobilisation in flexion performed by the clinician and by the patient at home

(Procedures 23 and 24), but improvements will not have been sustained. In this situation, as long as the pre-manipulative test procedure of rotation mobilisation in flexion again produces reduction, abolition or centralisation of symptoms, a manipulation should be considered. The manipulation is performed to the same side to which mobilisation was administered. This is the side that causes a decrease, centralisation or abolition of symptoms, regardless of whether this is achieved with movement towards or away from the painful side.

Only one manipulative procedure should be performed at one session. If a sustained improvement has not occurred after two sessions, further manipulative treatment should be abandoned as it is unlikely to provide any benefit.

18: Patient Management

Introduction

Patient management refers to the organisation, supervision and implementation of the strategies to be applied for the successful education and treatment of the patient. The patient has been questioned regarding the history of their complaint and their concerns and beliefs regarding their problem. An examination of appearance and function of the spine has been performed. The findings from the history and the examination allow the patient to be classified according to one of the mechanical syndromes or a non-mechanical diagnosis. From this classification the therapeutic management can be structured to suit the patient's requirements. The therapeutic management will consist firstly of an educational component and, secondly of an active mechanical therapy component. The educational component provides the patient with an understanding of their problem and the role of movement in their rehabilitation, while the active therapy component provides instructions in the appropriate exercises.

This chapter discusses the importance of information provision in the patient-clinician relationship in general and the value of educational interventions in back pain. However, patient management not only concerns what is done during the clinical encounter, but also how we as clinicians interact with the patient. Our ability to communicate well with the patient is a vital component of the clinical interaction. A breakdown in communication can nullify the clinician's input; some attributes of the clinician-patient interaction are thus considered. As this should be understood against the background of patient satisfaction, dimensions of this are also described.

Sections in this chapter are as follows:

- educational component of management
- educational interventions for back pain
- educating patients
- active mechanical therapy component
- compliance or therapeutic alliance?

- to treat or not to treat?
- communication
- patient satisfaction.

Education component of management

All patients have certain needs for information about their disorder from health professionals (Charles *et al.* 1997). In a group of patients attending their general practitioner in London, an 'explanation of the problem' was the most requested information during the consultation (Williams *et al.* 1995). However, doctors frequently underestimate patients' desire for information about their condition (Ong *et al.* 1995). Failure to provide adequate information is a common cause of dissatisfaction among patients in general (Hall and Dornan 1988; Locker and Dunt 1978). Lack of adequate information is one of the most common causes of dissatisfaction that back pain patients have with medical professionals (Greenfield *et al.* 1975; Deyo and Diehl 1986; Cherkin and MacCornack 1989; Cherkin *et al.* 1991; Fitzpatrick *et al.* 1987). Patients' expectations of a visit to a back pain clinic include the provision of information about back care, prognosis, investigations and discussion of any personal worries (Fitzpatrick *et al.* 1987).

Patients with back pain have preferences for certain management styles, especially those that encourage greater self-care, give them a better understanding of the problem and show more empathy (von Korff *et al.* 1994; Overman *et al.* 1988). Information must be individualised according to the patient's needs and concerns, but certain issues are key. Patients want to have information about these concerns (Williams *et al.* 1995; Hall and Dornan 1988; Cherkin *et al.* 1991; Fitzpatrick *et al.* 1987; May 2001):

- the problem itself
- how they can self-manage
- tests, diagnosis and interventions
- prognosis.

Patients' specific requirements for information will vary between individuals and between presentations. The depth and detail of information given should be appropriate for that patient, but must

cover the essentials that are necessary for the self-management of their problem. It is important to calm patients' anxieties and fears about the pain and encourage the earliest possible restoration of function. Failure to provide the appropriate information is likely to result in patient dissatisfaction and will seriously undermine the patients' ability to manage their problem in the optimum way. It is equally important to ensure that any information that we provide is not the source of increased fear or anxiety in the patient. We must be part of the solution, not part of the problem – for instance, in giving information about diagnosis.

"Abnormal diagnostic behaviour leads some patients into sick role behaviour. Patients become afraid, asking, 'Can you cure degenerative disc disease?' Ill-defined labels help to produce a person who cannot cope, leading to illness behaviour, which in turn might lead physicians and surgeons to perform 'abnormal' treatment" (Nachemson 1999a, p. 475).

Patient education should not be seen as a 'nice extra', but as an effective treatment in itself. In the field of arthritis, for instance, individual studies document the ability of patient education to decrease pain, depression, disability and tender joints (Lorig 1995; Mazzuca *et al.* 1997). Evidence suggests these health care gains can be sustained long-term (Lorig *et al.* 1985, 1993). Current meta-analyses suggest that pain, tender joints, depression, exercise, coping behaviours, disability and health service use can all be improved by educational programmes for rheumatology patients (Lorish and Boutagh 1997; Mullen *et al.* 1987).

Educational interventions for back pain

At first glance, the evidence about the role of education in the management of back pain is rather negative. Some reviews of the formal back school approach have not endorsed the intervention (Cohen *et al.* 1994; Linton and Kamwendo 1987). Some have weakly endorsed it, with caveats about short-term gains only, cost effectiveness, the need to be conducted in an occupational setting and to be linked with a rehabilitation programme (van Tulder *et al.* 1999b; Koes *et al.* 1994; Di Fabio 1995). Individual studies using educational pamphlets have also shown these to have no effect upon symptoms, function, health care visits or work absence, even when

the pamphlet stressed activity resumption and attitudinal advice (Hazard *et al.* 2000; Cherkin *et al.* 1996b).

Certain studies *have* shown the value of information provision and a self-management approach in reducing sick leave, anxiety and fear–avoidance and improving self-care and function (Indahl *et al.* 1995, 1998; von Korff *et al.* 1998; Little *et al.* 2001; Moore *et al.* 2000). Booklets have had the effect of reducing health care-seeking and referrals and have been shown to reduce fear–avoidance behaviour and functional disability (Roland and Dixon 1989; Burton *et al.* 1999).

Several factors seem important in achieving these beneficial effects. One is improved self-efficacy, which is the belief that people have about being able to control aspects of their life (Lorig *et al.* 1993). Another is internal health locus of control, which is the belief that an individual can affect their health without the help of 'external' others (Indahl *et al.* 1998). Another is the reduction of fear–avoidance behaviour, which has been identified as a factor influencing long-term outcome (Burton *et al.* 1999; Klenerman *et al.* 1995).

In other words, often the effectiveness of educational programmes appears to come from their ability to alter patients' attitudes, beliefs and behaviours, rather than the provision of didactic information per se. It is not enough to present dry facts about spinal anatomy or pathology; information about 'crumbling spines' or discs bulging onto nerves may actually undermine peoples' ability to cope. Patients must be able to use the information to affect their problem in a positive way. Information should be provided that reassures patients about particular concerns, disabuses them about pain – meaning harm – and encourages them to start self-care strategies. Some of these studies suggest that alterations in pain beliefs can be translated into improvements in function and pain behaviours. Ultimately, to ensure the success of self-management, clinicians must convince patients to adopt the cognitive and behavioural strategies that are appropriate.

The value of *Treat Your Own Back* (McKenzie 1997) as an educational tool has been demonstrated in a sample with chronic back pain (Udermann *et al.* 2000, 2001). Nine months after reading the book, in those who could be contacted, 87% were still exercising regularly, and 91% were still focusing on using good posture. Sixty percent were pain-free, mean pain severity had dropped from 1.3 to 0.44 on a five-point scale and number of episodes from 4.1 to 1.0 per annum.

Changes were still significant when those lost to follow-up were analysed in a worst-case scenario. Although there was no control group in this study, with a ten-year mean duration of pain in this very chronic sample, the group served as its own control. At eighteen months these improvements were maintained and individuals were still using the information.

This study demonstrates the practical value of a self-management programme involving exercise and posture even in those with very persistent symptoms. These individuals were given the opportunity to have greater control over their back pain problem using strategies that reduced symptoms. They then maintained compliance with those strategies as they were successful in decreasing pain. Major improvements were made in symptoms and episodes of back pain with a relatively minimal intervention.

Educating patients

Assessment and treatment, or mechanical diagnosis and therapy, if provided according to the principles described in this book, are totally educational and informative for the patient and provide a learning platform of immeasurable value. Nevertheless, the following extracts from *The Human Effect in Medicine* (Dixon and Sweeney 2000) highlight the importance of the quality of the information provided, which does not rest on the message only. *"Indeed our potential to alter the patient's perception of his/her disease may indirectly have far more beneficial consequences than any more direct therapeutic attempts to alter the course of disease. There is an increasing concentration in modern general practice on information, but information can do nothing if it does not affect the attitude, motivation and emotions of the patient. All of us witnessed, first hand, the effects of an inspirational teacher but their effects were not created by black and white information but by energy, enthusiasm, and wisdom, which they used to produce their message. It is not the message the clinician gives that matters but that which the patient receives"* (Dixon and Sweeney 2000, p. 61).

Information is a key factor in turning the patient from a passive recipient of care to an active participant in their health, and in shifting the health locus of control from clinician to patient. Information alone is not sufficient to change behaviour; it must be combined with strategies that can be used to harness that information. It must therefore be user-friendly and responsive to individual needs.

Giving advice should not render patients as passive recipients of expert knowledge. Simply telling them what to do can undermine their autonomy and may generate resistance. In attempting to bring about health behaviour changes, it is recommended that a patient-centred approach be adopted (Rollnick *et al.* 1999). To this end, Rollnick *et al.* (1999) suggest the following goals are worthwhile:

- encourage and allow patients to express concerns
- help them to be more active in the interview process
- allow them to articulate what information they require
- develop a therapeutic relationship in which treatment becomes a consultative process in which decision-making is shared.

The educational component of the clinician's input should not be left to the end of the consultation. At many points during the interview and physical examination, the patient may express concerns that need to be addressed and opportunities in which aspects of the patient's posture, work and leisure habits, general activity, beliefs, attitudes, investigations and so on can be discussed in a useful way. It is generally best to speak to these issues as they arise to ensure that communication is kept patient-centred and responsive to their needs. Part of the 'educational diagnoses' that the clinician should consider are the patient's readiness to learn, their willingness to take ownership of the treatment plan and their capacity to retain the details of the advice being offered (Jason 1997).

The communication of a message apparently depends mostly on voice sounds and body language; very little depends on the actual words. The way you say it may be more important than what you say. Between 37% and 54% of what doctors tell patients is forgotten (Dixon and Sweeney 2000). Information should be easily understood, repeated several times, and could be written to improve adherence. Giving the patient too much information, especially on the first visit, will often only confuse. What are the most important messages you wish to get across? Be clear about them. Repeat them several times. Use patient-friendly language and avoid being judgemental – this is likely to antagonise them, rather than make them receptive to your message.

When teaching exercises and appropriate symptomatic responses, show them what to do first, or demonstrate it yourself, and then get them to repeat it. Performance mastery of a task is the most effective

way of influencing self-efficacy and adherence (O'Leary 1985). Action – getting patients to perform an exercise – is a better teaching model than demonstration, information or persuasion. Talk through the pain response, giving the explicit assurance that it is all right if it hurts a bit. Check that the patient knows precisely what to do and not to do before they leave.

The following have been suggested to be key to lasting patient learning (Jason 1997):

- knowledge of what motivates the patient
- the patient assuming 'ownership' of the management plan
- repetitive practice
- reflection, thinking as well as doing
- feedback to the patient on the accuracy of their efforts
- a climate of trust.

However, if we give the patients all the answers, they become totally reliant and are less likely to maintain control of their problem after the intervention has finished. It is important to get the patients to problem-solve their own difficulties and dilemmas as much as possible and to work through how individual problems can be tackled. A way to do this is to give them options; for instance, alternative ways of avoiding postural stresses or overcoming problems concerning maintenance of an exercise regime. Patients who are reluctant to follow a programme can be offered the choice of doing it and changing their present state or not doing it and remaining as they are. The more they can cope without medical intervention, the more their independence is enhanced.

"Our actions may...undermine the patient's self-healing powers by taking things over and actually depriving the patient of the very control that he or she needs so desperately. If there is one common theme in most consultations, it is that patients are hoping for some sort of control over the situation that they find themselves in. Much better that they find this control themselves" *(Dixon and Sweeney 2000, p. 101)*.

In effect, the episode of therapy should be a process during which information and strategies are provided and control is gradually

transferred to the patient, so that at discharge they feel confident that they can manage their problem effectively and independently.

Active mechanical therapy component

Patients wish to have both an understanding of the problem and to know what they might do to help themselves. Once an explanation has been provided of the role that exercise has in restoration of normal function, it is then necessary to show the patient the active mechanical therapy component of their self-management. The specific exercises must be described or demonstrated to the patients, and they must practice these. The expected pain response must be explained, as well as any warnings against lasting aggravation of their condition or peripheralisation.

Necessary progressions and alterations should be given. In certain chronic conditions, a temporary exacerbation of symptoms is possible prior to any improvement. In most circumstances exercises should be repeated about ten times on each session. In general, sessions should be performed every two to three hours; obviously this may need to be tailored around the patient's day, especially if they are working. Avoid the temptation to give the patient more than one hour of exercise at any one time. Posture correction in addition to that exercise will, however, be essential.

Always make sure the patient has practised the most appropriate exercise several times before leaving and that they are clear how many times it should be performed and how regularly. If several different repeated movements have been done, ensure that they are aware which one is to be done as the home programme. Exercises are the key component of mechanical therapy, and this emphasis must be made clear to the patient by concentrating on them. Multiple interventions that also include an exercise component dilute the emphasis that should be placed on the active patient-generated part of therapy.

Following on from the history-taking and physical examination, it should be possible to select a principle of treatment that provides symptom reduction. This may involve a *temporary* avoidance or reduction of certain postures and activities that appear to be aggravating symptoms, as well as the performance of repeated movements or sustained positions that reduce, abolish or centralise symptoms.

Regarding temporary avoidance of certain postures, it is important not to make patients fearful of normal activities and always to ensure full restoration of function. If a directional preference cannot be found and the full effects of mechanical therapy have been explored, the patient should be encouraged to start a functional restoration programme.

Large numbers of patients respond when well informed and provided with the appropriate exercise and postural advice. On occasions, if improvements are not sustained or are too slow in coming, patient-generated forces may need to be supplemented by clinician-generated forces within the same principle of treatment. At all times, though, patient-generated forces must be maintained and the prime importance of this component of active mechanical therapy stressed. The various procedures to be used in the different treatment principles – extension, flexion and lateral – are listed in the chapter on procedures, and their use is discussed in the chapters on derangement and dysfunction.

Compliance or therapeutic alliance?

When self-treatment programmes are discussed, the issue is often raised of patient compliance or non-compliance. Patients are frequently seen as reluctant followers of medical or physiotherapy advice. Various studies have found that average compliance with medication regimes is only shown in 50 – 60% of patients, while compliance with physical therapy programmes was consistently worse at about 40% of patients (Deyo 1982). Factors that have been found to be important in non-compliance with exercise regimes are the barriers that patients perceive or encounter in doing the regime, the lack of positive feedback and the degree of perceived helplessness (Sluijs *et al.* 1993). Other factors that have consistently appeared to influence non-compliance are patients who have an external health locus of control, complex regimes, regimes that are not tailored to the patient's daily routine, unclear explanations and lack of explanation concerning the reason for performing the programme (Sluijs *et al.* 1993). It is better to teach the patient one specific pain-reducing exercise for reduction of a derangement and one only for posture correction to maintain reduction. This keeps it simple and gains the patient's involvement.

As compliance is defined as the extent to which patients adhere to health advice, it implies passive submission to the health professional on the part of the patient. An alternative has been proposed, termed *therapeutic alliance*, which implies negotiation and shared decision-making (Brady 1998). In ongoing disease states it is proposed that active patient involvement rather than passive submission to health instructions is the most appropriate model. *"The passive sick role is incongruent with rheumatology findings that control beliefs, such as self-efficacy and helplessness, are important influences outcomes"* (Brady 1998).

Patients and health care providers are each partially knowledgeable, and appropriate decisions can only be made with the active participation of the patient in the decision-making process. Clinicians must inform the patient about the condition and self-management strategies, but can only evaluate the effectiveness of those strategies with the patient. In turn, the patient with a chronic problem has certain responsibilities, these being:

- to learn about the condition and its management
- to take responsibility for self-management and joint decision-making
- to evaluate the health care experience.

Active self-management is the preferred outcome. This can only be achieved if the patient is sufficiently informed and educated. It is the clinician's responsibility to try to create this state of mind.

To treat or not to treat?

Treatment is usually perceived as doing something to patients – patients may feel cheated if something is not 'done to them', and clinicians may find it difficult to resist the urge to use 'hands on' techniques or make use of some modality. These influences are especially strong in a climate of financial exchange. The evidence at present available makes clear that the value of many of these interventions is largely unproven. However, it is clear that many patients wish to be informed about their condition and what they can do about it. Stronger evidence exists for the value of patient-centred therapies, such as exercise or appropriately constructed educational approaches.

The need for treatment also depends upon the status of the patient. If they are improving, no treatment is necessary; if unchanging, instigate treatment; if worsening, careful monitoring is necessary. Not everyone should be treated, but all need explanation and education.

In physiological terms, regularly repeated movements strengthen and normalise healing tissue and restore to normal function tissue that is abnormal, oversensitised or deconditioned – only patients can do these exercises. In psychological terms, patients' involvement in treatment has the ability to decrease fear–avoidance behaviour and allow patients to have greater control over their problem. Optimum treatment of many musculoskeletal conditions is founded on patient involvement. Thus the first and most powerful management option that should be used is educational. It is not always necessary to do something to the patient – this should in fact only occur if other avenues have been exhausted. Treatment can and should always comprise the provision of advice, information, encouragement and monitoring of patient-managed progress.

Communication

One of the key aspects of health care that affects patient satisfaction is the relationship between the patient and the clinician. Of particular importance are communication, empathy and reassurance (Sitzia and Wood 1997; Wensing *et al.* 1994). It is important that we make the patient as comfortable and as relaxed as possible. Patients enter all clinical situations with their own expectations, feelings and fears (Brown *et al.* 1986). It is important that the clinician recognise these issues, allay fears and answer questions. It is important to avoid the use of medical words or phrases that may be foreign to the patient. Conversation should be conducted using terms and phrases with which the patient will feel comfortable.

From the outset, listening is a key skill to clinicians, without which the seeds of truth that the patient can give will fall on deaf ears – the clinician's understanding of their condition will be limited and their management will not be optimal. It is only with good questioning and listening skills that the clinician grasps an understanding of the patient's problem and their attitude towards it. Communication with a patient is not simply about what we say, it is also the way we say it and the underlying attitude and behaviour of the clinician.

The best advice and management strategies are to no avail if the clinician's attitude or manner generates resistance or antagonism in the patient and leads to a failure in communication. *"If the pursuit of patient concerns and meaning is left behind, communication breakdown will follow, usually in the form of resistance from the patient"* (Rollnick *et al.* 1999, p. 30).

Various studies into doctor–patient interaction have highlighted the impact of communication on outcomes. Elements of the interaction have been seen to affect patients' health behaviour, their evaluation of professional competence and their compliance with medical advice, as well as being a major determinant of patient satisfaction (Heszen-Klemens and Lapinska 1984; Ben-Sira 1982; Stewart 1984; Murphy-Cullen and Larsen 1984).

Active listening is an integral part of the communication process – that is, to understand the underlying meaning behind the words used. Hearing is a passive act, whereas listening is an active process. The following techniques are recommended to maintain a patient-centred approach and effective communication (Rollnick *et al.* 1999; Haswell and Gilmour 1997; Jason 1997):

- simple open-ended questions
- active listening
- being non-judgemental
- adjusting vocabulary to individual patients
- providing 'normative permission' (suggesting that the patient's situation or attitudes are common, making them comfortable discussing their problems)
- encouragement with verbal and non-verbal prompts (non-verbal cues can convey 70 – 90% of your message)
- clarifying and summarising in order to bridge the gap between the patient's meaning and the clinician's interpretation:
- paraphrasing to encapsulate what the patient has said
- asking further probing questions
- summarising to give an overview of the patient's comments.
- reflective feeling – reflecting and exploring emotional aspects of the patient's problem.

Patient satisfaction

Issues of education, self-care strategies and interaction with clinicians should be seen against the background of patient satisfaction, which embraces these, as well as other dimensions. Patient satisfaction in general is a multidimensional concept that includes factors relating to three key areas (Gray 1997; Wensing *et al.* 1994):

- interaction with health professional
- clinical outcome/professional performance
- organisation and environment of care.

The dimensions that patients consider important in a satisfactory episode of physiotherapy care have been explored in a qualitative study in the UK (May 2001). Thirty-four past back pain patients were interviewed, and following are the main themes revealed as fundamental to an acceptable experience of therapy.

Table 18.1 Dimensions that patients consider important in an episode of physiotherapy

Dimension	*Key examples*
Personal manner of clinician	Empathetic Listening Respectful Friendly
Professional manner of clinician	Inspired confidence Skilled Thorough
Provision of information	The problem Self-management Process of treatment Prognosis
Treatment as a consultative process	Patient involved in evaluation of treatment Responsive to patient's questions Responsive to patient's self-help needs
Organisation	Short waiting list Open access Enough time with clinician
Outcome of an episode of care	Treatment effectiveness Gaining self-management strategies Ordered discharge

Source: May 2001

Patients have a wide range of different issues that contribute to overall satisfaction, of which outcome is only one. Patients frequently make clear qualitative judgements about what they consider to be good standards of physiotherapy care and notice both the presence and absence of these characteristics. They appreciate a personal manner that is friendly and empathetic and a professional manner that inspires confidence. Information is very important, including information about self-care. They want their questions answered, their individual problems addressed, and to be consulted about the efficacy of treatment. Although pain relief is a key outcome, gaining a good understanding of their problem and learning strategies of self-management is a highly satisfactory outcome for many patients.

Conclusions

In this chapter some of the key aspects of patient management have been considered, and it has been shown that this concerns more than the physical intervention itself. Although this is a vital part of the overall management, and is considered in depth in other parts of the book, this chapter has stressed different aspects of the patient–clinician interaction. As well as advice on active mechanical therapy, clinicians must also put energy into educating patients about activity, posture, pain, general exercise and whatever issues arise, and addressing their individual questions, needs and problems. Education cannot be considered a nice extra, but is an intrinsic part of the management of musculoskeletal disorders. None of this is possible without a good working relationship with the patient. This is established by good communication, with the clinician actively listening to the patient's concerns and problems and responding to them. Patients are far more likely to follow advice if they feel that their agenda has been recognised. Issues of compliance will not arise if the patient feels part of a therapeutic alliance addressing his or her problem. When taking into account the patient's satisfaction, it is insufficient to focus only on the outcome; the process of health care by which that outcome is reached must also be considered. The process must be patient-centred and holistic.

19: Follow-up Evaluations

Introduction

The review process is an essential part of patient management. We must be able to evaluate the management strategies that have been recommended to see if they are having the desired effect or if the patient is unchanged. According to this response, the strategy will be continued or amended. In Chapter 16 the aspects of symptomatic and mechanical responses that help us to evaluate management are presented in detail. It may be helpful to review that chapter before you read this one. In this chapter the specific way that the review should be conducted is presented.

To be able to conduct the review properly, it is essential to have gathered sufficient detail at the initial assessment. *If baseline details are inadequate, it will at times make it impossible to conduct a thorough review*. It is also too late to remedy this deficiency in retrospect.

Sections in this chapter are as follows:

- reaching a conclusion
- review process
- implications.

Reaching a conclusion

The confidence with which a patient can be given a syndrome classification on the first day and the management strategy applied varies. Sometimes it is very definite that symptoms are centralising and the mechanical presentation improves in one session, while at other times the response is less clear. The conclusion made on day one can sometimes be provisional; confirmation of the classification and the appropriateness of the chosen management strategy are made at follow-up. If the response is still equivocal, further testing may be necessary. Sometimes a period of three or four days with several sessions may be necessary to confirm a directional preference or lack of it. Diagnostic classification should be completed within five sessions, but is usually achieved more quickly than this.

Where uncertainty exists concerning the correct management approach, the patient should be reviewed every day until the appropriate treatment principle is confirmed. If the patient is unable to attend, then the review should be conducted by telephone. Once symptoms are resolving and the patient is successfully managing alone, review sessions should be further apart so that the patient can demonstrate independence and gain confidence.

Where uncertainty exists, several strategies may be used to reach a more definite conclusion. These include force progressions (overpressure or mobilisation), force alternatives (for instance, lateral forces or sustained procedures), ensuring end-range and testing effects of repeated movements over several days. For application of overpressure when uncertainty exists, see section *Testing inconclusive* in Chapter 15.

If symptoms have been present for some time, it is more likely that the response may be equivocal. It is often helpful at this point to get the patient to test a specific procedure for one or two days and gauge the response following this. Other ways of facilitating the diagnostic process when it is unclear are listed in Table 19.1 (from Van Wijmen 1994).

Table 19.1 Different methods of clarifying symptom response

- test provocative or reductive procedures over two to three days
- apply mechanical forces more vigorously (introduce clinician techniques)
- sustain postures
- lateral forces
- increase the number of repetitions
- increase the frequency of repetitions
- ensure that movements are to end-range
- use alternate starting positions
- stress the joints in one direction and check the effects on pain and movement range in the opposite direction.

Review process

On the second day and at each subsequent visit, a structured, logical and informative review process must be conducted. This is to determine what the patient has been doing regarding previous instructions, the immediate effect of any procedures being done and

if there have been any overall changes. We need to know from the patient as a result of following instructions if there has been any change.

- The first question asked should be, *'With the exercises and postural correction over the last day(s), overall are you better, worse or the same?'*

If the patient is better, there is no need to change management in any way, and they should continue with more of the same as long as improvement continues. The patient should be questioned and examined thoroughly to ensure that they are actually 'better' than the previous occasion.

- The second question should be, *'Are you definitely improved, or only perhaps improved?'* Patients sometimes like to please the clinician, and this question exposes uncertainty.
- The third question for further clarification should be, *'In what way are you improved?'* This is important, for it can identify which component of the treatment protocol has been most effective. For example, the answer might be, *'I had no pain lying in bed last night'*, indicating that correction of sleeping position has effectively eliminated one of the factors in the overall equation. The answer might be, *'I am pain-free for two or three hours after exercising'*, indicating the need to apply exercises more frequently.

Other questions regarding pain and function clarify the situation. If their response is definitely improved and supported by symptom location change or symptom abolition and mechanical improvement, the classification is confirmed and the appropriate management strategy has been selected.

If at a later point in the episode the patient stops reporting improvement, but reports that symptoms are unchanging, then a change in management strategy may be necessary. This could involve force progressions or force alternatives.

If they are worse or unchanged, they must be questioned more closely about what they have been doing:

- *Have you been doing the exercises we discussed?*
- *How frequently?*

- *What exercise have you been doing?* Get them to show you; however clear you think you may have been, unfortunately patients frequently 'adapt' the exercise.
- *Are there any problems that limit your ability to do the exercises?*
- *What happens to the pain when you do the exercise?*
- *What happens to the pain when you use posture correction?*
- *Have you been sitting the way we talked about last time?*
- *Have you understood the reasons for the exercises and posture correction?*
- Check symptomatic presentation fully
- If there is a change, is it definite or doubtful?
- Check symptoms:
    - intensity
    - site for centralisation
    - frequency (constant or intermittent; if intermittent, what proportion of the day)
    - severity
- Check if there has been any change in functional problems
- Check mechanical presentation:
    - range of movement
    - pain on movement
    - deformity.

It will then be known how regularly they have been doing their exercises and if they are doing them correctly. Their technique may need correcting, but wait until you have heard how it affects their symptoms. They may need encouragement to be exercising more regularly, or, less commonly, they might have been doing too much. If patients are having problems with the demands of a regular exercise routine, the importance of doing it regularly needs to be emphasised.

Encouraging patients to 'problem-solve' difficulties with the regime or the exercise itself promotes self-management. Some patients are very reluctant to do things that hurt and are still very anxious about pain responses. They need extra reassurance 'that hurt does not equal

harm', that reduced activity is only briefly beneficial at the onset of pain, and that *the only way* to try to re-establish normal function is graded exposure to normal activity.

Table 19.2 Main elements of review process

1. symptomatic presentation
   - site of pain
   - frequency
   - intensity
2. mechanical presentation
   - range of movement
   - deformity
   - quality of movement
   - function
3. mechanical therapy
   - have they been exercising?
   - what exercises?
   - posture correction?
   - what is the response when they do them?

Implications

From the review, you will also know their symptomatic and mechanical response to performing exercises over a day or two – they will either be better, worse or unchanged. As outlined in Chapter 16, this gives a 'green light' for more of the same, a 'red light' or an 'amber light'. It is important to ensure that they are actually in these states. Keen questioning and close analysis of symptomatic and mechanical responses is sometimes necessary to elucidate the true picture.

If better, nothing needs to be changed, only encouragement given and the management strategy maintained unaltered. However, at some point the direction or level of force of mechanical therapy may need to be altered, especially if symptoms stop improving. Do not stick rigidly with one loading strategy because of an initial improvement. If worse, exercises and symptom responses need to be checked, but ensure the patient is actually worse, rather than simply that the exercises 'hurt'. When starting any unfamiliar exercise programme, new pains may be generated. This is not unusual, but may confuse the patient. If they are truly worse, the treatment principle or start position may need to be changed; a derangement may be irreducible,

or consideration may need to be given to non-mechanical syndromes. If unchanged, is the patient exercising regularly enough and doing the right exercise? If they have been, force progression may be necessary, or, if this has been attempted already, an alternative treatment principle should be considered. In the case of dysfunction no change would be expected, and they should be encouraged to continue.

In essence, if the patient is better, then the provisional diagnosis has been confirmed. The original management strategy was correct, and this should be continued unchanged. If the patient is genuinely worse, a misclassification has occurred – either the patient has been categorised in the wrong syndrome or they have been given the wrong treatment principle. Sometimes the treatment principle is right, but the start position is wrong. In chronic symptoms there can be a temporary exacerbation of symptoms when activation is started.

If the patient returns and after a thorough assessment of symptomatic and mechanical presentations they are genuinely unchanged, then further analysis is necessary. This takes the form of force progressions until a change occurs. If symptoms start to reduce, abolish or centralise, the directional preference is confirmed.

If symptoms worsen or peripheralise, alternative direction of end-range loading, both static and repetitive, are indicated. Complete re-evaluation will usually be required in these cases and cessation of all mechanical intervention is often necessary. Some patients, failing to respond to mechanical therapy, demonstrate remarkable improvement when exercise is stopped. It is worthwhile, in cases where improvement is at a standstill, to consider complete rest from exercise for two to three days.

Conclusions

In this chapter the way the review process is carried out has been presented. This is conducted at each session to determine if the appropriate management strategy is being implemented. Depending on the certainty of response, this is done with more or less of the detail presented here, but essentially the review involves enquiring about the patient's symptomatic and mechanical response and about the mechanical therapy component of management.

Check at follow-up:

1. Symptoms better, worse or no change?
2. Mechanical better, worse or no change? Why? Understanding compliance performance.

Symptoms	*Better*	*Worse*	*No Change*
	Intensity	Intensity ▲	Intensity
	Frequency	Frequency ▲	Frequency
	Intermittency ▼	Intermittency ▲	Intermittency
	Central	Peripheral	Central/Peripheral
Mechanical	*Better*	*Worse*	*No Change*
	ROM ▲	ROM ▼	Status Quo

20: Clinical Reasoning

Introduction

The management of patients involves more than examination processes and treatment techniques, although this is a large part of it. There needs to be a thought process that matches findings to patterns of clinical presentations that excludes red flags, considers which examination procedures are necessary and which are not, determines what to do if responses are atypical or unclear, addresses patients' concerns and so on. This reasoning permits an examination process that goes beyond routine and habit and enables clinicians to solve more complex problems – this is termed 'clinical reasoning'.

On a practical level, clinical reasoning is the process of deciding what problem the patient has, and, from your knowledge base, deciding what can be done about it. First this involves the discovery of the 'character' of that problem. The patient is the best witness to this, and interview skills must be capable of determining a clear 'big picture'. Second, a rounded knowledge base is needed to provide practitioners with an understanding of diverse factors, such as the variety of clinical presentations, the natural history of a condition, pathophysiological changes, management strategies, the evidence base, the effect of an intervention, etc. The third and vital element is the ability to reason between the practical reality of the patient's problem and the available knowledge base. This involves constant interplay between theoretical concerns and clinical issues and a logical analysis of the effect of intervention strategies on the problem.

Clinical reasoning is thus an essential element in the translation of clinical theory into clinical practice, and thus an exploration of its key elements is vital to make full use of any approach to a patient's problem. This chapter discusses some of the aspects involved in clinical reasoning and then presents a clinical example in which a reasoning process is given. The sections in this chapter are as follows:

- clinical reasoning
- elements that inform the clinical reasoning process
- data gathering

- knowledge base
- clinical experience
- errors in clinical reasoning
- example of clinical reasoning process.

Clinical reasoning

Clinical reasoning is the cognitive and decision-making process involved in clinical practice used in the diagnosis and management of patients' problems (Terry and Higgs 1993; Jones *et al.* 1994). Two methods for clinical reasoning have been proposed, based either on pattern recognition or on a process of hypothetico-deductive reasoning (Jones 1992; Terry and Higgs 1993).

Hypothetico-deductive reasoning describes a process of hypothesis generation based on information gathered from the patient. The hypothesis is then tested out or further ones generated – "try it and see" – until a management pathway is clearly defined. Because hypotheses must be confirmed by responses to treatment, the process involves continual reassessment. In effect, every treatment is a form of hypothesis testing and has been described as 'best guess management'.

The alternative model is based on pattern recognition gained from certain features in a clinical presentation, which remind the clinician of previously seen clinical problems. In this model management strategies are derived from previous experience rather than an experimental 'try it and see' method.

Pattern recognition is only possible with a well organised knowledge base and plentiful clinical experience; thus it is only available to experienced clinicians. In the face of atypical problems, when pattern recognition is not possible, the expert reverts to hypothesis testing. The novice clinician tends to always have to use hypothesis testing (Jones 1992). In the first edition of this book, McKenzie provided for the novice clinician, the benefit of thirty years of cumulative pattern recognition.

The description of the three syndromes – derangement, dysfunction and posture – appearing in this edition are as originally described in the first. These form the entire base upon which the experience of pattern recognition can be immediately acquired by the novice clinician.

Elements that inform the clinical reasoning process

Certain factors inform the clinical reasoning process – namely data gathering skills, aspects of the knowledge base available to the clinician, clinical experience and meta-cognition skills (Terry and Higgs 1993; Jones 1992; Jones *et al.* 1994). It is thus a complex and cyclical process, as suggested by the model of clinical reasoning for physiotherapy proposed by Jones (1992). At every stage in this process errors may occur that could affect the reliability or validity of the reasoning process (Jones 1992).

Some of the aspects of clinical reasoning are briefly discussed in the following sections. Many of these issues are considered in more depth in the appropriate chapters, but this is an attempt to pull the disparate elements together. The main aspects to be considered are as follows (Jones 1992; Jones *et al.* 1994; Terry and Higgs 1993):

- data gathering
- knowledge base
- clinical experience
- meta-cognition.

Data gathering

Data gathering is the process of discovery about the patient's problem undertaken during the history-taking and the physical examination. The patient has available the essence of the problem; the skill is in accessing it. However, patients do not know the pieces of information that clinicians need to know, and the unwary may be flooded with a large amount of irrelevant information by some patients. Data gathering requires empathy and active listening by the clinician. Unless the situation is relaxed, friendly, respectful and non-judgemental, the patient is unlikely to tell his or her true story.

During the history-taking, considerations and hypotheses are raised and then rejected or retained for further probing. Does the patient have any features suggestive of serious spinal or nerve root pathology? Does the problem sound mechanical? Is there suggestion of a directional preference, and what features are there to give an idea about prognosis? Has enough information been gathered on baseline symptomatic and functional levels against which to judge later changes? These and other questions should be considered and

reflected on during the patient interview. From the history-taking an overall picture of the patient's condition should have been gained, and the direction that the examination is to proceed in should be suggested.

The physical examination is not a routine series of tests performed uniformly on every patient, but follows on directly from the data gathering and hypothesis testing of the history-taking. Often findings from the physical examination merely confirm what is strongly indicated by the history. Again, it is important that sufficient baseline mechanical data is collected against which to make later comparisons. Data collection continues until a decision can be made about management strategies. Once a directional preference is confirmed or possibly indicated, further testing is unnecessary. The decision may be tentative, in which case further data is gained at the next session and from the patient's response to the proposed management strategy.

Data gathering does not stop at the end of the first session, but continues on all subsequent occasions to ensure that optimal management is being maintained. If it is thought that force progressions or alternatives, further procedures or investigations become necessary, then these are instigated.

Knowledge base

Clinical practice requires a wide-ranging breadth of knowledge from different fields. For knowledge to be of value in the elucidation of the patient's problem, it must have clinical relevance. What is learned in training may have only a slight bearing on what is done in the clinic. Theoretical knowledge must have practical significance for it to be of worth.

Jones *et al.* (1994) list the following topics as relevant to the knowledge base of physiotherapy – anatomy, physiology, pathophysiology, procedures, patterns of clinical presentation and concepts. It is also stated that developing a hypothesis happens in six key areas: the mechanism of symptoms, differential diagnosis, predisposing or contributing factors, precautions or contraindications, management and prognosis (Jones and Butler 1991; Jones 1992; Jones *et al.* 1994). Information from other areas is needed to complete this list. Clinical practice also needs to be informed by knowledge about epidemiology, the research evidence, psychological issues and communication with patients.

Any of these factors, and more, may provide useful clinical information on different occasions. However, their value rests on their clinical application rather than their theoretical strength.

Clinical presentations

There are numerous clinical presentations that are relevant to back problems: a wide variety of ways that derangements may present, and the more consistent and less common patterns of dysfunction and postural syndrome. Beyond the mechanical syndromes, such entities as serious spinal pathology, nerve root pathology, hip joint problems, spinal stenosis and others must also be considered. Awareness of common patterns of clinical presentation will assist in differential diagnosis and selection of management strategies. Also, awareness of atypical presentations should alert clinicians to the likelihood of red or yellow flags.

Concepts

Using the approach of mechanical diagnosis and therapy requires an understanding of the mechanical syndrome classification. The postural, dysfunction and derangement syndromes describe clinical presentations and responses to specific mechanical loading strategies. Their clinical value, gained once the clinician is able to recognise them (using the tools of data gathering and cognition), is their use in proposing management guidelines. Confirmation of a proposed syndrome hypothesis is gained by expected responses to the applied loading strategy. Derangements are encountered most frequently in the lumbar spine. For the proper use of mechanical diagnosis and therapy, an understanding of the concepts and application of centralisation, force progressions and directional preference is also needed. Establishment of directional preference is the key to be pursued in management.

A logical analysis of the symptomatic and mechanical response to different end-range loading strategies will help to classify the problem and lead to the formulation of a management plan. A clear directional preference is not always established at the first session. If this is the case, the patient can continue the mechanical testing over the next three or four days, and/or force progressions can be used at the second session to produce a clearer reaction. If the patient is unchanging, and particularly if worsening, they should be seen daily until the management strategy is confirmed.

Communication with the patient

Gaining the patient's confidence on day one is essential. Even if that is all that is achieved, this is an important beginning. Self-management requires support, encouragement and explanation. Assessment and re-evaluation requires monitoring of symptomatic and mechanical presentations. All this can only be done if good communication is maintained with the patient.

Psychosocial factors

Certain psychosocial factors have been identified as risk factors for the development of chronic musculoskeletal problems – these are termed 'yellow flags'. Key factors, such as fear–avoidance behaviour, anxiety, depression, low self-efficacy, external health locus of control and passive coping need to be addressed by an approach that encourages activity, provides reassurance and explanation and offers some control in managing their problem. The attitudes and beliefs of the patient can be more important than any biomechanical pathology. Sometimes these factors are so dominant that a multi-disciplinary approach may be necessary. However, although patients may have inappropriate attitudes and beliefs, they may also have a straightforward mechanical back problem. If their anxieties and concerns can be placated, a normal episode of mechanical therapy can ensue.

Fortunately these problems are present in only a small percentage of patients. Identification and assessment of these 'yellow flags' is beyond the scope of this text. This issue is considered at length elsewhere (see Watson 2000; Watson and Kendall 2000; Kendall and Watson 2000).

Mechanism of symptoms

Pain may be predominantly mechanical in origin or predominantly chemical. Symptoms may be modulated strongly by pain behaviours dictated by the higher centres. Neurophysiological changes in the central nervous system may be the cause of persistent symptoms when peripheral tissue damage is resolved. Patients with these different problems describe different presentations and respond differently to mechanical loading.

Pathophysiology

Some pathophysiological concepts are essential to an understanding of musculoskeletal conditions. The normal healing process, the necessity of remodelling to regain full function, the problematical nature of tendon healing and the recognition of non-mechanical

factors in chronic pain are examples of such concepts that have wide relevance. Knowledge of typical areas of pain that emanate from pathology at certain sites gives a focus for the physical examination. An understanding of the difference between somatic and radicular pain and the pattern and type of disc herniation are specific pathologies that have relevance to spinal problems.

Contributing factors

Various factors may have a role in the causation or prolongation of the patient's problems. These may be static or dynamic postural forces that can be interrupted, such as occupational, recreational or domestic stresses. A key contributing factor in many back pain problems is the effect of sustained postures, especially sitting. Pre-existing overuse or degenerative changes may also be contributory factors that are less amenable to alteration. Psychosocial 'yellow flags' have been identified as risk factors for chronic pain.

Procedures

Both patient and clinician procedures are given in this text. . Management strategies will always use a patient-centred exercise programme, which may need to be supplemented with clinician procedures. These are classified by the treatment principle to which they belong – extension, flexion or lateral. Sometimes a combination of sagittal plane and lateral forces is required. Once a directional preference for certain movement is established, then that becomes the treatment principle. Procedures from the same treatment principle can be used should the need for force progressions or force alternatives arise.

Clinical experience

Clinical reasoning requires clinical experience. It is only having seen hundreds of patient presentations that patterns will be recognised and skills of data gathering will be mastered and focused into generation and confirmation of a hypothesis.

However, clinical experience can also lead to rigid thinking and failure to countenance unfamiliar presentations. Pattern recognition is not about squeezing square pegs into round holes, but continually re-evaluating data to confirm or deny a proposed hypothesis. Clinical experience of itself does not necessarily lead to improved clinical reasoning.

Errors in clinical reasoning

Errors in the thinking process may occur at any stage during data gathering, analysis, hypothesis generation and testing. These may be errors of perception, inquiry, interpretation, synthesis, planning or reflection (Jones 1992).

As a means of determining management strategies, clinical reasoning based on pattern recognition can have drawbacks. Failure to fully explore all options and bias towards one's favourite diagnosis can encourage premature foreclosure on alternative hypotheses. Pattern recognition, on its own, may be insufficient if it ignores certain complicating factors, such as exaggerated fear–avoidance.

Typical errors of clinical reasoning are making assumptions without further checking; prematurely limiting hypotheses under consideration; failure to gather enough information; attending to those features that accord with a favoured hypothesis while ignoring contradictory information; and gathering redundant information. Failure to listen carefully to a patient may lead to ignoring a key piece of information and creating a false trail of hypothesis generation. Focusing on the traumatic onset of a condition may lead to the assumption that the stages of inflammation, repair and remodelling must be gone through, ignoring the fact that a derangement may be the cause of symptoms. Doing every available test is a common way of gathering redundant information that the clinician is unable to use to fashion a treatment direction. It is important to question openly and listen without making assumptions. The use of the form focuses the data gathering on certain key areas, which should be sufficient in most cases, and thus avoids the gathering of redundant information that will not help in decision-making.

