DEVELOPING, TESTING AND REFINING A PHYSIOTHERAPY MODEL OF CARE FOR ACUTE LOW BACK PAIN

A Thesis submitted for the degree of Doctor of Philosophy

By

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ABSTRACT

This thesis is concerned with the physiotherapy management of acute low back pain. Various national guidelines contain conflicting views regarding the role of physiotherapy in the management of acute low back pain. The discrepancies involve primarily the content and timing of physiotherapy intervention. There is a need to place the physiotherapy management of acute low back pain on a more firm research base. A comprehensive literature review was undertaken to develop a best practice model of care for acute low back pain. This model was tested in a randomised controlled trial. Subjects involved in the treatment model demonstrated significantly better short-term outcomes than subjects given advice only. Furthermore, subjects treated early demonstrated significantly better long-term outcome than subjects who waited six weeks for their treatment. Changes in pain and physical function were found to be the factors most closely associated with good outcome in the short-term. Good outcome in the long term was associated with improvement in a number of physical and psychological variables. It is recommended that changes be made to the treatment model to facilitate improvement in pain relief and maintenance of physical and social function to further enhance treatment effectiveness.

I hereby declare that this submission is my own work and that it contains no material previously published or written by another person except where acknowledged in the text. Nor does it contain material that has been accepted for the award of another degree.

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Benedict M Wand

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CHAPTER ONE

Introduction

This Thesis is concerned with the physiotherapy management of back pain. Defining back pain is not without difficulty. A variety of taxonomies have been applied, none of which independently describe the whole experience of back pain (Spitzer et al 1987). The three most commonly used taxonomies are temporal, anatomical and aetiological. In this thesis only acute back pain will be considered, that is back pain of less than six weeks duration (Long 1999). The anatomical definition to be used is pain between the shoulder blades and the folds of the buttocks, with or without leg pain (NHS Center for Reviews and Dissemination 2000), commonly termed **low** back pain. It is the aetiological categorisation of back pain that is most extensive and most controversial (Long 1999). Of the many diagnostic systems in use the classification proposed by Waddell (1998) is one of the simplest and most widely accepted (Koes et al 2001). Waddell (1998) categorises low back pain as, simple low back pain, nerve root pain, or serious spinal pathology. This classification will be applied for the majority of this thesis, in which only simple low back pain will be considered. Further consideration will be given to the controversies of diagnosis and categorisation in Chapter Three.

Low back pain is a health problem of vast dimensions, both in terms of the proportion of people affected and the subsequent cost to the NHS and Social Services (Clinical Standards Advisory Group (CSAG) 1994). The point prevalence in Britain has been estimated at around fifteen percent (Mason 1994), and the lifetime prevalence at about eighty percent (Dionne 1999). Some researchers have suggested that low back pain is so common that it should be considered a normal human occurrence (Waddell 1987). The direct health care costs attributable to low back pain are estimated at £1.6 billion per year and the cost to society at £10.7 billion (Maniadakis and Grey 2000). Most dramatic has been the escalation in these costs in recent years (CSAG 1994, Waddell 1998, also see Chapter Two).

These startling figures have prompted the health authorities of a number of countries to formulate guidelines for the management of acute low back pain (Waddell 1998,

Bronfort 1999, Koes et al 2001). This process began in the UK with the publication of the Clinical Standards Advisory Group (CSAG) back pain report in 1994. This report offered a framework of care for the patient with acute low back pain based on available evidence and the consensus opinion of a multi-disciplinary committee. The CSAG report attracted some criticism for its reliance on anecdotal evidence (Feder and Hemmingway 1995). In 1996 the Royal College of General Practitioners (RCGP) formulated evidence-based guidelines for the management of acute low back pain, which were updated in 1999 (Waddell et al 1996, 1999). These guidelines synthesised the international literature on the management of acute low back pain and provided a rating system of the evidence for a range of diagnostic and management procedures. Algorithms of care were then derived from this research base. Most recently the National Health Service (NHS) Centre for Reviews and Dissemination produced a document summarising the research evidence on the effectiveness of conservative treatment for simple low back pain (NHS Center for Reviews and Dissemination 2000).

Each of these guidelines provides quite contrasting views on the role of physical treatment in the management of acute low back pain. The earliest more anecdotal guidelines offered strong support for the use of physical therapies. Symptomatic pain relief, manipulation, rehabilitative exercise and education were all described as important parts of acute low back pain management and recommendations were made for an increased role for physical therapists (CSAG, 1994). The RCGP guidelines (Waddell et al 1996) originally offered support for early manual therapy in the management of acute low back pain. In the revision of these guidelines (Waddell et al 1999) manual therapy was recommended only if patients failed to return to normal activities or needed additional help with pain relief. Both the 1996 and more recent RCGP guidelines do not recommend reactivation/rehabilitation within the first 6 weeks. Specific back exercises are not recommended at any stage in the management of acute low back pain. The recommended physical treatment in the acute stage was limited to manipulative therapy. Finally, The NHS Center for Reviews and Dissemination review (2000) contains no physical intervention in its summary of effective treatments for acute low back pain.

These discrepancies in the UK documents are mirrored in comparisons between the guidelines of different countries. The American (Bigos et al 1994) and New Zealand

(Accident Rehabilitation and Compensation Insurance Corporation 1997) guidelines both recommend physical treatment for acute low back pain patients. The Dutch (Faas et al 1996) and the draft Australian guidelines (Bogduk 2000) take a 'wait and see' approach to physical treatment and do not routinely recommend its use in the first 6 weeks.

There is a lack of consensus in the international literature on the role of physiotherapy in the management of acute low back pain. This relates primarily to the content of a physiotherapy treatment model and the timing of physiotherapy intervention. It is this uncertainty that principally drives this thesis, the broad objective of which is to place the physiotherapy management of acute low back pain on a firm research base. To achieve this objective four primary research aims will be attended to. These are:

1. To develop a best practice, research based model of physiotherapy care for acute low back pain (Chapter 3)
2. To test the efficacy of this model (Chapter 4)
3. To investigate the effects of the timing of delivery of the model (Chapter 4)
4. To try and more fully understand the mode of action of the treatment model so as to enable further refinement of physical treatment for acute low back pain patients (Chapter 5).