Data gathering skills vary with different presentations. Sometimes close questioning concerning symptomatic responses to loading strategies is necessary to determine the correct management strategy. At other times, in chronic patients, a close focus on pain is less relevant, and attention should be on function. Failure to find a favoured presentation should not lead to trying to squeeze patients into diagnostic boxes that they do not fit. Do not make clinical decisions without sufficient information. If pattern recognition is not immediately available, revert to hypothesis generation tactics. Use repeated movements and progressive loading to determine the appropriate

management strategy. Equally, do not change a management strategy without having first fully explored the patient's response.

Example of clinical reasoning process

In the following illustration some examples of clinical reasoning are given in italics.

A 45-year-old man is referred by his GP. He is a computer technician with a job that involves some driving and sitting, but is also reasonably varied and active. He scores twelve out of twenty-four items on the Roland and Morris disability questionnaire (Roland and Morris 1983) and indicates his pain at six on a 0 – 10 visual analogue scale. He is not off work with the present episode. He has stopped his usual sporting activities – running and climbing – because of back pain, but he is keen to resume them. On the last occasion when he tried to run, his leg pain was severely exacerbated for several days.

His age puts him in the range when mechanical low back pain is common. His job involves a lot of flexion loading (contributing factors). Being still at work and wishing to resume abandoned sporting activities suggests motivation will not be a problem. His responses to the functional disability questionnaire and visual analogue scale are consistent with each other and do not appear to be exaggerated (psychological factors). Returning him to his sporting activity will be part of the goal of treatment, as general exercise has a protective effect.

His symptoms have been present for about three months. They came on for no apparent reason and are now unchanging. They consist of aching that radiates from his back and left buttock all the way down the back of his thigh and leg to his ankle. Sometimes he has noted pins and needles in the outer edge of his foot.

Initial triage classification suggests that his problem has nerve root involvement (pathophysiology). Initial mechanical classification would be a reducible derangement, but other possibilities are adherent nerve root or an irreducible derangement (concepts). This will become clearer once his response to different loading is discovered. Treatment pathways will be selected for pain below the knee.

Symptoms commenced in his back and spread into his leg after several weeks. The intensity of the pain is the same in the back and leg. In the back, symptoms are constant, but in the limb they are intermittent. He estimates that he feels the ache in the thigh about 80% of each day and in the leg about 50% of each day. The pins and needles in his foot are less frequent, but do occur every day, when the pain is at its worst.

The intermittency of leg symptoms and the ongoing presence of his back pain suggest a good prognosis and excludes the likelihood of an irreducible derangement. The fact that the paraesthesia is not constant also suggests the condition may be easily reversible (clinical experience). A left postero-lateral derangement seems likely, but adherent nerve root cannot be ruled out yet (clinical presentations). In terms of mechanism of symptoms, his constant back pain may be caused by constant mechanical deformation; it is unlikely at this stage to be inflammatory. The intermittent symptoms are probably the product of increasing and decreasing mechanical deformation that affects the nerve root (mechanism of symptoms).

He has provided a clear picture of his symptomatic baseline presentation against which to judge change. This includes a description of the extent and frequency of referred and local pain (data gathering).

He reports that his symptoms are made worse and in time peripheralise by bending, sitting, walking, driving and as the day progresses. He prefers being on the move, and his symptoms are also better when he lies down and in the morning. His sleep is not disturbed.

As commonly occurs with nerve root problems, positions of loaded flexion make his symptoms worse. However, he is also made worse by walking, a position of loaded extension (contributing factors). The only mechanism for relief from his symptoms at this point appears to be unloading the spine. Although he is describing peripheralisation of symptoms with both flexion and extension loading, his response to unloaded extension may be more favourable. If this is not the case, a lateral component may need to be considered early on.

He relates that he has had several previous episodes of back pain over the last ten years, but no leg pain before. Previous episodes have lasted a few weeks and then spontaneously resolved, with more

recent episodes tending to be longer in duration. He has not sought treatment before.

He gives a typical history of episodic derangement, with this latest episode the worse, both in terms of referral and duration of symptoms and status. From an epidemiological perspective, his story is common with several previous episodes and a gradual deterioration. The role of self-management is important both now and for the future (epidemiology).

He reports no disturbance of bladder function and no altered gait, but sometimes experiences increased buttock pain on coughing and sneezing. He has not had x-rays, has had no surgery, nor has he been involved in any accidents, and his weight is stable. He reports his general health is excellent with no ongoing medical conditions.

His negative response to these 'red flag' questions excludes the consideration of serious spinal pathology. This confirms earlier suggestions of an essentially mechanical back problem, although with probable nerve root involvement (diagnosis and red flags).

He sometimes takes analgesics, up to about four a day. These dull the pain temporarily, but as they are rather ineffective, he only uses them a few days a week. When he first saw the GP he took a course of anti-inflammatory tablets, but their effect was also negligible.

Medication, including anti-inflammatory medication, has little value, which is not uncommon when a problem is predominantly mechanical (mechanism of symptoms). At this point certain provisional conclusions can be drawn about classification, prognosis and directional preference (concepts and cognition). He displays every indication of having a reducible derangement with minor nerve root involvement that is responsive to mechanical loading. There are no indications of the presence of 'red flags' relating to serious spinal pathology or 'yellow flags' relating to inappropriate pain behaviours (red flags and psychosocial factors).

The intermittency and variability of symptoms suggests a good prognosis. At this stage there are no clear indications of directional preference – it is obvious that flexion worsens his condition and prolonged exploration of this will not be required. However, extension – at least when loaded – also appears to make him worse.

His response to unloaded extension exercises needs to be explored, but this aspect of his history could indicate a relevant lateral component. This will become clearer with exploration of repeated movements (clinical experience and cognition).

To ensure that the clinician has an accurate understanding of the problem, the clinical presentation is briefly
'You have had back and leg pain for three months, which is now unchanging. Back pain is constant, thigh and leg pain are there at least half of the day, and you also occasionally have pins and needles on the outer edge of your foot. Symptoms are aggravated by sitting, driving and walking, and the only position of ease is lying down.' The patient confirms this brief outline of their problem (data gathering).

He sits slouched on the treatment couch. When he is asked what has happened to his symptoms during the interview, he reports that the pain has spread into his thigh while they have been talking. On attempting posture correction, the thigh pain is increased. He stands with a flattened lumbar spine and without a lateral shift.

His response to posture correction confirms what is already known about his response to loaded extension. Although at this stage it is not known if he has a relevant lateral component, he does not have a lateral shift. It is not necessary to immediately embark on the lateral principle; it will be appropriate to test out his response to unloaded extension first (clinical experience).

His pain status in standing is back and thigh pain, with no symptoms in his leg. He displays a moderate loss of flexion, reaching to his upper shin, which increases his thigh pain. Normally he can reach his feet on forward flexion. He also displays a major loss of extension, which produces calf pain after one movement that abates after a few minutes – this movement is not tested further. Side gliding is asymmetrical with nil loss of right side gliding, but a major loss of left side gliding.

In line with the symptomatic presentation collected during the history-taking, he also displays a clear mechanical presentation of restricted movements. All these provide a useful base line against which to judge the effects
Although he displays losses in the sagittal plane, he also shows an asymmetrical loss of lateral movements, which further suggests consideration of a relevant lateral component.

A relevant neurological examination is conducted. Resistance testing of his calf muscles, extensor hallucis longus and dorsiflexors are the same on both sides, and there is no apparent loss of sensation around the lateral edge of his foot, big toe or medial part of his leg.

The neurological examination reveals no definite impairment of nerve conductivity. Although variable nerve root irritation may be present, this is not persistent enough to cause an outright neurological deficit. This further confirms a good initial prognosis (pathophysiology).

His pain status in lying is back and thigh pain again. Extension in lying produces calf pain after several repetitions, and so again he is stopped from performing further movements.

It is clear from his symptomatic response to the pure sagittal plane that further testing of these movements is unnecessary, which confirms the information collected during the history. It was already known that his symptoms worsened during loaded flexion, and so it was unnecessary to test repeated flexion. He also reported loaded extension activities to worsen his pain, which indeed it was found to do and so was not pursued. Repeated movements have shown that unloaded extension also causes peripheralisation, so no further testing is done in the pure sagittal plane. There have been several clues already that there may be a relevant lateral component, and a fuller exploration of this should now be conducted (procedures).

The patient's hips are shifted to the right as he lies prone on the plinth. The clinician stabilises his hips in the off centre position while the patient performs extension in lying. During repeated movements of this kind he reports a lessening of symptoms in the thigh. After two sets of ten repeated movements, he reports that the pain is no longer to his knee, but is now just below his buttock. When he stands after performing two more sets of repetitions, he reports only left-sided and central back pain.

This patient has demonstrated a clinically induced symptom change. His symptoms have centralised in response to extension/lateral movements. Pure extension forces caused him to worsen. This man had a relevant lateral component that required a combination of extension and lateral forces to effect change. The ease with which symptoms were centralising suggests a favourable and rapid

prognosis. There is no need for further testing or hypothesis-generation at this point as enough information has been gained during the history and physical examination upon which to base an initial management strategy.

Session two

He is not able to return for two days. When he returns he is asked, 'As a result of what you have been asked to do, are you better, worse, or the same?' *He reports he is better, and is questioned about the five possible dimensions of improvement:*

- has pain location changed?
- has pain frequency changed?
- has pain intensity changed?
- is there more movement for less pain?
- has function improved?

He reports that he has had neither calf pain nor pins and needles since the initial consultation. The thigh pain is mostly now in the top of his thigh and is present much less frequently. The back pain is still constant and is slightly more noticeable. Movement is easier and certain activities that were painful cause less or no pain now. He reports that he has performed the extension in lying with hips off centre movement regularly, at least every two hours. Every time he performs the procedure any symptoms present in his thigh are abolished, and symptoms in his buttock are reduced. Overall he rates himself at least 50% better already. He is very satisfied with progress and continuing to improve.

Re-assessment of his symptomatic presentation demonstrates he is responding to treatment. Pain is referred less far down his leg, it is there less frequently and is less severe. There is no problem with his motivation to be involved in treatment. He is performing the active mechanical therapy component of management both regularly and effectively. He reports this to have been very successful in easing his symptoms at the time and overall feels a lot better (data gathering).

On checking his mechanical presentation, extension displays a minimum loss and there is now only a minor loss of left side gliding. His technique is checked and he is performing the procedure correctly.

The mechanical loss of movement has also improved. Thus all features of the symptomatic and mechanical presentations have improved compared to baseline measures taken on day one. The day one provisional diagnosis of derangement is confirmed. This at present is responding to extension/lateral forces. No further assessment is needed at this time. No force progressions need to be considered as he is making rapid improvements with patient-generated forces only.

He is not able to attend for five days, but is encouraged to continue with the present management as long as it produces the same response. He is warned that if improvements do not continue, he may need to do straightforward extension in lying exercises.

Session three

He is pleased upon his return, but also feels that no further improvement has occurred in the last two days. In that time he has only experienced an ache in the back, which is present about 50% of the day. There have been no symptoms in his thigh or lower leg in the last forty-eight hours. The exercise has little effect on the remaining back pain. He has not felt any need to take tablets at all since starting treatment.

Good improvements have been made and distal symptoms have been abolished. However, change has stabilised and the present management is not abolishing his back pain. This requires further exploration (cognition).

On further questioning, he reports that back pain returns mostly when he is sitting or driving. He is generally free of symptoms when walking about. He reports some back pain as he sits in the clinic. This is abolished with posture correction. His range of flexion has now returned to normal, and his side gliding movements are equal right and left. He has a minor limitation of extension that produces his back pain. Repeated extension in standing begins to increase the back pain, which goes when he stops the movement. Extension in lying also produces back pain, but this is reduced and then abolished on repetition. Afterwards extension in standing is pain-free and full.

From the history and symptom response, it is clear that a change in management strategy is required. He is now responding to pure sagittal plane movements. Over the next few days he is instructed to maintain

posture correction with the use of a lumbar roll when driving or sitting for long periods and to regularly perform extension in lying (procedures).

Session four

He has had virtually no symptoms at all in the last few days. Occasionally, if he sits poorly symptoms return, but he is rapidly able to abolish these with a change in position. Extension in lying has either been pain-free and full, or if pain is present on first performing the exercise, it is soon abolished. He has been for a two-mile jog at a gentle pace with no ill effects. He indicates no functional loss on the Roland and Morris disability questionnaire (Roland and Morris 1983), and between 0 – 1 on the pain visual analogue scale. All his movements are examined, including repeated flexion in lying, and then repeated flexion in standing. All movements are full and no symptoms are produced.

He is considered to have made a full recovery, and restoration of function is unnecessary as movements are full range and pain-free. He is encouraged to make a gradual increase in his sporting activity. The issue of relapse, the importance of general fitness and the use of the same exercises, as long as they generate the same response, are discussed. He is happy to be discharged.

Conclusions

This chapter has considered the elements that contribute towards clinical reasoning – the thought process that underlies the therapeutic process. Essentially this involves gathering data about the patient's problem and considering this data in the light of theoretical issues, concepts and clinical experience. Certain errors may undermine this cognitive process, which is cyclical and ongoing during an episode of therapy. A case study has also been presented in this chapter as an example of the clinical reasoning process.

21: Recurrences and Prophylaxis

Introduction

As discussed in Chapter 1, recurrences, episodes and persistence of symptoms are very common in the back pain population. The strongest known risk factor for future back pain is a history of past back pain (Croft *et al.* 1997; Shekelle 1997; Smedley *et al.* 1997). At least 50% of those who have a first episode of back pain will have further episodes. Many recurrences are common and a quarter of the back pain population have a long-term problem (Croft *et al.* 1997; Evans and Richards 1996; Waddell 1994; Papageorgiou and Rigby 1991; Linton *et al.* 1998; Brown *et al.* 1998; Szpalski *et al.* 1995; Heliovaara *et al.* 1989; Toroptsova *et al.* 1995). For many, *"low back pain should be viewed as a chronic problem with an untidy pattern of grumbling symptoms and periods of relative freedom from pain and disability interspersed with acute episodes, exacerbations, and recurrences"* (Croft *et al.* 1998). Back pain should be viewed from the perspective of the sufferer's lifetime – and, given such a perspective, the logic of self-management is overwhelming.

Any education or assistance that the patient can be given to prevent recurrences, reduce the number or length of episodes or improve their ability to manage recurrences is an essential part of management. Provision of such education and encouragement of patients to problem-solve their own difficulties should be part of treatment. Supervision of patients must, in the light of the epidemiology of back pain, involve the nurturing of self-management strategies. This should be done from day one, and those strategies need to be individualised according to the patient's circumstances.

Primary prevention refers to risk modification to decrease the susceptibility for an event to occur (Lahad *et al.* 1994). Goals of secondary prevention in musculoskeletal problems could be to prevent or decrease the number of new episodes, shorten the duration of episodes, enhance self-management strategies, decrease the need for seeking health care or to decrease the need for time off work (Linton 1996). Given that no intervention has successfully been shown to reduce the prevalence or incidence of back pain, primary prevention appears unrealistic. Secondary prevention is perhaps a more realistic goal.

Sections in this chapter are as follows:

- preventative strategies
- patient's perspective.

Preventative strategies

Mobility and posture

There are suggestions in the literature that lack of flexibility and a reduced lumbar lordosis may be associated with future episodes of back pain (Takala and Viikari-Juntura 2000; Adams *et al.* 1999). The wide variation of what is normal precludes using range of movement for screening purposes, but may imply that patients should be encouraged to maintain general flexibility.

If patients have responded to self-management strategies in the resolution of derangements, their own role in the prevention of future episodes and management of any that do occur should be further emphasised. If, for instance, patients have responded to an extension principle programme, the appropriate use of extension in lying and extension in standing should be discussed. The standing exercise is very effective in the prevention of back pain, especially after prolonged sitting; however, once back pain has actually recurred, extension in lying is generally most effective. At the first sign of recurrence, the patient should immediately commence the procedures that previously led to recovery. Although back pain can commence suddenly and without warning, many patients get twinges or other slight symptoms that are a precursor to more serious back pain. If this type of warning is experienced, the patient may prevent more serious symptoms from developing if the appropriate procedures are applied.

Postural stresses, driving, and frequent bending and lifting have been identified as risk factors for back pain (Kelsey 1975; Kelsey *et al.* 1984a, 1984b; Frymoyer *et al.* 1983; Damkot *et al.* 1984; Krause *et al.* 1997; Massett and Malchaire 1994; Mundt *et al.* 1993; Punnett *et al.* 1991; Videman *et al.* 1984; Marras *et al.* 1993; Waters *et al.* 1999; Zwerling *et al.* 1993). Sitting, bending, driving and sedentary positions in general are very common aggravating factors when people have back pain (McKenzie 1979; Painting and Chester 1996; Biering-Sorensen 1983b; Boissonnault and Di Fabio 1996). Such postural factors are the predisposing and perpetuating influences that can most easily be altered and controlled.

Patients should be warned about the deleterious effects of sustained postures, and that while single movements of flexion are relatively harmless, maintaining a flexed posture for a lengthy period may precipitate an episode of back pain. During prolonged sitting or driving, maintenance of a good posture, using a lumbar roll, and regular interruption of that position is necessary. Walking around for a few minutes or performing a few repeated extensions in standing achieves this. Two recent studies (Harrison *et al.* 1999, 2000; Pynt *et al.* 2001) have reviewed the evidence relating to the optimal sitting and driving position. Both confirm McKenzie's 1979 assertion that the best sitting posture is one of lordosis, coupled with regular interruption of the sustained position. Likewise, activities involving prolonged stooping require interruption of that position and regular restoration of the lordosis by a few repetitions of extension in standing. The value of restricting activities of flexion, even in patients with chronic back pain, has been demonstrated by Snook *et al.* (1998, Snook 2000).

A specific and common situation that may need addressing in some instances is pain experienced after vigorous athletic activity. Commonly such individuals state that their pain appeared after participation in their sport, and a health care provider may have reinforced the belief that the activity was the cause of their symptoms. This assumption is often mistaken, as closer questioning reveals that the back pain actually commenced while sitting with a slouched posture adopted by the tired athlete *after* their vigorous activity. A consequence of sporting activity can be to make the individual more susceptible to back pain from the effects of postural stresses. Such persons need advice about the best posture in which to sit or rest after their vigorous sport, not advice to forego their sport.

Exercise

The usefulness of different interventions in secondary prevention has been evaluated by a number of reviews of the available literature (van Poppel *et al.* 1997; Gebhardt 1994; Lahad *et al.* 1994; Karas and Conrad 1996; Zimmerman 1998; Minor 1996; Maher *et al.* 1999; Maher 2000; Linton and van Tulder 2000, 2001; Jellema *et al.* 2001). These reviews considered one or all of the most commonly used interventions, namely exercise, back school classes, ergonomics advice and lumbar supports. They reached broadly the same conclusions, as follows.

Exercise has been shown to have a protective effect against back pain and its consequences. Exercise programmes have decreased the prevalence, severity and duration of back pain episodes, and have decreased time off work due to back pain. There is consistent evidence that exercise may be effective in preventing back pain (Larsen *et al.* 2002. Can Passive Prone Extensions of the Back Prevent Back Problems? Spine 2002 27.2747-2752).

General aerobic exercise appears to be as effective as specific trunk-strengthening exercises. Specific strengthening of abdominal muscles was found not to be effective in primary prevention of back pain over a two-year follow-up (Helewa *et al.* 1999). More general physical activity, for at least three hours per week, reduces the lifetime, one-year and point prevalence by at least 10% in middle-aged individuals (Harreby *et al.* 1997). In the year following a rehabilitation programme, those patients who maintained regular exercise habits had fewer recurrences of persistent pain ($P=0.03$) and less absence from work ($P<0.01$) compared to those who were physically inactive (Taimela *et al.* 2000).

We are clearly justified in urging our patients to return to sporting or recreational hobbies that they have stopped or to take up regular activity such as walking, swimming, jogging or gym activities, depending on their capabilities. New or unfamiliar exercise should always be started gently, but can be increased gradually over time as fitness and confidence improves. *The message that normal exercise is not only* not *bad for backs, but also actually helps in their recovery, is an important one to convey to patients.*

Lumbar supports, back schools

There was no evidence for the effectiveness of back belts and little for purely educational programmes. There is consistent evidence that lumbar supports and back schools are not effective in preventing back pain, and there is no good quality evidence on the effectiveness of ergonomics or risk factor modification.

Table 21.1 Key points to patients in prophylaxis

- Remember the importance of posture in looking after your back.
- Compensate for periods of prolonged stooping or sitting by standing erect and bending backwards a number of times.
- Regularly perform the exercises that led toyour recovery, especially extension in lying.
- Remember the importance of frequent changes of activity and limiting the time you remain in one position.
- The fitter, more active and more supple you are, the less likely you are to have pain and the better you will cope with it if it returns.
- Keep on top of your back problem by exercising regularly.
- When you start to increase your fitness, do so in a gradual way. Start with an easy level of exercise for you and do more as you feel able.
- A healthy back is a flexible back.

Patient's perspective

Patients are keen for an understanding of their problem, and lack of adequate information is one of the most common causes of dissatisfaction that back pain patients have with medical professionals (Greenfield *et al.* 1975; Deyo and Diehl 1986; Cherkin and MacCornack 1989; Cherkin *et al.* 1991; Fitzpatrick *et al.* 1987). Information must be individualised according to the patient's needs and concerns, but a key area relates to the provision of self-management strategies.

Qualitative studies provide valuable insights into patients' beliefs and attitudes about medical problems and treatment options. Of fifty-two patients with back pain who attended primary care in the UK, twenty-nine claimed to be actively working on the problem and consciously making alterations in their lifestyle in order to manage it (Skelton *et al.* 1996). Usually several preventative strategies were used: adopting a particular body posture when sitting, lifting and bending; taking light exercise; rest; and performing trunk-strengthening exercises. Four patients were in a contemplative stage, beginning to recognise an ongoing problem and the need to do something about it. Sixteen patients adopted a minimalist or sporadic approach to secondary prevention, despite having some knowledge about appropriate strategies for back care.

In a study of thirty-four mostly chronic back pain patients, acceptance of a certain level of symptoms was common (May 1998). Recognition

that they had an ongoing problem made many patients interested in finding out what they could do to help themselves and cope better with the condition. Many believed that their participation was an essential part of management and wished to be appropriately advised. Patients had a wide range of strategies to help themselves with the pain and to retain function, principally through exercises and postural and ergonomic awareness. The consciousness of having a back problem and being 'back aware' was a key component in self-management.

In a study of seventy-two elderly people with chronic pain, the majority of which was caused by musculoskeletal conditions, preferred strategies for management included self-administered physical interventions such as heat, and informal cognitive strategies such as various social or recreational activities (Lansbury 2000). Non-preferred strategies included medications, which were found to be ineffective or made them ill, and physiotherapy. Often physiotherapy treatment had not helped at all and they felt anxious about being reprimanded for not being 'better', but more importantly, it failed to give them any long-term strategies for management of chronic conditions. Awareness of the importance of a daily exercise programmes was widespread, but not commonly practised because of anxieties about its performance. They expected pain as part of the ageing process and often accepted their chronic conditions, but they were most interested in self-management strategies that they could use to cope with the problems from day to day.

These studies provide insights into patients' attitudes, beliefs and behaviours when confronted with persistent back pain and their expectations about management. Information should be a key component of any interaction with a health professional, addressing the problem, self-management strategies, treatment and investigations, and the future. Many patients are actively working on self-management strategies, accept this to be a necessary part of coping with the problem, and are keen to receive advice about this from health professionals. Commonly used strategies include general or specific exercise and postural adaptations. Smaller numbers of patients are not so ready to adopt the necessary behavioural changes, while some are at a contemplative stage prior to actually adopting self-care strategies.

Post-treatment surveys of patients who have been managed by mechanical diagnosis and therapy have demonstrated its value in secondary prevention. In a survey of 318 patients, 87% felt they had been shown how to prevent future attacks of back pain, and 75% stated that they had reduced recurrences by over 50% (McKenzie 1979).

The value of a purely educational approach, using *Treat Your Own Back* (McKenzie 1997), has also been explored in sixty-two volunteers with chronic back pain, of whom 81% were available for follow-up nine months after reading the book (Udermann *et al.* 2000). At this point 87% were still exercising regularly, 91% still used good posture, 82% noted less back pain and 60% were pain-free. Mean pain severity had dropped from 1.3 on a four-point scale to 0.44 and mean number of episodes from 4.1 to 1.0 per annum. Over 70% had found extension exercises to be most beneficial. Although there was no control group in this study, with a mean length of duration of back pain of over ten years prior to the intervention, this chronic sample served as its own control. Further improvements had been made in this group when followed up at eighteen months (Udermann *et al.* 2001).

In summary, many patients wish to be involved in their back care management, to practice preventative strategies or to be informed. Patients place considerable emphasis on information from clinicians about self-management strategies and prognosis. The most commonly adopted strategies, and therefore presumably the most useful, relate to exercises and posture.

Conclusions

Patients should be made aware that recurrence of back pain is common; many will have this knowledge from past experience anyway. This should not be allowed tm engender a fatalist attitude towards back pain, but be used to reinforce the importance of ongoing self-management. Most patients are interested in information about this.

It is the health care professional's responsibility to provide information and strategies that the patient can use in order to try to gain some independence of care. The value of general exercise and fitness applies in all cases. Patients should be encouraged to participate in some form of regular exercise at an appropriate level. If patients have had a derangement, awareness of sustained postural loads and the appropriate reductive exercise is important.

Information and advice to the patient about looking after their back in the future should not be regarded as a final part of the treatment package. These issues should be raised and discussed at any opportunity that is appropriate. The more the patient can problem-solve their own predicament, rather than be entirely tutored by the clinician, the more likely it is that future independence will be assured.

22: Derangement Syndrome – Characteristics

Introduction

Derangement syndrome is by far the most common mechanical spinal disorder – between 80% and 90% of patients may be so classified (McKenzie 1981; Rath *et al.* 1989 in Robinson 1994; Razmjou *et al.* 2000a). This chapter gives an overall introduction to the derangement syndrome and its distinguishing characteristics. Following chapters describe the varied clinical presentations of derangement and the management of the derangement syndrome.

Certain characteristics distinguish derangements from the other mechanical and non-mechanical syndromes. The distinguishing characteristics of the derangement syndrome are listed in the table below and are described in more detail in this chapter.

This chapter covers the following topic:

- characteristics of derangement syndrome.

Table 22.1 Characteristics of derangement syndrome

- sensitivity to loading strategies
- dynamic
- symptomatic and mechanical presentations and responses
  - centralisation and peripheralisation
  - deformity, impairment, loss of function
- continuums
- reducible or irreducible
- directional preference
- different treatment principles and loading for reduction of derangement
  - extension
  - flexion
  - lateral
  - force progressions
  - force alternatives

Characteristics of derangement syndrome

Sensitivity to loading strategies

Sensitivity to different loading strategies means that symptoms vary during the course of a day depending on activities and postures. Derangements respond to mechanical loading strategies in the form of repeated movements or sustained postures. This sensitivity to postural loads will be noted first during the history-taking, when, for instance, patients report a worsening or peripheralising of pain when sitting or bending and a lessening or abolition of pain when walking or lying. In the presence of such a history, very often repeated movements confirm the preferred direction of treatment to be extension, while flexion worsens the symptoms. Although sometimes it is single movements that cause a symptom change, much more commonly this is caused by sustained positions. This time factor can be a potent source of symptom aggravation and relief and is sometimes used in therapeutic loading.

History-taking is not always revealing, and may in some instances be paradoxical. For instance, in large derangements with constant symptoms and major impairment, patients may have found that temporary ease can be gained by opening the joint space and moving away from the painful obstructed movement. For instance, a flexed posture brings temporary relief of symptoms in a posterior derangement, as does leaning away from a lateral derangement. However, maintenance of such postures only perpetuates the displacement and overall worsens symptoms. Although providing temporary relief while opening the joint space, reduction will not be achieved until movements or positions are adopted that close the joint space by moving into the painful obstructed movement. Thus, the information gained from the history-taking can sometimes be confusing. Ultimately it is always the symptomatic and mechanical responses to loading strategies performed over twenty-four to forty-eight hours that dictate the management.

Dynamic

Derangements are dynamic. Pain may change during the day as the patient performs different activities and sustains certain postures, and derangements may change over time. The site of a patient's pain may change; symptoms may spread distally and proximally as the condition waxes and wanes. As symptoms worsen with a particular activity, movement may become more difficult, and as they ease

impairment may improve. This causes the inconsistent pattern of signs and symptoms that is typical of derangement and is a product of the sensitivity to loading strategies.

Treatment principles, which are detailed later, may also be dynamic. A patient's symptoms may reduce and then resolve with a loading strategy entirely in one plane. However, the need for a change in loading strategies during the reductive process may also occur.

Symptomatic and mechanical presentation and response

Derangements show both a symptomatic and mechanical presentation – that is, the patient complains of pain and displays some disturbance of normal movement and function. Pain may be spinal, with or without radiating or referred pain, and may be accompanied by paraesthesia. With the exception of nerve root adjerence, which is a dysfunction, radiating or referred pain occurs only in the derangement syndrome. Pain may be either constant or intermittent, but is likely to vary during the course of the day depending upon activities undertaken and sustained positions. Besides these symptomatic characteristics of derangement, the patient also demonstrates certain aspects of a mechanical presentation. This could include loss of normal range of movement, deviation on movement, inability to move fully in one plane of movement or an abnormal posture.

As symptoms improve, the movement loss and/or deformity should lessen also. If symptoms worsen, the obstructed movement is likely to become more impaired. The symptomatic and mechanical presentations behave in parallel. The symptomatic and mechanical presentations provide clues as to preference for a treatment direction.

During the physical examination or during an extended mechanical assessment, the patient with derangement demonstrates characteristic symptomatic and mechanical responses that allow the classification of that patient. In response to the appropriate therapeutic loading strategies, there will be reduction, abolition or centralisation of pain and there will be an improvement in range of movement. It is these characteristic symptomatic and mechanical responses that define the clinical presentation of derangement.

Centralisation and peripheralisation are two examples of symptomatic behaviour; deformity is an example of mechanical presentation – these are all unique characteristics of the derangement syndrome.

Centralisation and peripheralisation

A key element in the symptomatic presentation is the movement of pain proximally (centralisation) and distally (peripheralisation) in response to therapeutic loading strategies. Movement of pain in this way occurs during the natural history of derangements, but can be clinically exploited to bring about rapid and lasting changes, with centralisation strongly associated with a good prognosis. If pain is centralised, this refers to the explicit reduction, then abolition of distal pain in response to therapeutic loading strategies. This only occurs in derangement, and reduction of the derangement and centralisation happen together. The occurrence of centralisation is the strongest favourable symptomatic response to loading strategies and gives a clear indication of appropriate management. Conversely, the occurrence of peripheralisation indicates loading strategies to be avoided. These phenomena are discussed at greater length elsewhere.

Deformity

The most visible and dramatic, if infrequent, example of the mechanical presentation is the deformity. As a result of a significant derangement, the patient is locked in an asymmetrical posture and is unable to self-correct. In this situation, movement in the opposite direction is obstructed, or if the patient is able to correct the posture, they are unable to maintain the corrected position. A patient may be locked in a position of lumbar kyphosis and be unable to extend. They may be locked in a position of lateral shift to the right and cannot straighten or laterally flex to the left, or if they can do so they cannot maintain the correction, or they may be locked in extension and be unable to flex. Such deformities that occur as a result of derangements *are easily recognised.* They occur as a result of significant displacement and produce a very evident clinical presentation. The importance of recognising this phenomenon is that *inappropriate loading strategies can substantially worsen the patient when applied in the presence of the deformity.* For instance, in a patient who has a lateral shift, the application of extension forces may result in a severe worsening of the underlying pathology, the symptoms and the health of the nerve root.

Acute deformities of kyphosis, lateral shift or lordosis are substantial deviations from normal anatomical alignment and are clearly visible at presentation. They need to be recognised as they will determine treatment. Patients may also present with barely perceptible shifts in the lateral plane, with asymmetrical movement loss, and with

deviations on movement – these are not deformities. Although some in this group will also need lateral forces, many will respond to sagittal plane loading, and this should be explored first. If sagittal forces are inappropriate, a worsening or peripheralising of symptoms occurs and the need for lateral forces is exposed.

Continuums

Derangements describe a continuum in their symptomatic and mechanical presentations at one end of which is gross impairment of movement, severe pain and deformity, at the other end mild symptoms and a minimal mechanical presentation, such as a minor loss of movement. At one end clinician forces may be needed to correct deformity and promote self-treatment, at the other end minimal loading strategies, such as change of posture, may be needed to improve symptoms. Irreducible derangements represent the extreme end of the pathological continuum in which loading strategies can no longer exert a lasting effect on symptoms. Thus there is a wide spectrum of ways that derangements may present in clinic.

Continuums will also be noted in the varying responses to different loading strategies. Some reductions can be achieved with limited patient input, others need strict adherence to the management strategy; some manage on patient forces only, whereas others need clinician forces; derangements that require lateral forces need this to be applied in varying degrees of flexion or extension.

Reducible or irreducible

Reduction describes the process by which the derangement is progressively lessened. During this process, symptomatic and mechanical presentations are gradually improved; centralisation, decrease or abolition of symptoms occurs and movement is restored. Centralisation only occurs in the derangement syndrome and is intimately connected with reduction, the two phenomena occurring together. When the derangement is fully reduced, pain is abolished and full-range, pain-free movement is regained. This ideal outcome is not always attained; for instance, if there has been a long history or in the presence of a dysfunction.

The majority of derangements are reducible. Loading strategies will be found that improve the symptoms as well as other loading that worsens them. Sometimes worsening of symptoms is related to sustained postures – this may only be evident from the history, while

little change occurs during the physical examination. A few derangements are irreducible, in which case only loading strategies that peripheralise, worsen or do not affect symptoms are found. This is more likely in those with more marked symptomatology, at the extreme end of the pathological continuum. Some can be reduced relatively easily, while the reduction of other derangements requires strict adherence to exercise and postural management.

Maintenance of reduction is variable. Most reductions are stable in a short period of time and with a limited application of loading strategies, while others need a strict application of loading strategies over a more protracted period to bring about and maintain reduction. Some reductions are so unstable that simply a change in loading causes re-derangement.

Directional preference

Derangements generally show a preference for a treatment direction. Certain movements or positions cause symptoms to increase or peripheralise and movement to worsen. As noted earlier, when sustained loading is significant, the history rather than the physical examination may indicate this. However, the opposite movements or positions can cause symptoms to decrease or centralise and movement to improve. This is termed *directional preference* (Donelson *et al.* 1991). Reduction of the derangement often requires the temporary interruption or cessation of the aggravating factors, as well as the promotion of the particular reductive forces for which that derangement has a directional preference. However, as in all continuums, some patients only require minimal intervention, such as regular interruption of the aggravating factors, while others require a very strict adherence to postural and exercise strategies.

For instance, and most commonly, many derangements have a directional preference for extension. Symptoms decrease, abolish or centralise with extension procedures, and the mechanical presentation improves. Conversely, these same derangements worsen symptomatically and mechanically with sustained postures or repeated movements of flexion. Some derangements have a directional preference for flexion, others for lateral procedures, and some need a combination of sagittal and lateral forces. Some derangements require a change of forces during the reduction.

Different treatment principles and loading for reduction of derangement

When derangements are reducible, they will often respond to a single direction of movement. However, directional preference is not always stable during the reductive process and changes in therapeutic movements may be needed during the reduction. The majority of derangements respond to movements into extension, or a combination of lateral and extension, and smaller proportions require flexion or lateral forces. These loading strategies provide the treatment principles that guide management. In some derangements an initial loading of extension and lateral forces is required, which produces centralisation, and thus a requirement for purely extension movements. This process may occur in one treatment session or over several days.

In those who require the extension principle, some require posture correction only while others initially cannot even tolerate prone lying. Some patients have a favourable response to extension in standing, but most require extension loading to be performed in lying. The concept of *force alternatives* is used to describe the options in therapeutic loading that are available. Alternatives include different start positions, different directions of movement, repeated or sustained loading, and the varying degrees of flexion or extension used in lateral forces. An example is the use of sustained or repeated extension loading; many patients respond to extension in lying (a repeated movement), but some initially need the time factor provided by prone lying and prone lying in extension (sustained or static procedures).

Within each treatment principle a continuum of responses among different patients can be found. Some respond to exercises alone, while others need the addition of clinician forces. For those who require the lateral principle to bring about reduction, some require clinician-generated shift correction while others with a relevant lateral component can achieve this with patient-generated forces alone. The concept of a *force progression* is used to describe the escalation of therapeutic loading strategies from patient-centred movements to clinician-centred mobilisation or manipulation. The minimum force that produces reduction of the derangement is the preferred loading strategy. In most instances this will be achieved independently by patient-centred forces only; less commonly, clinician forces may be required.

Identification of directional preference is ultimately decided by the patient's symptomatic and mechanical response to loading strategies

over a test period, although clues are gained during the history-taking. It is this principle of a directional preference for loading strategies that guides subsequent management. Over the episode of care, these responses can sometimes change; the treatment principle therefore is not necessarily stable throughout the reductive process.

Conclusions

This chapter summarises the major characteristics that will be encountered in patients with derangement syndrome. The distinguishing characteristics outlined in this chapter provide the means of clinical recognition of the syndrome. Some of these features are unique to derangement – they are not all present in all cases, but their presence attests to derangement. The characteristics of the clinical presentation and management are discussed in more detail in the following chapters.

The detail provided in this and the following chapters is summarised in the form of criteria and operational definitions contained in the Appendix – these are necessary pre-conditions for identification of the different syndromes.

23: Derangement Syndrome – Presentation and Classification

Introduction

The variable nature of derangement

The clinical presentation of derangement can be highly variable because of several factors. There can be degrees of derangement, and thus there is a continuum of different presentations. Derangements may present at any point on a spectrum. Minor derangements cause minimal symptoms and disruption of normal function and resolve speedily and spontaneously. Major derangements may present with severe and persistent symptoms, including radicular pain and paraesthesia. The spectrum of derangement is wide.

Derangements are a dynamic pathology, rather than static and unchanging entities. In one individual they may vary over time and during the day, depending on loading strategies adopted. Derangements may, for instance, present at different points with back pain only or with back and referred pain.

Most important clinically, there are sub-groups within the derangement syndrome based on directional treatment principles. Not all derangements respond to the same management, and the treatment principle is not always static throughout treatment.

For these reasons the clinical presentation of derangement is much more varied than the other mechanical syndromes, and inconsistency may be noted during assessment. For instance, sometimes a particular activity hurts and sometimes it does not; sometimes the pain is on the right and sometimes on the left; sometimes the pain is referred down the leg and sometimes it is only felt in the back. The variable nature of derangement means that recognition may be problematical, but equally this variability is a key feature in identification. When the patient reports that 'sometimes' an activity hurts or 'sometimes' it is difficult to do a certain movement, think derangement. In this chapter some of the typical ways that derangements may present are outlined.

Sections in this chapter are as follows:

- clinical presentation
- treatment principles.

Clinical presentation

The great majority of patients presenting with back problems do so because of derangement, with a small minority attending with dysfunction or posture syndrome. On most occasions a high index of clinical suspicion for derangement classification can be made during the history-taking. Often there are enough clues in the history both to suspect the presence of a derangement and to give indications of the required treatment principle. Such clinical indications should lead to a focused physical examination that will often confirm initial suspicions.

Symptoms

Pain from derangement may be felt around the spine only or can radiate or be referred into the thigh and leg. Paraesthesia, numbness and myotomal weakness can accompany the pain. Thus symptoms may be either somatic, or neurogenic only in origin, or a combination of both. In the three mechanical syndromes, only in derangement are symptoms referred into the thigh or calf. The exception is adherent nerve root – a type of dysfunction that involves referred symptoms. This is found sometimes after a derangement resolves and sometimes as a complication following surgery (Chapter 29).

In derangement syndrome, symptoms may change over time. For instance, the patient may report that at onset the pain was felt around the spine, but over a period of weeks has spread into the lower limb and is now accompanied by tingling in the toes. This history represents a deteriorating presentation. Conversely, a patient may report that initially pain was felt in the whole leg, but in the last few weeks has only been present in the low back – this represents a presentation that has improved. In derangement symptoms may also cross the midline – the patient may report that initially pain was to the left of the back, but now is on the right. Occasionally it is reported that the patient first had sciatica several years ago in one leg and a few weeks or months ago developed the same symptoms in the other leg.

The symptoms of derangement also vary during the course of the day or week. For instance, a patient may report that pain is worse during the early part of the morning, on first rising, and also in the evening when 'relaxing' on an easy chair. Conversely, during the day when the patient is active and moving about at work, the symptoms are much less severe. A patient may report that symptoms are worse during the weekdays when they are at their desk performing their

clerical work, but at the weekend, when they are active and involved in a variety of pastimes, it troubles them less.

A patient may report that the back pain is most noticeable during prolonged sitting or driving, but considerably better when walking about or when on the move. Another patient may report that both walking and sitting aggravate leg symptoms – a relevant lateral component with or without a lateral shift should be suspected. Such variable symptom behaviour is very typical of derangement syndrome; a considerable range of such presentations will be heard from patients with this problem. They all demonstrate the mechanical sensitivity of the condition to different loading strategies. The details of which postures or movements aggravate the problem and which relieve the problem provide vital clues about the appropriate management strategy.

The phenomena of peripheralisation and centralisation, when pain moves distally or proximally in response to different postures or movements, only occur in the derangement syndrome.

In derangement syndrome, symptoms may be either constant or intermittent. Frequently pain is constant in nature; there may be no position in which they can get relief. In the three mechanical syndromes, constant pain is only found in derangement syndrome; constant pain is never present in dysfunction or postural syndrome. There may be a dull constant ache that is aggravated by certain movements and positions and eased by different postures. Some histories can suggest an irreducible derangement. For instance, if pain is constant and severe no position of ease can be found, and different positions only exacerbate symptoms; or if a relieving posture is found it only provides a short-lived respite, and a further change in position is then required.

However, not all patients with derangement have constant symptoms. Some patients present with intermittent symptoms, reporting that certain activities bring on the pain that then persists for a time. Other activities may reduce or abolish their symptoms. A common pattern is the production of symptoms with prolonged activities of flexion, principally sitting. Once they change from the aggravating posture and walk about, symptoms are reduced or abolished and remain absent or lessened until they sit for a prolonged period again. Again, the history guides us in determining the appropriate management strategy. This sort of presentation sounds superficially like postural

syndrome (Chapter 30). Whatever the underlying pathology may be, postural factors can confuse the clinical picture; until they are eliminated, the picture remains unclear. However, other features are characteristic of derangement. Once pain has appeared, it persists beyond the period that the posture is maintained and restricted movements accompany symptoms.

History

Derangement may arise either from an event or for no apparent reason. No apparent reason for onset of back pain is most common. Although trauma is sometimes involved in the onset, derangements frequently arise from an activity or movement that has been performed thousands of times previously without incident. The patient may report that a normal bending or lifting movement initiated symptoms. They may report that symptoms came on for no apparent reason; however, when questioned more closely, the patient may reveal an extensive period of sitting or driving, a prolonged bending activity, or an unaccustomed episode in the garden prior to the onset of symptoms. They may report a minor 'twinge' of pain, following which they 'rested' on the sofa for several hours – when they came to get up they were unable to straighten up fully and the pain was considerably worse.

Thoroughly questioning the patient about the onset of this episode of back pain may reveal an obvious or obscure postural stress that has initiated symptoms. This is helpful in determining the appropriate management strategy. Most commonly, flexion activities appear to be the key initiating and aggravating postural strains. However, frequently it is impossible to determine any causative event; pain appears to have come on for 'no apparent reason'. In this case the factors that aggravate and relieve symptoms may help elucidate management, or else this must be determined solely by the physical examination.

Patients with derangement may present at any stage of the disorder, from acute to chronic. If seen at the acute stage, these patients may have developed considerable functional disability in a matter of hours or days. This sudden onset of disabling symptoms only occurs in derangement syndrome. Major losses of range of movement, deformity or the presence of neurological signs and symptoms may accompany the onset of symptoms.

Frequently patients with derangements have had episodes of pain in the past. This may be a repetition of past episodes, or else symptoms may be gradually getting worse, with greater duration of episodes or extent of referred pain.

Physical examination

Sitting posture is frequently poor in the derangement syndrome; that is, the patient sits in a flexed posture with absent or reduced lordosis. Posture correction (Procedure 4, Chapter 17) may decrease, abolish or increase any pain that is present at the time. In standing, the lumbar lordosis is often reduced, and sometimes a deformity of kyphosis or lateral shift is present. Rarely is an accentuated lordosis seen. Symptoms may also alter between sitting and standing, which also provides clues about the postural component of a derangement.

There is nearly always a loss of movement, although the degree of loss is variable. In a few patients there may be no detectable loss, but end-range pain reduces on repetition. Most commonly, sagittal plane movements are markedly affected, with both flexion and extension being limited. Another common pattern is a marked loss of extension, but only a minor reduction in flexion. Sometimes sagittal plane movements are relatively mobile and side glide movements are most affected. The loss of side glide movements is often asymmetrical. The plane of movement that is most affected is usually an indication of the plane that needs to be explored for symptom-reducing movements. Sometimes, when symptoms are especially severe, all movements are substantially limited.

In derangement there may also be a departure from the normal pathway of movement, so that as the patient bends forward they deviate to the right or left. Less commonly this may also occur in dysfunction or adherent nerve root. Deviations may also occur on extension.

The response to repeated movements in the derangement syndrome is extremely characteristic and helps to distinguish the syndrome. Some movements may cause a worsening or peripheralisation of pain, but the opposite or other movements may cause a centralisation, abolition or decrease of symptoms. Equally, the appropriate loading strategy will lead to an improvement in range of movement.

Flexion activities frequently aggravate symptoms. If the patient has already indicated this during the history-taking, especially in acute

patients, an over-vigorous testing of repeated flexion movements should be avoided. This may simply exacerbate symptoms and tell you nothing that was not already known.

If the patient has a deformity of lateral shift, the lateral component must be addressed first. The deformity is a substantial and obvious lateral deviation from normal alignment that has come on with the pain, which the patient is unable to self-correct, or if able to correct swiftly falls back into the shift position. Attempting to restore extension in the presence of a lateral shift could result in a severe aggravation of symptoms.

In all other circumstances, sagittal plane movements are examined first. Even in patients with unilateral or asymmetrical pain, there is often a response to sagittal plane movements. If at any point symptoms are peripheralised or made worse afterwards, further testing of that movement may require that static positioning be explored or the movement is abandoned. Movements should always be explored in the loaded and unloaded starting position. It is not unusual for extension in standing to aggravate symptoms, but extension in lying eases them.

During the assessment of repeated movements, it is important to closely monitor symptom response. In this regard the presence and extent of spinal and referred pain should be established prior to movement testing in one direction. Symptom response during movements is noted, but of most importance is the status of symptoms a few minutes after testing.

The mechanical presentation as well as the symptomatic presentation may alter during testing. A patient who describes a significant increase in pain during testing often exhibits an increase in obstruction to movement and may develop or increase a deformity. Conversely, a decrease in pain should be accompanied by a reduction in obstruction to movement and decrease in any deformity that is present. Sometimes the mechanical response is clearer than the symptomatic response. For a fuller description of analysis of symptomatic and mechanical responses, see Chapter 16.

In derangements, repeated movements can have a rapid and lasting effect upon symptoms and mechanical presentation. Movement in one direction or sustained postures may peripheralise, increase or

produce pain, which remains worse after testing. Conversely, movements in the opposite direction may centralise, decrease or abolish pain, and the patient remains better afterwards. Equally, sustained postures or repeated movements may cause a loss or improvement in mobility. Rapid and lasting changes in the condition, such as these, which occur as a result of repeated movements or sustained positions, only occur in derangement. These changes can occur within a few minutes in some cases or a few days in others.

In certain circumstances repeated movements may not cause a lasting change in symptoms. Particularly if the problem has been present for some time, an immediate response to repeated movements may not be forthcoming during the initial assessment. Nonetheless, there may be clues in the history and in the physical examination to a possible preferred direction of movement. For instance, the patient may report a worsening during prolonged sitting or may demonstrate a blockage to extension while flexion remains comparatively free, or the extension range may improve during or after repeated extension. All of these instances indicate that extension is the preferred direction of movement. Even if there has been no lasting change in symptoms, such characteristics give a good indication of the direction of movement to be pursued over the forthcoming few days.

At other times, patients with chronic symptoms may report minimal mechanical sensitivity of the problem. During repeated movement testing symptoms may increase, but are no worse afterwards. Following the physical examination, they remain unchanged. Although the prognosis of some patients with chronic symptoms is poor, the length of time that they have had the problem should not be seen as a bar to a thorough mechanical assessment. Frequently such patients, whose initial response may appear discouraging, improve when repeated movements and postural advice is applied over several days. These patients should always be given a chance to improve the self-management of their problem.

Irreducible derangements can be aggravated by repeated movements and sustained postures, and no movement or position has a lasting beneficial effect. Irreducible derangements are generally at the severe end of the continuum. These are often specific nerve root conditions: disc herniations with clear-cut dermatomal pain patterns, sensory and motor deficit, nerve tension signs, major loss of movement and deformity. Symptoms and neural deficits are constant. These patients

are possible surgical candidates, which may provide short-term better outcomes if all appropriate criteria are met. Mechanical diagnosis and therapy is useful in detecting these patients, but conservative therapy is unable to improve symptoms, although generally time will.

Table 23.1 Derangement syndrome – criteria

History:

- local or referred pain, possibly paraesthesia
- variable symptoms: centralisation/peripheralisation, increased/decreased, changed sides
- constant or intermittent
- mechanical pain – varies with different postures
- problems with curve reversal after prolonged sitting or bending
- acute through to chronic
- onset can be associated with considerable functional disability.

Physical examination:

- poor posture (slouched sitting)
- postural correction affects symptoms
- reduced lordosis
- deformity of lateral shift, kyphosis or accentuated lordosis (uncommon, severe cases)
- loss of range of movement (major loss in severe cases)
- positive neurological testing (severe cases)
- repeated movements or sustained positions produce, worsen or peripheralise symptoms
- opposite repeated movements or sustained positions abolish, make better or centralise symptoms
- repeated movements or sustained positions decrease range of movement
- opposite repeated movements or sustained positions increase range of movement.

Treatment principles

For the purpose of establishing treatment principles, a series of clues must be identified. All these factors should be used in the continuing analysis of the patient's response. These factors not only provide reliable indicators or predictors of appropriate loading strategies, but they also provide essential ongoing analysis of their therapeutic effect. These factors are important in predicting patient response and in evaluating response to management.

Table 23.2 Dimensions in analysis of derangements

- history-taking – clues to directional preference
- observation – presence of deformity, loss of lordosis
- single movement testing – range, deviation, curve reversal, blockage to movement, pain
- response to loading strategies – postural correction, sustained posture or repeated movements to establish directional preference.

These factors are also discussed in the chapters on history and physical examination, but some examples are presented here.

Patients frequently report that symptoms are worsened during or after prolonged sitting or bending, while concurrently their symptoms improve or are abolished during periods of walking (history-taking). Such patients are describing directional preference for extension, and prolonged testing of flexion is unnecessary – they have already told you it makes them worse. In such a patient, flexion may be reasonably well maintained but extension is severely limited (single movement testing). Response to posture correction and repeated movements is likely to confirm the picture (response to loading strategies). This may be apparent during the first session or could need a day or two of repeated loading strategies to confirm.

On another occasion a patient may report that the ache never totally abates, but is made worse by activities that involve extension or flexion, such as walking and sitting (history-taking). They enter the room with a severe alteration to their normal sagittal alignment (observation), and their movement in all directions is minimal or non-existent (single movement testing). In such an individual, the lateral component must be addressed immediately; attempting extension exercises can severely worsen symptoms. Performing the procedure of correction of a lateral shift causes centralisation of pain and reduction of the postural deformity (response to loading strategies).

It is important to be aware that in certain acute presentations the patient can actually provide contradictory or 'false testimony' concerning directional preference. For example, the patient has limited or no extension, and every time they attempt to stand straight the pain is exacerbated; they feel easier, but are not made better in some degree of flexion. Such a patient sounds as though they have a directional preference for flexion. This ignores the key mechanical finding of a major obstruction to extension. It is the extension that

needs to be targeted – an unloaded starting position is required. Such 'false testimony' is most common in instances of deformity, in which patients find temporary relief in the causative position, but are made no better by it.

These illustrations are at different points on the continuum, but they exemplify the need to incorporate all aspects of the history-taking and physical examination into decision-making about management strategies. During the history-taking the patient provides clues as to directional preference or lack of it. Observation may reveal a clear lateral shift or no such gross deformity. Single movement testing frequently reveals an obstructed direction, which is also painful – and is generally the direction that needs to be explored during repeated movements. Response to loading strategies often confirms previous clues, and *ultimately symptomatic and mechanical responses provide the justification for the loading strategy adopted*. Treatment principles are grounded in symptomatic and mechanical responses to therapeutic loading.

Table 23.3 Main treatment principles for derangement syndrome by directional preference

- extension
- flexion
- lateral
- combination
- irreducible.

Conclusions

This chapter outlines some of the features of history and physical examination that may be present in typical derangements. The variable nature of derangement should be remembered, which means that there are numerous different ways in which the syndrome presents. Features of the history and physical examination and response to therapeutic loading strategies determines the appropriate treatment principle. Assessment of these features should be conducted at each treatment session to ensure that the appropriate management strategy is being recommended. The key distinction is to determine the appropriate directional preference that will bring about the reduction of that derangement. A great number of all patients will respond to extension forces, or a combination of lateral and extension forces,

with fewer requiring flexion or lateral forces. It is necessary to determine whether the problem is reducible – that is, can mechanical loading strategies have a lasting effect on symptoms. Irreducible derangements are more likely at the extreme end of the continuum – those with radicular pain and neurological signs and symptoms.

24: Derangement Syndrome – Management Principles

Introduction

This chapter considers the general principles of management of derangement syndrome. The principles considered are the stages of management in derangement: reduction, maintenance of reduction, recovery of function and prevention, as well as the treatment principles: extension, flexion and lateral. Factors involved in the identification of irreducible derangements are also considered.

Sections in this chapter are as follows:

- stages of management
- treatment principles
- irreducible derangements.

Stages of management

Successful management of derangement involves a mixture of education and mechanical therapy. Four stages are involved in treatment of the derangement: reduction, maintenance of reduction, recovery of function and prophylaxis. Reduction of the derangement is key and takes priority; however, in practice, the stages overlap. Prophylaxis is not left to the end, but should be discussed at any and every appropriate opportunity. Encouraging self-management for the present, and any future problems, needs discussion and information provision from the beginning of an episode of care.

Initially the key goals are reduction of the derangement and maintenance of the reduction. Reduction can occur during the first session, but may take several management sessions and weeks of home self-management. Reduction, by itself, may be of little value if the patient does not know how to maintain improvements or avoid provoking factors. Once reduction of the derangement is maintained, it is important that the patient's mobility and confidence in activity is fully restored. Finally, patients should be informed about the high rate of recurrence of back pain. There should be discussion about what they can do to try to reduce the risk of recurrence, and what to do should back pain nonetheless recur.

Table 24.1 Stages of management of derangement

1. reduction of derangement
2. maintenance of reduction
3. recovery of function
4. prevention of recurrence.

Reduction of derangement

The treatment principle is selected according to the directional preference of the individual. This is based upon the movements that abolish, decrease or centralise symptoms, and restore mobility and function. The treatment principles are categorised as extension, flexion or lateral. Reduction is often attained using patient-generated forces only, although sometimes clinician-generated forces must supplement these.

Reduction describes the process by which the derangement is progressively lessened. The reductive process is continuing when peripheral pain is reported to be progressively centralising or decreasing, or if pain located in the back is centralising (felt more in the spine than all across the back), decreasing or ceasing. During this process, centralisation occurs, movement is restored and symptomatic and mechanical presentations are gradually improved. When the derangement is fully reduced, pain is abolished and full-range pain-free movement is usually regained.

Reduction is complete only when the patient reports none of the original back or referred pain when undertaking normal daily activities and pain-free movement is restored. During the process of reduction, the patient may undertake certain activities that impede or reverse the process and cause symptoms to reappear. With cessation of the aggravating positions and performance of the appropriate end-range movements, symptoms should once again start lessening or centralising.

As noted in the classification algorithm (see Appendix), the decision that a patient has a reducible derangement should be made within five visits, but often it will be made on the first visit. In many patients reduction occurs rapidly over days or weeks, but in some patients this process may take several weeks, especially if avoidance of aggravating factors is not strictly adhered to. Chronic derangements and non-mechanical factors can also elongate the reductive process.

Procedures that are achieving reduction need not be supplemented in any way, but should be continued until symptoms are abolished, mobility is full and pain-free, and function has returned to normal. If improvements plateau at any point, force progressions, which may include clinician-generated techniques, are added. The minimal force necessary to achieve reduction is used at all times; this ensures that patient involvement and independence is always maximal.

Usually repeated movements are involved in the reductive process, but when a time factor is important, sustained procedures are more important, at least initially.

Patients should be aware what to expect from the exercises. Movements may initially generate increased back pain, but reduce leg pain. The increased spinal pain can sometimes be quite disconcerting to the patient, so they must be reassured about this. Just as they should be told what to expect, patients must also be informed about indicators to stop the exercises. The main reasons for stopping the programme are a worsening of distal pain or peripheralisation of pain. In such instances patients should be told to stop the exercises and return for review. For the exercises to be held responsible for a worsening of pain, this must occur at the time of performing the exercises, not several hours afterwards. If symptoms appear two hours after exercises are completed and when the patient is 'relaxing', their return is due to the posture at the time, not the exercise.

It is common for patients who are unused to exercise to experience 'new pains' after starting the programme of exercises. These may be felt in the thoracic spine or shoulders and arms after performing extension in lying. These result from adopting new positions and unaccustomed movements, and generally wear off within a week provided the exercises are continued.

When reduction is complete or nearly complete, the patient may report that the original pain felt on a particular movement is gone, but that they now experience a strain or stiffness. Patients generally recognise this as normal, but occasionally may need to be reassured that this is usual. Thus when patients report that they experience pain at end-range of extension, it is necessary to clarify the true nature of the problem. They should be asked, *'Is this pain or strain?'* Patients mostly find it easy to differentiate. It is usually only necessary to

make this distinction at the latter stages of reduction or when reduction is complete. It should be remembered that this might occur during the initial assessment, so this question should be routinely asked in all circumstances.

In many derangements the reductive movements restore full mobility in all directions. Thus, often in a derangement requiring the extension principle, extension procedures remove the obstruction to extension, but also restore full-range flexion. However, sometimes due to the derangement and avoidance of the aggravating movement, some adaptive changes can occur. This only occurs when symptoms have persisted for six to eight weeks or longer. When this happens it is in the opposite direction to that which has been used in reduction of the derangement. Thus, in a derangement requiring the extension principle, flexion may become restricted and tight. The derangement is reduced, but pain on end-range movement, which may be limited, persists because of a dysfunction. This may be recognised only after the derangement has been stabilised for several days. Sometimes it is not recognised at all, and in the case of a flexion dysfunction, the clinician thinks the derangement is still present because of pain on flexion. This situation is addressed in recovery of function.

Reduction – key aspects:

- identification of treatment principle that centralises, reduces or abolishes symptoms and restores function
- regular performance of self-management exercise until symptoms are abolished and function fully restored
- force progressions only necessary if no initial improvement or improvement ceases
- re-evaluation of treatment principle only necessary if improvement ceases.

Maintenance of reduction

During the reduction process and once reduction is achieved, this situation must be maintained. This involves avoidance of the aggravating postures or movements that are likely to reverse the improvements that have been made.

In posterior derangement, the most common aggravating factor is flexion. While single movements of bending are often tolerated,

sustained flexed postures, such as sitting or driving, or repeated flexed movements, are commonly found to cause a return of symptoms. Instruction in posture correction (Procedure 4, Chapter 17), the use of a lumbar roll, interruption of prolonged sitting, and avoidance of repetitive flexion are thus important elements of maintenance of reduction. During maintenance of reduction it will also be necessary to maintain the exercises that were used in reduction of the derangement. Patients should be advised to continue these exercises for several weeks after resolution of symptoms and to initiate them more regularly should symptoms return.

The first time maintenance of reduction may be important is standing after performance of extension in lying and other reductive procedures. The patient may abolish symptoms, which return as soon as they stand. The patient should then be shown the correct method for getting off the plinth. They move to the side of the plinth, perform an extension to maximum, then drop one leg and then the other to the floor. At the same time they use the fingertips of both hands to keep the trunk more and more erect as the upright weight-bearing position is regained. This should ensure maintenance of reduction; if it does not, and symptoms return immediately, the reductive process may be unstable.

In anterior derangement, the most common aggravating factor is extension. Sustained postures of extension such as standing or walking are commonly found to aggravate symptoms or cause them to return. Instruction in slumped sitting, interruption of sustained erect stances and frequent flexion exercises as in toe touching are thus important elements of maintenance of reduction. During maintenance of reduction it is always necessary to maintain the exercises that were used in reduction of the derangement. Patients should be advised to continue these exercises for several weeks after resolution of symptoms and to initiate them more regularly should symptoms return.

In derangements that require lateral forces in their reduction, instructions for maintenance are often similar to those given for posterior derangements. Activities of flexion often aggravate, so avoidance of sustained flexed postures is usually necessary. Some elements may need to be individually tailored for the patient; for instance, posture correction may need to be gradually introduced as reduction occurs.

Maintenance of reduction is variable. Some reductions are stable in a short period of time and with a limited application of loading strategies, while others need a strict application of loading strategies over a more protracted period to bring about and maintain reduction. Some reductions are so unstable that simply a change in loading causes re-derangement.

The importance of posture in the reductive process and maintenance of reduction is especially important in derangements that require the extension principle. While extension procedures may reduce the derangement relatively easily, successful reduction can often be short-lived if attention is not paid to postural stresses. In particular, sustained sitting can prolong pain from a derangement and is generally far more potent a cause of symptom aggravation than bending forward a few times.

For this reason, the patient must be taught the importance of postural correction and use of a lumbar roll for maintenance of the lordosis when sitting. Often they will have identified sitting as an aggravating factor already, so they are receptive to advice on sitting postures. It is often impossible to maintain an upright lordotic posture on a sofa, settee or lounge chair. Patients should be encouraged to use upright chairs, maintain the lordosis with a lumbar roll and regularly interrupt the sitting posture. If symptoms recur, the reductive procedure should be performed. For patients who are at risk of developing pain on sitting or for those who have a history of recurrent or persistent back pain with prolonged periods of sitting or driving, lumbar rolls should be available for loan or inexpensive purchase.

Patients may return for review stating that the exercises work in abolishing their symptoms, but the pain returns after a while, and overall they are no better. Further questioning reveals that the return of symptoms occurs when they sit. Another patient may report that generally they are much better and symptom-free, but every time they sit or drive for more than half an hour the symptoms return. Another patient may report that they are now generally free of pain during the day when they are active and moving about, but in the evening, when 'relaxing', symptoms return. In all such instances, force progressions should never be used. In effect, the patient is reporting successful reduction of derangement – the problem is in maintaining it. Further discussion must be had about the troublesome

nature of sitting and the ways to avoid its effects, especially reinforcing the procedures of slouch-overcorrect and posture correction, and use of the lumbar roll. Instigating clinician procedures at this point will not help the patient deal with the postural stresses of normal activity when they arise.

Maintenance of reduction – key aspects:

- regular performance of the reductive procedure
- use of postural correction, including lumbar roll if sedentary
- avoidance of aggravating factors, especially sustained postures
- regular interruption of sustained postures.

Recovery of function

During maintenance of reduction, patients will have been avoiding certain movements and as a consequence adaptive shortening may have occurred in certain structures that have not been regularly stretched. Even in the absence of adaptive changes, the patient may have become overcautious about the aggravating movement, so it is also important to restore their confidence in its use. Recovery of function thus concerns the restoration of restricted movement after the reduction of the derangement and the restoration of the patient's confidence in normal use of the lumbar spine. *Failure to recover function after an acute episode can be a potent factor in nurturing fear–avoidance beliefs, thus predisposing patients to chronic symptoms.*

Most commonly, the restricted movement is flexion following the reduction of a derangement with the extension principle. Flexion in lying and in standing should be routinely checked following full and stable reduction. Often no problem will be found, or a minor degree of tightness that rapidly improves with repeated movements. Sometimes a greater degree of restriction is found, which is an incipient or actual flexion dysfunction. Because restoration of this movement involves the postural stress that previously aggravated the derangement and thus may cause it to return, an over-vigorous recovery of flexion should be avoided initially. Recovery of function is commenced once the reduction of the derangement is stable (see Table 24.2). However, it should be noted that once derangements are fully reduced, very often there is no 'underlying' dysfunction. In anterior derangements, recovery of extension is virtually never required.

Because the flexion programme that is being used in the remodelling process could cause recurrence of posterior derangement, certain precautions should be observed. This is especially important if the derangement has only recently been reduced. The stability of reduction can be ensured by the following precautions. A timeframe is presented as a rough guide for general purposes, although this may need tailoring in individual circumstances.

Table 24.2 Recovery of function – ensuring stability of derangement

Determining if derangement is stable:

- monitor symptomatic response to repeated flexion in lying:
  - symptoms may be produced at end-range, no worse
  - symptoms may become less painful on repetition
  - symptoms must not be produced, and remain worse
  - symptoms must not become more painful on repetition
  - symptoms must not peripheralise
- monitor mechanical response – range of movement and response to extension must remain unchanged following repeated flexion.

Introducing flexion:

- commence with less stressful flexion procedures and progress as that procedure is easily tolerated or when it brings no further improvements:
  - slouch-overcorrect (day 1 onwards)
  - flexion in lying (day 1 – 2)
  - flexion in sitting, gradually straighten legs (day 3 – 7)
  - flexion in standing (day 8 onwards)
- perform new exercises less frequently, initially only 5/6 repetitions 5/6 times a day
  - avoid over-vigorous or prolonged flexion procedures within first few hours of waking – during this time period the disc is likely to be under increased pressure as a result of nocturnal re-absorption of fluid
  - initially perform ten repetitions of flexion from midday on, every three hours, until retiring. If the derangement appears stable, the patient may commence the exercise a little earlier in the day and repeat it every two hours. It is inadvisable to perform flexion in standing immediately on waking.

Recovery of function – key aspects:

- all movements must be made full range and pain-free after reduction of derangement
- patients should be made confident to bend and perform other normal activities
- restoration of flexion should proceed in stages, as above
- mobility into extension should remain unchanged after repeated flexion
- recovery of function rarely required in anterior derangement.

Note – avoidance of flexion

Although it is important to be cautious when introducing flexion procedures in certain circumstances, it is important also not to exaggerate the 'danger' of what are normal daily movements. We must not be guilty of making our patients overanxious or fearful of certain movements. In the past some clinicians have been overly concerned about the effects of flexion, and this anxiety has been conveyed to the patient. Making patients fearful about normal movements is not helpful for a full return to function. The patient should be given the timetable for recovery of function, as detailed above, so they can be assured about normal recovery of function in that timeframe.

Prevention of recurrence

Advice concerning back care in the future is always given to the patient during the treatment episode and prior to discharge. This should include discussion of the recurrent nature of back pain, avoiding prolonged aggravating postures, practice of prophylactic exercises and the importance of general fitness. Discussion and education about prophylactic concepts should commence at the first session and continue on each patient visit. When the pain starts to centralise, the usefulness of this phenomenon to guide treatment can be explained and the patient can be advised that the procedure that achieves this is their 'first aid' for the future. 'This is the exercise you must do at the first sign of recurrence.' Numerous questions, opportunities and teaching tools will arise during an episode of care, and these must be fully exploited to maximise patient understanding and independence. See Chapter 21 for more detail about prophylaxis.

Prevention of recurrence – key aspects:

- continuance of exercise programme for six weeks to maintain full mobility, flexion and extension
- beware of sustained postures
- lifelong use of lumbar roll
- importance of general fitness
- use of exercises if back pain re-occurs.

Treatment principles

The key to correct management is the identification of the appropriate loading strategy, which reduces the derangement and improves signs and symptoms. Not all derangements respond to the same loading strategy, and what may work in one case may cause the situation to worsen in another case. The sub-groups of derangement syndrome depend on the direction of reductive movements. Mechanical evaluation is used to determine this preference for loading strategy in a certain direction. Frequently the opposite movement worsens the condition. Favouring a particular postural or movement loading is termed *directional preference* (Donelson *et al.* 1991).

The fundamental distinction is between derangements that are reduced by movements or postures of extension, flexion or a lateral direction. Within these three sub-classifications, there is considerable variety of response to the same procedure. For instance, in derangements with a directional preference for extension, a few respond in standing while others are worsened by this procedure, and most require extension in lying. Many improve in prone lying, but those with a kyphotic deformity will not tolerate this position initially. They need to be positioned to accommodate their deformity, and then gradually and slowly resume the prone lying position. Similar qualitative decisions are required in derangements that have a directional preference for flexion or lateral movements. Just as derangements themselves are continuums, their reduction by different directional preferences should be seen as a continuum also.

Treatment principles are not necessarily stable throughout the reduction of a derangement, although they may be. In one situation it may occur that a patient reduces and then resolves symptoms with a loading strategy entirely in one plane. However, situations may also arise in which initial loading in extension exposes a relevant lateral component with a worsening of peripheral symptoms. The introduction of a combination lateral and extension loading produces rapid centralisation. The patient then requires extension to abolish the remaining central back pain. In a few minutes the patient has required extension, a combination of lateral/extension, and extension loading. In another instance a patient initially has an obstruction to extension and improves with extension loading. However, after a few days of this loading force, an obstruction to flexion is created

and a brief period of flexion loading is required to resolve symptoms. Such dynamic responses to loading strategies are not universal; many patients will reduce a derangement with loading in a single plane. However, the need for variable loading strategies during the reduction of a derangement occurs frequently enough to need constant awareness of this possibility.

Table 24.3 Treatment of derangement syndrome by directional preference

- extension
- flexion
- lateral
- combination
- irreducible.

Extension principle

This is the most commonly demonstrated directional preference displayed by derangements. Whether the patient has symmetrical, asymmetrical or unilateral symptoms, or back, thigh or leg pain, *it is still necessary in most cases to explore sagittal plane movements*. Sagittal plane forces, that is extension and/or flexion, are in most instances the only loading strategies to be examined, at least initially. However, there are times when frontal plane movements, that is lateral forces, need to be explored, and these situations will be outlined.

There are certain situations when the sagittal plane is avoided or its use deferred (Table 24.9). There is one indication for avoiding the sagittal plane altogether on day one, at least initially. There are two situations in which exploration of the sagittal plane is abandoned. All of these are situations when lateral forces should be explored.

1. *Only in the case of a lateral shift deformity should there be no testing at all of the response to extension. In this case, lateral forces are immediately applied.*
2. Secondly, if at any time while exploring unloaded sagittal plane movements, both dynamic and sustained, there is peripheralisation or a worsening of unilateral or peripheral pain, this plane should be abandoned and lateral forces should be explored. Extension forces should not be abandoned if symptoms are worsened or peripheralised in standing without first having explored unloaded extension.

3. Thirdly, if there has been no lasting improvement of unilateral or asymmetrical symptoms after sagittal plane forces have been thoroughly explored, lateral forces should then be explored. Before sagittal plane forces are abandoned, in this instance there should be a progression of force, including extension mobilisation.

Following the application of lateral forces, sagittal forces may subsequently become necessary.

Except for the one absolute exclusion criterion of an obvious and clinically relevant lateral shift, repeated movement testing would always start using sagittal plane forces. Often the history has already provided clues as to likely directional preference, and the physical evaluation simply confirms this. If symptoms are reduced, abolished or centralised with extension forces, or if the mechanical presentation improves, management proceeds with the use of extension.

Table 24.4 Clues as to the need for extension principle – not all will be present

History:

- onset may relate to flexion activity
- activities of flexion produce or worsen symptoms
- activities of extension decrease or abolish symptoms
- problems arise with curve reversal after prolonged sitting or bending

Physical examination:

- poor posture – slouched or slumped sitting
- posture correction affects symptoms
- lumbar lordosis reduced
- loss of flexion
- greater loss of extension
- extension is obstructed and painful
- side glide movements minimal or no loss, and little pain
- repeated flexion produces, worsens or peripheralises symptoms
- repeated flexion worsens mechanical presentation
- repeated extension abolishes, reduces or centralises symptoms
- repeated extension improves extension range
- kyphotic deformity – patients fixed in flexion and unable to stand erect (uncommon, very severe presentation)
- extension cannot be tolerated
- flexion appears to bring temporary relief.

If symptomatic and mechanical responses are not fully elucidated by initial testing in the sagittal plane, then force progressions may be necessary in order to make the situation clearer. This can either be done on day one using overpressure, or else the patient can perform many sessions of repeated movements over a twenty-four- to forty-eight-hour test period. Many patients will respond over this time. To enable them to resolve their problems using self-mobilisation only, it is undesirable to use clinician techniques on the first occasion unless absolutely necessary. If at review the situation is still unclear, then force progressions, including extension mobilisation, should be definitely included at this point. *Unless the sagittal plane is fully explored, the correct reductive movement may not be found.*

In some instances, to clarify uncertainty, symptom provocation can be applied using repeated movements to provoke symptoms. If the patient tests this out over twenty-four hours, they must remain alert to those circumstances that provoke symptoms. Once it is clear that certain loading, such as flexion, provokes symptoms, this activity is restricted and the opposite principle, in this case extension, is applied. This tactic is generally not used in patients with acute symptoms.

It is important to note that force alternatives may be necessary, with considerable variation needed in the degree and timing of loading strategies. Some patients require no more than posture correction and advice about maintaining this and about frequent interruption of the aggravating position. Often extension in standing is initially found to worsen symptoms, while extension in lying improves them. If patients present with symmetrical back pain and the deformity of kyphosis, too rapid or vigorous an application of extension force will worsen them. Initially they will not even tolerate prone lying; they will be fixed in flexion and need gradual exposure to the prone position over time before they can benefit from more advanced extension loading strategies.

On other occasions, patients with unilateral asymmetrical symptoms are unchanged or worsened by pure extension procedures, but respond when a lateral component is introduced. Thus different starting positions (loaded/unloaded), degrees of extension, loading strategies and the time factor should be considered when establishing the correct level at which to commence extension forces.

Force progressions and force alternatives may be necessary to establish directional preference and maintain improvements. Minimal intervention is always best – the simpler the intervention, the easier for the patient to accomplish themselves and the more likely they are to gain independence of management. Progression does not imply a necessary starting point; patients will enter the spectrum of treatment choices at different levels. Nor does it imply an absolute need to progress – many will respond at minimal levels and no progression of force is required. Force progression should sometimes be seen as force alternatives, and it is essential to establish the appropriate starting position.

Table 24.5 Force progressions and force alternatives in extension principle (procedures are described in Chapter 17)

Extension principle: force progressions

- lying prone (Procedure 1)
- lying prone in extension (Procedure 2)
- extension in lying (Procedure 5)
- extension in lying with patient overpressure (Procedure 5)
- extension in lying with clinician overpressure (Procedure 6a)
- extension mobilisation (Procedure 7)
- extension manipulation (Procedure 8).

Extension principle: force alternatives

- posture correction (Procedure 4)
- slouch overcorrect (Procedure 10)
- extension in standing (Procedure 9)
- sustained extension – only necessary in those fixed in flexion/ with kyphosis deformity (Procedure 3).

Extension principle with lateral component

- extension in lying with hips off centre (Procedure 11)
- extension in lying with hips off centre with clinician overpressure (sagittal or lateral) (Procedure 12)
- extension mobilisation with hips off centre (Procedure 13)
- rotation mobilisation in extension (Procedure 14)
- rotation manipulation in extension (Procedure 15).

Lateral principle

A proportion of patients need some degree of lateral forces. This will be in those with asymmetrical or unilateral back/leg pain. Indications for use of lateral forces or a combination of lateral force with extension relate to symptom response, to sagittal plane loading or the presence

of a lateral shift (Table 24.7). Clues as to the need for lateral force are found during the history-taking and the physical examination.

Two situations require lateral forces (Table 24.7). The more common is the relevant lateral component without a lateral shift. This is when sagittal plane forces either cause unilateral asymmetrical symptoms to worsen or cause no important change. The less common situation is the presence of a relevant lateral shift (Table 24.8).

Table 24.6 Indicators for consideration of lateral component

- Unilateral or asymmetrical symptoms, and 1 or 2:
  1. Indication for temporary abandonment of sagittal plane testing:
     - peripheralisation or worsening of peripheral symptoms in response to sagittal movements
     - non-response after full exploration of sagittal plane forces conducted over several days and/or including force progressions
  2. Indicators to avoid or defer testing of sagittal plane:
     - presence of clinically relevant lateral shift deformity.

In all instances sagittal movements may later be required.

In some patients the history-taking and early stages of the physical examination do not reveal a clear directional preference, although they may relate that symptoms are worsened by both flexion and extension activities. The need for lateral forces becomes apparent when loading strategies ape explored. Positions of extension or movements into extension cause a worsening or peripheralisation of pain – the need for lateral forces is predicted by the response to loading strategies. Often such patients respond to a combination of extension and lateral forces – that is, extension in lying performed with hips off centre.

In other patients, pure extension forces may not actually cause peripheral symptoms to increase and remain worse, but cause only an increase while being performed. Should this response fail to improve, but be repeated each time after multiple repetitions even with the inclusion of force progressions, a combination of extension and lateral forces would again be the next procedure of choice. Many such patients would, after a brief use of lateral forces, subsequently require pure sagittal extension.

In some a very apparent lateral shift or blockage to full movement is found – the patient may be fixed with their hips shifted to the right and be unable to reverse the curve. In such an example, the need for lateral forces is easily predicted in order to correct the shift. With the presence of a 'hard' deformity the patient is unable to achieve this alone, and clinician assistance is required. In some the obstruction to movement is 'soft' and yields to repeated patient-generated forces. In the presence of a lateral shift, pure lateral forces are used.

Table 24.7 Lateral shift – definitions

Lateral shift:

- upper body is visibly and unmistakably shifted to one side
- onset of shift occurred with back pain
- patient is unable to correct shift voluntarily
- if patient is able to correct shift, they cannot maintain correction
- correction affects intensity of symptoms
- correction causes centralisation or worsening of peripheral symptoms.

Right and left lateral shift:

- A right lateral shift exists when the vertebra above has laterally flexed to the right in relation to the vertebra below, carrying the trunk with it. The upper trunk and shoulders are shifted to the right.
- A left lateral shift exists when the vertebra above has laterally flexed to the left in relation to the vertebra below, carrying the trunk with it. The upper trunk and shoulders are shifted to the left.

Contralateral and ipsilateral shift:

- Contralateral shift exists when the patient's symptoms are in one leg and the shift is in the opposite direction – for instance, right leg pain with upper trunk and shoulders laterally shifted to the left.
- Ipsilateral shift exists when the patient's symptoms are in one leg and the shift is to the same side – for instance, right leg pain with upper trunk and shoulders laterally shifted to the right.

Table 24.8 Clues as to the need for lateral principle – not all will be present at once

History:

- unilateral or asymmetrical symptoms
- activities of both flexion and extension aggravate symptoms
- worse with standing and walking
- preference for side lying
- patient reports sudden recent onset of postural misalignment.

Continued next page

Physical examination:

- lateral shift – patient is locked in a position of lateral deformity and is unable to straighten, or if they correct they cannot maintain it (uncommon clinical presentation)
- significant difference between sides in frontal plane movement loss
- lateral movement is asymmetrical, with major loss in one direction
- symptoms centralise or are made better by lateral movements
- symptoms peripheralise or worsen with prone lying or extension in lying
- symptoms are overall unchanged after several days' application of extension protocol.

Lateral forces are described in the chapter on procedures according to the extension or flexion with which they are combined. Some procedures are primarily ones of extension combined with a small lateral component; others are primarily flexion procedures with a lateral component. These procedures are described in the extension and flexion principles in Chapter 17, and examples are given in columns two and three, Table 24.10. Pure lateral movements involve the side glide or shift correction procedures listed in column four. In each type of force, procedures may involve patient- or clinician-generated activity, or sometimes a combination of the two.

Table 24.9 Procedures used when a relevant lateral component is present

	*Extension/lateral**	*Flexion/lateral**	*Lateral**
Patient-generated force	Extension in lying with hips off centre (11)	Flexion in step standing (22)	Self-correction of lateral shift/side gliding (16)
		Rotation in flexion (23)	
Combination	Extension in lying with hips off centre with clinician overpressure (12)		
Clinician-generated force	Extension mobilisation with hips off centre (13)		
	Rotation mobilisation in extension (14)	Rotation mobilisation in flexion (24)	Manual correction of lateral shift (17)
	Rotation manipulation in extension (15)	Rotation manipulation in flexion (25)	

O/P = overpressure
* numbers refer to procedures in Chapter 17

Flexion principle

A smaller proportion of derangements improve with flexion forces. In a study that looked at centralisation response to sagittal plane movements (Donelson *et al.* 1991), 40% had a clear preference for extension and 7% preferred flexion.

Generally patients present only with back pain or back and anterior thigh or inguinal pain, without any neurological signs or symptoms. They may have symmetrical, asymmetrical or unilateral pain. As in all derangements, a continuum is present. There may be few distinguishing features denoting derangements that respond to flexion. If they are able to slouch, they are often better after sitting and slowly worsen on standing and walking. The history may be unclear.

On physical examination they may present with a severe loss of flexion, fixing them in lordosis so that they are unable to slouch or bend forward very far. Despite this major mechanical presentation, they have comparatively minor symptoms. Major losses of flexion are also found in posterior derangements; however, symptomatic presentation is greater, accompanied by a major loss of extension, and the obstruction to flexion is because of pain. In flexion principle patients or anterior derangements with a major loss of flexion, often the patient remarks that it is not so much pain that prevents the movement as it 'simply won't go'. Also there is usually a surprisingly well-maintained lordosis, even during lumbar flexion, and range of extension.

Often the clues in this group are gained during the single test movements. There may be a major loss of flexion, which appears inappropriate to the degree of symptoms and is limited by stiffness rather than pain, and does not fit with the well-maintained range of extension. In effect, there is a range of ways this group may present, and when the derangement is minor these clues may be missing. In this case, *ultimately symptomatic responses provide the justification for the loading strategy adopted.*

As in any situation when management strategy is unclear, the diagnostic pathway is often elucidated by repeat movement testing in one direction and observation of the mechanical effect on the opposite movement. Response to repeated extension may be equivocal, rather than causing a worsening of symptoms or causing the lordosis to lock in extension. The surest way to provoke both signs and symptoms and confirm an anterior derangement is by

sustaining the patient in end-range extension for four to five minutes. This will have the likely effect of provoking pain and will certainly cause an obstruction to flexion.

Table 24.10 Clues as to the need for flexion principle

History:

- worse with activities of extension
- pregnant or post-partum (possible risk factor)
- rear-end motor vehicle accident (possible risk factor)
- prone sleepers or sunbathers (possible risk factor).

Physical examination:

- accentuated lordosis
- lordosis maintained in attempting flexion in standing
- major loss of flexion
- no loss of extension
- deviation of flexion possible
- repeated or sustained extension causes or increases loss of flexion
- repeated flexion abolishes, reduces or centralises symptoms
- repeated flexion in lying may increase flexion range
- repeated flexion in sitting increases flexion range.

Derangements requiring flexion principle with symmetrical symptoms requires pure sagittal procedures. If symptoms are asymmetrical or unilateral, especially if deviation in flexion is present, flexion procedures with a lateral component may be required.

Table 24.11 Force progressions and force alternatives in flexion principle (procedures are described in Chapter 17)

Flexion principle – force progressions:

- flexion in lying (Procedure 18)
- flexion in sitting (Procedure 19)
- flexion in standing (Procedure 20)
- flexion in lying with clinician overpressure (Procedure 22).

Flexion – force alternatives:

- flexion posture correction – slouched sitting.

Flexion principle with lateral component:

- flexion in step standing (Procedure 22)
- rotation in flexion (Procedure 23)
- rotation mobilisation in flexion (Procedure 24)
- rotation manipulation in flexion (Procedure 25).

Irreducible derangements

In patients with constant sciatica and signs and symptoms of interference with nerve root integrity, a poor response is common. They may also have a lateral shift or sciatic scoliosis that responds to attempts at correction only by aggravation of referred symptoms. Once again, aspects of the history and physical examination will expose clues to prognosis (Table 24.13).

Although an irreducible derangement may be suspected on day one, this failure to respond must be confirmed over several sessions. Sometimes a patient appears with what initially seems to be a poor prognosis, but on re-examination demonstrates a favourable response. *The patient with suspected irreducible derangement must always be examined over at least three sessions to confirm this classification.*

Table 24.12 Clues to irreducible derangements – not all need be present

History:

- patient reports truly constant symptoms that extend to foot
- patient reports constant paraesthesia or numbness
- patient reports leg pain with no back pain
- patient reports no position relieves pain
- patient reports back pain ceased with the onset of leg pain.

Physical examination:

- patient displays weakness in relevant myotome
- patient displays gross loss of all movements
- patient displays lateral shift – patient is locked in a position of lateral deformity and is unable to straighten or maintain correction
- all attempts to correct lateral shift result in worsening of peripheral symptoms
- all loading strategies result in worsening of peripheral symptoms
- no movement or position reduces or centralises.

Conclusions

This chapter has described the principles of management involved in patients with a derangement. The stages of management involve reduction of the derangement, maintenance of reduction, recovery of function and prophylaxis. Reduction of derangement is achieved using the directional treatment principles of extension, lateral, flexion

or a combination of these therapeutic loading strategies. There will be occasions when all loading strategies fail to reduce, abolish or centralise symptoms from a derangement, and in such instances the derangement is irreducible.

Subsequent chapters describe the management and treatment of derangements in more detail according to the site of symptoms. The description of derangement management will be based on two aspects of the symptomatic presentation. First consideration is given to the central or lateral nature of symptoms, and then the referral of symptoms is considered.

25: Management of Derangements – Central Symmetrical

(Previously Derangements 1, 2, 7)

Introduction

Previous chapters described the conceptual model, the distinguishing characteristics of derangement, their typical presentations and the general principles involved in management. When a derangement is present, the key clinical issue is to detect movements or positions that cause symptoms to be decreased, abolished or centralised. These therapeutic loading strategies occur in purely sagittal or frontal plane movements, or some combination of the two.

Management is considered relative to certain pain patterns; namely, the symmetrical or asymmetrical nature of the pain, and secondly the degree of referred or distal pain in those with asymmetrical symptoms.

Sections in this chapter are as follows:

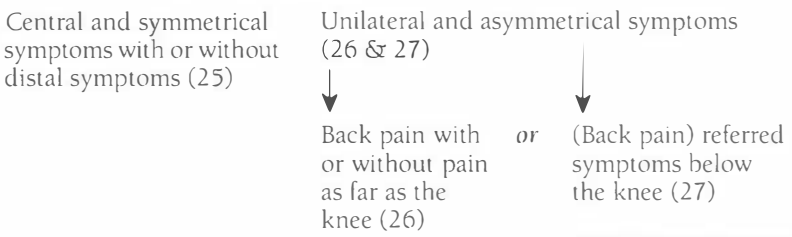
- treatment pathways in derangement
- management of derangement – central/symmetrical pain
- extension principle – history and physical examination
- extension principle – management guidelines
- extension principle – review
- flexion principle – history and physical examination
- flexion principle – management guidelines
- flexion principle – review.