The discrepancies between guidelines exist despite the fact that they are drawn from the same literature base. Bjordal (1998) explicitly addressed this issue and suggested a number of reasons why the evidence has been interpreted differently by different authors. The reasons highlighted included such things as reviewer bias, inadequate literature search, errors in data synthesis and differing methodological filters. Of particular importance to this thesis is what Bjordal (1998) termed the focus of the review. This essentially refers to the homogeneity of the clinical condition under review and the appropriateness of the clinical interventions to the problem. This is particularly relevant to low back pain research (Foster 1999, Golby 1997). In an attempt to mitigate this problem a comprehensive best practice model of care will be developed that considers not only the clinical trials on low back pain but also the philosophy of physiotherapy and the literature on the nature of the low back pain experience. Integrating knowledge from these three area enables consideration to be given not just

to what is done, an issue explicitly addressed in the empirical research literature, but also why it is done, a subject less often considered.

The physiotherapy model of care will be developed from literature review. A number of sources of information can be used to develop and inform the content of treatment. Bogduk and Mercer (1995) suggest three sources of evidence. These are a convention axis, a biological axis and an empirical axis.

The convention axis will consider the philosophy and scope of practice of physiotherapy. This is an important initial step, which aims to answer the fundamental question of whether physiotherapy is suitably placed to manage low back pain. Furthermore, the convention axis will help set the philosophical framework of the model and define the boundaries of what the treatment model may involve.

The biological axis will consider, essentially, what is wrong with someone with low back pain. This information will help decide what the priorities of physiotherapy intervention should be and what the intervention should be trying to achieve. This is important information to help shape the focus of the empirical review as well as provide information to help resolve discrepancies within the clinical research base.

Finally the empirical axis will consider the clinical trials of physiotherapy intervention, providing data on what has been shown to be useful. Evidence from these three areas will be synthesised to develop a comprehensive physiotherapy model of care for acute low back pain.

Once developed, this model and the timing of its delivery will be tested. A single blind randomised controlled trial has been designed that enables both questions to be answered concurrently. All eligible patients will be examined, given an explanation of the examination findings and advice on staying active. Subjects will then be randomised to either enter immediately into the physiotherapy treatment programme or commence treatment six weeks after their referral was received. As the control subjects only enter into the active physiotherapy programme after six weeks a comparison between groups at this time point enables the treatment model to be evaluated against

advice to stay active. Analyses of group differences at longer term follow up will enable comparisons to be made between early and late intervention.

The purpose of clinical research should be to add to the evidence base, inform clinical practice and extend the theoretical appreciation of the clinical condition. The current understanding of low back pain precludes the application of research designs that fully address all these issues. Further analysis needs to be undertaken to understand the mode of action of the intervention. This information can be used to help further refine the treatment model and gain greater insight into the clinical course of acute low back pain. Chapter five will address these issues. Exploring the relative and combined potential of physical and psychosocial parameters in explaining low back pain related disability, and the relationship between changes in each of these parameters and disability over time, will provide a comprehensive model of the determinants of low back pain disability. This analysis will elucidate the process by which good outcome is achieved and provide insight into the possible mode of action of effective treatment. The synthesis of this data will enable further refinement of the treatment model.

Before looking at these four primary aims it is important to review the epidemiology of acute low back pain. Epidemiological evidence has been largely responsible for driving the amount and direction of research into acute low back pain, and has provided valuable information in shaping the current understanding of the low back pain experience.

CHAPTER TWO

The Epidemiology of Low Back Pain

It is unusual to read anything written on low back pain in the last ten years that does not begin with a description of the epidemiology of back pain. This reflects the vast contribution epidemiological research has played in the understanding and management of low back pain. The enormous prevalence and cost of low back pain have contributed to the current practice of guideline development and the funding of research into low back pain management. The escalation in low back pain disability and the elucidation of the myriad of psychological and social variables that have contributed to this have led to a paradigm shift in the understanding of low back pain and how it should be managed (Waddell 1987, 1998).

Medical Epidemiology is the study of the distribution and determinants of disease frequency in human populations (Greenberg et al 1999). This definition encompasses the three key components of epidemiology. These are: distribution, determinants and frequency (Hennekens et al 1987). Investigating these key components of low back pain is not without difficulty. The issue is complicated by the problems of defining low back pain. Low back pain is not strictly a disease but rather a symptom or set of symptoms that may be due to a number of different causes (Bogduk 1997). This allows for enormous variability in describing the population to be studied. Studies have not only used different anatomical definitions but have also differed in how they have defined symptoms. In some instances back pain is recorded only if it lasts a certain period of time, in other cases only if it led to work loss or health care utilisation. These latter two measures more correctly represent back pain related disability and the costs of back care. The epidemiology of these parameters is quite different to the epidemiology of low back pain, as we shall see below. The effect of low back pain

definition on the data obtained is most dramatically demonstrated in the work of Tsui-Wu and Deyo (1987). They defined low back pain as 'pain in the lower back on most days for *at least 2 weeks.* This definition yielded a lifetime prevalence of low back pain of only thirteen percent, about seven times less than is generally reported in the literature (see below).

Epidemiological surveys can also be open to subjective bias as they are dependent on subjects own reporting of pain (Nachemson et al 2000). In studies where subjects are asked about their back pain history over a long period of time there is also a potential for recall bias. A recent paper that surveyed the same subject group three years apart found that ten percent of subjects had forgotten about an episode of low back pain reported three years previously (Waxman et al 2000). Additionally, Nachemson et al (2000) suggest that people with more severe trouble may be more likely to include earlier information than those with less severe symptoms, giving a false impression of average symptom severity and duration. Finally, there is also the potential for sampling bias. The group under investigation may not be representative of the general population.

Enough studies are available which attempt to control for bias and use definitions of low back pain in keeping with those used in this thesis to enable the synthesis of a coherent picture of the epidemiology of low back pain. These studies will now be considered in a brief review of the frequency, determinants and distribution of low back pain.

Frequency of Low Back Pain

Definitions

Frequency involves quantification of the occurrence of a disease (Hennekenns and Buring 1987) and can be defined as a function of its incidence, prevalence and recurrence (Dionne 1999). Incidence is the percentage of a population with new symptoms appearing over a given time, indicating the rate of low back pain (Shekelle 1997). Prevalence is a proportion referring to the percentage of people who have low back pain over a particular period of time. The proportion will vary depending on the

time scale investigated. A number of common time scales are used. Point prevalence is the percentage of people who have the disease now, at the time of investigation, while one month, one year and lifetime prevalence are the percentage of people who have pain at some time during that time period (Waddell 1998). Recurrence is a particularly important parameter in the investigation of low back pain (Croft et al 1998). The natural history of low back pain demonstrates a number of interesting patterns, which will be considered below. Finally, consideration of the frequency of the consequences of low back pain enables a greater appreciation of the low back pain experience.