Treatment pathways in derangement

The first consideration is the location of pain and the next is the extent of distal symptoms. This is decided by the patient's report of present symptoms, which is defined as the pain that is their present problem. This applies to all symptoms being experienced, even if not actually present at the time of assessment. There are two groups of patients: those with central or symmetrical pain and those with unilateral or asymmetrical symptoms. In those with symmetrical symptoms, there may also be referral of symptoms into the thighs or legs. In those with asymmetrical or unilateral back pain, there may

be referral of symptoms as far as the knee or below the knee – this may include paraesthesia. Management is described in terms of these three groups (Figure 25.1). This should include all pain patterns; however, if the patient's description of their pain does not clearly fit one of these patterns, consideration should be given to the closest equivalent.

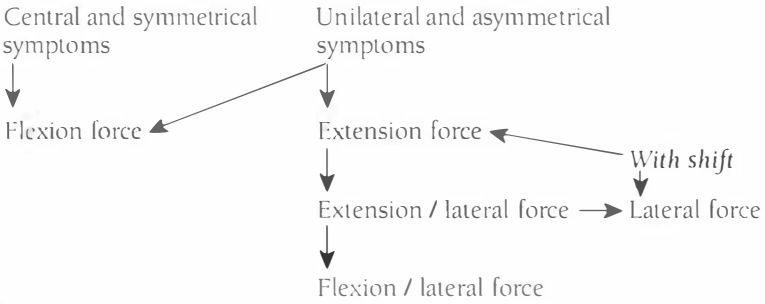
Figure 25.1 Derangement – management considerations (relevant chapter)



Patients with central symmetrical symptoms are those previously classified as derangements 1 and 2. Patients with unilateral asymmetrical symptoms as far as the knee are those previously classified as derangements 3 and 4. Patients with unilateral asymmetrical symptoms below the knee are those previously classified as derangements 5 and 6 (McKenzie 1981). Patients previously classified as derangement 7 will be found in the first two categories.

If symptoms are central or symmetrical across the back, then management is nearly always in the sagittal plane, principally involving extension, and occasionally flexion. Sometimes patients in this group have distal symptoms. As long as these are relatively equal bilaterally, management is conducted in the sagittal plane. Broadly speaking, with the presence of more distal and unilateral symptoms, it is likely that non-sagittal treatment procedures may be required. However, as many in this group will respond to sagittal plane forces, this plane is always explored first. Failure to respond or unfavourable symptom response means other loading strategies are explored – first a combination of extension and lateral forces, then other lateral forces. Symptom response always decides management. If a clinically relevant lateral shift is present,t$hen attention focuses on the lateral direction immediately. Response to loading strategies is dynamic and can change during an assessment or episode of treatment. The clinical reasoning pathways in derangement according to the different treatment principles are shown in Figure 25.2.

Figure 25.2 Derangement treatment principles and symptoms



Management of derangements – central/symmetrical pain

Patients' symptoms will be central or symmetrical across the back, and may include radiating symptoms equal bilaterally into both buttocks. This group involves those patients formally classified as derangements 1 and 2 (McKenzie 1981). Occasionally symptoms may be referred down both thighs and legs – as long as these are relatively equal bilaterally, they are still considered to have symmetrical symptoms. This is a non-specific somatic disorder (Chapter 3 for pain terms). Most derangements in this category will respond to the extension principle, with a small minority responding to the flexion principle.

Extension principle – history and physical examination

Aspects of the history often provide clues about the effect of different loading patterns (Table 24.5). For instance, the activity at the time of onset may have involved flexion – the patient may describe a lifting incident, or pain arising after prolonged sitting or driving. The aggravating and relieving activities may indicate that flexion activities produce or worsen the pain, while activities of extension reduce or abolish it. Problems with curve reversal after prolonged sitting or bending are an indicator of the unfavourable effect of prolonged flexion. Thus history may often be more indicative of directional preference than the effect of a small number of repeated movements.

After prolonged sitting, restoring the lordosis may centralise, reduce or increase the pain. The lordosis is usually reduced. In extreme cases the patient may have been forced to adopt the deformity of kyphosis.

This is a particular but uncommon posterior derangement and is discussed below in the section on *Kyphotic deformity*.

The response to repeated flexion may be increased and worsened symptoms. They may radiate further from the spine, or extension movement may be made more restricted and more painful. However, repeated movements performed in the clinic may only cause the symptoms to increase, not worsen. As mentioned above, in such cases the effect of prolonged sitting, as learned during the history-taking, may be more indicative of the appropriate management.

In some instances patient's pain may centralise, decrease or abolish with extension in standing. If this is the case, this procedure should be used in management as it is easy to perform during normal daily activities or when at work. However, it is not unusual for extension in standing to cause symptoms to increase temporarily, or even worsen. It is useful to compare this to the response after the performance of extension in lying. If easier, it is further confirmation of the appropriateness of this strategy.

If symptoms are severe and constant, and/or if there is an initial increase in symptoms when the patient assumes the prone lying position, then prone lying and prone lying in extension are always necessary procedures to be used before extension in lying is attempted. These procedures are sometimes more important and effective than extension in lying, at least initially. In these instances a time factor exists, which is important in allowing the restoration of extension. These procedures are particularly important where time is a factor in the production of symptoms, so time is a factor in resolution.

Symptoms that are present initially on lying prone, but which slowly abate and eventually cease altogether, give a strong indication of the need for extension. In response to repeated extension in lying, often symptoms centralise, decrease or abolish during the initial clinical visit, or end-range extension produces pain, which is decreased and then abolished on repetition, and/or the mechanical response may improve. The range of extension might increase, or a movement in standing that was painful or obstructed can be re-tested for change. With the other clues gained during the history and physical examination, these responses all confirm that the extension principle is appropriate.

In some instances, despite many indicators that a patient requires the extension principle, their initial response to repeated extension is unclear. Pain is increased, not worsened, or pain is decreased, but does not remain better (see Chapter 16 for terms used in symptom response). Very often in such instances a beneficial response to the extension principle is confirmed at review twenty-four to forty-eight hours later, and a clear improvement in symptomatic or mechanical presentation is noted. This confirmation of classification at review is quite usual. Much can be achieved using patient-generated forces in the first few days, and in this period the patient can realise and experience the extent to which self-management can improve the problem. ***Therefore it is generally undesirable to use clinician-generated forces during the first session.***

However, if confirmation of the appropriateness of the extension principle is required, the addition of overpressure may do this. Overpressure can be applied in prone lying or during extension in lying. When this pressure is applied, the patient is asked, *'With more pressure, is there more pain or less pain?'* A 'less pain' response to this question confirms the appropriateness of the extension principle for both the clinician and the patient. After either procedure it is also useful to re-examine their response to extension in lying.

It is sometimes the case that following the evaluation the patient experiences a return of symptoms within ten to fifteen minutes. After decrease or abolition of symptoms, it is sometimes useful to make the patient walk about in the corridor and monitor the symptoms. If symptoms recur, the reductive process should be repeated, and care should be taken that the patient maintains the lordosis as they get up from the plinth. If symptoms do recur, an assessment should be made of the effects of repeating extension in lying over the following twenty-four hours. Repeated every two hours while awake over this period, a more stable reduction may occur and the patient report that symptoms remain improved following each exercise session. This twenty-four-hour response confirms the mechanical diagnosis and treatment programme.

Extension principle – management guidelines

Photo 100: Posture correction.

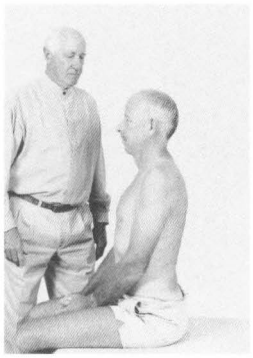


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Photo 101: Extension in standing.

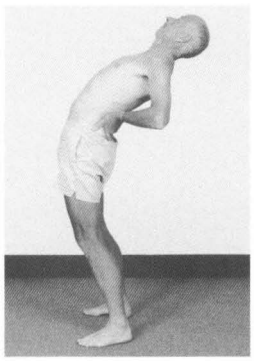

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Photo 102: Lying prone.

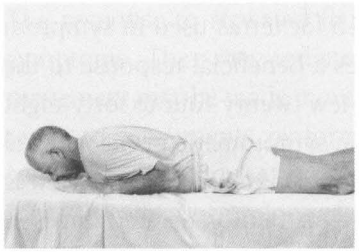

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Photo 103: Lying prone in extension.

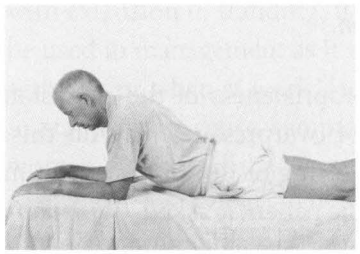

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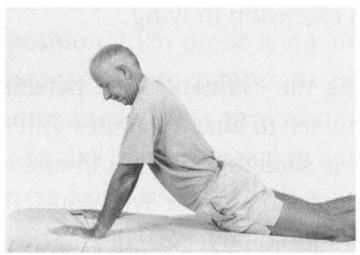
Photo 104: Extension in lying.



104

Procedures that may be needed in first few days are listed below.

- Lying prone (Procedure 1) – when symptoms present, should always be performed; may be omitted if not present. This is an essential procedure when a time factor has occurred in the development of the symptoms – pain may initially be present in this position, but then reduce or disappear over four to five minutes
- Lying prone in extension (Procedure 2) – may be recommended as a position of rest, for instance for reading or watching television as an alternative to lounging on the sofa. This is an essential procedure when time factor is important – pain may initially be present in this position, but then reduce or disappear over four to five minutes
- Extension in lying (Procedure 5) – essential
- Maintenance of lordosis is important as the patient regains the upright posture (see Procedure 5)
- Posture correction (Procedure 4) – essential
- Extension in standing (Procedure 9) – may be useful.

Regularity:

- Ten times every 2 – 3 hours

Expected response:

- centralisation, decrease, abolition of pain
- possible temporary increase of pain centrally
- increase in range of movement extension, flexion and side gliding
- may cause temporary new pains.

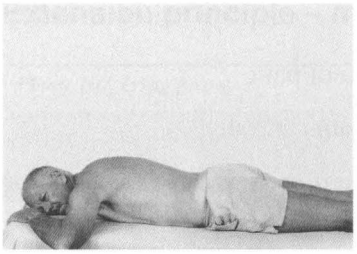
Kyphotic deformity

In all instances of those patients requiring the extension principle, there is an obstruction to extension. This obstruction to range of extension is on a continuum from minor obstructions that are easily reduced to major obstructions that prevent the patient from even adopting an upright posture. In between are those with a flat rather than a lordotic lumbar spine, who initially find it difficult to tolerate the prone lying position.

Those with a kyphotic deformity are forced to adopt a flexed posture and cannot tolerate any attempts to resume an extended position. Any attempt to do so results in severe pain and the patient is forced to return to the flexed position in which temporary relief is found. This is one of the instances in which the patient provides contradictory or 'false' testimony concerning the effects of loading or directional preference. Such a patient gives the impression that they have a directional preference for flexion. This ignores the key mechanical finding of a major obstruction to extension. They must be managed in a particular way, for if handled incorrectly the condition can be made worse and produce peripheral symptoms.

Unloading and time are the key treatment factors in these derangements, and only a very gradual resumption of extension will be tolerated. Initially the patient must be positioned prone in a way that accommodates the deformity; that is, in slight flexion. This can be done over pillows or on a plinth. By changing the angle of the plinth or removing pillows, the prone lying position is gradually regained. Once this is achieved, then active movements to increase the range of extension can be attempted. If this cannot be tolerated, extension must be increased more slowly and passively. This is done by steadily raising the end of the plinth. As the process is usually lengthy and painful, regular resumption and brief rests in the prone lying position are often necessary. Time and a careful monitoring of the symptomatic response are essential for this procedure.

Photos 105, 106, 107: Allow several minutes to pass before making the next progression (105). Raise the end of the couch in small increments. Allow symptoms to guide progress (106). Having regained this amount of extension, do not progress further (107). This is sufficient to enable the patient to self-treat.



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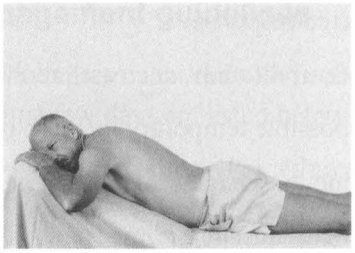

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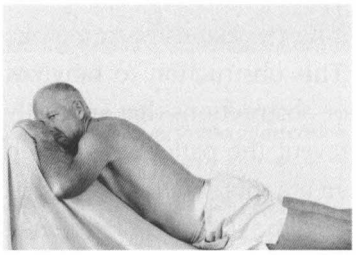


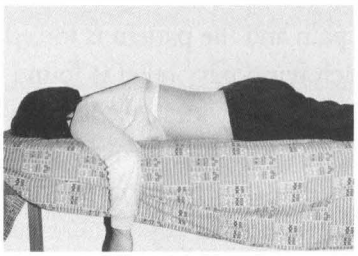
107

Procedure:

- Sustained extension (Procedure 3).

See Chapter 17 for a detailed account of this procedure.

Photos 108, 109, 110, 111: Lying prone (108). Lordosis restored (109). Lateral view (110). Close up (111).



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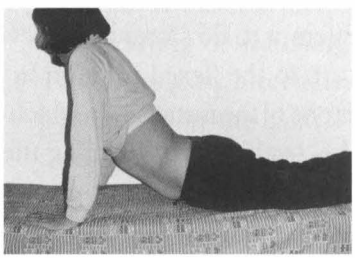

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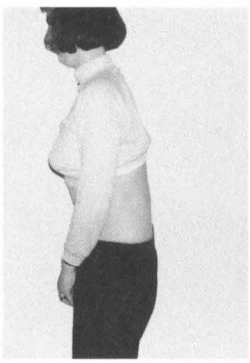

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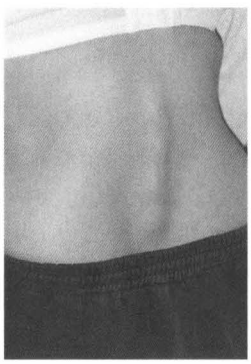

111

Extension principle – review

When the patient returns for review they will be improved, worsened or unchanged. Ensure that the status reported by the patient is their true state. Patients may report their symptoms to be worse when wide-spread pain has centralised to the middle of the spine; they may report themselves to be unchanged when in fact pain that was constant has become intermittent. Some patients, keen to please and to get better, report an improvement that is difficult to confirm. See Chapter 16 for details of how to analyse clinical presentations and Chapter 19 for the structure of a review process.

Patient is better

If there is improvement in the symptomatic and/or mechanical response at review, management strategy should not be changed. It is unnecessary to supplement present procedures with any other techniques or interventions if the patient is getting better. Management continues in the same way unless there is a change in status.

Patient is worse

There are certain instances that the patient may interpret as being 'worse', which we would not consider as such. When centralisation occurs there can be an accompanying and temporary increase in central pain. When patients with longstanding derangements and an associated obstruction to extension commence the necessary extension principle procedures, there can sometimes be an initial temporary increase in symptoms. The patient may be performing the procedures incorrectly, or may have misinterpreted instructions and be doing different exercises. New pains may have appeared in the upper back or arms as a consequence of performing the exercises, which has made the patient reluctant to continue. Do their symptoms improve with the exercises, but get worse later because of insufficient attention to posture? Did they initially get better with the instructions, but then worsened because they increased their activity level too soon? All these instances should not be considered a worsening scenario.

If really worse, pain is usually more widespread. It should be ensured that the patient definitely has symmetrical symptoms. If a condition is truly worsening, the patient should be advised to stop the exercises – patients sometimes improve with this step. If unequivocally worse, their response to flexion principle should be explored. If there is still a worsening response to all procedures, non-mechanical conditions should be considered, or see *Mechanically Inconclusive* (Chapter 13).

Patient is unchanging

First it should be ensured that the patient is performing the right exercises correctly and with enough regularity and that they are abiding by postural correction. If this is not the case, further instruction and discussion is necessary to ensure that the patient understands the procedures and their own role in managing the problem. It is best to see the patient daily until certainty of management is established and the patient is confident about their management strategy. If the patient cannot be reviewed regularly, this can create problems. In such cases, attempts to review by telephone are desirable.

If they have been following the treatment principle correctly, but still no improvement has occurred, then force progressions should be implemented. These are done in the order demonstrated in the photographs, with subsequent force progressions applied only if there is failure to improve. If at any point improvement does occur, further progression is unnecessary. Whatever progression is used, the patient must continue to perform the appropriate exercises at home with suitable regularity. Following a force progression, the effects of this procedure should be evaluated at the next review. Force progressions can be repeated on up to two occasions before they should be abandoned if no change ensues. Do not instigate clinician techniques unless it is clear that improvement cannot be achieved by any other means.

Photo 112: Extension in lying with clinician overpressure.

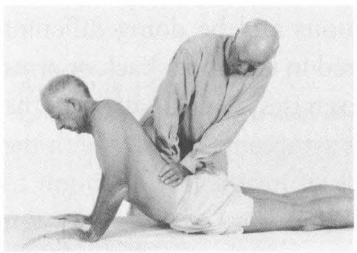

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Photo 113: Extension mobilisation. Rhythmical pressure.

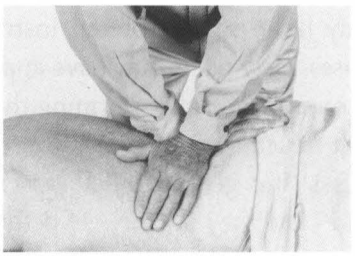

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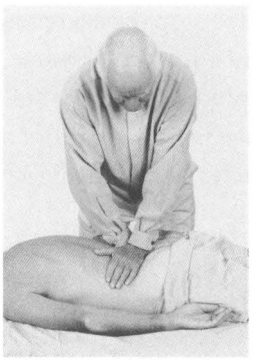
Photo 114: Extension manipulation. High velocity, short-amplitude thrust.



114

Progressions:

- extension in lying with clinician overpressure (Procedure 6a)
- extension mobilisation (Procedure 7)
- extension mobilisation further into physiological range of extension
- extension manipulation (Procedure 8).

This last progression is only applied if the effect of extension mobilisation is to decrease symptoms, but with no lasting improvement – see Chapter 17 for full description of use.

Flexion principle – history and physical examination

The history provides few definite clues as to the need for the flexion principle (Table 24.7). The patient is likely to relate that they are worse walking and standing, and have obstruction and increased pain on

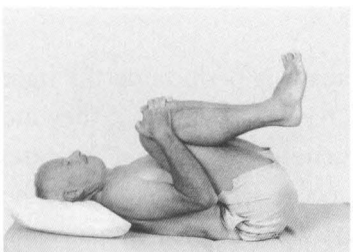
bending forward when standing or sitting. They often improve during prolonged periods of sitting. Sometimes these patients are fixed in lordosis even when they sit, and they are unable to find a relieving position.

The distinguishing characteristics of anterior derangements are detected during the physical examination. The lordosis is accentuated, often fixed in extension, thus causing a major loss of flexion. The patient may be unable to slump when sitting, and even when they bend forward, the lumbar spine remains lordotic. Major losses of flexion are also common in posterior derangements requiring the extension principle – certain features differentiate the two. When the flexion principle is appropriate, symptoms are generally less severe and when flexion is attempted, often patients remark it is not pain that stops them, they are simply unable to bend. The key difference is range of extension, which in patients with anterior derangement is well maintained.

If the symptom response is unclear, the mechanical response to repeated movements or sustained positions usually provides convincing proof of the directional preference. This can be evaluated in two ways. First, during flexion in lying there is often an improvement in range. Following the performance of repeated flexion in lying, re-assessment of flexion in standing often shows a dramatically improved range. Secondly, if the patient is positioned in sustained end-range extension for two or three minutes, this provocative test procedure causes a dramatic reduction of their range of flexion.

Flexion principle – management guidelines

Photo 115: Flexion in lying - applying overpressure.



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Procedure:

- flexion in lying (Procedure 18) – it is common that flexion in lying ceases to be painful during first or second cycle of ten. If that happens immediately, progress on to flexion in sitting, which is easier to perform during normal activities.

Photo 116: Flexion in sitting.

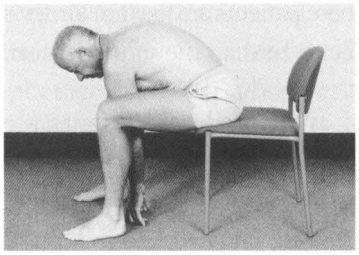

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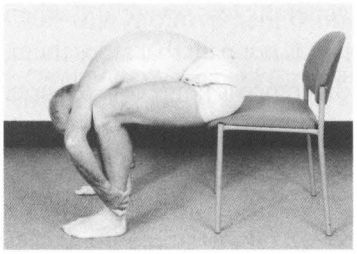
Photo 117: Flexion in sitting with patient overpressure.



117

- flexion in sitting (Procedure 19).

Regularity:

- ten times every 2 – 3 hours.

Flexion principle – review

When the patient returns for review, they will be improved, worsened or unchanged. Ensure that the status reported by the patient is their true state. See Chapter 13 for a detailed analysis of assessment and the review process.

Patient is better

If there is improvement in the symptomatic and/or mechanical response at review, management strategy should not be changed. It is generally the case, however, that flexion in sitting and standing will be needed for full reduction.

Patient is worse

If really worse, pain is usually more widespread. It should be ensured that the patient definitely has symmetrical symptoms. If a condition is truly worsening, the patient should be advised to stop the exercises – patients sometimes improve with this step. If unequivocally worse, their response to extension principle should be explored. If there is still a worsening response to all procedures, non-mechanical conditions should be considered.

Patient is unchanging

First it should be ensured that the patient is performing the right exercises correctly and with enough regularity and that they are abiding by appropriate postural instruction. If this is not the case, further instruction and discussion is necessary to ensure that the patient understands the procedures and their own role in managing the problem. It is best to see the patient daily until certainty of management is established and the patient is confident about their management strategy. If the patient cannot be reviewed regularly,

this can create problems. In such cases, attempts to review by telephone are desirable.

If unchanging and the patient stands for prolonged periods, they should be advised to bend forward frequently: 'Tie your shoe laces every ten minutes'. If they have been following the treatment principle correctly, but still no improvement has occurred, then force progressions should be implemented.

Progressions – usually required after a few days:

- flexion in sitting (Procedure 19) – if not already introduced on day one
- flexion in standing (Procedure 20)
- flexion in lying with clinician overpressure (Procedure 21).

Photo 118: Flexion in sitting.

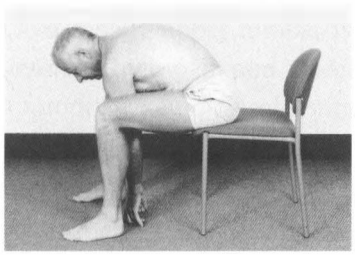


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Photo 119: Flexion in sitting with patient overpressure.

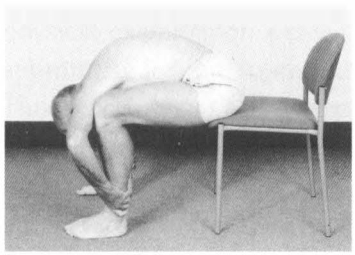

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Photo 120: Flexion in sitting with knee extension.

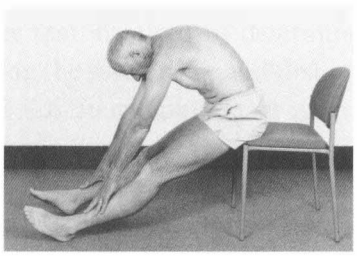

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Photo 121: Flexion in lying with clinician overpressure.

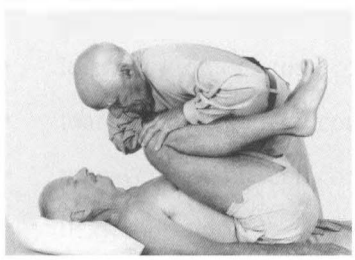

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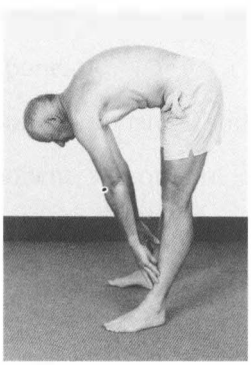
Photo 122: Flexion in standing.



122

26: Management of Derangements – Unilateral Asymmetrical Symptoms to Knee

(Previously Derangements 3, 4, 7)

For overview of treatment pathways in derangement, see first section in Chapter 25 and Figures 25.1 and 25.2.

Patients in this category have unilateral or asymmetrical back pain; distal or referred symptoms may also be present, possibly extending as far as the knee. This group mostly encompasses patients with non-specific somatic pain in which the pain-generating mechanism may cause limited referral of symptoms (see Chapter 3 for pain definitions). Occasionally upper lumbar nerve root problems may present with anterior thigh pain and associated paraesthesia and weakness, and appropriate neurological testing is then necessary. This group involves those patients formally classified as derangements 3, 4 and 7 (McKenzie 1981).

The key decision in all those with unilateral asymmetrical symptoms is determining the appropriate loading strategy – sagittal or with a lateral component (Figure 25.2). There will be clues in the history (Tables 24.5 and 24.9). The first section of this chapter details the physical examination procedures that are used to determine the appropriate loading strategy during the assessment and first review. This details the way to determine if a lateral component is present. The criteria for identifying a lateral component are then reviewed. The management of patients with a lateral component, including force progressions and alternatives, is then detailed – this is relevant to those with symptoms to the knee and below the knee (Chapter 27).

Sections in this chapter are as follows:

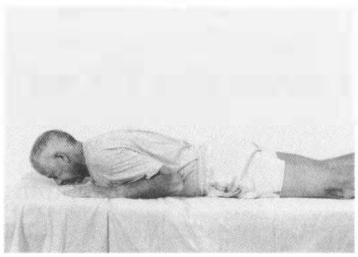
- assessment – determining the appropriate strategy
- identification of lateral component
- management – lateral component, no lateral shift
- management – lateral component, soft or hard lateral shift
- flexion principle.

Assessment – determining the appropriate strategy

Many patients with unilateral or asymmetrical symptoms respond or begin to respond to extension exercises performed as described for those with symmetrical pain. However, if there is not an early response to extension, then the lateral component should also be explored. Worsening of symptoms in response to extension leads to an early introduction of lateral forces.

Management using the extension principle is exactly as described above. The same starting procedures and force progressions, if needed, are used. Procedures applied on day one:

Photo 123: Lying prone.



123

Photo 124: Lying prone in extension.

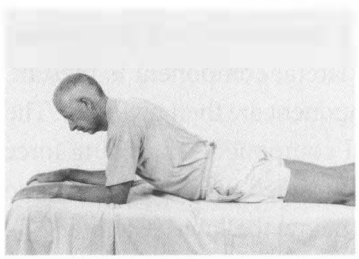


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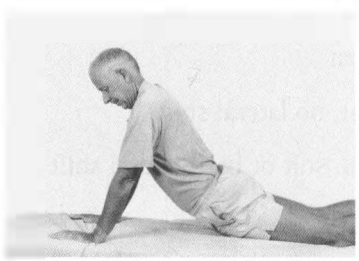
Photo 125: Extension in lying.



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- Lying prone (Procedure 1) – when symptoms are present, should always be performed; may be omitted if symptoms are not present. This is an essential procedure when time has been a factor in the onset of the symptoms. Pain may initially be present in this position, but then reduce or disappear over four to five minutes.
- Lying prone in extension (Procedure 2) – should be recommended as a position of rest; for instance for reading or watching television as an alternative to lounging on the sofa. This is an essential procedure when time has been a factor in the onset of the symptoms. Pain may initially be present in this position, but then reduce or disappear over four to five minutes.
- Extension in lying (Procedure 5) – essential.

Photo 126: Posture correction.

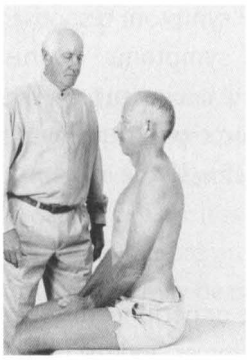

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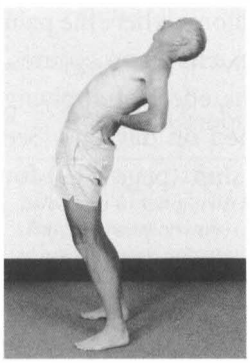
Photo 127: Extension in standing.



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- Posture correction (Procedure 4) – essential.
- Extension in standing (Procedure 9) – may be useful.

Regularity:

- ten times every 2 – 3 hours

Expected response:

- centralisation, decrease, abolition of pain
- may temporarily increase pain centrally
- increase in range of movement extension and flexion
- exercises may cause a new pain or strain.

Response to extension

In response to extension forces, unilateral symptoms may respond in one of three ways, each with different management implications (Table 26.1). They will either be better, worse or unchanged.

Table 26.1 Response to extension forces in unilateral asymmetrical and implications

Response to extension forces	*Implications*
Decrease pain	Continue with extension forces
Abolish pain	
Centralisation	Continue with extension forces
Increase distal pain	Introduce lateral component
Peripheralisation	See *Management – lateral component, no lateral shift* for procedures (page 610)
Indeterminate response Increase, not worse	Progress forces and explore lateral component and then decide on the most appropriate loading strategy

Better

In the first instance, it is very apparent that extension forces are appropriate because there is a rapid favourable symptom response, with decrease, abolition or centralisation of symptoms. In this situation, management would be conducted according to the extension principle, including any necessary force progressions, as long as improvements continued (Chapter 25). If the response changed, a review would be necessary.

Worse

Likewise, in the second instance a rapid increase or peripheralisation warns of the inappropriateness of pure extension forces, and the lateral component is introduced. A relevant lateral component has been determined from symptomatic response.

In patients with asymmetrical or unilateral symptoms where the pain is worse as a result of the initial assessment with extension procedures, or has increased laterally or peripheralised, the effect of applying forces to the lateral compartment is determined on day one. See *Management – lateral component, no lateral shift* (page 610) for procedures that may be considered.

Unchanging

When the symptom response is indeterminate, the management strategy is less clear. In these instances it is important to compare symptom response to sagittal movements to the symptom response of movements applied to a lateral component. This determines if there is a clear directional preference. Determining the best strategy requires applying a clinical reasoning process. Overpressures and mobilisation in both planes may be considered on day one to help determine the appropriate loading.

Extension

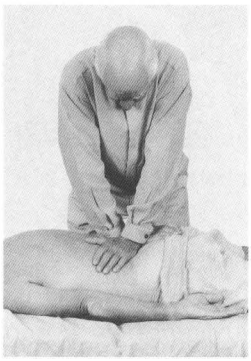
If, following force progressions in extension, purely sagittal procedures cause peripheralisation or worsening of distal symptoms, exploration of the lateral component must ensue.

Overpressure may be applied in extension to see if this generates a favourable response. The patient lies prone and relaxed; therapist hand placement is as for extension mobilisation (Procedure 7) and a gradual slow increase of extension pressure by weight transference is made until the patient is able to identify a response. 'When I apply more pressure, do you feel more pain, less pain, or is there no effect?'

A favourable response indicates extension, otherwise the lateral component should be explored.

If testing extension is inconclusive, determine if more pressure causes more or less pain. Apply an extension overpressure. If more pressure causes less pain, progressions are indicated. If more pressure causes more pain, other levels may be tried or force alternatives may be required.

Photo 128: Extension overpressure.



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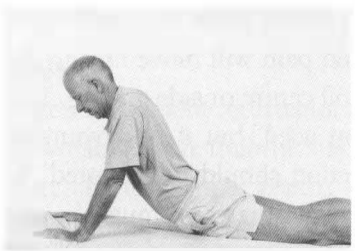
To further explore the sagittal plane, one may progress through:

- extension in lying with patient overpressure (Procedure 5)
- extension in lying with clinician overpressure (Procedure 6a)
- extension mobilisation (Procedure 7).

Photos 129, 130, 131:
Extension in lying with patient overpressure (129). Extension in lying with clinician overpressure - if more pressure produces less pain, extension force is used. If more pressure causes more pain, another level or the lateral component is explored (130). Extension mobilisation (131).



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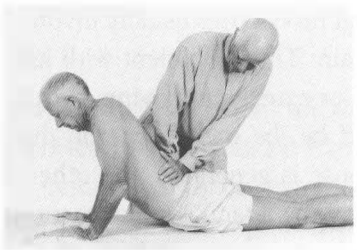

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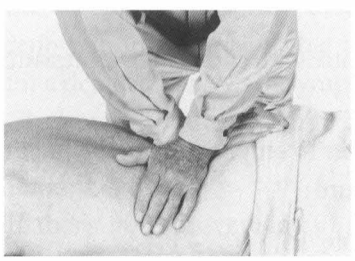

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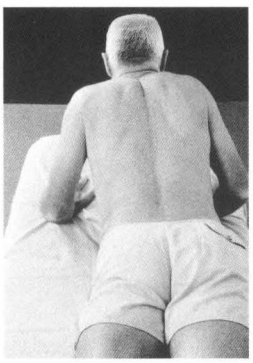
Photos 132, 133, 134, 135, 136, 137:
Extension in lying with hips off centre – when extension in lying is inconclusive. Hips are most commonly shifted away from the pain (132). Extension in lying with hips off centre with extension overpressure (133). Extension in lying with hips off centre with lateral overpressure – to increase lateral force, hold patient's hips shifted to one side (134). Side glide in standing – alternatively, apply lateral force in standing (135). Rotation mobilisation in extension – pressure on alternate sides (136). Rotation mobilisation in extension – unilateral pressure on appropriate side (137).



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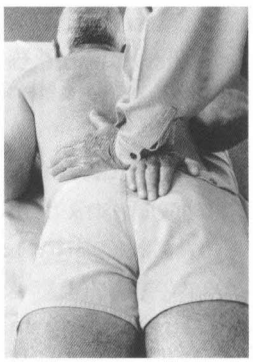

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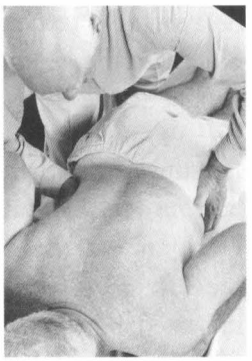

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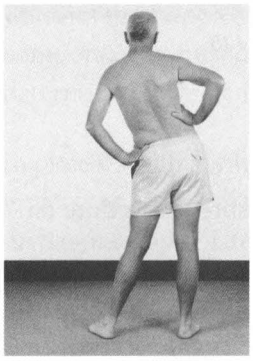

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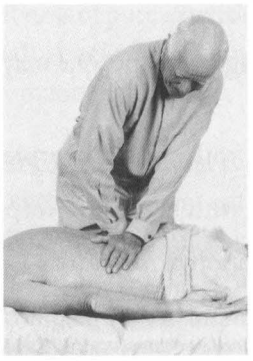

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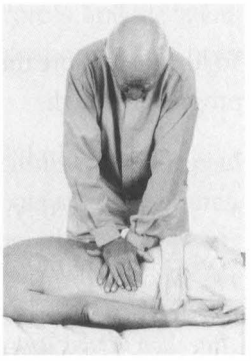

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Lateral

Lateral procedures usually involve movement of the hips away from the pain. Thus, a patient with left-sided pain will move hips to the right for extension in lying, with hips off centre or side gliding. This would be the most common direction used, but if no favourable response is generated, the other direction should be explored. To explore the lateral compartment, one may progress through:

- extension in lying with hips off centre (Procedure 11), usually shifted away from the pain
- extension in lying with hips off centre with clinician overpressure (Procedure 12)
- overpressure may be applied either to emphasise the sagittal or lateral component of the procedure
- side gliding in standing (Procedure 16)
- extension mobilisation with hips off centre (Procedure 13)
- rotation mobilisation in extension (Procedure 14)
- following use of lateral forces symptoms may centralise completely, and pure sagittal plane forces are then reconsidered.

Depending on symptomatic and mechanical responses, either extension in lying (Procedure 5) or extension in lying with hips off centre (Procedure 11) is given as the home exercise, with appropriate postural correction.

Those patients who report a reduction or abolition of pain during activities of extension, such as walking (Table 24.5), may indicate in their history a positive response to extension forces. A trial of posture correction with extension in lying for twenty-four hours may generate a clearer symptomatic response than several sets of repeated movements. Likewise, other patients may report a worsening of symptoms to both flexion and extension forces in both sitting and walking (Table 24.9).
revealing than repeated movements.

In summary, for patients with unilateral asymmetrical symptoms who do not show immediate benefit using extension forces, the lateral component should always be explored. Overpressures and even mobilisation can be used to help determine the appropriate loading strategy. In many patients, the addition of the lateral component or force progressions clarifies the preferred loading. Sometimes, however, a trial over twenty-four hours is more revealing than repeated movements during the clinical examination.

Review

When the patient returns for review, they will be better, unchanged or worse. Ensure that the status reported by the patient is their true state. Patients may report their symptoms to be worse when widespread pain has centralised to the middle of the spine; they may report themselves to be unchanged when in fact pain that was constant has become intermittent. Some patients, keen to please and get better, report an improvement that is difficult to confirm. See Chapter 16 for a detailed analysis of symptomatic and mechanical responses and Chapter 19 for the structure of a review appointment.

Patient is better

If there is improvement in the symptomatic and/or mechanical response at review, management strategy should not be changed. It is unnecessary to supplement present procedures with any other techniques or interventions if the patient is improving. Management continues with the same loading unless there is a change in symptomatic or mechanical responses.

If patient has been using extension in lying with hips off centre (Procedure 11) or other lateral force, and symptoms have centralised, use of pure sagittal plane force is reconsidered.

Patient is unchanging

If no centralisation or clear improvement has occurred by the review and the patient has been compliant, then force alternatives and progressions should be considered as outlined above. This may involve sagittal plane procedures and procedures with a lateral component. It will definitely involve overpressure and therapist mobilisation in order to determine the appropriate loading strategy. Some of these should already have been applied on day one. At all times a clinical reasoning process is used, and the clinician is alert to an unfavourable response that means a procedure should be abandoned at that time.

Force progressions in the sagittal plane are as indicated earlier. Lateral force progressions are as indicated in the section below: *Management – lateral component, no lateral shift*.

It is important that the sagittal plane is not abandoned prematurely. Sagittal plane procedures should always include the use of sustained positioning, and unless there is a clear worsening of symptoms, overpressure and mobilisation. Worsening means that symptoms are peripheralised or that distal symptoms remain worse; worsening is not indicated by a temporary increase in response to a limited number of repeated movements. Equally, if there is no lasting response to extension forces only, a lateral component should be introduced at an early point. See below: *Management – lateral component, no lateral shift*.

Patient is worse

When centralisation occurs there can be an accompanying temporary increase in central pain. When patients with longstanding derangements and an associated obstruction to extension commence the necessary extension principle procedures, there can sometimes be an initial temporary increase in symptoms. The patient may be performing the procedures incorrectly or may have misinterpreted instructions and be doing different exercises. New pains may have appeared in the upper back or arms as a consequence of performing the exercises that make the patient reluctant to continue. These instances should not be considered a worsening scenario.

In patients with asymmetrical or unilateral symptoms, a worse scenario is indicated when symptoms have changed from intermittent to constant, increased in intensity, increased laterally or peripheralised. Lateral forces or a combination of extension and lateral forces may be required. Pure extension forces should no longer be used unless there is a change in symptom response. If extension/lateral procedures have been used, flexion/lateral procedures may be considered with caution. See below, *Management – lateral component, no lateral shift*.

Identification of lateral component

This section reviews the clinical presentation that may indicate that a relevant lateral component is present; these have already been considered in Chapter 24.
examination provide clues as to the possibility of a relevant lateral component (Table 24.9):

- unilateral or asymmetrical symptoms
- activities of both flexion and extension aggravate symptoms
- lateral movement is asymmetrical, with major loss in one direction
- symptoms centralise or are made better by lateral movements
- symptoms peripheralise, worsen or are unchanged with prone lying or extension in lying
- symptoms are overall unchanged after several days' application of extension protocol.

Identification of a relevant lateral component, which requires the lateral principle, occurs in three ways:

1. Peripheralisation or worsening of peripheral symptoms in response to extension in lying, or a force progression in the extension principle. As long as sufficient time was allowed for extension procedures and force progressions were not introduced too rapidly.
2. Non-response to sagittal plane forces. When there is no benefit from extension procedures, the lateral component should be explored. It is important to assess the response to lateral forces early when this seems appropriate. Equally, it is important on other occasions to make sure that the sagittal plane is not

abandoned prematurely and that an extended mechanical evaluation with force progressions is conducted.

3. Presence of lateral shift deformity:
    - The 'soft' lateral shift – the lateral shift will have accompanied the recent onset of back pain. The patient presents with a very visible lateral deformity that they are initially unable to self-correct. On repetition of side gliding techniques, some patients can achieve self-correction without clinician assistance.
    - The 'hard' lateral shift – the lateral shift will have accompanied the recent onset of back pain. The patient presents with a very visible lateral deformity that they are unable to self-correct. They are unable to bring their shoulders and hips back to the middle, or, if they can, are not able to maintain correction. These patients need clinician assistance.

Management of a relevant lateral component is approached differently depending on the presence or absence of a lateral shift. If there is no lateral shift, the symptomatic response to sagittal plane forces will have determined the appropriate use of lateral forces or some combination of extension and lateral. In the first two situations, the lateral principle is adopted after a worsening or unchanging symptomatic response to sagittal plane evaluation as described above. In the presence of a deformity of lateral shift, lateral forces are adopted immediately. Management is described below in two sections:

- lateral component, no lateral shift
- lateral component, soft or hard lateral shift.

Management – lateral component, no lateral shift

This section describes the management strategy adopted for unilateral or asymmetrical pain without a clearly discernible lateral shift that has not responded to pure sagittal plane procedures. During the initial and possibly second assessment, extension procedures and progressions will have been explored as described above. These will either have caused a worsening of symptomatic or mechanical presentations or caused no substantial change. Worsening refers to a change from intermittent to constant pain, an increase in intensity

or peripheralisation. Thus, the sagittal plane has either generated an unfavourable response or, despite progressions, has made no significant change.

Lateral procedures usually involve movement of the hips away from the pain. Thus a patient with left-sided pain will move hips to the right for extension in lying with hips off centre or side gliding. This would be the most common direction used, but if no favourable response is generated, the other direction should be explored.

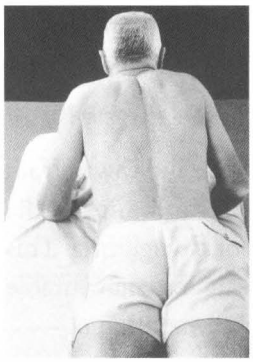
Procedures are performed in the following order, with subsequent force progressions applied only if there is failure to improve. If at any point improvement does occur, further progression is unnecessary. Whatever progression is used, the patient must continue to perform the appropriate exercises at home with suitable regularity. Following a force progression, the effects of this procedure should be evaluated at the next review. Force progressions can be repeated on up to two occasions before they should be abandoned if no change ensues.

Do not instigate clinician techniques unless it is clear that improvement cannot be achieved by any other means. On occasions, however, the application of clinician overpressure during a clinic session helps to determine the appropriate therapeutic loading strategy. After the application of lateral principle procedures, extension procedures frequently become necessary.

The progressions are listed below in the order that most frequently generates a favourable clinical response. However, in determining the appropriateness of loading strategies, some flexibility in the application of procedures may be required. Application of force progressions and force alternatives should always be conducted with due consideration given to clinical reasoning and attentive interpretation of symptomatic and mechanical responses.

Force progressions and alternatives – *when relevant lateral component is present.*

Photos 138, 139, 140, 141, 142, 143:
Extension in lying with hips off centre; hips are usually shifted away from the pain (138). Extension in lying with hips off centre with extension overpressure (139). Extension in lying with hips off centre with lateral verpressure (140). Side glide in standing; hips are usually shifted away from the pain (141). Rotation mobilisation in extension - pressure on alternate sides (142). Rotation mobilisation in extension - unilateral pressure on appropriate side (143).



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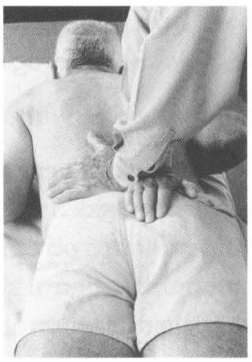

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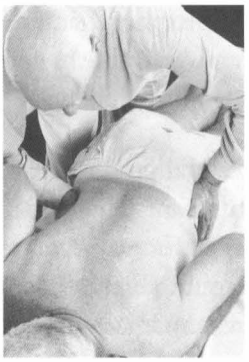

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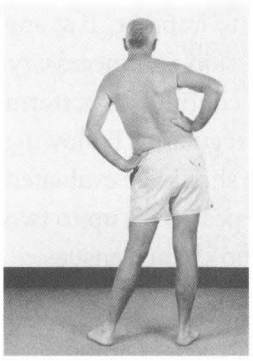

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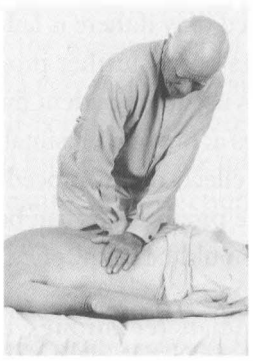

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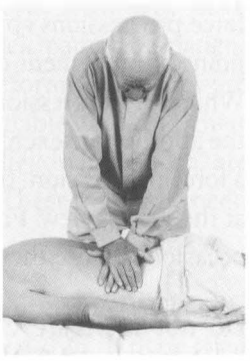

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Photo 144: Rotation in flexion.

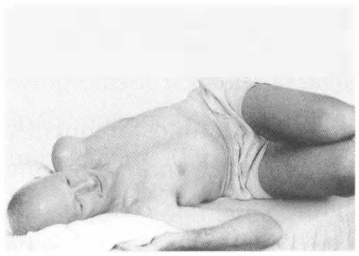

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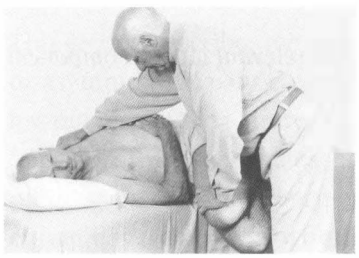
Photo 145: Rotation mobilisation in flexion.



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- extension in lying with hips off centre (Procedure 11)
- extension in lying with hips off centre with clinician over-pressure (Procedure 12)

Force alternatives – some patients respond better to a loaded procedure

- side gliding in standing (Procedure 16)

Further force progressions – if all preceding procedures have generated no clear response

- extension mobilisation with hips off centre (Procedure 13)
- rotation mobilisation in extension (Procedure 14)

If the patient is worse with extension/lateral procedures or pure lateral procedures, flexion/lateral procedures are considered.

- rotation in flexion (Procedure 23)
- rotation mobilisation in flexion (Procedure 24)
- flexion in step standing (Procedure 22).

The management strategy adopted is that which causes symptoms to be decreased, abolished or centralised with the greatest degree of patient independence. To help determine the appropriateness of different loading strategies, it may be necessary to apply overpressure. In this way the favoured loading can be confirmed.

These different procedures produce different biomechanical effects on the joints of the lumbar spine. Most of these procedures use a lateral component that is combined with different amounts of flexion or extension. Thus different symptomatic and mechanical response are forthcoming from the different procedures. They should be explored in the order above, but in unilateral symptoms, especially pain below the knee, a certain amount of careful experimentation may be necessary to unearth the reductive force.

Review

When the patient returns for review, they will be better, worse or unchanged. Ensure that the status reported by the patient is their true state. See Chapter 16 for a detailed analysis of symptomatic and mechanical responses and Chapter 19 for the structure of the review process.

Patient is better

If there is improvement in the symptomatic and/or mechanical response at review, management strategy should not be changed as long as a favourable response continues. When symptoms centralise, assess ability to return to use of sagittal plane movements.

Patient is worse

If the patient is worse with extension/lateral procedures or pure lateral procedures, flexion/lateral procedures are considered.

- rotation in flexion (Procedure 23)
- rotation mobilisation in flexion (Procedure 24)
- flexion in step standing (Procedure 22).

Patient is unchanging

First it should be ensured that the patient is performing the right exercises correctly and with enough regularity and that they are abiding by appropriate postural instruction. If this is not the case, further instruction and discussion are necessary to ensure that the patient understands the procedures and their own role in managing the problem. It is best to see the patient daily until certainty of management is established and the patient is confident about their management strategy. If the patient cannot be reviewed regularly, this can create problems. In such cases attempts to review by telephone are desirable.

If they have been following the treatment principle correctly, but still no improvement has occurred, then appropriate overpressure and mobilisation should be applied as in the order of force progressions indicated above.

Later force progressions

If, following rotation mobilisation in extension, symptoms are decreased or abolished but soon return, manipulation may be considered.

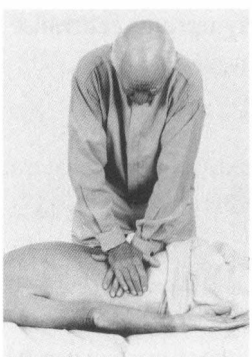
- rotation manipulation in extension (Procedure 15)

If, following rotation mobilisation in flexion, symptoms are decreased or abolished but soon return, manipulation may be considered.

- rotation manipulation in flexion (Procedure 25).

If, after all of the above progressions have been attempted, the symptoms remain unchanged or worse, then the derangement may be irreducible or other pathology may be present. The patient is unlikely to respond to mechanical therapy and other management considerations or further investigation should be contemplated.

Photos 146, 147: Rotation manipulation in extension (146). Rotation mobilisation in flexion (147).



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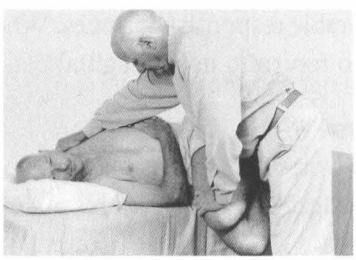

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Management – lateral principle, soft or hard lateral shift

These patients are identified by their obvious lateral deformity, which they are unable to self-correct, that is of recent onset with their symptoms; or, if they can bring their hips back towards mid-line, they are unable to maintain this correction. Often there is also a loss of the lumbar lordosis, and flexion and extension are also reduced. The patient appears to be shifted sideways from the level of the lesion and above. The deformity is either termed a *contralateral shift* if away from the painful side, or an *ipsilateral shift* if towards the painful side. McKenzie (1972) found 96% of a series of 500 patients to have a contralateral shift.

Table 26.2 Criteria for a relevant lateral shift

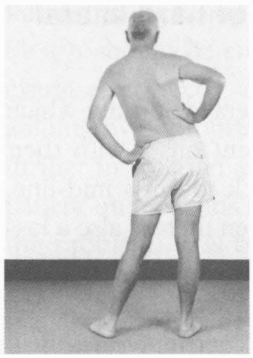
- upper body is visibly and unmistakably shifted to one side
- onset of shift occurred with back pain
- patient is unable to correct shift voluntarily
- if patient is able to correct shift they cannot maintain correction
- correction affects intensity of symptoms
- correction affects site of symptoms.

The application of extension in the presence of the lateral deformity can significantly worsen or peripheralise symptoms. The lateral principle is always adopted immediately and repeated sagittal plane movements are not explored.

Some patients with a deformity, who initially appear to be fixed in a lateral shift, can in fact with repeated side gliding movements begin to self-correct and start to regain the lost movement. If patients respond in this way, they should be encouraged to continue with self-correction of lateral shift as long as the symptomatic presentation is improving in line with the mechanical presentation. Such 'soft' deformities should start to show some change within two sets of repeated movements. If after this time nothing is altered, clinician force should be applied.

Patients with a 'hard' deformity are completely unable to modify the mechanical and symptomatic presentation in any way. They are truly fixed in the lateral shift position, and clinician assistance is needed to correct the deformity and regain mobility.

Photos 148, 149, 150, 151, 152, 153, 154, 155, 156: Side gliding. Normally hips are moved away from the side with pain (148). Teaching self-correction of lateral shift (149). Teaching self-correction of lateral shift (150). Self-correction of lateral shift – against wall (151). Manual correction of lateral shift – stages (26). Clinician stands to side of shift (152). Patient's arm at right angles (153). Encircle patient with hands on iliac crest (154). Gradual correction (155). Over-correction (156).



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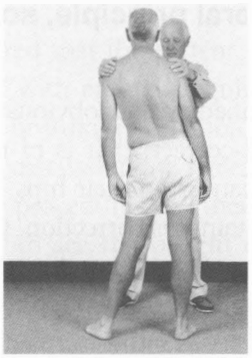

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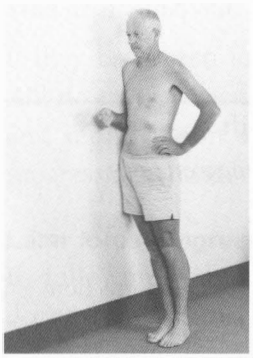

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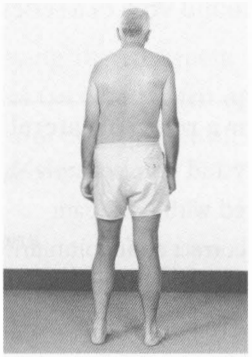

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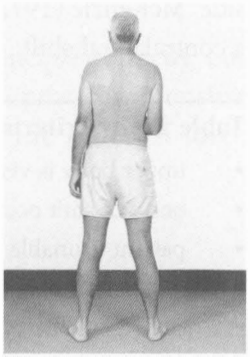

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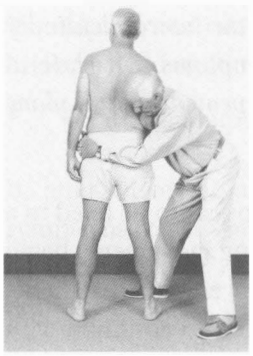

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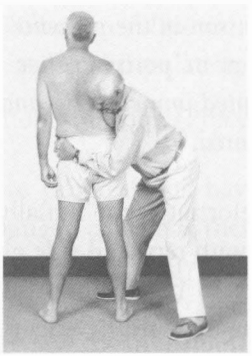

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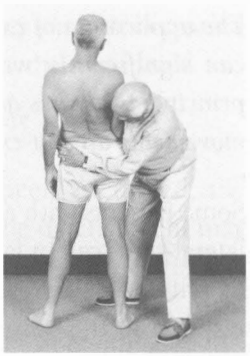

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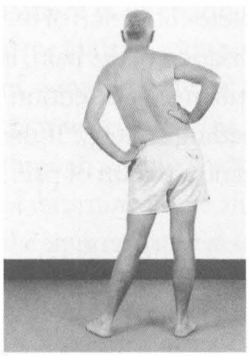
Time is often very important in this procedure, which must not be rushed. At all times there is careful monitoring of distal symptoms. The procedure involves four stages and is described in detail in the chapter on procedures (17):

- correction of lateral deformity
- over-correction of side gliding
- restoration of lordosis
- instruction in self-correction of lateral shift.



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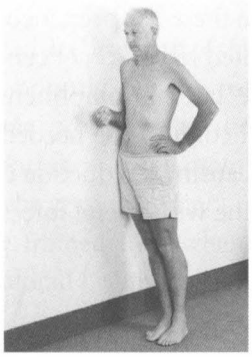

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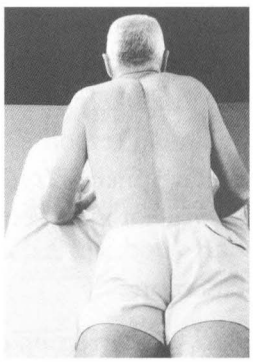

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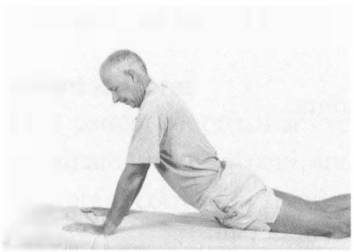
Photos 157, 158, 159, 160, 161: Side gliding (157). Self-correction of lateral shift – against wall (158). Exten in lying with hips off centre (159). Extension in lying (160). Posture correction (161).



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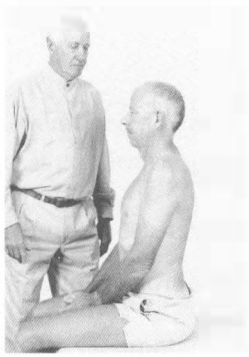

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Procedures:

- self-correction of lateral shift (Procedure 16) – 'soft' deformity
- manual correction of lateral shift (Procedure 17) – 'hard' deformity.

The home programme to be used after the first session depends on whether there has been a partial or complete correction of the deformity as well as symptom response. If on the first session the deformity is completely reduced and either the patient is pain-free or has only low back pain, and that is responding to the extension principle, then they are instructed as follows:

- self-correction of lateral shift (Procedure 16)
- extension in lying with hips off centre (Procedure 11)
- extension in lying (Procedure 5)
- posture correction (Procedure 4).

Regularity:

- ten to fifteen times every 2 – 3 hours.

If after the initial session there has been incomplete correction of the deformity and only limited reduction or centralisation of the pain, it is most likely that the lateral component still needs attention. Symptom response will confirm the needed loading strategy. If the lateral principle is still causing a reduction or centralisation of pain, the patient must continue with lateral forces.

Procedure:

- self-correction of lateral shift (Procedure 16)
- extension in lying with hips off centre (Procedure 11).

Regularity:

- ten to fifteen times every 2 – 3 hours.

Expected response:

- continuing decrease or centralisation of pain
- continuing improvement of mechanical presentation.

Review

Review is best done on a daily basis until reduction of the derangement is stabilised. When the patient returns for review they will be better, unchanged or worse. Ensure that the status reported by the patient is their true state. See Chapter 16 for a detailed analysis of symptomatic and mechanical responses and Chapter 19 for the structure of a review appointment.

Patient is better

If there is improvement in the symptomatic and/or mechanical response at review, management strategy should not be changed as long as a favourable response continues. If pain is centralised or much decreased, there should be a parallel improvement in the deformity. This should now be absent or minimal, and side gliding movement should have improved.

If symptoms are unilateral or asymmetrical and responding to lateral forces, continue with this management as outlined in section above, *Management – lateral component, no lateral shift.*

If symptoms are central and symmetrical, responding to extension forces and the lateral shift is abolished, continue with management in the sagittal plane as outlined in Chapter 25.

Patient is unchanging

If the initial session resulted in correction of the shift and centralisation of symptoms, albeit temporarily, but the patient is now back to as they were, then the same procedure as the previous day is gone through again. The importance of maintaining posture correction, of returning home after the session and not to work and performing the appropriate exercise immediately when they arrive home and regularly thereafter should all be emphasised.

If improvement ceases, force progressions become necessary. For force progressions with the lateral principle, see *Management – lateral component, no lateral shift*.

Patient is worse

If the patient reports that every attempt to correct the lateral shift exacerbates the distal pain, and this is confirmed in the clinic, further attempts at correction should not be made.

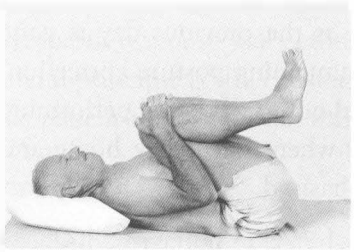
- rotation in flexion (Procedure 23)
- rotation mobilisation in flexion (Procedure 24) – these procedures can give some patients temporary relief.

Failure to reduce derangement and improve symptoms is not uncommon in this group, especially under the following circumstances (McKenzie 1972):

- symptom duration for longer than twelve weeks
- ipsilateral shift
- constant sciatica with neurological deficit.

If there is no change in symptoms, or the patient is worse, then the patient is a non-responder – the derangement is irreducible. The patient is unlikely to respond to mechanical therapy and other management considerations or further investigation should be contemplated.

Photos 162, 163, 164, 165:
Flexion in lying (162). Flexion in sitting (163). Flexion in standing (164). Flexion in sitting with patient overpressure (165).



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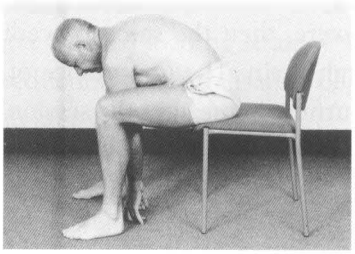

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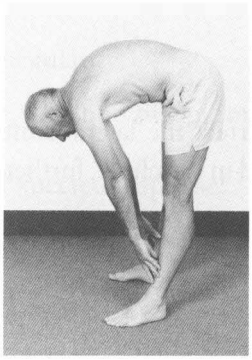

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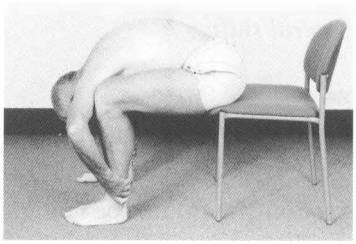

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Flexion principle

Uncommonly, some patients with unilateral or asymmetrical symptoms with pain to the knee require flexion forces in their reduction. Management using the flexion principle is exactly as described in Chapter 25. The same starting procedures and force progressions, if needed, are used. In some instances of unilateral or asymmetrical symptoms, flexion techniques with a lateral component may be required.

Procedure:

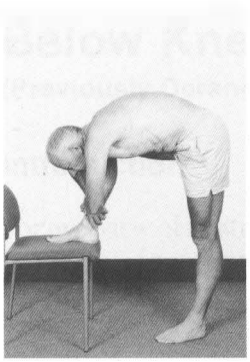
- flexion in lying (Procedure 18).

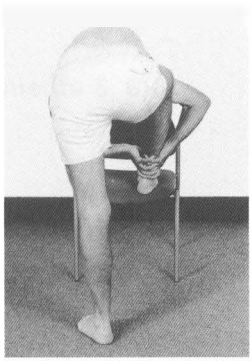
Regularity:

- ten times every 2 – 3 hours.

Progressions – usually required on the first day or after a few days:

- flexion in sitting (Procedure 19)
- flexion in standing (Procedure 20).





Photos 166, 167, 168, 169:
Flexion in step standing (166/167). Rotation in flexion (168). Rotation mobilisation in flexion (169).

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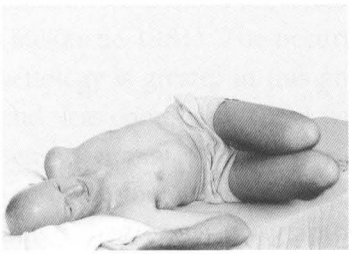

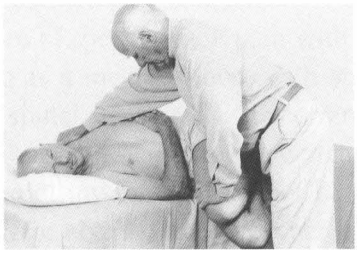

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Alternatives with lateral component:

- flexion in step standing (Procedure 22)
- rotation in flexion (Procedure 23)
- rotation mobilisation in flexion (Procedure 24)
- rotation manipulation in flexion (Procedure 25).

27: Management of Derangements – Unilateral Asymmetrical to Below Knee

(Previously Derangements 5, 6)

Introduction

For overview of treatment pathways in derangement, see first section in Chapter 25 and Figures 25.1 and 25.2.

This chapter describes the management of patients with distal leg or calf pain with and without neurological signs and symptoms. These patients were those previously classified as derangements 5 and 6 (McKenzie 1981). The occurrence of specific back pain with clear aetiology is greater in this group as it includes those with sciatica and, less commonly, those with spinal stenosis. The occurrence of sensory or motor deficit or neural tension signs attest to irritation of the nerve root. However, nerve root involvement cannot be ruled out if these signs or symptoms are absent, as the occurrence of objective neurological signs is a variable, not absolute, accompaniment of nerve root irritation. Although this chapter concentrates on specific pain, namely sciatica, it should be recognised that not all distal leg pain results from nerve root irritation (Rankine *et al.* 1998). Somatic pain mechanisms, such as intra-discal, zygapophyseal and sacro-iliac joint pathology can also cause symptoms below the knee (Schwarzer *et al.* 1994b, 1995a, 1995d).

Management is described according to whether symptoms have been present for more or less than three months, and whether symptoms are constant or intermittent. Management of reducible derangements is the same in these different situations and is as described for unilateral asymmetrical symptoms to the knee (Chapter 26). The sagittal plane is initially explored; if there is an unfavourable or lack of response to extension procedures, the lateral component is introduced. This process of determining the appropriate loading strategy and the appropriate force progressions is reviewed in the section *First twelve weeks – constant sciatica, reducible derangement*. What is different within these different timescales and frequency of symptoms are the differential diagnosis and the prognosis.

Sections in this chapter are as follows:

- differential diagnosis

- management of derangement – unilateral asymmetrical below knee
- management – first twelve weeks or three months
    1. constant sciatica – first twelve weeks
    2. intermittent sciatica – first twelve weeks
- management – sciatica after twelve weeks
    3. constant sciatica – after twelve weeks
    4. intermittent sciatica
- differential diagnosis derangement, nerve root adherence and nerve root entrapment.
- repeated movements.

Differential diagnosis

Established musculoskeletal causes of nerve root problems, which may be suspected clinically but need paraclinical investigations to be confirmed, are as follows. These entities are described in more detail elsewhere:

- disc herniations
- central canal stenosis
- lateral canal stenosis.

Disc herniations may be classified as follows (Chapter 6):

- protrusion – reducible
- protrusion – irreducible
- extrusion – irreducible
- sequestration – irreducible
- entrapment – irreducible.

The most common cause of sciatica or nerve root irritation is a disc herniation (Spitzer *et al.* 1987). These are predominantly postero-lateral, implicating flexion as an aetiological and aggravating mechanical factor (Ninomiya and Muro 1992). Less than 10% of herniations are purely lateral in direction. The plane of fissures and direction of herniation routes has important implications for appropriate management. See Chapter 4 on intervertebral disc for more detail. Successful conservative management depends on

whether it is reducible rather than irreducible. The other well-established cause of distal leg pain with
stenosis (Chapter 13). This differential diagnosis is considered there (see Table 13.20).

The natural history of sciatica allows many, although not all, patients to recover eventually (see Chapter 5). In many patients, resolution of pain and restoration of function may be accelerated with appropriate mechanical procedures. However, the recovery period for others may sometimes be extremely prolonged. A few patients may fail to obtain lasting relief and these may develop significant long-term physical and psychological disability. Both disc herniations and spinal stenosis may be managed surgically, and this is discussed in Chapter 13. Here we are concerned with the conservative management of derangements with distal leg symptoms, with without accompanying neurological signs and symptoms. The key clinical distinction is between those conditions that will respond to conservative management – that is, a reducible derangement – and those that will not – an irreducible derangement or spinal stenosis.

Sciatic pain, for the first twelve weeks following onset, arises directly from the displaced discal tissue in contact with or adjacent to the nerve root complex. There may be indirect tension or direct compression of the nerve root. Symptoms may be generated by direct mechanical stresses, or indirectly, inducing swelling around the root and causing ischaemia. Alternatively, symptoms may be due to chemical irritation of the nerve root. Over the first few months there may be a reduction of swelling at the site of the displacement and a gradual lessening of distal pain and pain intensity. This is usually accompanied by a gradual improvement in function. Many patients' symptoms show signs of recovery within the first few months. When a reducible derangement is present, mechanical therapy can speed up the natural recovery, but when there is an irreducible derangement, mechanical therapy cannot affect the natural history.

While sciatic symptoms persisting beyond twelve weeks from onset may still be caused by the disc herniation itself, there is the possibility that secondary complications may be responsible for continuing leg symptoms. Nerve root entrapment, due to the disc or to bony changes in the spinal canal or intervertebral foramen, or nerve root adherence due to scar formation, may now be the cause of the symptoms. The key clinical distinction is between an active pathology that we are

able to influence with mechanical therapy – that is, a derangement or reducible herniation – and an inactive pathology that will only change slowly over time, if at all. The latter is due to tethering of the nerve root or compression or tension of the nerve root from an irreducible disc herniation or from bony changes.

Management of derangement – unilateral asymmetrical below knee

For the sake of clarity, the description is divided into the management of the disorder in the first twelve weeks or three months from onset and after twelve weeks from onset. From about this time symptom persistence may be due to secondary complications of the original disc herniation. Onset in this context refers to the time since commencement of leg symptoms and not from the onset of coexisting back pain, which may have preceded sciatica by many months. In the following sections management is described in the categories of patients shown in Table 27.1 (also see Figure 27.1).

Table 27.1 Management of sciatica

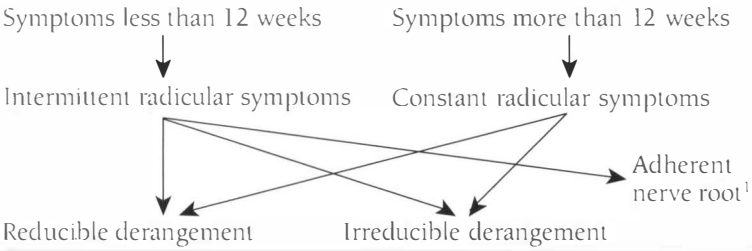
Management first twelve weeks:

1. Constant sciatica
   a) reducible derangement
   b) irreducible derangement
2. Intermittent sciatica
   a) reducible derangement

Management after twelve weeks:

3. Constant sciatica – improving
   a) resolving derangement – unchanging
   b) reducible derangement
   c) irreducible derangement / nerve root entrapment
4. Intermittent sciatica
   a) reducible derangement
      Development of adherent nerve root
   b) adherent nerve root (Chapter 29)

Figure 27.1 Classification pathway for sciatica



[1] See Chapter 29 for detail

Management – first twelve weeks or three months

1. Constant sciatica – first twelve weeks

In the early stages, leg symptoms are constant in many patients. Sciatica may remain constant for eight to twelve weeks and even longer in extreme cases. The presence of constant sciatica indicates that the volume and location of displacement causing compression and or tension of the nerve root is unremitting and the patient is unable to find any movement or position that gives lasting relief. Although many such patients will respond favourably to mechanical therapy, a large number will have pathology that will not be amenable to conservative management.

When irritation of the nerve root is constant, neurological deficit is more likely. This may present as constant numbness in the big toe or lateral border of the foot, and/or as marked weakness of dorsiflexors, extensor hallucis longus or gastrocnemius. When neurological motor deficit is present, the chance of success using mechanical procedures is less. The presence of sensory deficit alone is not necessarily a poor prognostic indicator. Assessment should proceed with care; if at any point a significant increase in peripheral pain or paraesthesia is provoked, it is unwise to continue with mechanical testing in that direction.

The pain of sciatica can be significantly exacerbated if patients with these symptoms are subjected to an over-vigorous physical examination. Flexion has the possible effect of causing further displacement, and repeated movement testing in this direction can quickly worsen the condition. From the history itself and from observation of the patient's restriction of general mobility, it should be possible to identify those patients with severe symptoms who require particular care. The mechanical evaluation of such patients must be limited to the

application of essential elements only. In the early stages of applying the mechanical evaluation and during the treatment of the patient with acute constant sciatica, close attention should be paid to symptom response.

In patients with constant sciatica, it is impossible to predict the likely outcome of mechanical therapy until the static and dynamic mechanical evaluation has been completed, usually over several days. Our ability to effect beneficial change in the volume and location of displaced intradiscal tissue is entirely dependent on the integrity of the annulus fibrosus. A ruptured or incompetent annulus will not allow any lasting alteration in the location or volume of displaced tissue when reductive pressures are applied to the affected segment. In contrast, an intact competent annulus not only restrains further displacement, but also allows a reversal of existing displacement.

1a. Reducible Derangement

Assessment

From the history and physical examination of the patient with constant sciatica, it may become apparent that a decrease or centralisation of pain is possible. This conclusion can be confirmed when a decrease, abolition or centralisation of symptoms occurs with extension or lateral procedures and lordotic sitting postures. Should the symptoms remain improved following extension procedures, the diagnosis can be confirmed. The conclusion can also be confirmed if extension postures and movements worsen distal symptoms, but lateral forces decrease, abolish or centralise them.

It is sometimes the case that, following the evaluation, the patient experiences a return of symptoms within ten to fifteen minutes. After decrease or abolition of symptoms, it is sometimes useful to ask the patient to walk about in the corridor and monitor the symptoms. If symptoms recur, the reductive process should be repeated, and care should be taken that the patient maintains the lordosis as they get up from the plinth. The patient should be advised to repeat the reductive movements over the following twenty-four hours and to be especially attentive to maintenance of lordosis after performance of the exercises. In this way a more stable reduction may be achieved and the patient report that symptoms remain improved following each exercise session. Thus the mechanical diagnosis and management programme is confirmed.

Management

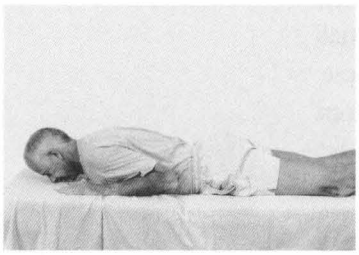
Identification of the treatment principle that abolishes, decreases or centralises symptoms and improves mobility and function is the key to management. Many reducible derangements have a directional preference for extension. However, a sizeable proportion of these problems with symptoms below the knee need additional lateral forces in their reduction, and so awareness of this option needs to be maintained

Any position or movement that enhances sciatica should be identified and temporarily avoided during the reductive process and during maintenance of reduction. In this regard, as has been noted already, flexion can easily aggravate symptoms due to its ability to enhance posterior displacement. This effect can be particularly marked with sustained postures.

Assessment thus proceeds as outlined in the previous chapters:

1. If a deformity of lateral shift is present, immediate consideration is given to lateral forces. Sagittal forces are contraindicated initially, when lateral procedures are always used. Sagittal forces may become necessary later. See section Management – lateral component, soft or hard lateral shift (Chapter 26, page 615).
2. If no deformity of lateral shift is present, exploration of sagittal plane forces proceeds as outlined in Chapter 26.
3. If a deformity of kyphosis is present, the procedure of sustained extension is necessary in order to ensure a gradual resumption of the lordosis. In these instances if extension is attempted too quickly, symptoms will be dramatically aggravated. On most occasions the presence of kyphosis in sciatica indicates an irreducible derangement.
4. Sagittal movements are explored initially. Search for clues in the history that may suggest directional preference (see Tables 24.5 and 24.9). If distal pain worsens or pain peripheralises, introduce a lateral component. Use force progressions as necessary and to confirm directional preference. See section Assessment – determining the appropriate strategy (Chapter 26, page 602) for clinical reasoning process.

Photos 170, 171, 172, 173, 174, 175, 176, 177, 178:
Lying prone (170). Lying
Extension in lying with clinician overpressure (176). Extension mobilisation (177). Posture correction (178).



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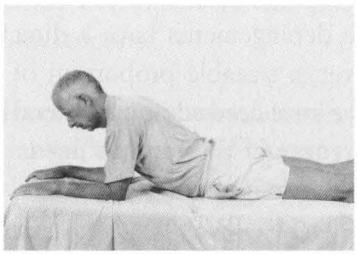

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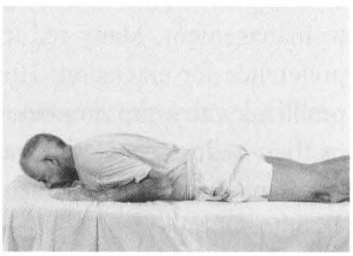

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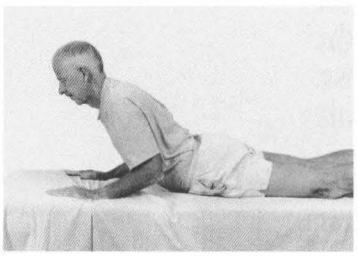

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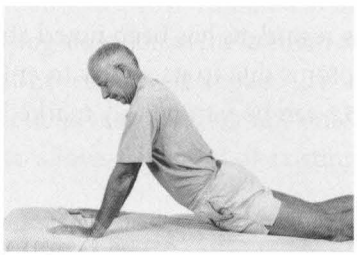

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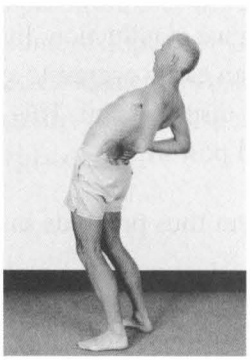

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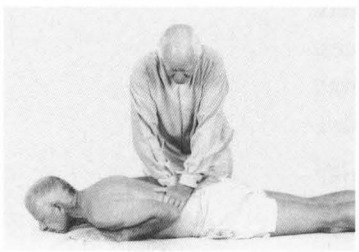

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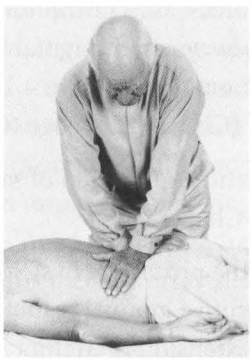

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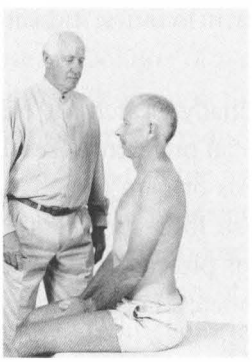

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- lying prone (Procedure 1)
- lying prone in extension (Procedure 2)
- extension in lying (Procedure 5)
- posture correction (Procedure 4)
- extension in standing (Procedure 9)
- extension in lying with clinician overpressure (Procedure 6a)
- extension mobilisation (Procedure 7).

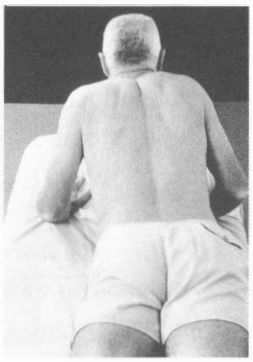
5. If during extension procedures there is a worsening of peripheral pain or peripheralisation of pain, sagittal plane forces are abandoned and exploration of lateral forces is conducted as outlined in the previous chapter. Equally, if symptoms are unchanged, the lateral component is explored. *When the symptom response is indeterminate, it is important to compare symptom response to sagittal movements with the symptom response to movements addressing the lateral component and determine if there is a clear directional preference.* Determining the best strategy requires applying a clinical reasoning process, and overpressures and mobilisation may be considered on day one, and definitely used on day two, to help determine the appropriate loading. See section Assessment – determining the appropriate strategy (Chapter 26, page 602) for clinical reasoning process to determine appropriate management.

Lateral procedures usually involve movement of the hips away from the pain. Thus a patient with left-sided pain will move hips to the right for extension in lying with hips off centre or side gliding. This would be the most common direction used, but if no favourable response is generated, the other direction should be explored. See section Management – lateral component, no lateral shift (Chapter 26, page 610) for more detail. Consideration of the lateral component may include the following procedures:

- extension in lying with hips off centre (Procedure 11)
- extension in lying with hips off centre with clinician overpressure (Procedure 12)
- side gliding (Procedure 16)
- extension mobilisation with hips off centre (Procedure 13)
- rotation mobilisation in extension (Procedure 14)
- rotation in flexion (Procedure 23)
- rotation mobilisation in flexion (Procedure 24)
- flexion in step standing (Procedure 22).

The progressions are listed in the order that most frequently generates a favourable clinical response. However in determining the appropriateness of loading strategies some flexibility in the application of procedures may be required. *Application of force progressions and*

Photos 179, 180, 181, 182, 183, 184, 185, 186: Extension in lying with hips off
away from the pain (179). Extension in lying with hips off
overpressure (180). Extension in lying with hips off centre with lateral overpressure (181). Side glide in standing, hips usually shifted away from the pain (182). Rotation mobilisation in extension - pressure on alternate sides (183). Rotation mobilisation in extension - unilateral pressure on appropriate side (184). Rotation in flexion (185). Rotation mobilisation in flexion



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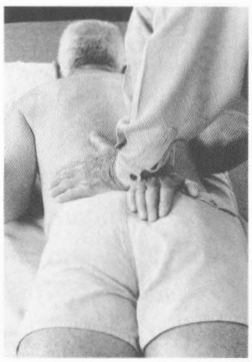

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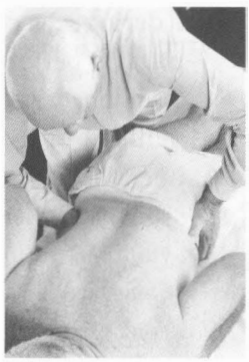

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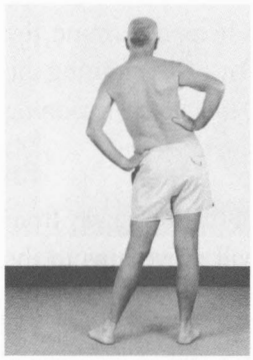

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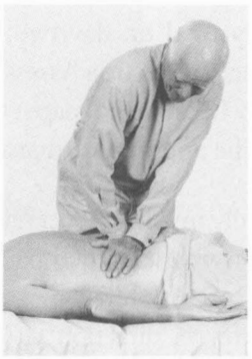

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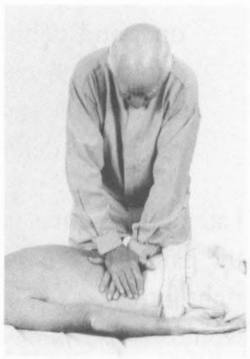

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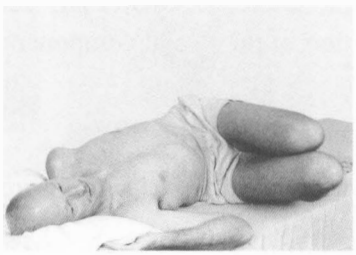

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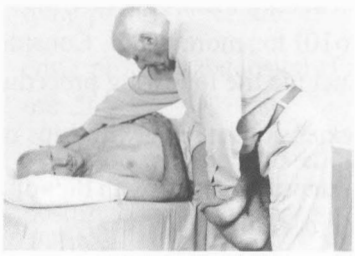

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force alternatives should always be conducted with due consideration given to clinical reasoning and attentive interpretation of symptomatic and mechanical responses.

The procedure that is found to abolish, decrease or centralise pain is the procedure that is adopted in management. This is altered if symptom response changes.

After reduction of derangement, flexion procedures should be introduced to ensure stability of reduction and full recovery of function (Chapter 24, page 568)

1b. Irreducible derangement

Assessment

When, during the initial mechanical evaluation of patients with constant sciatica, all movements cause an increase in radiating pain and *no position can be found to provide lasting relief*, it is likely that the derangement is irreducible. Should further evaluation on successive days confirm that finding, additional attempts at reduction should be abandoned.

Derangements causing constant sciatica are often irreducible by mechanical means during the first few weeks after onset. Clues to distinguish a potentially reducible protrusion from an irreducible extrusion or sequestration can be gained from the history and physical examination (Table 27.2). The conclusion that a derangement causing nerve root compression and or tension is irreducible will be made over up to five sessions during which period the findings obtained from the initial mechanical evaluation will be unchanged and, in some cases, symptoms may be exacerbated.

Table 27.2 Distinguishing between sciatica due to a protrusion or an extrusion/sequestration – features are variable

Disc protrusion	*Disc extrusion/sequestration*
LBP =/> thigh / leg pain	Leg pain >> LBP / No LBP Distal pain ++
Gradual onset leg pain Onset leg pain LBP remains the same	Sudden onset leg pain Onset leg pain LBP eases or goes
Postural variation ++ Variable back and leg pain	Less postural variation
Intermittent / constant pain	Constant pain
Intermittent / constant tingling	Constant numbness
Variable deformity	Constant deformity or no deformity
Variable weakness	Motor deficits
Moderate / variable tension signs	Major, constant tension signs Crossed straight leg raise positive
Movements able to decrease, abolish or centralise symptoms	Movement increases distal symptoms No movement able to decrease, abolish or centralise symptoms in a lasting way
	Severe restriction walking capacity
Possible related neck pain	

LBP = low back pain

Source: Kramer 1990; Brismar *et al.* 1996; Beattie *et al.* 2000; Pople and Griffith 1994; Vucetic *et al.* 1995; Uden and Landin 1987; McKenzie 1981; Jonsson *et al.* 1998; Jonsson and Stromqvist 1996b; Zanoli *et al.* 2001

It is sometimes the case that certain movements can be found to temporarily reduce or sometimes even abolish distal symptoms in patients with irreducible derangement. This usually occurs with procedures performed in the unloaded position. However, the symptoms do not remain better and return immediately when loading is reapplied or when a change of position occurs. The movement is insufficient to provide a lasting reduction of displacement, or else the annular wall is incompetent or breached and is unable to provide a barrier to protect the nerve root. In such cases it is likely that any reduction will be unstable and if after a week or two no improvement results, further attempts at reduction are probably unwarranted.

Assessment will proceed as normal first in the sagittal plane, with an examination of the effects of flexion, extension and curve reversal with posture correction. The effects of lateral forces will then be explored. Most repeated movements will cause an increase and temporary worsening of peripheral symptoms, although sometimes certain movements may have no effect.

Management

Where irreducible derangement is the cause of severe constant sciatica, one to three weeks of relative rest and pain medication may assist in the reduction of pain and allow a more stable repair during this early period. Subsequent evaluation could be considered after sufficient time has passed, but a course of treatment can be justified only if it is possible now to affect the symptoms or if the symptoms have changed to become intermittent.

Bed-rest has been shown to be no more effective than 'watchful waiting' in acute sciatica (Vroomen *et al.* 1999). The value of NSAID therapy for sciatica is unproven, according to one systematic review (Koes *et al.* 1997), and has been shown to be ineffective in one trial (Weber *et al.* 1993). Saal and Saal (1989) reported on an aggressive treatment programme, including exercises and injections, for sixty-four patients with herniated discs. Functional recovery and return to work was achieved by over 90%; a sub-group with extruded discs had a 100% return-to-work rate. Sick leave was substantial, averaging four and a half months, and six patients came to surgery.

Sciatica alone is not a sufficient indication for surgery. Segmental signs and symptoms and a demonstrable disc herniation from an imaging study are also usual requirements. Furthermore, surgery is

usually only considered after failure of four to six weeks of conservative treatment (Hoffman *et al.* 1993). If improvement is perceived, the decision to surgically intervene should be further delayed. Because of greater symptomatic severity from non-contained disc herniations, they are more likely to come to surgery earlier than contained lesions. However, if such patients can tolerate the first two months, which is the worst period, conservative management can be successful (Ito *et al.* 2001). The patient should be encouraged to remain active and commence a programme of general exercise during the period of recovery so that function is maintained.

2. Intermittent sciatica – first twelve weeks

2a. Reducible Derangement

Assessment

When sciatica is intermittent, symptoms and signs are usually less severe – as in the protrusion column, Table 27.2. Intermittent symptoms of sciatica suggest a much better prognosis than constant symptoms. This may be so even if the patient presents with associated neurological signs or symptoms or nerve tension signs. Patients may describe fluctuating symptoms in their leg and back, or constant back pain and variable leg pain. The variability of leg pain may present in a variety of ways, for instance that it is absent in the morning, but returns in the afternoon and evening. Other patients may describe that their sciatica, which was constant, is now intermittent.

This history suggests that spontaneous resolution of the derangement is occurring and we should identify procedures that will accelerate recovery. We should also ensure that nothing is done that will interfere with the spontaneous resolution already under way. The patient's description of activities that aggravate and improve the condition will lead to the appropriate procedures for treatment.

When the patient describes intermittent sciatica since onset, it is most likely that the derangement is rapidly reducible. They may explain that the leg pain ceases or decreases during certain activities and positions. It then remains better for hours until it is aggravated again by other activities. Some patients are unable to relate the variability of symptoms to particular activities and report that symptoms fluctuate for no apparent reason. If symptoms of sciatica can change in intensity or disappear for periods during the day, it is

likely that aggravating and relieving mechanisms can be easily exposed from the history and/or the dynamic mechanical evaluation.