Incidence

In studies conducted on the general population, one-year incidence rates vary from 1.4% to as high as 11%. The low figure represents data obtained from the Quebec Workers Compensation Database (Abenhaim et al 1987). This is likely to under report the true incidence of low back pain as it only includes those who sustained a back injury at work. The high figure represents the one-year incidence for 30 year olds amongst a sample of Danish adults. This age group probably over represents the low back pain incidence across the full age spectrum as the incidence of low back pain reaches its peak around this age.

Three UK studies, which sampled the general adult population, report yearly incidence rates of 4.0% (Papageorgiou et al 1995) 4.0% (Waxman et al 2000) and 4.7% (Hillman et al 1996). These figures are similar to those obtained from studies on specific populations. Venning et al (1987) demonstrated an annual incidence of 4.9% amongst nurses, and Bigos et al (1992) a 2.3% annual incidence among manufacturing workers. In summarising a number of epidemiological studies, Shekelle (1997) concluded that between two and five percent is a reasonable estimate of the annual incidence of low back pain.

Prevalence

There is far more data available on the prevalence of low back pain. Point prevalence of low back pain in the general adult population ranges from 14% to 33% (Mason 1994, Skovron 1994, Hillman 1996, Cassidy 1998, Nagi 1973). Similar figures are seen when specific populations are sampled. Chiou (1994) found 14% point prevalence amongst

nurses, a figure of 17% was noted amongst male school students (Ebrall 1994) while Bergenudd (1988) demonstrated a point prevalence of 30% amongst 55 year olds. At any given time as much as 33% of the adult population can be suffering from low back pain.

Figures for longer time periods further highlight the extent of the problem. The one-month prevalence amongst the general adult population in the United Kingdom (UK) has been estimated at 39% (Papageorgiou et al 1995). The six-month prevalence of a Canadian survey was 49% (Cassidy et al 1998) and in the United States (US) 41% (Von Korff et al 1988). The data from a number of large UK studies report very similar figures for prevalence over one year. Hillman et al (1996) found a one-year prevalence of 39% amongst the general adult population, Mason (1994) 37% and Walsh (1992) 36%. These figures suggest a consistent prevalence across the country as well as stability in this figure over a number of years. Finally, the figures for lifetime prevalence range from 58% to 85% (Cassidy et al 1998, Papageorgiou et al 1995, Skovron et al 1994, Leboeuf and Lauritsen 1995, 1988, Walsh 1992, Heliovarra 1989). The two UK studies both calculated a lifetime prevalence of 58% (Walsh 1992, Papageorgiou et al 1995).

Several authors have explicitly investigated the trends in low back pain frequency over time. Leino et al (1994) surveyed subjects in Finland every year from 1978 through to 1992. The amount of low back pain reported remained largely unchanged over this period. The findings of this study have been cited as evidence that the prevalence of low back pain has changed little over time.

Waxman et al (2000) provide evidence to support this view. They sampled the same UK population 3 years apart. In this time the one-year prevalence of low back pain in this group rose only slightly. However, they noticed a 50% increase in the number of low back pain patients reporting needing help with activities of daily living. This increase was seen despite there being no change in pain levels, duration of episode or co-morbidity. This discrepancy between the epidemiology of low back pain and low back pain related disability will be considered below.

Palmer et al (2000) used a slightly different methodology and noted almost reciprocal findings. They administered the same questionnaire on similarly derived populations 10 years apart. Over this time the one-year prevalence of low back pain rose from 36% to 49%. Interestingly, there was negligible change in their measure of low back pain related disability over this time (ability to put on hosiery). This finding prompted the authors to suggest that cultural change may have lead to a greater awareness of more minor back symptoms and a greater willingness to report them.

Interrogation of social security and workers compensation databases has also provided information on the historical trends in low back pain frequency. Murphy and Volinn (1999) undertook a retrospective analysis of low back pain claims from 3 separate databases. These included a workers compensation provider, the Washington State Department of Labour and Industry and the U.S Bureau of Labour Statistics. The annual rates of low back pain decreased over time in all three databases. The results showed a 34% decline over 9 years for the U.S bureau, a 19% decline over 5 years for the Washington data and a 22% decline over 4 years for the compensation company. Disability was also investigated by calculating the time lost from work. The authors report a 65% decrease in the number of claims for more than 90 days work loss. Both low back pain and low back pain related disability were found to be in decline.

These figures are in stark contrast to the available UK data. Between 1978 and 1992 there was a 208% increase in the number of days of sickness and invalidity benefit paid due to low back pain (CSAG 1994). Murphy and Volinn (1999) note these discrepancies and suggest that the long-term historical picture has certainly been one of increasing low back pain related disability, but the more recent trend may be one of reversal. Further consideration will be given to low back pain related disability below.

A few epidemiological studies investigating the frequency of low back pain have also provided data on the duration of symptoms, thus giving a fuller picture of low back pain frequency. Taylor and Curren (1985) noted that 14% of low back pain patients in their study had been in pain for more than 30 days. Brattberg (1989) found that 20% of patients reporting low back pain in their sample had been in pain for more than 6 months. Mason (1994) provides probably the fullest picture of low back pain duration.

Subjects were asked to specify the length of any low back pain episode during the previous year. The results are summarised by Waddell (1998) in table 2.1.

Table 2.1 Duration of episode of low back pain during previous year. (From Waddell 1998)

Duration	Prevalence
< 1 week	16%
1-4 weeks	34%
1-3 months	16%
3-12 months	13%
Whole year	20%

While the majority of low back pain episodes last only a short time, a substantial number of people have low back pain for considerable periods. These data challenge the notion that low back pain is a short duration, self-limiting condition.

Natural History

The final parameter to consider in understanding the frequency of low back pain is the rate of recurrence of the problem (Dionne 1999). With respect to low back pain this is usually considered under the heading of natural history. Low back pain has traditionally been viewed as a self-limiting disorder with a very favourable prognosis. Various studies have estimated that between 80% and 90% of patients return to work within a few weeks (Dionne 1999). The favourable view of the natural history of low back pain is quite different when return to work is substituted for pain or disability (Dionne 1999).

Von Korff and Saunders (1996) reviewed a number of papers and different sources of data on the natural history of low back pain. At one-month post consultation about 33% of patients still had moderate intensity pain, while up to 25% still had substantial limitation of activity. At one year only about 20% of patients were back pain free, with 20% still reporting substantial activity limitation.

Papers subsequently published have confirmed these findings. Croft et al (1998) followed up 463 patients who consulted their general practitioner (GP) with low back pain. Twelve months after consultation only 25% of subjects had completely recovered in terms of pain and disability. Van den Hoogen et al (1997) also followed patients for one year. While they reported that 90% of patients were pain free by 12 months, 76% of patients had a recurrence of their low back pain in this time. Schiotz-Christensen (1999) reported that at one year 46% of low back pain patients were still in pain and 15% had experienced recurrent episodes in that year.

Waddell (1998) provides information on an unpublished study of osteopathic patients. One year post consultation 79% of subjects were still reporting some symptoms. Over a four year period 71% of acute patients, 80% of sub-acute patients and 95% of chronic patients had experienced a relapse. These data support the view that low back pain is not a self-limiting disorder. Although the risk of developing severe disabling low back pain is low, the risk of developing chronic low back pain is high, and recurrence is a common feature of the low back pain experience.

The data presented on the frequency of low back pain highlights how common an affliction it is. Furthermore, a significant number of people with low back pain are affected for a considerable period, and the recurrence rate is common and frequent. The majority of data supports the view that the frequency of low back pain has remained relatively constant over time, though some recent data suggests that industrial low back pain, in the US at least, might be on the decline (Murphy & Vollin 1999).

Consequences of Low Back Pain

The presence of low back pain has consequences for that individual and society, and it is these consequences that are the most important characteristics of the low back pain experience (Dionne 1999). In contrast to the relative stability in the incidence and prevalence of low back pain, the consequences of low back pain are increasing dramatically. The consequences of low back pain are generally considered under four headings: disability, work loss, health care utilisation and costs (Dionne, 1999). The

data considered in this section deals with both acute and chronic low back pain. The consequences of chronic low back pain are still important to this thesis as the aim of acute intervention should be to prevent the development of chronicity.

Disability

Disability considers the impact that low back pain has on an individual's life and is most simply defined as a restriction in activity (Nachemson et al 2000). Most surveys reviewed share the common problem of offering a very narrow often one-dimensional view of disability.

In 1997 it was estimated that almost twelve million Americans were disabled by back pain (Frymoyer and Durett 1997). Kelsey and White (1980) found low back pain to be the most common cause of activity limitation amongst adults under the age of 45 and to account for about 10% of all chronic health conditions. More recently Taylor and Curren (1985) established that 14% of US adults reported some activity limitation due to low back pain in the previous year.

The figures for Britain are similar. Approximately 10% of the adult population reported a one-month prevalence of activity limitation due to low back pain (Mason 1994). In their 1994 UK survey Waxman et al (2000) noted 24% of respondents reported that low back pain had interfered with activities of daily living in the previous year. This figure had risen to 38% in the 1997 survey. Papageorgiou et al (1996) found that 8% of their sample had rested in bed because of low back pain at some stage during the previous year. The most complete picture of disability in low back pain comes from Walsh et al (1992). A disability score out of 16 was calculated from the responses to eight questions about ADL. The one-year prevalence for a disability score greater than 8 out of 16 was about 10%.

Work Loss

The majority of data on work loss is derived from Social Security and compensation body databases, so probably under reports the true figures as not all time off work is recorded on these central databases (Nachemson et al 2000).

In 1990/91 the Department of Social Security (DSS) in the UK recorded 67 million days of incapacity due to low back pain, which accounted for 13% of the total working days lost (Klaber-Moffett et al 1995). This figure rose to 106 million in 1993/94 (CSAG 1994), and to 116 million days in 1994/95 (Maniadakis and Gray 2000). Interestingly, between 1986 and 1992, work loss due to low back pain rose by 104% whereas work loss for other reasons rose only 60% (Klabber-Moffett 1995).

Hillman et al (1996) reported a one-year prevalence of time off work due to low back pain of 13.5%. Walsh et al (1992) report both one-year and lifetime prevalence figures. For men the figure for one-year prevalence is about 10% and for women about 7%. The lifetime figures were 34% and 23% respectively.

Data from North America demonstrates similar trends. Back pain accounts for the greatest number of days lost from work in the US (Lawrence 1998). In 1988 there was an estimated 150 million days of work loss attributable to low back pain (Guo et al 1995). In Quebec low back pain accounts for about 10% of all compensated injury claims (Spitzer et al 1987). It has also been demonstrated that the longer a person is off work the less likely it is that they will return to work (Frank et al 1996, Spitzer et al 1987).

Health Care Utilisation

The use of health care services represents such things as visits to health professionals, imaging, surgery and hospital admissions. Hart estimates that low back pain accounts for up to 4.5% of GP visits and for as many as 14% of new-patient consultations (Frymoyer 1988). In the UK the incidence of GP consultation was estimated at 6% (Papageorgiou et al 1995) and the yearly frequency ranges from 6% (Klaber-Moffet et al 1995) to 12% (Walsh et al 1992).

The CSAG report (1994) suggested a figure of 12 million GP consultations for the year 1993. While Maniadakis and Gray (2000) calculated about 8.5 million consultations for 1997/98. These consultations resulted in an estimated 5.3 million drug prescriptions and about 1.7 million X-rays (Maniadakis and Gray 2000). In this same period there were an estimated 10.9 million physiotherapy sessions, 4.3 million osteopathy sessions and

1.7 million chiropractic sessions (Mandiakis and Gray 2000). In 1994/95 there were 55,677 day cases and 69,535 hospital admissions related to low back pain in England (Mandiakis and Gray, 2000).

Costs

The costs attributable to low back pain, both in terms of health care utilisation and cost to society have risen considerably in recent times. In 1995 Klaber-Moffett et al (1995) calculated an annual cost to the NHS of up to £383 million. Mandiakis and Grey (2000) estimated that this had risen to £974 million by 1998.

Societal costs show the same trend. In 1992 the costs attributable to work loss was suggested to be between £1.2 and £1.74 billion (Klaber-Moffet et al 1995). The most conservative figure for work loss in 1998 was £3.4 billion, with an upper estimate of £9.1 billion (Mandiakis and Grey 2000). In 1979 the number of days of paid sick leave was estimated at 26 million. This had risen to 81 million by 1992 and further increased to 106 million by 1994 (CSAG 1994). The economic burden of low back pain in the UK is higher than any other disease for which an economic analysis has been undertaken (Mandiakis and Grey 2000).

Figures from the US are comparable. The cost of compensation for low back pain in 1989 was estimated at $4.6 billion. By 1997 the figure had risen to $11.4 billion (Shekelle 1997). Though now quite old, Webster and Snook's (1994) analysis of a 1989 workers compensation data base provide interesting information on the costs of work related low back pain. While low back pain accounted for only 16% of the number of workers compensation cases, it represented 33% of the total cost. What is more, over the previous three years the cost had risen twice as fast as the consumer price index. The average expenditure per case was $8,300 while the median cost was only $400, indicating a considerably skewed distribution. In fact 96% of the costs were incurred by only 25% of the cases.