In some cases the patient may have only brief periods in the day when no pain is felt. The fact that the leg pain may cease totally for only ten to fifteen minutes is an indication that the location or volume of displacement, although significant, can be affected under certain specific circumstances. Usually it is a position rather than a movement that achieves such an effect. We need to know what circumstances cause the distal symptoms to appear and disappear; in other words, what loading strategy increases and what reduces the derangement?

Neurological deficit is less likely to develop, as even short periods without root compression or tension allow physiological recovery of the nerve root. Sensory or motor deficits will mostly be absent, but if present are likely be mild, variable or recovering. Tension signs, such as the SLR test, will frequently be negative, mildly positive or variable.

From the mechanical evaluation or more often during static loading while history-taking, it will be found that the effect of flexion is to aggravate symptoms. For instance, during static sitting or with repeated application of flexion performed in standing, distal symptoms will appear or, if already present, will progressively increase or peripheralise. The pain will be experienced during the movement itself and not just at end-range. In some patients the range of extension and perhaps other movements will become progressively reduced as obstruction increases consequent to further displacement. The increased pain remains worse as a result. However, this simultaneous change in pain and movement pattern also indicates that certain movements can still influence the location and or magnitude of the displacement.

This effect should be confirmed following the assessment of the effects of repeated extension in lying. This should cause the symptoms to decrease, abolish or centralise. Simultaneously, the range of extension should also improve.

If extension procedures cause a worsening of distal symptoms or peripheralisation, then lateral forces should be explored.

Management

For patients with intermittent sciatica, management is the same as those with constant sciatica due to a reducible derangement. The

directional preference must first be identified – treatment principles will generally be extension or lateral. Most of these conditions are aggravated by flexion activities.

Assessment thus proceeds as outlined previously:

1. If a deformity of lateral shift is present, immediate consideration is given to lateral forces and sagittal forces are contraindicated. See section *Management – Lateral component, soft or hard lateral shift* (Chapter 26, page 615).
2. If no deformity of lateral shift is present, exploration of sagittal and frontal plane forces proceeds as outlined above under *Reducible derangement* (section 1a, page 628).

For those patients with intermittent sciatica, progression of forces can be applied more confidently, rapidly and effectively than is the case where symptoms are constant.

Management – sciatica after twelve weeks

If patients attend for their initial assessment with sciatic symptoms that have been present for more than twelve weeks, differential diagnosis still includes reducible and irreducible derangements, but also should include adherent nerve root. If patients are already under successful management, and the twelve-week mark is crossed, there is obviously no need to reconsider treatment. Intervention at this point depends partly on the status of the condition – whether it is improving or unchanging. The type of intervention then depends on what is continuing to cause symptoms – is it the original derangement, or is it now scarring from the initial damage? If the derangement is still the cause of symptoms, is this reducible or irreducible?

3. Constant sciatica – after twelve weeks

3a. Improving constant sciatica

Assessment

Many patients with a resolving derangement experience improving back and distal symptoms beyond twelve weeks. These patients state that they have experienced a slow and gradual reduction in pain and an improvement in their general mobility, which continues at the present time. With a slow but progressive reduction in displacement or with progressive scarring in the region of injury, recovery from intervertebral disc herniation may continue for many months.

Management

Improvement may be occurring spontaneously or may be as a result of treatment. Whichever the case, nothing should be done to alter the conditions or environment conducive to the eventual resolution of the derangement. If improvement is occurring spontaneously, it is often better to allow nature to take its course rather than apply mechanical therapies that might disrupt this process. If improvement is apparently in response to treatment, it is unnecessary to use additional procedures. Progressive interventions may not always provide the expected response. If the patient wishes to speed up the process of recovery, or if improvement ceases, assessment and management should proceed as follows:

Deformity of either lateral shift or kyphosis is less likely to be present at this stage when the symptom status is improving. When the sciatica has actually resolved, some patients are left with a significant and visible lateral shift, which has become a dysfunction of lateral glide many months or years later.

Management is as described above for first twelve weeks (section 1a, page 628).

3b. Reducible derangement

Sometimes derangements do not demonstrate the good natural history that is commonly seen. Symptoms do not necessarily spontaneously improve over the first few months, but may nonetheless respond to mechanical therapy. A normal mechanical assessment as detailed above, to look for symptom response to loading strategies, should be carried out.

1. A relevant lateral deformity may unusually still be present at this point in time. If it is, both lateral and extension forces can be explored.
2. Management is as described above for first twelve weeks (section 1a, page 628).

3c. Irreducible derangement – nerve root entrapment

If constant sciatica – totally resistant to mechanical therapy – persists beyond twelve weeks from onset and shows no signs of improving, the cause is almost certainly a large irreducible discal hernia. The bulging annulus or displaced disc material, now in a state of fixation by fibrous repair, is incapable of moving and constantly causes tension

or compression of the nerve root. Inescapable entrapment of the nerve root results and thus sciatic symptoms are felt constantly. Because of the extent of the formation of scarring and fibrosis, resolution is unlikely. In essence this is a form of spinal stenosis, in which the narrowing about the nerve root is caused by discal tissue rather than bony tissue.

Assessment

Patients with nerve root entrapment describe leg pain and in many cases numbness that has been present for longer than twelve weeks. The pain and or numbness never cease; *the intensity of the symptoms reduced in the first month, but improvement stopped some weeks or months ago. Since that time there has been no change in either pain intensity or mobility*. The symptoms are aggravated by many activities, but subside to their former level within thirty to sixty minutes. The patient may have noticed that activity seems to improve mobility for a time, but difficulty, especially with forward bending, reappears soon after resting. They often report morning stiffness and worse pain in the morning.

The key test movement in the identification of nerve root entrapment is flexion in standing. At the commencement of the mechanical evaluation, the range of flexion performed in standing will always be restricted. Repeated application of flexion in standing will demonstrate a progressive increase in the range of motion. These may cause some increase of sciatic symptoms as each movement is performed. If pain is experienced, it will be felt during the movement and through to end range, but soon subsides to the original intensity, and is no worse afterwards. The distal symptoms are not worsened in a lasting way. In some instances there is a temporary decrease in symptoms as a result of the standing flexion test, but they soon return to their normal level of intensity. The key to identification is the mechanical response to repeated flexion in standing. The range of flexion movement will be significantly increased following completion of the standing flexion tests.

Should the patient then be instructed to move about or walk for five or ten minutes following the repeated application of flexion in standing, the symptoms will go back to their former intensity and the range of movement will return to its former restricted level. Irrespective of how often the procedure is applied, a temporary

increase in movement and a temporary change in pain follows, but the patient does not remain better as a result, nor does the practice of the exercise for weeks affect improvement. Excessive repetition may cause an increase in all symptoms, but this exacerbation usually subsides overnight.

Where entrapment by an irreducible derangement is the suspected cause of persisting symptoms, repeated extension in standing or lying will usually increase sciatic symptoms, but at this stage in the course of the disorder further displacement is unlikely. Because of the immovable displacement, repetitive extension will cause increased compression of the nerve with each repetition, resulting in an increase of distal pain. This increase usually occurs at a limited end-range and then subsides on return to the neutral position. There is little point in subjecting the patient to repeated extension exercise when the only outcome will be a temporary increase in pain.

Nerve root entrapment is an irreducible derangement with a characteristic type of presentation. Other irreducible derangements may display similarly inactive pathology that is not amenable to change. Repeated movements and sustained postures may increase symptoms, which will then be no worse. No movement will be found that is able to lastingly decrease, abolish or centralise the pain, nor change the mechanical presentation.

Management

If the patient has never undergone mechanical assessment or treatment, a two-week trial to identify any potential for improvement should be undertaken. If no change has occurred at the end of this period, it is unlikely that mechanical therapy will assist in the resolution of this disorder. Although remodelling of soft tissues over time by exercising is theoretically possible, it is not known whether specific exercise in this instance can accelerate this process. From clinical experience, four or five months of structured exercise programme failed to alter the symptoms in those patients willing to persevere with a quite painful treatment strategy.

Given the natural history of disc herniations outlined above, in which regression frequently occurs, especially with the larger herniations and extrusions, it would be hoped that spontaneous recovery might occur over time. During this period the patient should be encouraged to maintain activity and mobility. However, long-term studies of those with

severe sciatica, which might include this group of patients, reveal that persistent symptoms are common at one year and after thirteen years (Balague *et al.* 1999; Nykvist *et al.* 1995).

4. Intermittent sciatica

4a. Reducible Derangement

Sciatic symptoms persisting intermittently beyond twelve weeks may do so because of a recurring derangement. In cases where a weakened but still competent outer annulus resists further displacement, symptoms may appear and disappear according to the degree and duration of applied daily loading. Findings from history and physical examination as well as treatment remain the same as described for intermittent sciatica in the first twelve weeks.

Assessment thus proceeds as outlined previously:

1. Acute deformity is unlikely to be present at this time. If it is, both sagittal and lateral forces should be explored.
2. Management is as described above for first twelve weeks (section 1a, page 628).

4b. Nerve root adherence

A secondary cause of intermittent persisting sciatica is nerve root adherence. Constant sciatica becomes intermittent as adherence develops. Thus symptoms will have improved from onset, but will have become unchanging. It should be remembered that nerve root adherence may be developing or fully developed depending on the time since onset that the patient is assessed. The longer symptoms have been present, the longer remodelling will take; a developing nerve root adherence may resolve much more quickly.

If despite overall improvement the range of flexion in standing remains limited and unchanging, patients may well have developed nerve root adherence. Patients who remain cautious of resuming normal activity and movement are likely candidates for this complication. Adherent nerve root also occurs sometimes in patients who have had surgery for sciatica and have not received the appropriate rehabilitation exercise programme. Assessment should seek to differentiate between derangement and adherent nerve root. If adherence is the cause of the remaining symptoms, such patients should be provided with a structured exercise programme designed

to remodel any structures that are adherent or contracted. This management is detailed in Chapter 29 (page 675).

If sciatica persists beyond twelve weeks and is now felt intermittently rather than constantly, it is possible for the symptoms to arise from two causes:

1. a recurring derangement
2. the development of nerve root adherence or fibrosis.

Adherent nerve root is a form of dysfunction and the presentation and management is described in Chapter 29. A summary is presented below as adherent nerve root is one of the differential diagnoses to be made in patients with sciatic pain.

Differential diagnosis between reducible derangement, nerve root entrapment and adherent nerve root

The following summary and Table 27.3 will help to differentiate between patients whose sciatica is persisting because of a recurrent derangement and those symptoms persist because of nerve root entrapment or adherence.

Repeated movements

In reducible derangement:

Flexion

Repeated application of flexion in standing or lying, or prolonged slouched sitting, produce the distal symptoms or cause them to progressively increase or peripheralise. The pain is experienced during the movement or position, and the sciatica remains worse as a result. The patient's range of extension and perhaps other movements progressively reduce. The changes in both signs and symptoms indicate increasing derangement.

Extension

Repeated application of extension or maintenance of correct posture will cause the distal symptoms to decrease, abolish or centralise. The symptoms will remain better as a result. The patient's range of extension and flexion will improve. The changes in both signs and symptoms indicate reduction of derangement. Alternatively, symptoms may worsen or peripheralise with extension movements

and postures, in which case lateral procedures will decrease, abolish or centralise symptoms.

In entrapment

Flexion

During repeated flexion in standing, the range of movement will progressively increase. The distal symptoms will increase with each movement and then subside to their former level on completion. The pain is experienced during the movement itself, and the distal symptoms will not progressively worsen. In some patients symptoms may decrease during the repeated movement, but also return to their former intensity upon cessation of movement.

Such mechanical and symptomatic changes occurring in patients with entrapment are short-lived. Once the patient moves about or waits for five or ten minutes, the symptoms will return to their former intensity and the range of movement will return to its former level. Irrespective of how often the procedure is applied, a temporary increase in movement and a temporary change in pain follows, but the patient does not remain better as a result nor does the practice of the exercise for weeks effect improvement.

Very infrequently, repeated flexion in lying may affect the symptoms during the performance of the exercises, but the increase in range of motion and reduction of pain intensity that occur following the performance of flexion in standing do not result.

Extension

Repeated application of extension in standing or lying usually increase sciatic symptoms. The increase usually occurs at a limited end-range, but the intensity then subsides to its former level on return to the neutral position.

The patient with sciatica secondary to entrapment does not respond to mechanical therapy, but over many months resolution may occur as adjacent structures accommodate the intrusion. Many patients fail to recover.

In adherent nerve root

Flexion

Repeated flexion in standing may cause the distal symptoms to be produced towards and at the limited end-range of the movement, or may cause pain only in the back and tightness in the leg. The pain then disappears on return to the upright position. The pain is not experienced during the movement itself and does not worsen with repetition. The range of motion is limited, and does not increase or decrease with repetition.

Repeated flexion in lying will not produce distal symptoms, but may produce or increase localised symptoms in the lumbar spine or buttock.

Extension

Repeated extension in standing or lying has little or no effect on sciatica in the presence of nerve root adherence. Some temporary localised pain may be produced in the lower back or buttock at limited end-range.

Thus, using repeated movements, it is possible to distinguish between these apparently similar disorders of derangement and entrapment and identify the patient with nerve root tension signs caused by root adherence. It must be noted, however, that studies to confirm the reliability of this method of differentiation await completion. These and other distinguishing features of these different entities are summarised in Table 27.3.

Table 27.3 Differentiating between a reducible derangement, an irreducible derangement/nerve root entrapment (NRE), and adherent nerve root (ANR) in patients with persistent leg pain

Clinical presentation	*Reducible derangement*	*Nerve root entrapment*	*Adherent nerve root*
Stage	Acute to chronic	Chronic	Chronic
Status	Improving/worsening/ unchanging/varying	Unchanging	Unchanging
Symptoms	Constant/intermittent	Constant	Intermittent
Symptom Behaviour	Consistent/inconsistent Variable Better/worse/ centralising/ peripheralising	Consistent Activity increase, no worse	Consistent Tension position Produce, no worse

Continued next page

Clinical presentation	*Reducible derangement*	*Nerve root entrapment*	*Adherent nerve root*
Aggravating factors	Flexion activities *or* flexion and extension activities	All activities, temporary aggravation	Toe-touching, long sitting, driving, walking
Problems with curve reversal	Yes/no	No	No
Relieving factors	Extension or lateral activities Lying	No activities Some relief with movement	Avoidance of aggravating factors
Episodic	Yes/no	No	No
Physical examination			
Deviation in flexion	Contralateral > Ipsilateral	Contralateral / ipsilateral	Ipsilateral
Loss of flexion	Variable Minor to major	Moderate loss	Moderate to major loss
Loss of extension	Moderate to major major	Moderate to	Variable Nil to moderate
Repeated movements:			
Flexion in standing	Worsen or peripheralise PDM / ERP ROM worse	Increase, no worse *or* decrease, no better PDM Increase ROM for 5 – 10 minutes, then no better	Produce, no worse ERP ROM same
Extension in standing	Better or centralise ROM better *or* worse or peripheralise*	Increase, no worse ROM same	No effect *or* produce back pain, no worse ROM same
Flexion in lying	Response similar to flexion in standing, but often less severe	Increase, no worse ROM same	No effect *or* produce back pain, no worse ROM same
Extension in lying	Better or centralise ROM better *or* worse or peripheralise*	Increase, no worse ROM same	No effect *or* produce back pain, no worse ROM same

PDM = pain during movement ERP = end-range pain ROM = range of movement, and other mechanical presentation.

* with this response, lateral plane is investigated.

28: Dysfunction Syndrome

Introduction

In dysfunction syndrome, the mobility or function of soft tissues is reduced because of structural impairment. It is a painful disorder caused by loading or stretching tissue that is imperfectly repaired or has become adaptively shortened (McKenzie 1981, 1990). Structural impairment arises from contractures, adhesions, scar tissue or imperfect repair, which in turn is the result of trauma, poor postural habit, degenerative processes or derangement (McKenzie 1981, 1990). In dysfunction syndrome, structurally impaired tissue gives rise to pain with normal mechanical end-range loading.

In the lumbar spine, dysfunction is the second most common mechanical syndrome after derangement, but still is not common, with different studies classifying between 4% and 19% of patients in this syndrome (Razmjou *et al.* 2000a; Rath *et al.* 1989 in Robinson 1994). Derangement is the most common classification, but in some cases, following reduction, an 'underlying dysfunction' is revealed. The derangement is always treated first as the main source of symptoms. Any underlying dysfunction can then be addressed. Often after the derangement is reduced, no dysfunction remains to be treated.

Sections in this chapter are as follows:

- categories of dysfunction
- pain mechanism
- clinical picture
- physical examination
- management of dysfunction syndrome
- instructions to all patients with dysfunction syndrome
- literature on stretching
- management of extension dysfunction
- management of flexion dysfunction.

Categories of dysfunction

Dysfunction affects peri-articular, contractile or neural structures (McKenzie 1981, 1990; McKenzie and May 2000). In an articular dysfunction, end-range movement in one or more directions, which puts tension or compression on the affected structure, provokes the pain. In a contractile dysfunction, pain is experienced during movement that loads the affected tissue, which can be active or resisted. Contractile dysfunction occurs predominantly in tendons; muscle tissue, being well vascularised, in general heals uneventfully. The number of lengthy tendons taking heavy loads that exist in the limbs is probably the reason for the common occurrence of contractile dysfunction in peripheral musculoskeletal conditions. Contractile dysfunction is described elsewhere (McKenzie and May 2000). There is also a specific form of dysfunction involving the nerve root or dura complex known as an adherent nerve root (ANR); this is described in Chapter 29. In this syndrome, placing tension on the course of the involved nerve reproduces symptoms.

In extremity problems, it is relatively straightforward to distinguish articular from contractile dysfunction, whereas in the spine the distinction is not so clear. In the spine the syndrome presents as articular dysfunction, but involvement of contractile tissues cannot be ruled out.

In a spinal dysfunction, when normal movement is attempted the range is restricted and structurally impaired tissues are prematurely placed on full stretch or compression. End-range articular loading reproduces symptoms. Attempts to move further towards end-range results in pain. Mechanical deformation of free nerve endings within these tissues produces pain at a restricted range of movement. Once a painful restriction is reached, attempts to push further into range increases the pain being experienced as greater mechanical deformation occurs within the abnormal tissue. The pain is felt at the end of the existing range and ceases when the end-range stretch is released.

Dysfunction is classified by the direction that is limited and painful, so in flexion dysfunction there is pain and limitation of movement on attempting end-range flexion. In extension dysfunction there is pain and limitation of movement on attempting end-range extension, etc. Dysfunction of ANR is dealt with separately (Chapter 29).

Pain mechanism

Dysfunction syndrome can follow a traumatic event or a previous derangement, or may arise insidiously following poor postural habits or degenerative changes.

Trauma

Pain in the dysfunction syndrome should be seen in the context of the healing process following soft tissue injury (see Chapter 3 for description of the healing process). In ideal circumstances following tissue damage, regardless of the site of injury, healing passes through three stages (Evans 1980; Hardy 1989; Enwemeka 1989; Hunter 1994; Witte and Barbul 1997; Barlow and Willoughby 1992; Carrico *et al.* 1984). Each stage is necessary to restore the damaged structure to optimal function.

Several factors can operate to promote a less than optimal repair if remodelling is not properly implemented. The granulation tissue that repaired the damage can later act as glue to prevent movement between tissue interfaces. There may also be increased molecular cross-linkage – these processes may produce adhesion formation and impair collagen gliding (Hunter 1994; Donatelli and Owens-Burkhart 1981). Without the appropriate stresses, the scar tissue remains disorganised and structurally impaired.

Collagen repair contracts from the third week unless appropriate stresses are applied. Contracture of old scar tissue may in fact occur for years after the problem originated (Evans 1980; Hunter 1994). Failure to perform the appropriate tissue stretching leaves the repair process complete, but the remodelling stage incomplete – the individual may still be bothered by pain and limited function, and the tissue may be weak and prone to re-injury. The nerves, which infiltrated the tissue during repair, can now be sources of pain each time the scar is stretched or loaded.

It is generally not possible or even necessary to identify the specific structure that is at fault in dysfunction syndrome. All connective and muscle tissue heals in the same way by primary formation of granulation tissue and scarring. The only exception is cartilage, which is avascular and lacks the inflammatory response (Barlow and Willoughby 1992). Irrespective of where in the musculoskeletal system damage may have occurred – in ligament, muscle, intervertebral

disc, zygapophyseal joint capsule or aponeurosis – healing by fibrous repair eventually follows. Any of these structures may be a source of dysfunction; precise identification of the structure involved is not necessary to promote the appropriate remodelling strategy.

Derangement

Not every patient with dysfunction will present with a history of trauma; it may also follow a history of derangement. Typically the patient will have had an acute episode of back pain at some time in the past, which will have substantially improved, but not fully resolved. They are left with intermittent pain and a permanent restriction of movement. This is not due to the original derangement, but to tension or compression on the repair itself. Because stress of the repair is painful, the patient considers the injury to be still present and avoids the end-range movement that produces pain. Continuing avoidance of the painful end-range movement allows the structural impairment to persist and, without intervention, a general deterioration in the range of movement is inevitable. Continuing contracture of the fibrous collagenous scar tissue further limits mobility, and such inextensible repair will cause pain whenever the patient attempts full end-range movement.

The healing potential of intervertebral discs is generally considered to be limited. Being the largest avascular structure in the body, they have a very low capacity for repair and remodelling (Adams and Dolan 1995). Nonetheless, experimental animal models with artificially induced wounds show that the annulus fibrosus responds in the same way as other tissue to injury with the formation of scar tissue consisting of collagen and fibroblasts (Hampton *et al.* 1989; Kaapa *et al.* 1995; Smith and Walmsley 1951; Key and Ford 1948; Ahlgren *et al.* 1994). The repair process started in the first few days after the incision was made. There was no evidence of healing in the inner portion of the annulus where avascularity is total, only at its surface where a small vascular network exists. The normal lamellae structure was replaced by disorganised granulation tissue whose density increased over time. Notwithstanding the ultimately destructive effect of such tissue damage to the structure of the disc, and the subsequent degeneration that frequently appears, these experiments show that a normal reparative process occurs in part of the disc. The outer annulus fibrosus heals with fibrous repair tissue, is capable of scar and adhesion formation and thus may have a role in symptoms due to dysfunction.

Degeneration and poor postural habit

Dysfunction may also arise insidiously as part of the degenerative process. A common cause of reduced spinal mobility is poor postural habits maintained during earlier decades of life (McKenzie 1981). This is especially so when the individual is under-exercised, leads a largely sedentary lifestyle and their occupation is predominantly desk-bound or at the wheel of a vehicle.

There is a gradual reduction of spinal mobility that occurs with ageing, which is associated with changes in the motion segment (Twomey and Taylor 1994a; Taylor and Twomey 1994). However, there is considerable variability in the normal range of movement in the older population. It is likely that maintenance of activity and postural habits throughout life have a role in determining ultimate range of movement. Movements that are not performed regularly are likely to diminish. Later these movements may become uncomfortable, and are avoided as this is seen as the inevitable accompaniment to old age. Reductions are likely to be associated with considerable soft tissue adaptation, and the movement becomes impossible to perform without producing pain. The individual may assume that this is only the anticipated affects of maturity.

Physiologists estimate that up to half of what we currently know as usual ageing is a phenomenon of disuse (O'Brien Cousins 1998). There is abundant evidence that older individuals can positively affect their mobility and physical function by reversing the effects of a sedentary lifestyle and becoming more active (O'Brien Cousins 1998). The effects of stress deprivation on connective tissue are well known and include increased random deposition of collagen, increased collagen cross-links, formation of adhesions and contractures in and between the synovial membrane, capsule and other tissues, and generalised osteoporosis (Akeson *et al.* 1987; Bland 1993; Videman 1987). All these physiological changes reduce the available range of movement. Symptoms of stiffness and pain associated with a premature limitation of movement and related to contracture of the joint capsule are seen as part of the degenerative process (McCarthy *et al.* 1994; Threlkeld and Currier 1988).

Very often, because of normal activities of daily living and postural habits, flexion at the lumbar spine is better maintained, while extension becomes more and more difficult to perform. The individual may comment that they have been unable to lie prone, for instance

to sunbathe, for years. Due to inadequate extension in the lumbar spine, the capsule and ligamentous structures are placed on full stretch prematurely and pain is produced.

The situation can arise from similar restrictions of movement, but ones that remain painless. These are usually non-traumatic and related to a slow adaptation to a sedentary lifestyle. In such instances the contractures may be so extensive that it defies overstretching and the production of pain.

Clinical picture

Onset

Patients with dysfunction syndrome present with characteristic findings in the history-taking and physical examination. The individual will have a history of trauma or derangement, or in an older individual symptoms may have developed insidiously. In some people extensive contracture and loss of movement accompanying the ageing process and a sedentary lifestyle are present without pain. When severe back pain or significant trauma marked the onset of this episode, some time will have elapsed; at least six to eight weeks is probably necessary to allow dysfunction to develop. Since the onset the pain will have eased considerably, but is now unchanging. When the onset has been insidious the patient will be older, poor posture will be obvious, there may be an episodic history of back pain in the past, and symptoms and functional impairment may well be worsening gradually over time.

Pain from dysfunction sometimes develops in an episodic manner and appears to resemble derangement. These episodes of pain are triggered by excessive use, for example a vigorous afternoon in the garden. Overstretching of contracted soft tissues causes minor trauma and turns an intermittent pain into a constant ache. If the patient avoids the vigorous activity for a few days the ache subsides, but further scarring and contracture of repair tissue increasingly limits the available range of movement. A vicious circle is perpetuated unless treatment for a dysfunction is instigated.

Symptoms

Except in one instance, pain from dysfunction is felt locally around the spine with limited radiation. Only in the presence of an adherent nerve root (ANR) is pain referred to the thigh and/or calf. ANR is a

type of dysfunction that can follow the resolution of a derangement with referred symptoms. Other than this one exception, all dysfunctions present with back pain only.

Intermittent end-range pain

Pain is always intermittent in dysfunction syndrome – this is a key identifying characteristic. It will not persist for long periods, but is always associated with certain movements. Sometimes this is apparent to patients, and they comment that, for instance, every time they bend forward they produce their pain, but this goes once they are upright.

Whether apparent to patients or not, consistency of aggravating factors is another key identifying characteristic. It is always end-range movements that provoke symptoms in articular dysfunction; this is when the adaptively shortened tissue is stressed or compressed. This happens much sooner in a patient with dysfunction than in a normal person, and the greater the loss of function, the more often pain is provoked. The same movement(s) will consistently provoke their pain, with extension and flexion being the most commonly limited and painful movements. End-range pain, consistently brought on by the same movement(s) and not amenable to rapid change, is another key identifying characteristic of dysfunction.

The patient is usually also aware that they are less flexible than they were. Although painful, patients often also feel that 'it just won't go'. There is a noticeable resistance to movement at the same time as the pain.

Often the patient with dysfunction states that he or she feels no pain when they are active and moving about. During general activity, end-range stretch is mostly avoided, whereas at rest, end-range positions that provoke pain are more readily assumed.

The vital questions in identifying a dysfunction thus relate to consistency of the aggravating factor and relief from symptoms once the aggravating position is released. 'Does bending [for instance] always bring on your symptoms?' 'When you stop bending [for instance], does the pain go away or does it persist?' If the patient responds that *sometimes* a movement is painful and *sometimes* not, or that *sometimes* the pain persists for hours at a time, derangement is the likely diagnosis and a dysfunction classification should be discounted. If, however, they respond that *always* when they perform

that movement it is painful, but that *always* afterwards the pain goes, a dysfunction is more likely.

Physical examination

Poor posture may be noted. Slouched sitting, if at end-range, may provoke symptoms in a flexion dysfunction (painful limitation of flexion), in which case posture correction will abolish symptoms. Return to slumped sitting produces symptoms each time. In a neutral sitting or standing position the patient has no pain; only on attempting full movement is there pain.

There will always be reduced movement in spinal dysfunction. When dysfunction results from some discrete past event such as an accident or derangement, movement loss is likely to be asymmetrical. When dysfunction results from poor postural habit or spondylosis, movement losses are generally symmetrical in all directions and affect many segments. Sagittal movements will most likely be affected. Upon attempting the movement at premature end-range, pain will be produced.

In a dysfunction, repeating the painful movement *consistently* produces symptoms on every occasion at end-range, and there is no change in range or any other aspect of mechanical presentation. There is no pain during the movement, only when their full but restricted end-range is achieved. Once the repeated movements cease, pain rapidly abates. Within a few minutes of stopping the patient will be no worse and will return to a pain free state. Repeatedly performing the same movement will, without fail, every time reproduce their pain at end-range, which abates when movement ceases. Repeated movements in one direction have no effect on pain or range of the opposite movement. Unlike derangement, there are no rapid changes in symptomatic or mechanical presentations in dysfunction syndrome. Symptoms and mechanical restriction will persist for many weeks.

Table 28.1 Articular dysfunction syndrome – criteria (all will apply)

History:

- spinal symptoms only (except adherent nerve root)
- intermittent symptoms
- no symptoms if end-range avoided.

Physical examination:

- at least one movement is restricted, and the restricted movement consistently produces concordant pain at end-range only, and
- there is no rapid decrease or abolition of symptoms, and
- no lasting production and no peripheralisation of symptoms.

A dysfunction classification should be suspected during the history-taking, and the appropriate focused questions given above should be asked. The physical examination should merely endorse the initial clinical suspicions. The diagnosis should be confirmed at review after twenty-four to forty-eight hours' mechanical evaluation. If at any point there is doubt concerning the classification, hypothesis testing should focus on the diagnosis of derangement, which also frequently presents with end-range pain. This is the most common mechanical syndrome and is susceptible to aggravation if mismanaged. If a derangement is the classification, it is not possible at the outset to make a diagnosis also of underlying dysfunction. The derangement is always treated first as the main source of symptoms. These patients frequently also present with end-range pain, and it is not possible to know if there is an underlying dysfunction until the derangement is reduced.

Management of dysfunction syndrome

The treatment of adhesions, contractures or adaptive shortening as exists in an articular dysfunction essentially requires the application of movements that encourage the process of remodelling. Only with the application of such loading strategies will normal tissue function be re-established. Ideally such movements commence during the stages of repair and remodelling in the weeks after an injury (Evans 1980; Hardy 1989; Hunter 1994; Barlow and Willoughby 1992). If appropriate and graded tension is applied to injured tissue during the proliferative and remodelling phases of wound healing, adhesions and contractures will not form and dysfunction is prevented. The longer the time lapse between repair and the initiation of the recovery of full function, the more consolidated the scar tissue. Thus the task of remodelling will be more difficult and the time to recovery longer. Once the scar tissue is well consolidated, *the very nature of the abnormal tissue prohibits a rapid recovery of function.*

In such cases, the remodelling of collagen by applying a long-term structured exercise programme is necessary. By applying regular stress sufficient to provide tension without damage, collagen undergoes

chemical and structural changes that allow elongation and strengthening of the affected tissue. Because tissue turnover is slow, one must recognise it may be a slow process. If the contracture has been present for some time, the remodelling programme will have to be followed for several months; Evans (1980) reports that some patients may have to exercise for the remaining years of their life. Stretching of old injuries should be routinely practised, especially prior to participation in sporting activities (Hunter 1994). The animal experiment of Arem and Madden (1976) showed that 'old' scar tissue might be unresponsive to a remodelling programme. Well-established contractures, especially where the original healing process has been interrupted by repeated re-injury causing the production of more inflammatory exudate, may thus be resistant to improvement.

The process of recovery in dysfunction is thus lengthy and should be measured in weeks and months rather than days. During this period, because the nature of change is slow in this syndrome, patients may become frustrated due to the lack of apparent change. They must be encouraged to persevere as a programme of remodelling is the only solution, and should be warned early that a relatively lengthy period of rehabilitation awaits them.

Given the tendency of old scar tissue to contract over time, stretching must be performed frequently if remodelling is to occur. If the intervals between stretching procedures are too long, the length of time when no stretching takes place negates the effect of stretching. In dysfunction syndrome, exercises to restore movement and function must be performed repeatedly at two- to three-hour intervals throughout the day – each session should consist of ten to fifteen stretches.

To achieve a remodelling effect, exercise must be firm enough to cause change but not so excessive as to produce micro-trauma. If no strain pain is felt when the exercise is done, it is a waste of time. ***The pain that the patient complains of must be produced each time the stretch is performed.*** However, equally important, the pain must quickly subside when the stretching is completed. If pain persists long after the exercises are finished, either overstretching has occurred, with micro-trauma and further tissue damage, or else the original classification was wrong or has changed, and a derangement may be responsible for the exacerbation.

Instructions to all patients with dysfunction syndrome

Patients will be attending the clinic with pain. To be told that they must go away and regularly cause the pain that they are complaining about needs a very good explanation to gain their adherence to the programme. As long as patients are given a good justification for performing the exercises, most will follow the advice that is given. Most understand the idea of scar tissue that needs to be 'stretched' to recover full movement; that stretching the scar hurts, and on releasing the stretch the pain will abate. Reassure patients that when their pain is consistently reproduced they are affecting the necessary tissues – 'if it doesn't hurt, it isn't right'. Before giving patients the following guidelines, it is essential they understand the reason they are performing the exercises.

Table 28.2 Instruction to patients with dysfunction syndrome

- exercises must be performed regularly throughout the day, every two to three hours
- if patients are unable to exercise as regularly as recommended, recovery of full function is likely to take longer
- at each session, perform ten to fifteen stretches
- if the exercise does not produce their pain, it has not been performed properly
- the exercise must consistently reproduce their pain each time
- the pain should have subsided within ten minutes after the completion of the exercises; mostly it will abate much quicker
- if pain from the procedures persists constantly afterwards for a long period, either overstretching has occurred, in which case repetitions must be reduced, or the original classification was mistaken or has changed – in either case a review is necessary
- if the patient feels they are getting worse, they must stop exercising and return for a review appointment
- there will be no rapid changes in range of movement – if they experience a dramatic change in function or range, they must return for re-evaluation
- if there is a spread of pain distally or a rapid deterioration in their situation, they must stop exercising and return for a review appointment.

Particular caution should be applied if the patient has recently recovered from a derangement or has had regular episodes of back pain in the past and presents with a flexion dysfunction. In such instances, flexion exercises will have to be performed regularly; these, however, can constitute a risk in provoking a derangement. It should be emphasised to the patient that they should never be worse when

they have stopped the exercises, they must not provoke constant or peripheralising pain, and their ability to extend should be maintained.

Clinician techniques and passive modalities have little or no role in the treatment of dysfunction. Only the patient is able to perform the appropriate exercise with sufficient regularity to ensure remodelling takes place. Manipulation procedures may cause minor trauma and perpetuate the cycle of repair and failure to remodel. Mobilisation may possibly be able to generate the appropriate tissue tension, but without regular exercises, the timespan between remodelling sessions is totally inadequate to achieve a lasting change. Stretching two or three times a week or even once a day is insufficient. Heating tissues may make collagen more pliable, but again, the effect is short-term, the heating may not be deep enough for the affected tissues, and is in any case unnecessary to achieve improved function.

Literature on stretching

There is a large amount of literature on stretching that provides a variety of opinions. Several authors (Smith 1994; Safran *et al.* 1989; Wilkinson 1992; Shrier and Gossal 2000) have reviewed the topic. There are a plethora of individual studies that demonstrate rather contradictory results (Bandy and Irion 1994; Bandy *et al.* 1997, 1998; Bannerman *et al.* 1996; Magnusson *et al.* 1998b; McNair *et al.* 2000; Hubley *et al.* 1984; Wallin *et al.* 1985; Clark *et al.* 1999; Henricson *et al.* 1984; Taylor *et al.* 1995; Wessling *et al.* 1987; Lentell *et al.* 1992; Brodowicz *et al.* 1996; Funk *et al.* 2001).

Bybee *et al.* (2001) compared the ability of static or repeated stretching to affect lumbar extension range against a control group. The repeated stretch group performed 81% of their exercises in standing. Both groups showed significant increases in range compared to baseline and the control group, with a greater increase in range in the repeated stretch group.

Unfortunately, all these studies lack direct clinical utility as they concern asymptomatic volunteers. Their findings cannot be applied to a patient population with scarring, adhesions, contractures or imperfect repair in which the tissue state is so different. No directly applicable work has been done in this area, and there should be caution about extrapolating these results to the symptomatic population (Shrier and Gossal 2000).

Various studies have used animal models to examine the effect of stretching, but mostly these have examined the effect during the early stage of the healing process. These studies show the value of early regular mobilisation, starting towards the end of the first week. Motion started earlier than this or too aggressively can be detrimental to ultimate strength and function. Early motion leads to greater improvement in orientation of blood vessels and collagen fibres, greater flexibility and greater tensile strength (Gelberman *et al.* 1981, 1982; Arem and Madden 1976). In older scar tissue there is less potential to achieve improved flexibility (Arem and Madden 1976). These studies demonstrate the anatomical truism that form matches function. Cells, tissue and structures are sensitive and responsive to changes in physical load (Merrilees and Flint 1980; Gelberman *et al.* 1981). Their ultimate utility is predicated on use; simply put, 'use it or lose it'.

The value of early controlled repeated movements in improving function has been demonstrated in healing hand extensor tendons in humans (Evans 1989). A single case study of scarring around the lips due to acid burns has demonstrated the effectiveness of regular sub-maximal stretching to produce a new and longer tissue length (Bahnof 2000). Scarring was sufficient to restrict mouth opening; treatment consisted of passive manual stretching performed by the clinician several times a week and home self-stretching exercises several times daily, continued for six or seven weeks. Stretches were performed statically, held for ten to twenty seconds, and repeated ten to fifteen times. The restricted mouth opening improved from 28mm to 46mm over the time period. Low-load prolonged stretch has been found to be much more effective than high-load brief stretch at increasing range of movement in very elderly subjects with knee contractures due to immobility (Light *et al.* 1984).

Repeated stretches are necessary to overcome the inherent resistance in all connective tissue. Collagen fibres at rest assume a wavy shape, known as *crimp*. The first effect of a tensile force is to straighten this crimp, which occurs at low loads, after which further elongation is resisted more strongly. Creep is the progressive deformation of a structure under a sustained constant load due to the rearrangement of collagen fibres and proteoglycans and the expulsion of water (Bogduk 1997). Upon release from the force, as long as this has not been excessive, the structure begins to recover. Within a relatively short time full recovery can occur and the structure returns to its original shape as the fluid equilibrium is restored.

However, restoration of the initial shape of the structure occurs more slowly and to a lesser extent than the initial deformation. The different rate at which recovery happens between loading and unloading is known as *hysteresis* (Bogduk 1997). The structure may not immediately return to its original length, but remains slightly longer. This difference between initial and final length is known as *set*.

If the behaviour of collagenous structures to mechanical loading is extrapolated to imperfect repair tissue, certain assumptions about appropriate management can be made. Loading needs to overcome initial crimp and be sufficient to cause a creep in the tissue. Secondly, loading needs to be repeated regularly enough to bring about a lasting change in the tissue's flexibility and properties. A programme of exercises that is capable of remodelling imperfect repair or contracted tissue needs to be applied very regularly, over a period of many weeks, in order to produce a lasting change. Only with regular and repeated movements will there be enough loading to remodel the cellular structure. There will be occasions when attempts to remodel fail because of the dense nature of the repair itself.

Management of extension dysfunction

The most common form of dysfunction in the lumbar spine involves a loss of extension (McKenzie 1981). The loss of extension in some is quite marked and with it may come an inability to sit with lordosis, stand fully upright or lie prone. This generalised loss of extension can increase the likelihood of derangement, and unless actively treated, leads to a continuing deterioration of function.

As with all treatments, a thorough and convincing explanation to the patient is a prerequisite to gain their committed involvement. Procedures to be used in this syndrome all come under the extension treatment principle.

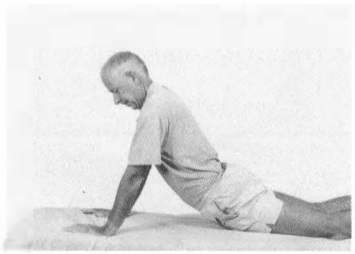
Procedures to be used:

- extension in lying (Procedure 5)
- extension in standing (Procedure 9).

Regularity:

- ten times every 2 – 3 hours.

Photos 187, 188, 189:
Extension in lying (187). Extension in lying with patient overpressure (188). Extension in standing (189).



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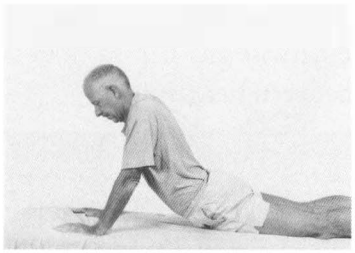

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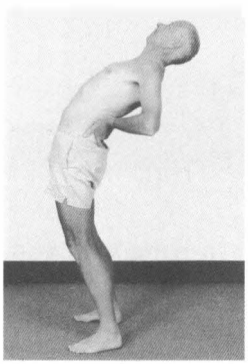

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Expected response:

- temporary (ten minutes maximum) localised back pain
- pain produced with exercise is concordant with patient's complaint
- pain occurs at limited end-range extension
- pain abates rapidly when out of this position
- all other movements asymptomatic and normal for them, and remain so
- new pains around thoracic spine and shoulders, due to new exercise
- improved function and pain takes 4 – 6 weeks.

Maintenance:

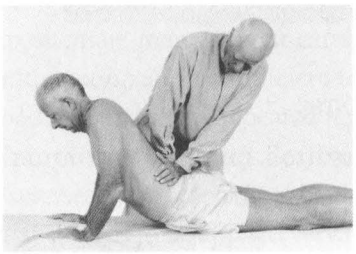
- once function and pain are improved, patients should be advised to maintain ten repetitions of extension in lying 1 – 2 times per day.

Progressions:

- if improvements slow down or cease, the following progressions, in the order given, should be considered:
- only use one new procedure at any one session
- always wait at least twenty-four hours to evaluate the response and before considering further force progressions

- repeat force progressions a maximum of two sessions if no definite improvements occur
- the patient must continue with the home exercise programme, otherwise any benefit from the force progressions will be lost between treatments.

Photos 190, 191, 192:
Extension in lying with clinician overpressure (190). Extension in lying with belt fixation (191). Extension mobilisation (192).



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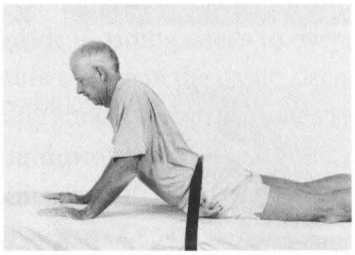

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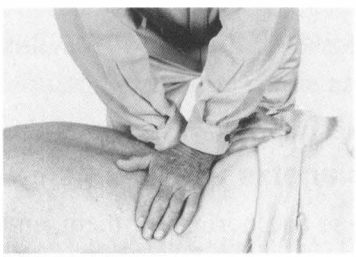


192

- extension in lying with clinician overpressure/belt fixation (Procedures 6a/6b)
- extension mobilisation (Procedure 7).

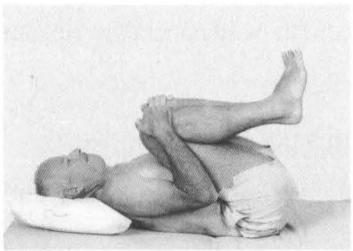
Management of flexion dysfunction

Loss of flexion is the second most common dysfunction to affect the lumbar spine (McKenzie 1981). Patients with this problem are limited in their amount of flexion. In a few, as they flex forward, with a reasonably normal range of movement, they unintentionally deviate to one side. This alteration from the normal sagittal pathway may be due to adhesions, and if the patient is forced to maintain a purely sagittal plane, will be found to have a much greater loss of flexion.

As with all treatments, a thorough and convincing explanation to the patient is a prerequisite to gain their committed involvement.

Procedures to be used mostly come under the flexion treatment principle:

Photos 193, 194, 195, 196, 197:
Flexion in lying (193). Flexion in sitting (194). Flexion in sitting with patient overpressure (195). Flexion in standing (196). Extension in lying (197).