The consequences of low back pain to the individual, to the health care system and to the wider community are substantial and rising at an alarming rate. This increase has occurred despite there being little change in the overall frequency or incidence of low

back pain symptoms. These findings have had major implications in the understanding of low back pain and how low back pain affects the individual and society. The traditional medical model has been unable to explain these findings. In fact some authors suggest that a medical approach to the management of low back pain might be in part responsible for the current situation (Zusman 1998). This information has acted as a catalyst for the development of alternative models to explain the low back pain experience and manage the many problems associated with low back pain (Waddell 1987, 1998).

The Determinants of Low Back Pain

Just as it was required to consider both low back pain and the consequences of low back pain to fully understand the frequency of low back pain, it is also necessary to regard the determinants of low back pain under two broad headings. Firstly it is requisite to consider the determinants of the onset of low back pain, commonly termed risk factors. Secondly, once low back pain is present the features influencing the progression of the problem are quite different to what determines onset. These factors will be considered under the heading of prognostic factors.

Risk Factors

The determinants of low back pain have often been investigated by the use of cross sectional or retrospective studies of low back pain patients. These methodologies make it difficult to distinguish cause from effect and do not tell us who, from a given pain free population, will go on to develop back pain. Some authors have attempted to answer this question by the use of prospective cohort studies on pain free populations. It is worth noting that in most cases it is not low back pain per se that is being measured but consultation or reporting of low back pain.

Strongly associated factors

The strongest and most consistent risk factor for the development of low back pain is a previous history of low back pain (Shekelle 1997). Shekelle (1997) contends that no other factor approaches previous low back pain in terms of the strength or magnitude of the association with future low back pain. This reflects the recurrent nature of the problem emphasised in the discussion of natural history above.

The characteristics of the previous episodes also have some predictive value. If there was associated leg pain, use of analgesics, medical consultation or compensation for previous episodes then the risk of recurrence is increased (Muller et al 1999, Ready 1993).

Age is also quite strongly related to the onset of low back pain. The prevalence of low back pain rises with age before decreasing or levelling off around the fifth decade (Papageorgiou et al 1995, Mason 1994). It is uncommon to develop the onset of symptoms before the age of 16 or over the age of 55 (Waddell 1998).

A number of studies have looked at work related risk factors for the development of low back pain. In terms of the strength of association it is generally agreed that psychosocial aspects of the work experience are more strongly associated with the onset of low back pain than biological or ergonomic factors. Job satisfaction has been clearly and consistently shown to relate to the onset of low back pain. Bigos et al (1991) found that poor job satisfaction was the strongest predictor for the subsequent reporting of low back pain among aircraft workers. A finding consequently supported by Skovron et al (1994) Papageorgiou et al (1997) and Bergenudd and Nilsson (1994) on general populations samples.

Different aspects of mental and emotional stress also appear to be related to the reporting of low back pain symptoms. Theorell et al (1990) coined the term *job strain,* which looks at the relationship between the demands of the job, and the control the individual has over those demands. A number of studies have shown relationships between this variable and reporting of low back pain (Bongers et al 1993, Nachemson and Vingard 2000). Weaker though still consistent findings have been found between

the reporting of low back pain and poor social support at work, poor relationships at work and stress at work (Bongers et al 1993, Nachemson and Vingard 2000).

The physical workload of a job still has some association with the development of low back pain. Heavy and repetitive lifting, especially in awkward postures, exposure to vibration and prolonged static loading are the best-documented occupational risk factors (Shekelle 1997, Dionne 1999, Nachemson and Vingard 2000). Nachemson and Vingard (2000) note that there would appear to a dose-response relationship between exposure to physical loads and the onset of low back pain, adding strength to the notion of an association.

Moderately Associated Factors

A number of other variables display inconsistent or weaker relationships to the onset of low back pain. There is a substantial amount of physiological data linking smoking with poor health and degeneration of spinal tissues (Nachemson and Vingard 2000). However, the data linking smoking to the onset of low back pain is contradictory. One problem lies with the potential for confounding factors. Smoking may simply reflect a set of demographic, psychosocial and lifestyle factors that alter the risk of low back pain (Waddell 1998). The balance of evidence from studies that have controlled for confounding variables suggests a consistent but weak relationship between smoking and the development of low back pain (Nachemson and Vingard 2000, Waddell 1998, Shekelle 1997). Interestingly this association does not seem to apply to smoking and sciatica (Nachemson and Vingard 2000).

The data on the association between physical fitness and low back pain paint a very similar picture. There is a substantial body of literature supporting the benefit of physical activity on musculoskeletal tissue (for review see Bland 1993), but the epidemiological data is equivocal. The classic and often quoted study from Cady et al (1979) noted a ten fold decrease in the onset of low back pain from the least fit to the most fit in a group of fire-fighters. The significant body of knowledge published since has failed to replicate these results. Nachemson and Vingard (2000) reviewed 22 papers on the topic, and only 5 offered support for a relationship between fitness and onset of low back pain.

Psychological factors have been investigated extensively with respect to their role in determining the clinical course of an episode of low back pain. Interestingly there is also a small amount of research that has investigated if psychological factors play a role in the development of low back pain. Symptoms of depression and psychological distress in pain free populations have been shown to have a weak association with the development of low back pain in hospital workers (Adams et al 1999, Mannion et al 1996) and the general population (Croft et al 1996). Low mood at baseline was predictive of which nurses would take sick leave for low back pain (Smedley et al 1997). Finally Linton et al (2000) demonstrated that subject's beliefs about physical activity, work and pain (fear-avoidance) and excessive concern about the possible negative consequences from a situation (catastrophizing) were also predictive of low back pain development.

Factors Not Associated

A vast array of other demographic, clinical, occupational and social factors have been investigated. The evidence from a number of extensive reviews on the topic suggests that gender, weight, height, build, strength, mobility, structural abnormalities, social class and intelligence offer little in the way of predicting the onset of low back pain (Nachemson and Vingard 2000, Waddell 1998, Shekelle 1997).

Prognostic Factors

The factors discussed so far have been concerned with the determinants of the onset of low back pain. It is also necessary to consider those factors that determine the clinical course of low back pain once it has started. This is an extensive area of research and one that will be considered in detail in Chapter Three, where these factors will be considered in the context of informing the content of physiotherapy treatment. At this point it is worth noting that prognostic factors differ as low back pain progresses. Those factors that predict outcome in the acute phase are different from the predictors in the sub-acute and chronic phases (Frank et al 1996a,b). Also, while both physical and psychosocial variables have been shown to have predictive value in determining the

clinical course of low back pain, it is the psychosocial factors that are more prognostic (see for e.g. Burton et al 1995).

Linton (2000) summarised the most strongly associated psychosocial prognostic factors as:

- Passive coping
- Fear avoidance
- Catastrophizing
- Depression, distress and anxiety
- Self perceived poor health

Waddell (1998) reviewed the main physical prognostic factors and suggested the most important to be:

- Gradual onset of pain
- Leg pain
- Limited straight leg raise (SLR)
- Root irritation signs
- Poor trunk muscle function

He notes further that the nature and severity of the original injury has remarkably little prognostic value.

These findings further emphasise the multifaceted nature of low back pain. A variety of biological, psychological and social factors both determine the onset of low back pain as well as influence its clinical course. While physical factors might be more important determinants of onset, psychological variables are dominant in determining prognosis.

The Distribution of Low Back pain

The final parameter to be considered in the epidemiology of low back pain is the distribution of the problem. The distribution of low back pain across different geographical areas and between different cultures has received scant attention in the literature. However, the evidence that is available reveals some interesting findings.

These generally reinforce the differences between the epidemiology of low back pain and the epidemiology of the consequences of low back pain, and further highlight the importance of non-biological factors in shaping these differences.

In an early investigation, Anderson (1984) studied the prevalence of spinal pain in rural Nepal and found it to be comparable to that of westernised countries. Likewise Honeyman and Jacobs (1996) found a comparable level of low back pain among a group of traditional Australian Aboriginals, though there was reluctance among this group to openly discuss their pain. Waddell (1998) relates his experiences as a visiting orthopaedic surgeon in Oman and concludes that low back pain was very widely spread among the communities he was working with.

In a review of the epidemiological surveys of different countries, Volinn (1997) noted very similar levels of low back pain between westernised countries and only slightly lower figures among the urban populations of middle and low-income countries. He did conclude that there might be larger differences between urban and rural populations, though it is not possible to determine if this is a difference in low back pain rates or differences in the reporting of low back pain. The evidence generally supports the view that low back pain is distributed uniformly between different countries and cultures.

In contrast to the homogeny of the prevalence of low back pain distribution, the consequences of low back pain display enormous geographical variations. Anderson (1984), Honeyman and Jacobs (1996) and Waddell (1998) all remark on the almost total absence of low back pain related disability in the traditional societies they studied. Moreover Waddell and Waddell (2000) note that urbanisation and rapid industrialisation seem to be associated with increased low back pain disability, though low back pain prevalence remains fairly stable.

Cherkin et al (1994) compared the spinal surgery rates across a number of western countries. There was an almost five-fold variation in the amount of back surgery performed between the highest (USA) and lowest (Scotland) countries. The surgery rate for the US was 40% higher than the next highest country. Even greater differences were seen when surgery rates in different parts of the US were analysed. Volinn (1992)

studied the surgery rates in different counties of Washington State. There was an almost fifteen fold difference in rates across the State.

Norland and Waddell (2000) compared the costs of low back pain between the UK, Sweden and The Netherlands. Despite almost identical prevalence rates there were a number of striking differences in the consequences of low back pain. The rate of visits to physicians for low back pain is almost three times higher in the UK, while the number of sickness days is three times higher in Sweden, and the use of inpatient hospital care in the Netherlands is double that of the other two countries. Such marked differences in culturally similar countries indicate that the consequences of low back pain on an individual are strongly dependant on the traditions of organising health care within these societies.

The distribution of low back pain would seem to be fairly uniform culturally and geographically. In contrast the consequences of low back pain display distribution patterns strongly influenced by geography and culture. These findings reinforce the data from other areas of epidemiological research that psychosocial factors most strongly influence the consequences that low back pain has for an individual. The expectations of the content and intensity of health care provision for low back pain are likely to be different between individuals of different cultural backgrounds.

CHAPTER THREE

A Physiotherapy Model of Care for Acute Low Back Pain

Introduction

The previous discussion on epidemiology highlighted the enormous cost and prevalence of back pain and back pain related disability. Not surprisingly, the management of patients with low back pain is a major part of physiotherapy practice, accounting for about 40% of the physiotherapy caseload (Beckerman et al 1993). The CSAG report (1994) calculated that physiotherapists within the NHS carry out some 7 million treatment sessions per year at an approximate cost of £63 million. A more recent review estimated that physiotherapists undertake about 10.9 million sessions per year at an annual cost of £151 million (Maniadakis and Gray 2000).

Despite these figures, there remains no clear consensus within the profession on how to best manage low back pain (Foster 1998, Pinnington 2001). Deyo and Phillip's (1996) comment that the medical management of low back pain has been a series of fads and fashions is equally applicable to physiotherapy. The physiotherapy literature is replete with theories on the nature of low back pain and accompanying treatment philosophies (for e.g. see Petersen et al 1999 and Riddle 1998).

Attempts by researchers to document current clinical practice clearly demonstrate this diversity in treatment approaches. Sullivan et al (1996) surveyed 155 American physical therapists on the treatments they used for low back pain. The therapists were given a list of 65 different treatment options. In the previous year therapists had used all but one of the treatments mentioned. Also in the US Battie et al (1994) surveyed 186 physiotherapists and asked them to comment on their treatment approach to three hypothetical low back pain patients. An equally large number of treatment modalities

were used. Furthermore the data indicated conflicting philosophies of care between therapists.

Li and Bombardier (2001) repeated this exercise on a group of Canadian physiotherapists. They were given three case scenarios and a list of treatment options. The therapists were asked to pick what treatments they would use as well as provide information on any other treatment preferences. While there was clear preference toward some interventions, all twenty-four listed treatment modalities were nominated as part of the treatment package, as well as a further six interventions not listed. It is also apparent from scrutiny of this data set that different therapists were offering very different models of care for the same case scenarios.

Jette et al (1994) investigated the physiotherapy treatment offered in over 2 300 health care facilities in the US. As well as providing data about the duration and cost of treatment, therapists were asked to list the three most important treatment goals and what they regarded as the three most effective treatment procedures for low back pain. The most interesting thing about these results is that only 10% of therapists listed improvement in function as an important treatment goal, well below such items as 'decrease spasm' and 'improve range of motion'. Furthermore only 5% of therapist nominated functional training as an effective treatment compared to 75% who nominated modalities. These findings are in direct contradiction to almost every evidence-based document published on low back pain management (Koes et al 2001).