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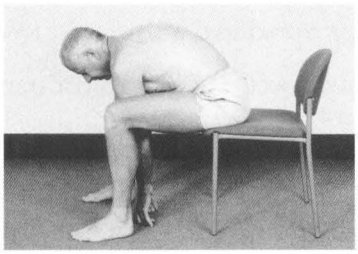

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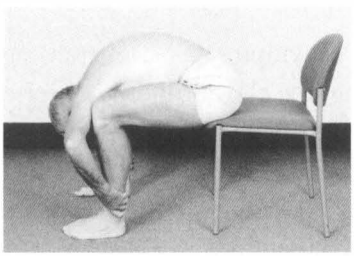

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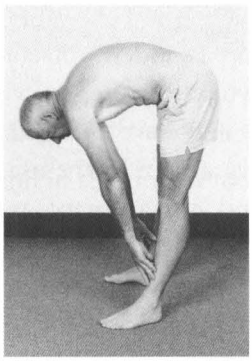

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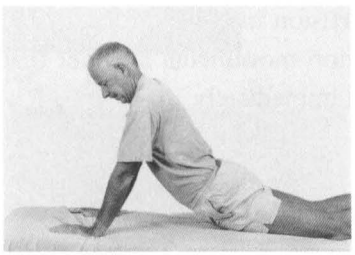


197

- flexion in lying – to be continued until full range and pain-free (Procedure 18)
- flexion in sitting (Procedure 19)
- flexion in standing (Procedure 20)
- extension in lying (Procedure 5) – for prophylaxis.

Regularity:

- flexion in lying – ten times every 2 – 3 hours
- flexion in standing – due to the effect of gravity, this procedure places more stress on the spine than flexion in lying and therefore should be introduced with care, especially when dysfunction results from *recent* derangement; this procedure can exacerbate symptoms
- initially: five repetitions, five times per day; after a few days, if no exacerbation: ten times every 2 – 3 hours
- extension in lying (Procedure 5) – ten times after repeated flexion.

Expected response:

- temporary (ten minutes maximum) localised back pain
- pain produced with exercise is concordant with patient's complaint
- pain occurs at limited end-range flexion
- pain abates rapidly when out of this position
- all other movements asymptomatic and normal for them, and remain so
- improved function and pain takes 4 – 6 weeks.

If patients report a sudden worsening of symptoms, peripheralising or persistent symptoms after exercising, the classification may have been wrong or may have changed. A derangement may have been provoked or aggravated by the use of flexion exercises. These must be stopped and management as for a derangement should be instigated. Likewise, it is important to be cautious when recovering flexion following a recent derangement (see Chapter 24, ***Recovery of function*** section for more detail). Extension in lying (Procedure 5) should always be used following flexion movements in order that any posterior disturbance is corrected immediately.

Progressions:

Force progressions that may be needed are all patient-generated:

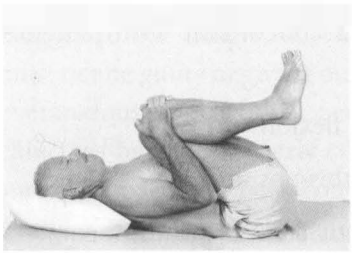
- flexion in sitting (Procedure 19)
- flexion in standing (Procedure 20)
- clinician-generated force progressions are rarely necessary.

Sometimes patients present with deviation on flexion. Due to adaptively shortened structures within the intervertebral segment, the patient is forced to deviate to one side during flexion, no matter how hard they try to maintain the normal sagittal pathway. In such instances, it may be necessary to use flexion procedures with a lateral component.

Procedures to be used:

- flexion in lying (Procedure 18) – to test effect of flexion over twenty-four hours; as long as no overall worsening or peripheralisation, then commence

Photos 198, 199, 200, 201, 202, 203:
Flexion in lying (198). Flexion in step standing (199/200). Flexion in standing (201). Rotation in flexion (202). Extension in lying (203).



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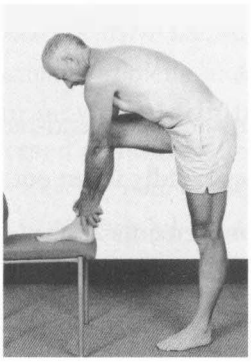

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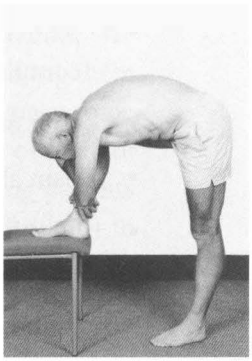

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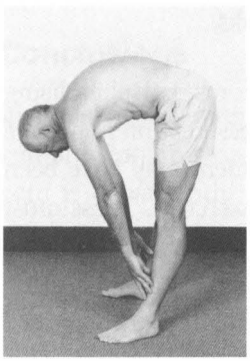
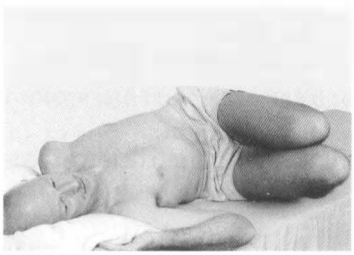


201

- flexion in step standing (Procedure 22)
- flexion in standing (Procedure 20)
- rotation in flexion (Procedure 23)
- extension in lying (Procedure 5) – for prophylaxis.



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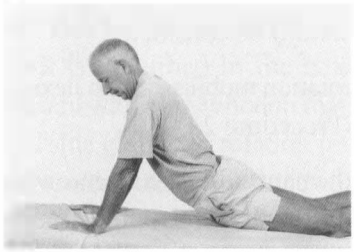


203

Regularity:

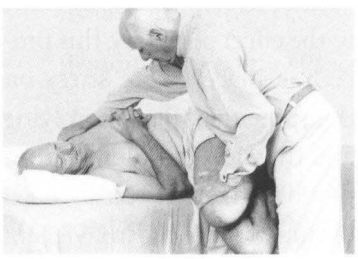
- flexion in lying – ten times every 2 – 3 hours
- flexion in step standing – due to the effect of gravity, this procedure places more stress on the spine than flexion in lying and therefore should be introduced with care, especially when dysfunction results from recent derangement; this procedure can exacerbate symptoms
- initially: 5 x 5 x per day; after a few days, if no exacerbation: ten times every 2 – 3 hours
- extension in lying (Procedure 5) – ten times after repeated flexion.

Expected response:

- temporary (ten minutes maximum) localised back pain
- pain produced with exercise is concordant with patient's complaint
- pain occurs at limited end-range flexion
- pain abates rapidly when out of this position
- all other movements asymptomatic and normal for them, and remain so
- improved function and pain by 4 – 6 weeks.

If patients report a sudden worsening of symptoms, peripheralising or persistent symptoms after exercising, the classification may have been wrong or may have changed. A derangement may have been provoked or aggravated by the use of flexion exercises. These must be stopped and management as for a derangement should be instigated. Likewise, it is important to be cautious when recovering flexion following a recent derangement (see Chapter 24, page 571 *Recovery of function* for more detail). Extension in lying (Procedure 5) should always be used following flexion movements in order that any posterior disturbance is corrected immediately.

Photos 204, 205:
Rotation mobilisation in flexion.



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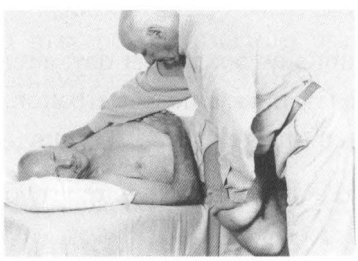


205

Progressions:

- clinician-generated force progressions are rarely necessary
- if improvements slow down or cease, the following progression should be considered
- rotation mobilisation in flexion (Procedure 24)
- the patient **must** continue with the home exercise programme; otherwise any benefit from the force progressions will be lost between treatments.

Note – avoidance flexion

Although it is important to be cautious when introducing flexion procedures in certain circumstances, it is important also not to exaggerate the 'danger' of what are normal daily movements. We must not be guilty of giving our patients problems and making them overanxious or fearful of certain movements. In the past some clinicians have been overly concerned about the effects of flexion, and this anxiety has been conveyed to the patient. Making patients fearful about normal movements is not helpful for a full return to function.

Conclusions

In spinal dysfunction syndrome, pain is due to premature stretch or compression on structurally impaired soft tissue. Thus the patient's pain is consistently reproduced when the appropriate end-range movement is performed. When the loading is released, the pain will abate. In order to rectify this situation, a remodelling programme must be instigated. Such a programme must regularly and repeatedly provoke the patient's pain. Recovery of normal range of movement and pain-free function will not occur rapidly, but over a period of weeks or months. In some patients with gross impairment, a remodelling programme may not be effective. Education and an appropriate regular exercise regime are the essential components in the treatment of dysfunction syndrome.

The lumbar spinal dysfunctions that are most commonly seen in the clinic are those relating to sagittal plane movements – extension, flexion and adherent nerve root (ANR). The management of the first two conditions has been described in this chapter; management of ANR is described in the next chapter. Occasionally patients may present with a dysfunction that is related to other planes of movement, and side gliding or rotation provokes the pain. In such instances, the movement that reproduces the patient's pain is the one chosen to regularly exercise, and the management takes a similar form to that described above.

The detail provided in this chapter is summarised in the form of criteria and operational definitions contained in the Appendix – these are essential for identification of the different syndromes.

29: Dysfunction of Adherent Nerve Root (ANR)

Introduction

Adherent nerve root (ANR) is a specific form of dysfunction that sometimes occurs following resolution of derangement with sciatica or after surgery. It is the only time in dysfunction when distal pain is experienced. It is also the only time when distal symptoms are repeatedly produced as part of management. Because the history and presentation of ANR are very distinct, this separate chapter has been allocated to its description. However, in every other way it behaves typically as a dysfunction. Pain is produced at limited end-range, which does not undergo any rapid changes, and pain abates once the end-range position is released. In this instance, any movement or position that causes tension of the sciatic nerve root w`ll provoke symptoms.

This chapter describes the development, clinical presentation and management of ANR. As patients with this syndrome present with thigh and/or calf pain, it should be a consideration in those with distal symptoms.

Sections in this chapter are as follows:

- development of adherent nerve root
- clinical presentation
- history
- physical examination
- management.

Development of adherent nerve root

These patients present with unilateral asymmetrical symptoms which may extend below the knee; sometimes pain is only felt in the thigh. A secondary cause of intermittent persisting sciatica is nerve root adherence. Constant sciatica becomes intermittent as adherence develops. Thus symptoms will have improved from onset, but have become unchanging. It should be remembered that nerve root adherence may be developing or fully developed depending on the

time since onset that the patient is assessed. The longer symptoms have been present the longer remodelling will take; a developing nerve root adherence may resolve much more quickly.

Experimental disc lesions made in dogs have led to the development of adhesions between the disc scar and the dura or nerve root (Key and Ford 1948). Adhesions between disc or posterior longitudinal ligament and nerve root or dura have also been noted at surgery (O'Connell 1943, 1951; Begg *et al.* 1946). Adherent nerve root may occur following a substantial disc lesion with nerve root symptoms when the original displacement has resolved, but symptoms remain due to tethering between the disc and nerve root and/or dura. *"The extradural nerve may be adherent to the posterior longitudinal ligament: this structure has lost its normal lustre or may be roughened by adhesions, and groups of small new blood vessels may be present in it. These findings probably indicate a healed or healing stage of a severe injury to the posterior longitudinal ligament and annulus, and tension-producing adhesions between nerve and ligament are frequently the only obvious cause of the symptoms"* (O'Connell 1951).

Cooper *et al.* (1995) reported that periradicular fibrosis associated with herniated intervertebral disc was a common finding at surgery and may well be the cause of persisting pain in many patients with chronic back and leg pain. Analysis of tissue removed from eleven patients at surgery revealed adhesions and perineural fibrosis. Patients all had proven disc herniations and had experienced symptoms for between one month and two years – mean duration of symptoms, eleven months. They discounted proposals that discal material could induce a local autoimmune reaction that would damage neural tissues. The authors also refer to the *"long held but unsubstantiated belief that an inflammatory component is etiologically involved in herniated intervertebral disc associated radiculopathy"*. In none of the tissue samples was there evidence of inflammatory cells. The results of this study relate the presence of perivascular and periradicular fibrosis to intraspinal vascular disruption, revascularisation and congestion caused by discal herniation.

Cadaver studies also attest to the presence of adhesions between the dura and the posterior longitudinal ligament (Yildizhan *et al.* 1991; Parke and Watanabe 1990). Parke and Watanabe (1990) examined fifteen spines and combined their findings with the earlier work by Blikra (1969), who investigated forty spines. Dense adhesions

between the dura and the posterior longitudinal ligament and the outer layer of the annulus fibrosus were found in 36% of specimens at L5, 40% at L4, 16% at L3 and 2% at L2. Nerves infiltrated the tissue taken from the adhesions. The origin of these adhesions is unknown, but their presence in newborn cadavers and in those without a history of back pain suggests that they are congenital rather than pathological (Yildizhan *et al.* 1991).

Nerve root adherence or fibrosis resulting from the repair consequent to discal herniation is not uncommon, but the majority of patients recover without developing this complication. Bed-rest for several weeks during the acute stage would appear to make the development of root adherence from sciatica more likely. In the case of a resolving derangement, the intensity of sciatica will gradually diminish and in many cases pain will become intermittent. During this period the range of flexion in standing should also increase, as will the range of straight leg raising. If the range of flexion in standing improves as the intensity of sciatica subsides, the patient will not develop nerve root adherence. During recovery of function, it is always essential to ensure the full and pain-free return of flexion mobility. During the recovery from sciatica, and certainly post-surgery, performing flexion in standing as far as pain permits once a day helps to achieve this.

If, despite overall improvement, the range of flexion in standing remains limited and unchanging, patients may well have developed nerve root adherence. Patients who remain cautious of resuming normal activity and movement are likely candidates for this complication. Adherent nerve root also occurs sometimes in patients who have had surgery for sciatica, and have not received the appropriate rehabilitation exercise programme. Assessment should seek to differentiate between derangement and adherent nerve root. If adherence is the cause of the remaining symptoms, such patients should be provided with a structured exercise programme designed to remodel any structures that are adherent or contracted. This management is detailed below.

Nerve root tension tests may be positive whether nerve root adherence or derangement is responsible for the persisting but intermittent symptoms. Information from tension tests, such as straight leg raising test, Lasegue's test, and slump test, is unhelpful unless a distinction is made between the syndromes of dysfunction and derangement. Is the test positive because of fibrosis and adhesions about the nerve

root, or because of tension or compression acting on the nerve root due to a disc herniation? Failure to identify derangement as the cause of pain has caused much unnecessary prolonging and aggravation of symptoms in patients where treatment has been applied to 'stretch' supposedly shortened or tethered neural structures. When a derangement is reduced, tension signs disappear immediately in much the same way as trigger points disappear after the reduction of derangement.

Clinical presentation

If sciatica persists beyond twelve weeks and is now felt intermittently rather than constantly, it is possible for the symptoms to arise from two causes:

1. a recurring derangement
2. the development of nerve root adherence or fibrosis.

Furthermore, if after surgery symptoms persist or recur, adherence as a result of surgical scarring may be the cause.

Fibrous repair following disc herniation or protrusion can cause adherence of the nerve root/dura complex to the disc wall, which in turn limits the mobility of the root itself. Any attempt to stretch the tethered structure produces increased tension and causes pain and tightness in the leg. Thus the patient cannot bend with straightened knees, nor sit upright with legs outstretched. Often the patient relates that when they attempt such a movement, they feel a tight band down their leg.

The change occurring in the nature and behaviour of perceived symptoms during the transition from tension or compression of the nerve by displacement of discal tissue to tension caused by adherence is slow and imperceptible. During this transition, the patient is usually unaware of the slow and subtle changes that are occurring to the nature and behaviour of his or her symptoms. This condition is a dysfunction and will behave in the consistent manner of all dysfunctions. It will not produce neurological deficit if the initial episode has not already done so.

Table 29.1. Adherent nerve root – clinical presentation (all will apply)

History:

- history of sciatica or surgery in the last few months that has improved, but is now unchanging, *and*
- symptoms are intermittent in leg
- symptoms in the thigh and/or calf, including 'tightness', *and*
- consistent activities produce symptoms – typically touching toes, long sitting, walking, *but*
- pain in leg does not persist on ceasing movement or changing position.

Physical examination:

- flexion in standing is clearly restricted and consistently produces concordant pain or tightness at end-range, *and*
- there is no rapid reduction or abolition of symptoms and no lasting production of distal symptoms, *and*
- moderate to major loss of flexion in standing
- flexion movement will improve if knee is flexed
- flexion in lying has no effect
- there will be no rapid changes in mechanical presentation with repeated movement testing.

History

Where nerve root adherence is the cause of intermittent persisting sciatica, the patient will describe an improvement from the pain and disability at onset, which occurred at least eight weeks previously, often much longer. However, the status of the condition is now unchanging. The symptoms always behave in the same manner on a daily basis. The same activities always provoke the pain, which may be felt in the calf only or in the thigh and calf. Some back pain may still be present, or appears in tandem with the leg symptoms. In addition to pain, the patient describes that they feel 'a tight band' running down the back of the leg when they stretch it. Any activity that exerts tension on the nerve root reproduces the symptoms. Forward bending from standing with knees extended and sitting upright with legs out straight, as in the bath, provoke the pain. Driving a motor vehicle and walking are other activities that can reproduce symptoms. The patient may have noticed that they have failed to regain their normal mobility of bending since the onset of back pain. By flexing the spine with the knees bent, a de-tensioning position, the patient may avoid pain for much of the time.

In some patients back pain may have persisted since the onset, which may be intermittent or constant. This does not respond to reductive movements, but may be temporarily worsened with prolonged sitting. Flexion in standing produces or increases back pain, which returns to its former status after testing. The back pain improves as the leg pain from the adherent nerve root resolves.

Adherent nerve root is a type of dysfunction and will behave consistently, as dysfunctions do. The patient reports that the same activities *always* produce their symptoms, but that the leg pain does not remain once they resume a neutral position. Similarly, the painful movement, flexion in standing, is *always* restricted.

Physical examination

Patients with nerve root adherence are able to flex the lower spine with little difficulty, provided the knees and hips are flexed. The nerve root is relaxed and not under tension when this manoeuvre is performed, and consequently no pain is experienced during the performance of flexion in lying or flexion in sitting with knees flexed.

The distal symptoms appear only at the now limited end-range of flexion in standing, or when the patient is asked to sit upright on the treatment table with the legs outstretched. Most patients with nerve root adherence are unable to sit in such a manner and usually must lean back supported with arms outstretched behind.

When the patient with nerve root adherence is standing, the nerve root is off tension and so pain status in the leg is always nil. When the patient performs flexion in standing, the range of motion will always be at least moderately restricted, and pain or tightness will result when the limitation is reached. In some cases this restriction can be extreme, the patient being unable to reach to the knees without bending them. The range of motion, irrespective of how often it is repeated or how vigorously it is performed, does not increase or decrease with repetition. Pain will not be felt during the motion itself, but only towards and at end-range. The symptoms will not worsen with repetition. On cessation of flexion in standing, the pain will subside in a few minutes.

As noted above, some patients with adherent nerve root have back pain that has persisted since onset. This pain may be intermittent or constant, and will be produced or increased with flexion in standing. Once testing stops, the pain will return to its former level.

In the presence of an adherent nerve root, even if several sets of repetitions in flexion are performed, the response will always be the same. The mechanical and symptomatic presentations remain unchanged, as will the range of and response to extension. However, if the symptoms are the result of derangement, end-range pain may also be produced. Flexion exercises, a necessity in management of adherent nerve root, have the potential to destabilise the repair if the symptoms arise from posterior derangement. It is therefore important to distinguish between the two entities. If symptoms were the result of derangement, repetition of flexion would cause the pain to peripheralise or worsen, and remain worse afterwards. The mechanical presentation may also change, with extension becoming obstructed following repeated flexion.

Table 29.2 Criteria definition for adherent nerve root (all will apply)

- history of sciatica or surgery in the past that has improved, but is now unchanging, *and*
- symptoms are intermittent, *and*
- symptoms in the thigh and/or calf, including 'tightness', *and*
- flexion in standing is clearly restricted and consistently produces concordant pain or tightness at end-range, *and*
- there is no rapid reduction or abolition of symptoms and no lasting production of distal symptoms, *and*
- flexion in lying does not produce distal symptoms.

Management

Nerve root adherence is a sequel to the repair process itself and is essentially a dysfunction. Management should provide a regular remodelling programme that will eventually alter the adherence and tethering that limit mobility. To remodel contracted or scarred fibrous structures, it is necessary to stress the affected tissue without disrupting it and causing further damage. At the same time, it is necessary to ensure that stress is applied with enough regularity and force so as to cause remodelling.

During the process of stretching, some sciatic pain and tightness or discomfort must be felt, but any discomfort produced should subside within ten minutes.

It should be noted that nerve root adherence is the only condition in which the deliberate provocation of distal symptoms can be permitted during the application of the treatment itself. Provocation of distal symptoms in the presence of derangement is unacceptable as has been demonstrated by patients suffering increased symptoms following administration of the 'slump test' by unwary therapists (McKenzie clinical records 1983).

Because the flexion programme that is being used in the remodelling process could cause recurrence of the derangement, certain precautions should be observed.

The stability of the reduction and repair must be determined before remodelling commences. This should be achieved by applying ten repetitions of flexion in lying (Procedure 18). This should immediately be followed by the repetitions of extension in lying (Procedure 3). The patient should be instructed to perform ten repetitions of each, four times per day for twenty-four to forty-eight hours, with instructions to stop should the condition worsen. If no aggravation of symptoms occurs, the patient may commence the remodelling programme.

Progress the procedures, commencing with the least stressful.

- flexion in lying (Procedure 18)
- flexion in sitting, gradually straightening legs (Procedure 19)
- flexion in standing (Procedure 20).

Always following flexion procedures with a few extensions in standing or lying.

- monitoring symptomatic response
    - symptoms may be produced, no worse
    - symptoms may become less painful on repetition
    - symptoms must *not* be produced, and remain worse
    - symptoms must *not* become more painful on repetition
    - symptoms must *not* peripheralise and remain so

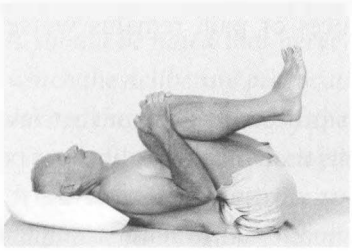
- monitoring mechanical response – range of movement and response to extension should remain unchanged following repeated flexion
    - if range of movement reduces or pain remains worse, suspect derangement
    - avoid over-vigorous flexion procedures within first few hours of waking – during this time the disc is likely to be under increased pressure as a result of nocturnal re-absorption of fluid
    - initially perform ten repetitions of flexion in standing from midday on, every three hours until retirement. If reduction of the derangement appears stable, the patient may commence the exercise a little earlier in the day and repeat it every two hours. However, it is inadvisable to perform flexion in standing on waking.

Table 29.3 Procedures for treating adherent nerve root

Procedure	*Regularity*	*Duration*	*Followed by*
FIL (Procedure 18)	5 – 6 x 5 – 6 x per day	One week	EIL
FISitt (Procedure 19) with increasing knee extension	5 – 6 x 5 – 6 x per day progress to 10 x 5 – 6 x per day	From week 2	EIL / EIS
FIS (Procedure 20)	5 – 6 x 5 – 6 x per day progress to 10 x 5 – 6 x per day	From week 2 – 3 for about 10 – 12 weeks	EIL / EIS

FIL = Flexion in lying
FISitt = Flexion in sitting
FIS = Flexion in standing
EIL = Extension in lying
EIS = Extension in standing

Photos 206, 207, 208, 209, 210, 211:
Flexion in lying (206). Flexion in sitting with partially extended knees (207). Flexion in sitting with both knees straight, reaching as far forward as possible is a progression (208). Flexion in standing (209). Extension in lying (210). Extension in standing (211).



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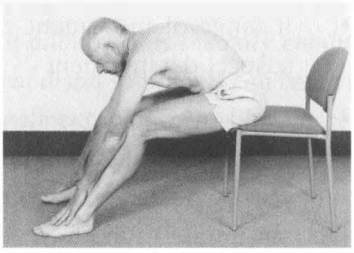

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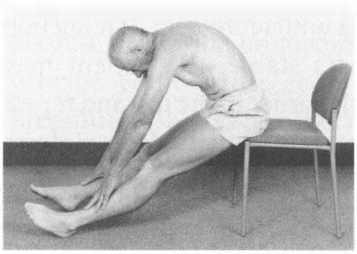

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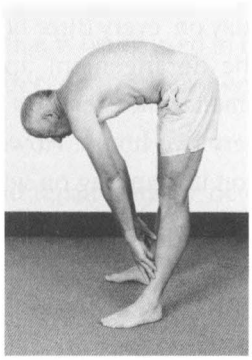

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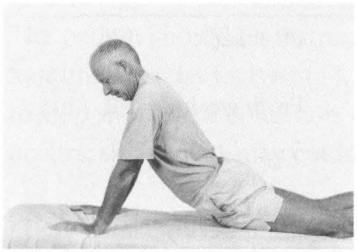

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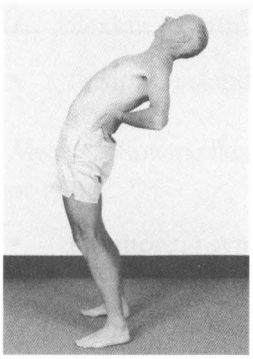
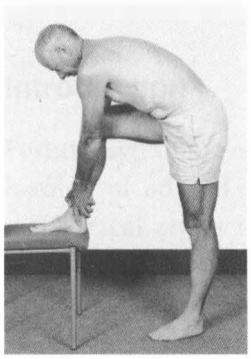


211

Patients with ANR may display deviation on flexion; significant adherence or tethering between the disc and root/dura complex may cause this to occur. Patients thus affected will always deviate to the side with symptoms. As well as the procedures outlined above, an additional flexion procedure with a lateral component may be applied:

Photos 212, 213: Flexion in step standing.



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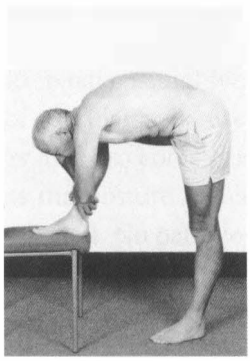


213

- Flexion in step standing (Procedure 22).

As remodelling of the adhesions about the nerve root/dura complex occur, the patient will experience the same amount of pain at end-range, but the range of flexion will slowly but steadily increase. It should be possible to assess progress from the mechanical presentation and daily activities. Fingertips will move further down the front of the patient's legs before the onset of pain; the patient can use this distance to judge progress, aiming at five millimeters per day. Sitting in the bath, driving the car and walking uphill will progressively cause less discomfort.

30: Postural Syndrome

Introduction

Postural syndrome is a painful disorder caused by prolonged static loading of normal soft tissues continued until the point when mechanical stress triggers discomfort (McKenzie 1981, 1990; McKenzie and May 2000). Pain from the postural syndrome is caused by mechanical deformation of soft tissues or vascular insufficiency arising from prolonged positional or postural stresses affecting the articular structures or the contractile muscles, their tendons or the periosteal insertions.

In spinal postural syndrome, pain arises from mechanical deformation of articular structures, while in extremity postural problems pain is as likely to come from vascular deprivation. Pain continues as long as the posture is maintained, but abates as soon as the posture is released. No pathology is present; as a consequence there is nothing to 'treat', and medicines or manipulation received for this syndrome are pointless and ineffective.

Many experience pain from this syndrome. It is especially common in schoolchildren and students who spend many hours sitting, bent over books or computer screens. Many people learn that a simple change in posture abolishes symptoms, and that it does not bother them when they are active and busy at other times. Consequently individuals rarely seek treatment with this problem, and patients with postural syndrome are rarely seen in clinical practice. It is the least common of the three mechanical syndromes, making up only a few percent of all back pain patients who seek treatment.

For instance, in a series of 319 patients, 2% were classified as having postural syndrome, 19% as having dysfunction syndrome and 79% as having derangement syndrome (Rath *et al.* 1989, in Robinson 1994). In a reliability study in which forty-five patients were assessed by two clinicians, only one patient (2%) was diagnosed with posture syndrome, while 4 – 9% were classified with dysfunction, and 87 – 89% were classified as having derangement (Razmjou *et al.* 2000). These studies did not provide sufficient detail to enable determination of the numbers of patients aged 25 and under who comprise the

bulk of patients affected by this syndrome. Generalisation of these percentages is therefore not necessarily applicable to the wider population. However, pain of postural origin frequently exacerbates and perpetuates symptoms in other mechanical problems and usually needs to be addressed.

Sections in this chapter are as follows:

- pain mechanism
- effect of posture on symptoms in normal population
- clinical picture
- physical examination
- postures involved
- management of postural syndrome
- posture syndrome – aggravating factor sitting
- posture syndrome – aggravating factor standing
- posture syndrome – aggravating factor lying
- management of postural syndrome
- consequences of postural neglect.

Pain mechanism

It is not necessary to actually damage tissue containing pain receptors in order to provoke pain (McKenzie 1981; Bogduk 1993). Pain of mechanical origin will be experienced as soon as mechanical forces applied to innervated structures are sufficient to *stress or deform* free nerve endings contained within. Pain disappears when the application of that force is terminated, and this often occurs by a mere change of position. This is termed ***physiological*** pain, and is related to the intensity of the stimulus in a measurable way (Woolf 1991; Woolf *et al.* 1998). It warns the body of potential damage and, as long as the mechanical stress is transient and interrupted before actual tissue damage occurs, no long-term sequelae will ensue. The stress fails to cause protracted symptomatology, as it is insufficient to cause tissue damage. Tissue damage always unleashes the inflammatory response (Levine and Taiwo 1994), in which case pain would persist after release from that position.

A good example is the pain incurred during prolonged slouched sitting, which disappears on standing upright. Similarly, when a finger is bent backwards slowly to end-range, first a strain is felt. If a position of strain is maintained for a few minutes, a mild aching results. If the finger is bent suddenly, by applying a brief, rapid, end-range force, the pain receptor system is activated immediately, but briefly. Thus pain can appear eventually, after moderate prolonged loading, or can appear immediately as a result of sudden short-lived overstretching. In both cases the pain will cease on release of stretching. In neither case, however, is damage to tissue necessary to cause the experience of pain.

Pain of postural origin in the lumbar spine is usually produced by mechanical stress at end-range, usually in flexion. It is most commonly a sustained end-range loading that eventually causes sufficient tissue deformation to provoke pain.

The greater the mechanical loading and deformation become, the greater is the intensity of pain. If the painful position is prolonged, the pain becomes more diffuse, widespread and difficult to define, and may expand distally (Harms-Ringdahl 1986). If such loading on return to a neutral position has caused no structural damage or displacement, the pain will become less diffuse, more localised to its point of origin, until it quickly subsides.

Increasing pain intensity at the end-range of movement indicates the beginning of overstretching. Further movement in the same direction may result in damage. As in the case of the finger, for example, the joint is obviously being moved in the wrong direction as the pain intensity increases, and in the corrective direction as the pain intensity decreases. This example demonstrates that the use of increasing and decreasing levels of pain provides a reliable guide in the choice of direction in which to apply therapeutic motion.

Effect of posture on symptoms in normal population

The study of Harms-Ringdahl (1986) has shown in the cervical spine the effect of sustained loading in symptom production. Volunteers without neck symptoms who maintained flexion of the lower cervical and thoracic spine and extreme upper cervical extension perceived pain within two to fifteen minutes. This increased with time, eventually forcing them to discontinue the posture, after which the

symptoms ceased. Pain was generally localised around the neck and upper scapulae, but radiated into the arms in a few individuals. The study demonstrates how individuals without spinal symptoms can have pain created by sustained loading, which eventually has a mechanical effect upon soft tissues.

During this sustained positioning in the protruded head posture, the levels of muscular activity in the trapezius, splenius, the thoracic erector spinae and rhomboids was generally very low. Consequently, it seems reasonable to conclude that pain due to extreme spinal positions is provoked by mechanical load on articular and peri-articular structures rather than sustained muscular activity (Harms-Ringdahl 1986). In studies conducted in the lumbar spine, it has also been shown that in kyphotic, supported or unsupported sitting positions, muscle activity is minimal (Andersson *et al.* 1975; Dolan *et al.* 1988). In relaxed sitting postures, the lumbar spine ahso is resting on articular and peri-articular structures.

Relevant to the lumbar spine, several studies have looked `t sitting postures and correlated positions with symptoms in the normal population. These demonstrate that sustained sitting postures often provoke symptoms, and that this is most likely to occur in slumped sitting. One study in asymptomatic individuals evaluated the effect of sitting for a two-hour period on ordinary canvas wheelchairs, compared with wheelchairs that had extra support added (Harms 1990). On the ordinary chairs, volunteers were in positions of flexion, while on the adapted chairs they sat with increased lordosis. Discomfort was strongly associated with the ordinary sling chairs, but not with the more supported sitting postures. Discomfort was felt in the mid-, upper and lower back and buttocks, and four subjects in the flexed posture group were unable to complete the full two hours because of low back pain.

Eklund and Corlett (1987) evaluated chair designs in a work environment with workers performing specific tasks for forty-five minutes. Consistently, chairs that allowed or encouraged a more flexed spinal posture caused increased discomfort. In another study, when given the chance to select a comfortable sitting posture, eighty individuals consistently chose a high, rather than a low seat (Mandal 1984). This had the effect of altering the position of the hips and pelvis so that the lumbar spine was more upright, compared to the flexed position of the spine on a low chair. Knutsson *et al.* (1966)

investigated the preferences in chair design of individuals with and without back pain. The most comfortable chair had a back support tilted back at 100 to 110 degrees, and gave a 1 – 2cm support in the lordosis. None of the seventy subjects found sitting in flexion to be comfortable for more than a short period.

McKenzie (1979) reported that almost without exception, patients with both acute and chronic back pain had less pain sitting with a well-formed lordosis supported by a lumbar roll.

Clinical picture

Patients with solely postural pain are usually under thirty years old, often much younger – schoolchildren may be brought to the clinic by concerned parents. Individuals are generally sedentary due to their occupation, studies or lifestyle. They may have back pain only, without referral, but may in addition describe pain in the thoracic and cervical regions that appears at the same time. Frequently they will have had symptoms for months, which have been gradually worsening – pain is now coming on more quickly and occurring more frequently. It is this gradual deterioration that is more likely to cause them to seek help rather than any dramatic onset of severe back pain.

A possible mechanism for this deterioration over time is the escalation in sensitivity that occurs when neural pathways are frequently generating the same pain signal. Alternatively, the increased sensitivity may be due to reduced thresholds to mechanical stimuli in nociceptors because of repeated mechanical exposure. Whatever the exact cause, unless patients begin to interrupt the aggravating end-range forces, with the passage of time symptoms are more easily provoked. Less mechanical stimulus is needed to produce the symptoms and they appear after a decreasing period of time.

Pain in the postural syndrome is always intermittent, and may sometimes not be present for days at a time. Pain is only brought on by static end-range postures – the most common being prolonged sitting and prolonged standing. If they sit for brief periods or are being generally active, they are pain-free. For instance, they may lead a more active lifestyle at the weekend and have no symptoms at this time. When constantly moving and changing position, they avoid the stresses arising from end-range static postures. If sitting is the

provoking posture, pain will not come on immediately, but only after a prolonged period of static positioning. Once the individual changes their position, or gets up and walks around, symptoms disappear. They will move fully and freely, and remain pain-free until they resume the same position for a period of time. The link between a particular posture, time and the onset of symptoms should be reasonably obvious in their history. Usually the patient has failed to make this connection.

Pain from the postural syndrome is never induced by movement, is never extensively referred and is never constant. There is no loss of movement, no sign of joint abnormality and no mechanical presentation. There is nothing to see other than the poor posture itself. There is no pathology; symptoms arise solely from prolonged mechanical loading.

Physical examination

The examination will be unproductive, except relating to one aspect of the presentation. There will be no deformity, no loss of movement and no response to repeated movements. Para-clinical tests will be negative. These 'negative' results in fact provide confirmatory data for diagnosis of posture syndrome.

The only relevant 'positive' result will relate to posture. The patient's sitting, and often standing, posture is poor. If enough time has elapsed during the history-taking and the patient is positioned in their provocative slouched sitting posture, their symptoms are produced. At this point, if they are encouraged to move from that position by correcting their posture, the symptoms are abolished and the relevance of the poor posture to their pain will have been demonstrated to the patient. Equally, once the patient stands, the symptoms abate.

Sometimes it may take longer, with a sustained posture for up to half an hour before the pain is felt. If this is the case, it may be necessary to ensure that the patient sits for this length of time in order to provoke the symptoms. Once the pain is present, it is simple to educate the patient in the relevance of posture to their problem. Posture correction or standing will abolish the symptoms. They thus learn the importance of their position, and how the way they sit can either provoke or relieve their symptoms. It is vital that the patient appreciates the intimate link between posture and pain in this syndrome; only then will they

be able to deal with it. Thus it is necessary to reproduce the symptoms in order to prove this link.

Table 30.1 Postural syndrome – criteria (all will apply)

History:

- local pain
- intermittent pain
- pain associated with time spent in a particular posture
- pain does not persist
- painless movement and activity.

Physical examination:

- poor posture
- full range of movement
- no deformity
- no problems with curve reversal
- repeated movements do not reproduce pain
- pain only produced by sustained loading in relevant position, which is then relieved on moving from that posture.

Those who are unfamiliar with the system of mechanical diagnosis and therapy can confuse the posture and derangement syndromes (Riddle and Rothstein 1993). In fact, the symptomatic and mechanical presentation of these two syndromes are very different, although both are affected by posture. In the case of a patient with derangement, there can be referred pain, pain on movement, deformity or blockage to movement, persistent pain after the posture is corrected or the aggravating position released, and in general a much more severe presentation.

Sitting is the most common cause of pain in the posture syndrome. It is also an extremely common cause of aggravation of pain in derangement, but pain behaviour in the two syndromes is distinctly different. If an individual is having back pain that is due to postural syndrome and is caused by sitting, there will be a clear association between the posture, when sustained for a sufficient period, and their pain – as frequently occurs in derangement. Upon rising and moving, however, the pain will rapidly cease, only reoccurring when they resume the sitting position for a sustained period, and when tested all movements will be full and pain-free. Pain in derangement has a vastly more significant effect in terms of pain and function. Another key difference between the two is prevalence in those seeking

health care. Postural syndrome is rarely seen in the clinic, while derangement syndrome is common.

Postures involved

Sitting is the most frequent cause of pain of postural origin, and many patients will name this as the only provoking factor. They may complain that pain is produced after spending a certain time, for instance quarter of an hour or so, in any sort of chair or when driving. Others may complain that the pain only comes on after working at the computer for awhile. Frequently bus, taxi and car drivers, pilots and passengers on aircraft complain about pain after some time in such seating.

Sometimes individuals complain of pain after prolonged standing, especially if working constantly bent forward or stooping. The same criteria as above must apply. To the inexperienced clinician this could be confused with derangement syndrome, which is frequently provoked by bending, but the impact of derangement is much greater. Other postures are less likely to produce pain of postural origin as they generally allow people a much greater opportunity to alter their position. Occasionally pain of postural origin is felt when the individual is lying down.

Management of postural syndrome

Once patients are made aware of the link between their posture and their pain, most find it relatively easy to self-manage this syndrome. However, it is vital that this correlation between their position and symptoms is made apparent to them. If the patient is finding this difficult to accept, positioning them in a sustained posture so that symptoms are provoked is usually sufficient to convince them of the cause. Once the link is well established, they need advice on correcting posture and avoiding or interrupting the aggravating factor. If they avoid end-range stresses to soft tissues for two to three weeks, the problem will resolve.

Repeated exposure to the aggravating posture over time leads to a gradual deterioration with increased frequency of symptoms. Conversely, correcting the posture enables the patient to remain pain-free for longer and longer time periods. If the causative posture is avoided, the sensitivity of tissue nociceptors to mechanical stimuli

diminishes over time. Thus, after two weeks of posture correction, resumption of the aggravating position is tolerated for longer before pain is triggered. Obviously this should not be encouraged, as it will set them once more on a deteriorating pathway.

As well as advice to avoid the causative posture, patients must be shown how to maintain a correct sitting position. Patients should be warned that the adoption of new postures might cause the temporary development of 'new' pains, which will subside within a week. Management is thus a combination of avoidance and performance – avoid the aggravating factor and performing the corrective procedures.

Table 30.2 Management of posture syndrome

- education on link between posture and pain
- education on posture correction
  - attain posture
  - maintain posture
- education on avoidance of aggravating posture
- posture correction (Procedure 4)
- slouch–overcorrect (Procedure 10).

Posture syndrome – aggravating factor sitting

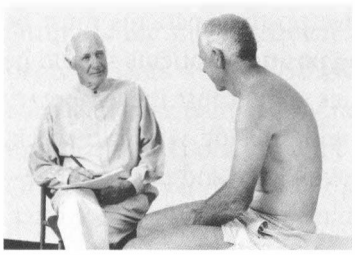
McKenzie (1981) described poor sitting posture as the number one predisposing perpetuating factor in low back pain causation.

The individual with poor posture is predisposed to this syndrome when exposed to long hours of sitting due to occupation, study, unemployment or hobby. It is important to establish patient's habit from the outset.

The postural habit when sitting for a period of time and the effect this may have on symptoms can be observed if the patient is seated without a back support as on the examination couch, rather than in a chair. The best opportunity arises while the history is taken. During this sometimes lengthy period, the effect of sustained relaxed sitting on the patient's posture may be noted. At the end of this part of the assessment, question the patient about the presence of pain. This may have developed during the interview, and if present, posture correction will rapidly abolish symptoms. *If a direct link between posture and pain can be clearly demonstrated to patients, their compliance*

to the management programme is rapidly achieved. For this reason, the patient should have the history taken while sitting unsupported, as described.

Taking the history



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Allow the patient to relax unsupported to expose the true nature of their sitting posture.

Correction or further investigation of the slouched sitting position and its effects on pain mark the beginning of the physical examination. The rest of the physical examination will be normal, with full range of movement and no pain on repeated movements.

Correction of sitting posture

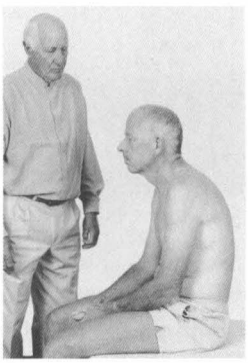
It should be explained to patients that when we sit, especially when preoccupied, a relaxed posture is adopted. The spine takes up the shape of the chair, or if sitting unsupported, eventually the slouched posture is adopted. Unless a conscious effort is made, or a well-designed chair with appropriate support is used, it is a universal phenomenon that within a short period of sitting individuals will have adopted a relaxed, slouched posture. This flexed posture will place ligaments, capsules and other peri-articular and articular structures under tension. If this posture is maintained, as creep occurs, greater tensile stress is placed upon these soft tissues. Eventually, if maintained without respite, enough mechanical tension can be generated to trigger nociceptor activity.

That simple mechanical tension will eventually become painful is easily demonstrated to the patient using the analogy of the 'bent finger', especially if the patient's own finger is used for educational purposes. By holding their finger in end-range extension, first a discomfort and then a dull ache is produced, but as soon as it is released the aching abates; within a minute the pain is completely gone, with no damage having occurred.

The patient needs to be convinced that the same process is at work in the spine. If pain is of postural origin, there is never any lasting repercussion – when the pain is provoked by sitting slouched, it will be abolished by correcting the posture. Hopefully during the first treatment

session the patient's symptoms will be produced after prolonged sitting – the period of history-taking allows fifteen to twenty minutes when this may occur. Upon completing the interview, the patient should be questioned about any symptoms that are now present. If they report the onset of symptoms during this period, then the effect of posture correction must be explored. If symptoms are due to posture syndrome, posture correction will abolish them. When pain is shown to be so clearly related to position, the patient will quickly accept the logic of correcting the sitting posture. If the pain does not abate when the posture is corrected, but increases, changes location or stays the same, another mechanical syndrome, derangement in all probability, is the cause of symptoms. Pain from derangement syndrome may also centralise or be reduced or abolished by posture correction.

If it is not possible to produce the patient's symptoms during the interview, then they must be instructed to test the effect of posture correction on the next occasion that symptoms develop. *'The next time pain occurs, can you abolish it by correcting your posture?'*



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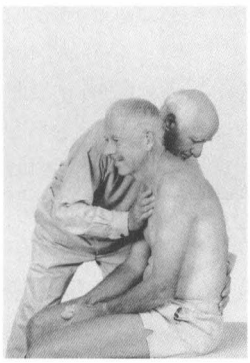

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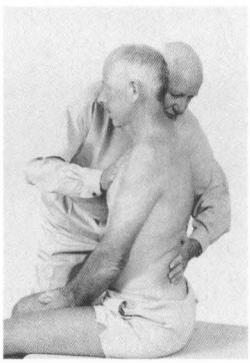

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Photos 215, 216, 217: Posture correction and symptom monitoring.
At conclusion of the history, ask about present pain status: 'In this position, what pain do you have'? (215) Press on the patient's upper sternum and pull the lower back into lordosis to correct the posture (216). Having corrected flexed posture, ask, 'What do you feel in this position?' (217)

Posture correction involves:

1. attaining correct sitting posture
2. maintaining correct sitting posture.

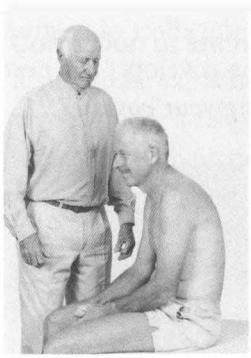
Attain correct sitting posture

To encourage the patient to attain and maintain the corrective posture, they must be convinced of its value. Most patients, when they are fully aware of the relationship between posture and the production of pain, quickly accept the need to alter their postural habits. They need to understand the correct sitting posture as well as recognise a poor posture, and they need to be able to control their trunk during

posture correction and maintenance. A 'good posture' is defined here as a position in which the lumbar spine is positioned in a moderate degree of lordosis, and the head and shoulders are evenly aligned over the pelvis.

To understand and attain the correct sitting posture, the 'slouch–overcorrect' procedure (Procedure 10) is introduced. This procedure allows patients to feel the difference between a poor slouched posture and a fully overcorrected posture. It is neither good nor desirable for patients to maintain this overcorrected position; prolonged excessive extension will eventually become as painful as prolonged flexion. The best sitting posture is gained by releasing the last 10% of the overcorrected sitting position. The lumbar lordosis should be similar when sitting to that which is present when standing.

Photos 218, 219, 220: Slouch-overcorrect. Extreme of bad position (218). Extreme of good position (219). Extreme of good position less strain (220).



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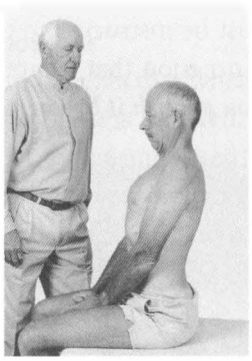

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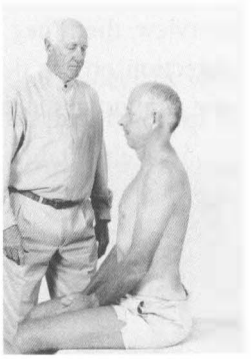

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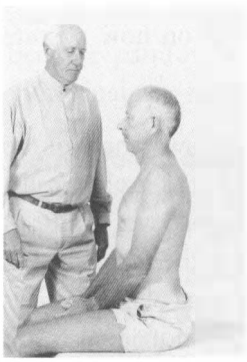
If the slouch–overcorrect procedure is practised three times daily, ten to fifteen times at each session, the patient will in a matter of a few weeks have re-educated their postural habit. They will no longer perceive the slouched posture as 'normal'; they will find that the corrected posture is now 'normal' for them. As well as practising slouch–overcorrect in order to retrain their postural 'habit' and to train their muscles to hold their trunk upright, the procedure should be done regularly whenever pain arises. Painful postures should be frequently and rapidly interrupted. The increased sensitivity to mechanical stimuli that has developed in the tissues from repeated exposure to those mechanical loads will gradually abate. Each time the slouched posture is resumed and pain re-triggered, then the threshold at which mechanical stimuli will cause pain will remain low. If the painful position is avoided altogether, sensitivity will return to normal and short periods of slouched sitting will no longer hurt.

Maintain correct sitting posture

When sitting for prolonged periods, it is essential that a lumbar lordosis be maintained at all times. The patient must be shown how to do this from the first day (posture correction – Procedure 4). The lumbar lordosis can be maintained in two ways:

1. actively, by muscular control, when sitting on a seat and not using a backrest
2. passively, with the use of a lumbar roll or support, when sitting in a seat with a backrest. The lumbar roll keeps the lumbar spine in moderate lordosis while driving, sitting at work or relaxing. Without it, the lordosis is lost if the person leans back in the chair or concentrates on something other than the maintenance of the lordosis.

Photo 221: Posture correction.



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Active control of the lumbar lordosis is demanding, and someone used to the slouched posture will have problems maintaining this position actively for long. However, it is good for the patient to practice this posture actively several times a day, holding it as long as they can for up to a few minutes. The performance of this regular active posture correction will improve their overall postural control. It has the additional benefit of strengthening the muscles responsible for maintaining the upright position.

Patients frequently complain about the effort to maintain the correct sitting posture, especially if they are actively maintaining the position. Many describe a strain pain or say that the new position is uncomfortable. These new postural stresses are to be expected, and if patients do not complain of 'new pains', it is likely that they have not been adequately practising or maintaining the correct posture. Adjustment to a new posture results in short-lived transitional aching, usually of a different quality and location than the original pain complained of. These are commonly felt further up the back in the thoracic region, and should not last longer than five or six days. Maintaining an erect posture is the best way to ensure the strengthening process continues throughout life.

A lumbar roll has a significant effect on the maintenance of the lordosis (McKenzie 1979). With increasing support producing increasing lordosis (Andersson *et al.* 1979). As long as the support is level with the lumbar spine, the exact level is less important as it does not much influence the ultimate angle (Andersson *et al.* 1979). It is most appropriate to place the support level with the lower lumbar spine, which is the area of greatest stress. The individual must push their pelvis to the back of the chair; otherwise the support is wasted. A cushion is not suitable as it simply moves the whole spine away from the chair without influencing the degree of lordosis of the lumbar spine.

A good sitting posture is often difficult to obtain on sofas or settees because the shape of the chair causes posterior rotation of the pelvis, which in turn reduces the lordosis (Keegan 1953). Good posture is easier to attain and maintain on a dining room-type chair. If any benefit is to be gained from a lumbar roll in a lounge chair, then cushions should be used first to prevent the support being absorbed by the upholstery. See Chapter 6 for more detail on how posture affects the lumbar curve.

In postural retraining, the problem lies in loss of awareness of the correct posture, not in an inability to assume it. Lumbar rolls, expensive office furniture and ergonomically designed work stations will all have no effect on postural habits unless the individual is aware of the correct posture. Likewise, strengthening of the muscles of the spine will have no effect on posture if the individual is not 'bodily' aware of the correct sitting position. No strengthening exercise can teach the patient the correct posture. Once the correct posture is attained, stronger muscles may help the patient maintain it. Regular use of the slouch–overcorrect procedure is a helpful way for the patient to learn how to attain the right posture. The patient will also be motivated to improve his posture as a result of improving symptoms. Actively maintaining the correct posture is the best way to strengthen the postural muscles and to enforce a new bodily posture. By regularly attaining and maintaining a better posture, this will become easier, and after three or four weeks a new postural habit will become normal.

Posture syndrome – aggravating factor standing

Prolonged standing is another position in which low back pain of postural origin can occur. There is a clear association between this activity when sustained for a sufficient period and the onset of pain. Thus, an occupation or hobby that involves extended periods of standing is likely to be a causative factor.

Two slouched standing positions are commonly seen. When observed, the patient may stand with an exaggerated lumbar lordosis and thoracic kyphosis and with the pelvis pushed forward, thus giving the appearance of a protruding abdomen. The other standing posture commonly adopted is obtained by taking all the body weight on one leg, with the other knee bent, causing the pelvis to droop to one side. In both postures the patient places the lumbar spine at end-range. The first involves end-range extension, the second end-range side gliding. If the patient is allowed to stand relaxed, they will adopt one of these extreme slouched postures, which if prolonged will become painful. In both these slouched standing postures, the individual is resting on articular and peri-articular structures.

Correction of standing posture

The patient must be made aware of the link between their posture and their pain. It may well be necessary to provoke the pain by requiring them to remain standing until it appears. Once this happens, postural correction rapidly abolishes symptoms.

Lifting the chest and thoracic spine, tilting the pelvis slightly anteriorly and gently tightening the abdominal muscles best achieves posture correction. The patient is then standing in a relaxed standing position rather than a slouched standing posture. Awareness of the position of the pelvis and control of this angle is essential in attaining posture correction.

If pain in standing cannot be reproduced on the first examination, the patient must be instructed to self-evaluate the relationship between posture and pain by postural correction the next time pain is felt. Therefore, in this situation the patient also needs instruction in postural correction and needs to practice this enough so that they are posturally aware of the difference between the slouched and relaxed standing postures.

Posture syndrome – aggravating factor lying

Lying is another position in which low back pain of postural origin can occasionally occur. There will be a clear association between prolonged recumbency and the onset of pain. Such patients will be woken by pain in the night, or wake with pain in the morning that was not present prior to retiring the previous night. Such pain abates soon after arising. As is usual in patients with posture syndrome, on examination nothing abnormal is found.

If resting through the night is causing pain, two factors need to be investigated:

1. The lying posture. This is different for each person and must be dealt with individually. Sleeping postures are habitual and can be difficult to influence. Concerning the lumbar spine, two extremes may be found. Individuals may lie in a very flexed position if they sleep curled up, in the 'foetal position', or if they lie with their legs straight out, the spine may be in an extended position.
2. The surface on which the person is lying. For the majority of people the mattress should not be too hard, whereas the base on which the mattress rests should be firm and unyielding. This gives adequate support without placing stresses on the spine. If the surface is too hard, due to the natural contours of the body the lumbar spine may be without sufficient support. If the bed is too soft or sags considerably, the sleep posture may be one of extreme flexion. Usually the surface on which one is lying is easily corrected or modified.

Modification of the lying posture

Patients can be encouraged to alter their sleeping posture if this is indicated, but it may be difficult to achieve. Three ways in which the lying posture can be modified are suggested. The position in which the person sleeps and the nature of the support provided by their bed need to be analysed in order to suggest the appropriate modification.

If the patient sleeps with legs extended and on a hard surface, lack of lumbar support may be the problem. If this is thought to be the cause, the patient should try a lumbar support roll. This is likely to work quickly or not at all, and should be tried for about three nights.

A beach or bath towel folded end-to-end and then rolled up usually fits around the average waist. Patients will need to experiment to find the correct size of lumbar support required for their particular case. The towel should be wrapped around the waist and the two ends attached to each other, for instance with a safety pin. If the towel is left loose, it will not remain in place and may move, leading to increased stresses on the lumbar spine.

If the mattress or the base of the bed are not firm enough and allow the spine to sag during sleep, this may be the problem. Rather than immediately going to the expense of new furniture, the mattress may be placed on the floor for a few nights. If firmer support is required, this should improve the symptoms. If after three or four nights there has been no change, it is unlikely that this is the answer to the patient's problem.

A small number of people require a sagging mattress. This can easily be created by placing pillows at both ends of the bed under the mattress. This may be tried for three or four nights to evaluate its effect on symptoms.

Conclusions

Only the patient can rectify pain of postural origin. No externally given treatment can alter the aggravating factor, which is their postural habit. To dispense treatment for a condition that can only be resolved through patient education is negligent health care. The essence of management for this condition is education and postural correction.

Management of postural syndrome

- education on link between posture and pain
- education on posture correction
    - attain posture
    - maintain posture
- education on avoidance of aggravating posture
- posture correction (Procedure 4)
- slouch–overcorrect (Procedure 10).

Pain of postural origin arises from postural neglect; through postural correction they can stop their pain and also prevent its onset. As long as the link between pain and posture has been clearly demonstrated to patients, and they have been adequately educated, most are well able to treat themselves.

When management by education is completed successfully, it should be explained to the patient that, although the present pain has been relieved, recurrence of similar symptoms is possible if postural care is neglected for extended periods. The consequences of postural neglect should be discussed.

Consequences of postural neglect

The effect of postural habits has long-term implications on the human shape (McKenzie 1981, 1990). The commonly observed posture of protruded head, rounded shoulders and flattened spine may become habitual. As age advances, permanent postural 'set' may occur – head protruded, shoulders rounded, dowager's hump, loss of lumbar lordosis and the erect posture replaced by a slight stoop. This is likely to be accompanied by considerable soft tissue adaptations. Positions that are frequently adopted, such as flexion, are maintained, while movements that are rarely performed, such as extension, become steadily more difficult to achieve. Long-term postural neglect can lead to adaptive tissue shortening, causing dysfunction syndrome.

As men and women age, their natural head position tends to progress to a more forward position; their ability to retract the head declines, while protrusion range is maintained, and there is an overall decline in antero-posterior mobility (Dalton and Coutts 1994). Between young adulthood and older age there is a reduction in all planes of cervical movements of 20 – 45% (Worth 1994), and a reduction in all planes of lumbar movements of about 30% (Twomey and Taylor 1994a, 1994b). Although a large part of this may be the natural effects of ageing, and is due to increased disc stiffness (Twomey and Taylor 1994a, 1994b), there is also an element of variability in the degree to which people become restricted in range of movement and in resting postures. The standard deviations to the mean range of sagittal plane movements in the lumbar spine constitute 43 – 47% of those mean values (Twomey and Taylor 1994a, 1994b). This demonstrates a considerable variability in the normal range of movement in the older population.

This means that protruded head positions and stooped postures are not simply an inevitable consequence of ageing. Movement that is lost because of tissue adaptation could have been retained had affected soft tissues been regularly stretched. If end-range movement is neglected, eventually that movement is lost forever. Postural 'sets' that arise from long-term postural neglect and tissue adaptation result from postural habit as much as the consequence of age. Loss of function can be prevented if end-range movements are regularly performed and posture corrected throughout life.

Thus, initially, poor postural habits produce pain of postural origin without loss of function. Prolonged postural neglect leads to adaptive shortening. If flexion is regularly performed but extension rarely, the anterior structures of the joints shorten and the posterior structures lengthen. Extension becomes more and more difficult to perform. Adaptive shortening implies loss of function and movement. Whenever shortened structures are placed on stretch, they will induce discomfort or pain. Furthermore, the decreased movement must inevitably lead to impairment of nutrition in the intervertebral disc, contributing to disc degeneration. *The shortening of soft tissue caused by poor postural habit and inadequate exercise can be prevented by regular postural correction and adequate performance of the relevant exercises* (McKenzie 1981, 1990).

The detail provided in this chapter is summarised in the form of criteria and operational definitions contained in the Appendix – these are essential for identification of the different syndromes.

Appendix

Classification and operational definitions

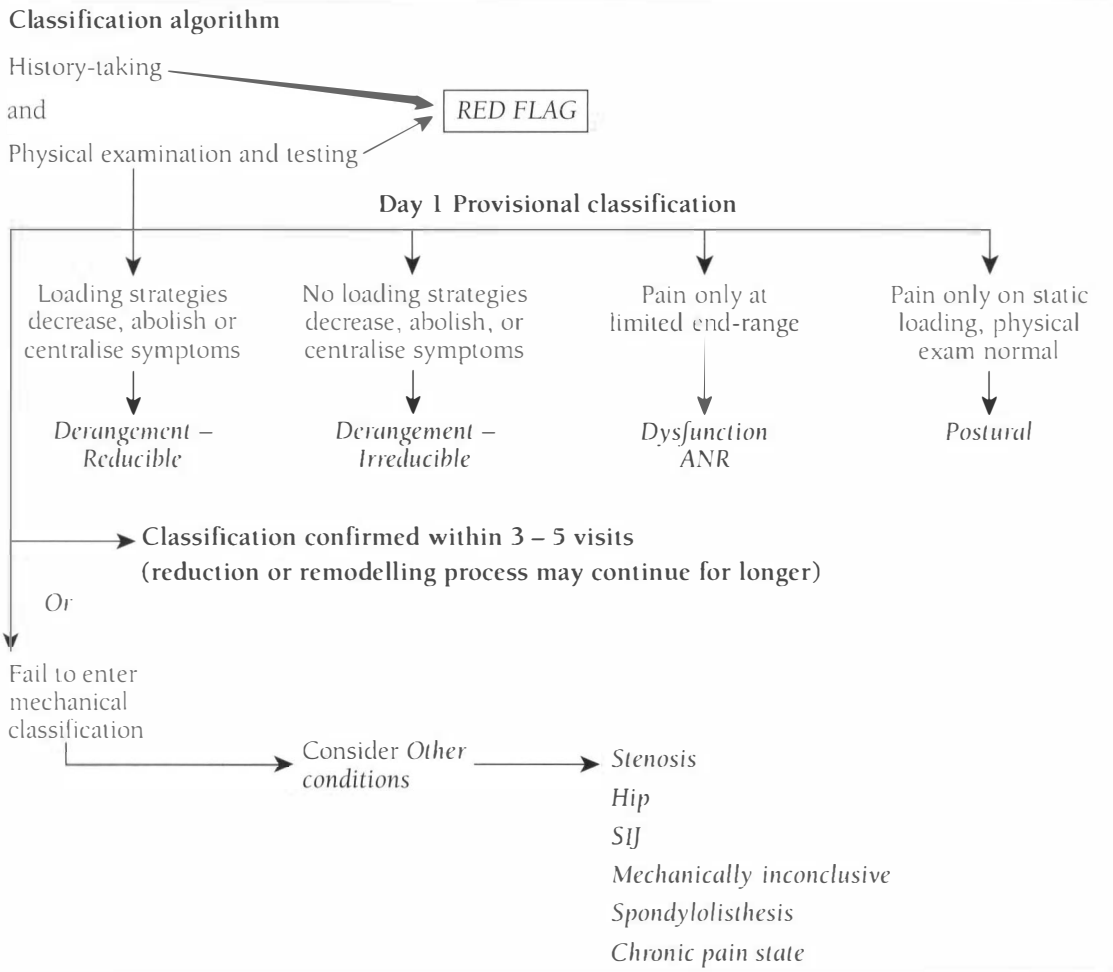
Category *Mechanical syndrome*	Definition	Criteria** *Symptom response*
Reducible derangement	Internal disc displacement with competent annulus	Centralisation Abolition Decrease
Irreducible derangement	Disc displacement with incompetent or ruptured annular wall	Peripheralisation Increase in peripheral pain No centralisation, reduction or abolition
Dysfunction	Soft tissue structural impairment	Intermittent pain when loading restricted end-range
Adherent nerve root	Adhesions producing functional impairment of nerve root or dura	Intermittent pain at limited end-range flexion in standing and long sitting
Postural syndrome	Prolonged mechanical deformation of normal soft tissues	Pain only with prolonged loading Physical examination normal
OTHER	*Exclusion of above*	*Lack of above responses, plus the following*
Spinal stenosis	Bony or soft tissue narrowing of spinal or foraminal canal causing neurogenic claudication May be associated *with* degenerative spondylolisthesis	History – leg symptoms when walking, eased in flexion Minimal extension Sustained extension may provoke leg symptoms
Isthmic spondylolisthesis	Slippage of vertebral body	Sports-related injury in adolescence Worse with static loading
Hip	Pain-generating mechanism due to mechanical, inflammatory or degenerative changes in or around hip joint	History – pain on walking, eased on sitting Specific pain pattern Positive 'hip' tests
SIJ	Pain-generating mechanism due to mechanical, inflammatory or degenerative changes in or around SIJ	Three or more positive SIJ pain provocation tests
Mechanically inconclusive	Unknown intervertebral joint pathology	Inconsistent response to loading strategies No obstruction to movement

Continued next page

Category *Mechanical syndrome*	Definition	Criteria** *Symptom response*
Chronic pain	Pain-generating mechanism influenced by psychosocial factors or neurophysiological changes peripherally or centrally	Persistent widespread pain Aggravation with all activity Exaggerated pain behaviour Inappropriate beliefs and attitudes about pain

Serious spinal pathology – suspected	Definition	Criteria
Cauda equina	Compression of sacral nerves by disc herniation or tumour	Bladder / bowel involvement Especially urinary retention Saddle anaesthesia Sciatica
Cancer	Growth of malignant tumour in or near vertebrae	Age > 55 History of cancer Unexplained weight loss Constant, progressive pain unrelated to loading strategy, not relieved by rest
Fracture	Bony damage to vertebrae caused by trauma or weakness due to metabolic bone disease	Significant trauma Trivial trauma in individual with osteopenia
Spinal infection	Infection affecting vertebrae or disc	Systemically unwell Febrile episode Constant severe back pain unrelated to loading strategy
Ankylosing spondylitis	One of the systemic inflammatory arthropathies affecting spinal and other structures	Exacerbations and remissions Marked morning stiffness Persisting limitation all movements No directional preference, but better with exercise, not relieved by rest Systemic involvement Raised ESR, + HLA B27

** The operational definitions provided below present the criteria in more detail. These give the symptom responses and timescale by which classification should be recognised.



Operational definitions

The operational definitions describe the symptom and mechanical behaviours and the timescale needed to document each category.

Reducible Derangement

Centralisation: in response to therapeutic loading strategies, pain is progressively abolished in a distal to proximal direction, *and*

- each progressive abolition is retained over time until all symptoms are abolished, *and*
- if back pain only is present this moves from a widespread to a more central location and then is abolished *or*
- pain is decreased and then abolished during the application of therapeutic loading strategies
- the change in pain location, or decrease or abolition of pain, remain better, *and*

- should be accompanied or preceded by improvements in the mechanical presentation (range of movement and/or deformity).

Timescale

A derangement responder can be identified on day one, *or*

- a derangement responder will be suspected on day one and a provisional diagnosis made. This will be confirmed by a lasting change in symptoms after evaluating the response to a full mechanical evaluation within five visits
- decrease, abolition or centralisation of symptoms is occurring but the episode may not have completely resolved within five visits
- aggravating factors may precipitate a deterioration in symptoms and a longer recovery process.

Irreducible Derangement

Peripheralisation of symptoms: increase or worsening of distal symptoms in response to therapeutic loading strategies, *and/or*

- no decrease, abolition, or centralisation of pain.

Timescale

An irreducible derangement patient will be suspected on day one and a provisional diagnosis made; this will be confirmed after evaluating the response to a full mechanical evaluation within five visits.

Dysfunction

Spinal pain only, *and*

- intermittent pain, *and*
- at least one movement is restricted, and the restricted movement consistently produces concordant pain at end-range, *and*
- there is no rapid reduction or abolition of symptoms, *and*
- no lasting production and no peripheralisation of symptoms.

ANR

History of sciatica or surgery in the last few months that has improved, but is now unchanging, *and*

- symptoms are intermittent, *and*
- symptoms in the thigh and/or calf, including 'tightness', *and*
- flexion in standing, long sitting, and straight leg raise are clearly restricted and consistently produce concordant pain or tightness at end-range, *and*
- there is no rapid reduction or abolition of symptoms and no lasting production of distal symptoms.

Timescale

- a dysfunction/ANR category patient will be suspected on day one and a provisional diagnosis made; this will be confirmed after evaluating the response to a mechanical evaluation within five visits
- if the patient fails to fit all criteria another category must be considered
- rapid change will not occur in this syndrome, and symptoms will gradually reduce over many weeks, as range of movement gradually improves.

Postural

Spinal pain only, *and*

- concordant pain only with static loading, *and*
- abolition of pain with postural correction, *and*
- no pain with repeated movements, *and*
- no loss of range of movement, *and*
- no pain during movement.

Timescale

- a posture category patient will be suspected on day one and a provisional diagnosis made. This will be confirmed after evaluating the response to a mechanical evaluation within two to three visits
- if the patient fails to fit all criteria, another category must be considered.

'Other' categories are only considered on failure to enter a mechanical diagnosis within five treatment sessions. To be designated into 'Other' category, patients will fulfil:

- 'other' criteria, *and*
- criteria for specific other category as listed below.

'Other'

- no centralisation, peripheralisation, or abolition of symptoms, *or*
- does not fit derangement, dysfunction or posture criteria
- no lasting change in pain location or pain intensity in response to therapeutic loading strategies, *and*
- fulfils relevant criteria in suspected 'other' pathology listed below.

Indicators for possible 'Red Flags'

Cauda equina

- bladder dysfunction (urinary retention or overflow incontinence)
- loss of anal sphincter tone or faecal incontinence
- saddle anaesthesia about the anus, perineum or genitals
- global or progressive motor weakness in the lower limbs.

Possible cancer

- age greater than 55
- history of cancer
- unexplained weight loss
- constant, progressive pain not affected by loading strategies, worse at rest.

Other possible serious spinal pathology

One of the following:

- systemically unwell
- widespread neurology
- history of significant trauma enough to cause fracture or dislocation (x-rays will not always detect fractures)
- history of trivial trauma and severe pain in potential osteoporotic individual
- sudden and persistent extremes of pain causing patient to 'freeze'.

Possible inflammatory disorders

- gradual onset, *and*
- marked morning stiffness, *and*
- persisting limitation of movements in all directions
- peripheral joint involvement
- iritis, psoriasis, colitis, uretheral discharge
- family history.

Stenosis

- history of leg symptoms when walking upright
- may be eased when sitting or leaning forward
- loss of extension
- possible provocation of symptoms in sustained extension, with relief on flexion
- age greater than 50
- possible nerve root signs and symptoms
- extensive degenerative changes on x-ray
- diagnosis confirmed by CT or MRI.

Hip

- exclusion of lumbar spine by mechanical evaluation, *and*
- pain worsened by weight bearing, eased by rest or worse first few steps after rest, *and*
- pain pattern – groin, anterior thigh, knee, anterior shin, lateral thigh, possibly buttock, *and*
- positive hip pain provocation test(s) – (concordant pain).

Symptomatic SIJ

- exclusion of lumbar spine by extended mechanical evaluation, *and*
- exclusion of hip joint by mechanical testing, *and*
- positive pain provocation tests (concordant pain) – at least three tests.

Mechanically inconclusive

- symptoms affected by spinal movements
- no loading strategy consistently decreases, abolishes or centralises symptoms, nor increases or peripheralises symptoms
- inconsistent response to loading strategies.

Symptomatic spondylolisthesis

- suspect in young athletic person with back pain related to vigorous sporting activity
- worse with static loading.

Chronic pain state

- persistent widespread symptoms
- all activity increases symptoms
- exaggerated pain behaviour
- mistaken beliefs and attitudes about pain and movement.

Other definitions

Definition of centralisation

- in response to therapeutic loading strategies pain is progressively abolished in a distal to proximal direction with each progressive abolition being retained over time until all symptoms are abolished
- if back pain only is present, this is reduced and then abolished.

Criteria for a relevant lateral shift

- upper body is visibly and unmistakably shifted to one side
- onset of shift occurred with back pain
- patient is unable to correct shift voluntarily
- if patient is able to correct shift, they cannot maintain correction
- correction affects intensity of symptoms
- correction causes centralisation or worsening of peripheral symptoms.

Right and left lateral shift

- a right lateral shift exists when the vertebra above has laterally flexed to the right in relation to the vertebra below, carrying the trunk with it; the upper trunk and shoulders are displaced to the right
- a left lateral shift exists when the vertebra above has laterally flexed to the left in relation to the vertebra below, carrying the trunk with it; the upper trunk and shoulders are displaced to the left.

Contralateral and ipsilateral shift

- contralateral shift exists when the patient's symptoms are on one side and the shift is in the opposite direction; for instance, right back pain, with / without thigh / leg pain, and upper trunk and shoulders displaced to the left
- ipsilateral shift exists when the patient's symptoms are on one side and the shift is to the same side; for instance right back pain, with / without thigh / leg pain, with upper trunk and shoulders displaced to the right.

Criteria for a relevant lateral component

- acute lateral shift deformity OR loss of frontal plane movements *and / or*
- unilateral / asymmetrical symptoms affected by frontal plane movements
- symptoms fail to improve with sagittal plane forces *or*
- symptoms worsen with sagittal plane forces *and*
- symptoms improve with frontal plane forces.

Glossary of Terms

Anterior compartment

The compartment of the intervertebral segment that is compressed with flexion forces.

Centralisation

The phenomenon by which distal limb pain emanating from although not necessarily felt in the spine is immediately or eventually abolished in response to the deliberate application of loading strategies. Such loading causes an abolition of peripheral pain that appears to progressively retreat in a proximal direction. As this occurs there may be a simultaneous development or increase in proximal pain. The phenomenon only occurs in the derangement syndrome.

Curve reversal/obstruction to curve reversal

In an asymptomatic state, individuals can move from an extreme position of flexion to an extreme position of extension without impediment; in derangement this can become difficult or impossible. Following a period of loading or repeated movements in one direction the opposite movement may become obstructed, and recovery is slow, gradual and/or painful. Thus, after spending a period of time in flexion, as in bending or sitting, or after repeated flexion, the patient is unable to regain the upright position immediately or without pain. They are forced to gradually and painfully resume the erect posture or movements into extension. In severe derangements patients may have difficulty straightening after one flexion movement.

Deformity

The patient experiences a sudden onset of pain and immediately or subsequently develops a loss of movement and a deformity so severe that they are unable to move out of the abnormal posture. The patient is fixed in kyphosis, lateral shift or lordosis and is unable to self-correct this very visible anatomical misalignment. If they are able to correct the deformity, they cannot maintain the correction. This phenomenon only occurs in derangement and must be immediately recognised as it determines treatment.

- *Kyphotic deformity* – the patient is fixed in flexion and is unable to extend.

- *Lateral shift* – the patient is fixed in (for instance) right lateral shift and is unable to bring his hips back to the mid-line or assume a position of left lateral shift. In the case of a 'hard' deformity, the patient will need clinician assistance to correct it, while in the case of a 'soft' deformity, the patient may be able to self-correct with repeated movements.
- *Lordotic deformity* – the patient is fixed in extension and is unable to flex.

Derangement syndrome

Rapid and lasting changes, sometimes over a few minutes or a few days, in pain intensity and location. Mechanical presentation can occur in this syndrome with the performance of movements or the adoption of sustained postures. Loading strategies produce a decrease, abolition or centralisation of symptoms. Opposite loading strategies may cause production, worsening or peripheralisation of symptoms if prolonged over a sufficient time. A distinguishing set of characteristics will be found during the history-taking and physical examination. The conceptual model involves internal articular displacement that causes a disturbance in the joint, which produces pain and impairment.

Deviation

There are two types of deviation: a) postural b) on movement.

a) Postural deviations – patients may prefer to hold themselves shifted to one side or in a degree of flexion because this brings temporary easing of their condition. However, they are capable of straightening, which distinguishes this group from those with a deformity. Both occur only in derangement.
b) Deviation on movement – for instance, as the patient flexes, they deviate away from the pure sagittal plane to left or right. This is indicative of either an adherent nerve root or a derangement.

Directional preference

The phenomenon of preference for postures or movement in one direction that is a characteristic of the derangement syndrome. It describes the situation when postures or movements in one direction decrease, abolish or centralise symptoms and often increase a limitation of movement. Postures or movements in the opposite direction often cause these symptoms and signs to worsen. This does not always occur, and may be a product of the length of exposure to provocative loading.

Distal symptoms

The symptoms located furthest down the leg; these may be radicular or somatic referred pain, or paraesthesia. During the evaluation of symptomatic responses to mechanical loading, the most distal symptoms are closely monitored. Movements that decrease or abolish these symptoms are prescribed, while movements that increase or produce them are avoided.

Dysfunction syndrome

Pain from the dysfunction syndrome is caused by mechanical deformation of structurally impaired soft tissues. This abnormal tissue may be the product of previous trauma or degenerative processes and the development of imperfect repair. Contraction, scarring, adherence, adaptive shortening or imperfect repair tissue become the source of symptoms and functional impairment. Pain is felt when the abnormal tissue is loaded. A distinguishing set of characteristics will be found during the history-taking and physical examination. In spinal dysfunction pain, is consistently produced at restricted end-range, and abates once the loading is released. Dysfunction may affect contractile, peri-articular or neural structures, with the latter two occurring in the spine.

Extension principle

This principle of treatment encompasses procedures, both patient- and therapist-generated, that produce extension of the lumbar spine. In a posterior derangement these will be used to abolish, decrease or centralise symptoms. In an extension dysfunction, the extension principle is used for remodelling.

Flexion principle

This principle of treatment encompasses procedures, both patient- and therapist-generated, that produce flexion of the lumbar spine. In an anterior derangement these will be used to abolish, decrease or centralise symptoms. In a flexion or ANR dysfunction, the flexion principle is used for remodelling.

Force alternatives

A change in the manner in which a force may be applied during the exploration of loading strategies to reduce derangements. For instance, alternative start positions (standing or lying), force directions (sagittal or lateral), dynamic (repeated movements) or static forces (sustained positions).

Force progressions

Within each principle of treatment direction (extension, flexion, lateral), there is a range of loading strategies available. These involve greater or more specific forces, but are still in the same plane of movement. For instance, sustained mid-range positions, end-range patient-generated movement, patient-generated force with clinician overpressure, clinician-generated force, or repeated movements over several days. Force progressions are used to determine the correct directional preference and when lesser forces are not able to maintain improvements.

Kappa

The Kappa coefficient is commonly used in studies to address the reliability of two testers to come to the same conclusion about a test. It takes account of the fact that there is a 50% probability of chance agreement even if random judgements are made. It reports a numerical value, with 1.00 being perfect agreement and 0.00 for agreement no better than chance. Negative values imply that agreement is worse than what would be expected by chance alone.

Guide to Kappa values

Kappa value	*Strength of agreement*
<0.20	Poor
0.21-0.40	Fair
0.41-0.60	Moderate
0.61-0.80	Good
0.81-1.00	Very good

Source: Altman 1991

Lateral compartment

The compartment of the intervertebral segment that is compressed with lateral forces. The lateral compartment becomes relevant if lateral forces influence the patient's symptoms.

Relevant lateral component

This refers to patients with derangement who have unilateral or asymmetrical symptoms that do not improve with sagittal plane forces. When the lateral component is relevant, asymmetrical forces are necessary to achieve centralisation or decrease of symptoms.

Lateral principle

This principle of treatment encompasses procedures, both patient- and therapist-generated, that produce an asymmetrical force on the lumbar spine. In postero-lateral or antero-lateral derangement these will be used to abolish, decrease or centralise symptoms.

Loading strategies

Describes
particular structures, and may be dynamic or static – dynamic would be a repeated movement; static, a sustained posture. The significant loading strategies, postures and repeated movements are those that alter symptoms.

Mechanical presentation

The outward manifestations of a musculoskeletal problem such as deformity, loss of movement range, velocity of movement or movement deviations. Very important in re-assessment of treatment efficacy.

Mechanical response

Change in mechanical presentation, for instance an increase or decrease in range of movement in response to a particular loading strategy.

Mechanical syndromes

Refers to the three mechanical syndromes as described by McKenzie – derangement, dysfunction and posture, which describe the majority of non-specific spinal problems.

Non-mechanical factors

Factors that are non-mechanical in nature that may influence a patient's experience of pain. For instance, in the acute phase of a problem, the pain-generating mechanism may be primarily inflammatory. In the chronic stage, various non-mechanical factors, such as central or peripheral sensitisation or psychosocial factors, may influence pain modulation.

Pain

Acute pain

Pain of recent onset of less than seven days. This includes some with pain of an inflammatory nature, but many will experience pain of a mechanical nature due to derangement.

Sub-acute pain
Pain that has lasted between seven days and seven weeks. In some this may represent an interface between inflammatory and mechanical pain, but again, mechanical factors are likely to predominate.

Chronic pain
Pain that has lasted for longer than seven weeks. In the majority this will be mechanical in nature, and non-mechanical in a minority.

Chronic pain states
Pain of long duration in which non-mechanical factors are important in pain maintenance. These factors may relate to peripheral or central sensitisation or psychosocial factors, such as fear–avoidance, etc. Symptoms are often widespread and aggravated by all activity, and patients display exaggerated pain behaviour and mistaken beliefs about movement and pain.

Chemical or inflammatory pain
Pain mediated by the inflammatory chemicals released following tissue damage, or due to systemic pathology, such as ankylosing spondylitis.

Mechanical pain
Pain that results from mechanical deformation of tissues. This occurs with abnormal stresses on normal tissues, as in the postural syndrome, and normal stresses on abnormal tissues, such as in derangement and dysfunction.

Constant pain
Constant pain describes symptoms that are present throughout the patient's waking day, without any respite, even though it may vary in intensity. This may be chemical or mechanical in origin, and may also exist in chronic pain states.

Intermittent pain
This describes pain that comes and goes during the course of the day. Commonly this relates to intermittent mechanical deformation that results in pain. Pain may be momentary or appear and linger for varying amounts of time, but does at some point during the day completely stop.

Site and spread of pain
The area in which pain is perceived in terms of the extent of referral into the limb. The most distal site of pain is important to monitor regarding centralisation and peripheralisation. This information provides important information during assessment and re-assessment of the symptomatic presentation.

Severity of pain
This provides important information during assessment and re-assessment of the symptomatic presentation. Either the patient is asked on a one-to-ten scale about the intensity of the pain on different occasions, or in retrospect is asked to compare present pain to when they first attended.

Peripheralisation
Peripheralisation describes the phenomenon when pain emanating from the spine, although not necessarily felt in it, *spreads* distally into, or further down, the limb. This is the reverse of centralisation. In response to repeated movements or a sustained posture, if pain is produced and remains in the limb, spreads distally or increases distally, that loading strategy should be avoided. The phenomenon only occurs in the derangement syndrome. The temporary production of distal pain with end-range movement, which does not worsen, is not peripheralisation, as this response may occur with an adherent nerve root.

Posterior compartment
Describes the compartment of the intervertebral segment that is compressed with extension forces.

Postural syndrome
Mechanical deformation of normal soft tissues arising from prolonged postural stresses, affecting any articular structures and resulting in pain. A distinguishing set of characteristics is found during the history-taking and physical examination. If prolonged sitting produces pain, it will be abolished by posture correction. Range will be full and pain-free, and repeated movements have no effect.

Red flags
This refers to features of the history-taking that may indicate serious spinal pathology, such as cancer, cauda equina syndrome or fracture. If possible 'red flag' pathology is suspected, further mechanical therapy is contraindicated and the patient should be referred to a specialist.

Reliability
This is the characteristic of a test or measuring tool to give the same answer in different situations. Inter-tester reliability examines the degree of agreement between different clinicians on the same occasion; intra-tester reliability examines the degree of reliability of a single

tester on different occasions. Results are presented in several ways: as a percentage agreement, correlation coefficients, or Kappa values.

Sensitivity

This is a characteristic of a clinical test used to diagnose a problem. The sensitivity is the ability of the test to be positive in all who have the problem. When a test is 100% sensitive, it is able to detect all who have the condition of interest. The sensitivity is the true positive rate. When sensitivity is extremely high (>0.95 or 95%), a negative test response rules out that disease. Poor sensitivity indicates a test that fails to identify many of those with the disease of interest.

Specificity

This is a characteristic of a clinical test used to diagnose a problem. The specificity is the ability of a test not to be positive in those who do not have the problem; it is thus the true negative rate. When a test is 100% specific it is able to identify all those who do not have the condition of interest. When specificity is extremely high (>0.95 or 95%) a positive test result gives a definite positive diagnosis. Poor specificity indicates a test that fails to exclude many individuals without the disease of interest.

Stage of condition

All musculoskeletal conditions can be anywhere on the continuum from acute to sub-acute to chronic. These stages are often of more significance to management than a structural diagnosis.

Standardised terms

These are used to make consistent descriptions of symptomatic responses to different loading strategies to judge their value for self-treatment. The description of symptoms during and *after* loading is significant in determining the management strategy to be applied. These are the words used to describe symptom response during the physical examination.

During loading:

Increase	Symptoms already present are increased in intensity.
Decrease	Symptoms already present are decreased in intensity.
Produce	Movement or loading creates symptoms that were not present prior to the test.
Abolish	Movement or loading abolishes symptoms that were present prior to the test.

Better Symptoms produced on movement, decrease on repetition.

Centralises Movement or loading abolishes the most distal symptoms.

Peripheralises Movement or loading produces more distal symptoms. No effectMovement or loading has no effect on symptoms during testing.

End-range pain Pain that only appears at end-range of movement disappears once end-range is released, and in which the range does not rapidly change. In end-range pain due to derangement, increased force reduces symptoms, while with end-range pain due to dysfunction, increased force will increase symptoms.

Pain during movement

Pain produced during the range of movement, but then subsides or remains when the individual moves further into the range of movement. In the three mechanical syndromes in the spine, this only occurs in derangements.

After loading

WorseSymptoms produced or increased with movement or loading remain aggravated following the test.

Not worse Symptoms produced or increased with movement or loading return to baseline following the test.

Better Symptoms decreased or abolished with movement or loading remain improved after testing.

Not better Symptoms decreased or abolished with movement or loading return to baseline after testing.

Centralised Distal symptoms abolished by movement or loading remain abolished after testing.

Peripheralised Distal symptoms produced during movement or loading remain after testing.

No effect Movement or loading has no effect on symptoms during or after testing.

State of tissues

This describes the different conditions that tissues could be in. They may be normal or abnormal. Abnormal tissues may be injured, healing, scarred or contracted, with healing suspended, hypersensitive to normal loading due to changes in the nervous system, degenerated or painful due to derangements.

Status of condition

This describes the direction of the condition relative to recovery. It may either be improving, worsening or unchanging. Its status is significant in decisions concerning management.

Symptomatic presentation

This describes the details of the patient's complaints and can be assessed and re-assessed regarding site, intermittency/constancy, diurnal variation, severity, consequent analgesic/NSAID consumption and self-reported functional disability. This is very important in re-assessment of treatment efficacy.

Symptomatic response

The behaviour of pain in response to a particular loading strategy, for instance centralisation, peripheralisation, worse or better.

Traffic light guide

Identification of patient's responses to loading strategies, using standardised terminology, determines the appropriateness of a management direction. If the patient remains worse afterwards, this is a 'red light' to that procedure; if the patient remains better, this is a 'green light' for that exercise; if there is no change, an 'amber light', a force progression or force alternative *may* be required. An 'amber' response is also a 'green light' in the presence of a dysfunction.

Treatment principle

The treatment principle defines the force direction used in management; they are termed extension, flexion or lateral. Each principle of treatment contains patient- and clinician-generated force progressions. In a derangement, the treatment principle is determined by the direction that causes a decrease, abolition or centralisation of pain. In a dysfunction, the treatment principle is determined by the direction that reproduces the relevant symptom.

Validity

This is the ability of a test to diagnose or measure what it is intended to diagnose or measure. There are various dimensions of validity, but criterion validity is critical to the accuracy of a diagnosis. This is the ability of a test to determine the presence or absence of a particular pathology. The value of a test is judged by its ability to diagnose the pathology compared to a 'gold standard'. The validity of the 'gold standard' is meant to be about 100%. Validity is measured by sensitivity and specificity.

Yellow flags

Term used to describe psychosocial risk factors for developing or perpetuating long-term disability or sick leave as a consequence of musculoskeletal symptoms. They include factors such as the attitudes and beliefs of the patient about their problem, their behavioural responses to it, compensation issues, inappropriate health care advice, information or treatment, emotions such as depression, anxiety and fear of movement, and relations with family and work.

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