Researchers have also documented physiotherapy practice in The Netherlands. Van de Valk et al (1995) analysed the treatments used by 83 physiotherapists in an attempt to explore the relationship between diagnosis and intervention. Patients were assigned a diagnostic categorisation dependant on the length of condition, and analysis was undertaken to see if treatment varied between these diagnostic groups. While there are some small differences, the treatments offered for the different diagnostic categories were very similar. The diagnostic features of the patients' presentation examined in this study did little to change the treatment offered by therapists. The same lists of standardised treatments were applied to patients regardless of the diagnosis.

Physiotherapists in the UK have also been surveyed on their current clinical practice (Foster et al 1999). Foster et al (1999) studied 813 therapists working with low back pain patients. The therapists were asked to identify which intervention they used most often in the management of low back pain. While there were clear preferences for particular types of interventions, eleven different treatment approaches were identified as the most commonly used intervention, as well as an additional five electrotherapy modalities. The study also details an extensive consultation and testing process to develop the questionnaire. It is interesting to note that in trying to understand how physiotherapists are currently managing low back pain the main emphasis was on what therapeutic manoeuvres and techniques the therapists preferred rather than describing the overall philosophy of management.

A more recent analysis looked at the actual treatments used on 200 consecutive low back pain patients at a single clinic (Jackson 2001). Fourteen different treatment modalities were used on the 108 patients who completed treatment.

These figures no doubt partly reflect the heterogeneous nature of low back pain patients and the difficulties of diagnosis in this group (see below), yet no study has identified a clear algorithm of care and there is little suggestion of a unified philosophical approach to the management of low back pain patients. In fact these studies all seem to emphasise recording various treatment techniques rather than exploring the underlying construct of physiotherapy treatment for low back pain.

The aim of this chapter is to put the physiotherapy management of acute low back pain on a more scientific footing and develop a best practice model of care for acute low back pain patients. Data from a number of areas can be used to inform clinical practice and assist in development of a treatment model. Bogduk and Mercer (1995) suggest that selection and application of treatment is dependant on information derived from three distinct, but complementary axes. These are convention, biological basis and empirical proof. These three areas answer the elemental questions of

1. What can physiotherapy do to help patients with simple low back pain (convention)?
2. What is wrong with someone who has simple low back pain (biological)?
3. And what has been shown to help these patients (empirical)?

This methodology will be used to develop a comprehensive physiotherapy treatment model.

Problems associated with the study of low back pain.

Before embarking on this process, there are two features associated with low back pain that require clarification. Both Foster (1998) and Deyo and Phillips (1996) identify several problems and pitfalls associated with the study of low back pain. Most germane to this chapter are the problems associated with aetiology and classification of low back pain.

Aetiology

Back pain arises when one of the innervated structures in the lumbar spine is nociceptively stimulated (Bogduk 1992, 1997). This can occur either mechanically, chemically or thermally (Poterfield and DeRosa 1998). The list of spinal structures in which nociceptive fibres have been identified is extensive and includes bone, disc, facet joints, sacroiliac joints, ligaments, muscles, and vascular and neural tissues (Bogduk 1992, 1997).

To be implicated as a source of back pain Bogduk (1997) suggests that any structure must also be capable of causing pain similar to that seen clinically, and that the structure should be susceptible to pathological processes that are known to be painful. An enormous number of diagnostic labels and pathological processes have been proposed (Long 1999). Nachemson and Vingard (2000) acknowledge as many as 50 definable disease entities that can cause back pain.

Despite this comprehensive and logical approach to studying spinal pain and the variety of sophisticated methods of evaluating the anatomy and function of the spine, a definitive diagnosis can only be reached in ten to fifteen percent of patients presenting with low back pain (Deyo et al 1992, van den Hoogen 1996, Deyo 1998, Nachemson and Vingard 2000). Less than one percent of low back pain is due to serious spinal

disease such as malignancy, infection or fracture (Deyo et al 1992, CSAG 1994, Nachemson and Vingard 2000). A larger group of patients have identifiable specific problems such as stenosis, spinal nerve pain or spondylolysthesis (Deyo et al 1992, Deyo 1998). However, definite identification of the aetiology of low back pain in the remaining eighty-five to ninety percent of patients is not possible (van den Hoogen 1996, Nachemson and Vingard 2000).

As a consequence the medical diagnosis of low back pain is limited to triaging patients into specific and non-specific categories based on identifying clinical syndromes derived from the patients history, clinical examination and the results of appropriate investigations (Waddell 1998). A system of triaging has been integrated into the clinical guidelines of most countries (Koes et al 2001). The most frequently adopted method categorises patients as having serious spinal pathology, nerve root pain or simple low back pain (Waddell 1998).

Simple low back pain is referred to as 'mechanical pain' (Waddell 1998) or 'activity-related pain' (Spitzer et al 1987), so there is the proposition that movement dysfunction is the elemental diagnosis in simple low back pain, though why movement is painful in an individual patient remains elusive. While this method probably represents the best approach given the current paucity of knowledge concerning the aetiology of low back pain, it is not without its deficiencies.

Simple low back pain is a description of symptoms rather than a diagnosis (Long 1999). Labelling the majority of patients with back pain in this way presents a number of problems. Firstly, 'simple low back pain' does not embody any explanation for these symptoms. The lack of consistent coherent theories to explain simple low back pain is a major barrier to effective management (Deyo and Phillips 1996, Foster 1998, Spitzer et al 1987). It does not always serve to allay the fears patients have about their pain, nor provide reassurance that their problem is understood (Deyo and Phillips 1996, Zusman 1997). Zusman (1997) further describes that the dissatisfaction patients are likely to feel with an inexact diagnosis may contribute to the development of chronic spinal pain.

Secondly, there is no clear and logical link between this diagnosis and a treatment response (Deyo and Phillips 1996, Spitzer et al 1987). Without a clear understanding of

the aetiology of the problem it is difficult to know where to begin the search for effective interventions.

Thirdly, the diagnosis of simple low back pain gives a false impression of homogeneity. Patients are grouped in this way not by what they have in common but by what they don't have, i.e. serious spinal pathology or nerve root pain. It is unlikely that this group has a common reason for low back pain, and so is unlikely to respond optimally to a single intervention (Golby 1997).

These features all contribute to the difficulty of researching low back pain. With particular reference to the aims of this chapter, failure to fully understand the aetiology of simple low back pain must be accounted for in any treatment model. The treatment model cannot be prescriptive. Instead it needs to be pragmatic and adaptive to individual patients needs (Golby 1997). The overall aims and philosophy of the model can be described but the implementation of this model must allow for individual variation. To facilitate such an approach it is important that some sort of system to classify patients is used.

Classification

Intimately related to the problems of aetiology is the issue of patient classification. The discussion above already introduced the classification of patients into specific and non-specific categories. It is worth now considering the idea of classification as applied to patients with non-specific low back pain. Both the Quebec Task Force (Spitzer et al 1987) and the International Forum for Primary Care Research into Low Back Pain (Borkan and Cherkin 1996) highlighted the lack of meaningful consistent patient classification as a major barrier to effective management.

The Quebec Task Force (Spitzer et al 1987) set out what they consider to be the characteristics of a useful categorisation system. These are

1. Plausibility
2. Exhaustive classification
3. Mutually exclusive
4. Reliable

5. Clinically useful
6. Simple to use

Wilson et al (1999) identified 35 different patient classification systems for low back pain, but commented that most fail to provide any guide to practical intervention. Both Peterson et al (1999) and Riddle (1998) have provided comprehensive reviews of the classification systems most applicable to physiotherapy. No system was found that adequately fulfilled the Quebec Task Force criteria, particularly with respect to clinical usefulness.

A number of researchers have recently advocated that the lack of a clear aetiology for simple low back pain need not preclude clinically meaningful classification of patients (Maluf et al 2000, Wilson et al 1999, van Dillen et al 1998 Fritz 1998, Fritz and Stevens 2000). These authors propose that rather than attempting to categorise patients into pathological groups, patients should be categorised with respect to the response of their pain to movement, an idea originally proposed by McKenzie (1981). Such a response is in keeping with the central idea that simple low back pain is a movement related problem and offers a categorisation system that guides treatment selection.

These more recent classification systems better match the Quebec Task Force criteria (Wilson et al 1999, van Dillen et al 1998, Fritz 1998, Fritz and Stevens 2000) and offer some interesting possibilities to meaningfully categorise patients with simple low back pain. However, At the time of writing it is felt that none of these systems is developed enough or has been researched enough to be incorporated into the treatment model.

Another solution to the problem of classification is offered by the work on clinical reasoning in musculoskeletal physiotherapy. Clinical reasoning refers to the thought processes and decision making associated with the examination and management of a patient (Jones 1997). For a number of years researchers in this field have advocated the adoption of hypothesis categories to facilitate the examination and management process (Jones 1992, 1995 Jones et al 2002). Hypothesis categories are a unit of information each relating to a particular aspect of the patients problem (Butler 1998). While the labels attached to hypothesis categories have changed over the years (Jones 1992, Jones et al 2002) the process is still essentially one of classifying aspects of the patient's presentation using a variety of sources of information. Furthermore, having 'classified'

some aspect of the patient's presentation, the therapist is encouraged to continually test or validate this knowledge (Jones et al 2002).

Recent research has shown that expert physiotherapists treating musculoskeletal patients do in fact reason in this way (Doody and McAteer 2002) and that expert clinical reasoning leads to superior outcomes in the management of chronic low back pain patients (Levsen et al 2001). While the reliability of this process is yet to be tested it offers probably the most complete solution to ensuring that a heterogeneous group of patients receive treatment that is appropriate to their individual presentation.

The problems associated with the aetiology and categorisation of low back pain presents a number of problems when attempting to develop a treatment model. When the target population is simple low back pain, a non-prescriptive pragmatic treatment model needs to be used. This necessitates some form of patient categorisation. A multitude of systems have been proposed but none fully meet the standards required for clinical use. To circumvent this problem the treatment model will have at it's core a clinical reasoning process. This enables a form of quasi-classification that encourages the treating therapist to individualise the treatment approach within the broad treatment framework. To facilitate this process, at the conclusion of the initial assessment the treating therapist will complete a clinical reasoning document (Appendix VII, VIII) to ensure integration of relevant information and to enable a logical comprehensive treatment plan to be developed.

This form is based on the model proposed by Jones (1994). The treating therapist is encouraged to consider the information gathered during the examination process in order to classify patients. Classification is considered under the headings of pain mechanisms (Gifford and Butler 1997), source of symptoms, nature of dysfunction, contributing factors as well as contraindications and precautions. A management plan is then formulated that best reflects the patient's classification array. Further justification for this model and the component parts will be considered below.

Convention Axis

The first area of information to consider in the development of the treatment model is the convention axis. Bogduk and Mercer (1995) define convention as the expert opinion and beliefs dominant within a profession. They argue that this is the most intellectually weak source of information, and one subject to considerable bias and misperception. While this is undoubtedly true for the definition they have used, and especially so when referring to a particular treatment modality, it is also possible to interpret convention in a more positive light.

For the purpose of this chapter the convention axis will consider the philosophical framework of physiotherapy and the scope of practice of the profession. These are important first steps in the development of a treatment model. Investigating the philosophy of physiotherapy explores the suitability of physiotherapists as managers of acute low back pain and shapes the fundamental agenda of the model. Consideration of the scope of practice helps to identify the possible contents of the treatment model. Rather than being used to evaluate one particular modality, convention here will be used to inform the framework and content of a model of care.

Philosophy

The progression of physiotherapy from an extension of the medical profession to an emerging, independent, applied science (Carr and Shepherd 1993) has been accompanied by several attempts to carefully enunciate the philosophical framework of the profession. In 1999 the World Confederation for Physical Therapy provided the following statement of the philosophy of physiotherapy,

> 'Physiotherapy is providing services to people and populations to develop, maintain and restore maximum movement and functional ability throughout the lifespan'.

Individual National bodies have also developed statements of philosophy. The American Physical Therapy Association (2001) described physiotherapy as 'the

diagnosis and management of movement dysfunction, which attempts to restore, maintain and promote optimal physical function, optimal wellness and quality of life as it relates to movement and health, as well as prevent the onset of movement dysfunction and disability'. The Chartered Society of Physiotherapists (1993) offers a similar definition of physiotherapy as 'the analysis of human movement based on the structure and function of the body, and the use of physical approaches in the promotion of health, and the prevention, treatment and management of disease and disability'.

There are five points contained within these definitions that are particularly important in the development of a physiotherapy model for acute low back pain. These are:

1. The central role of movement dysfunction in the philosophy of physiotherapy.
2. The recognition of the multifaceted dimension of health.
3. The emphasis on diagnosis.
4. The distinction between impairment and function.
5. The non-operational definition of intervention.

Each of these points will be considered in turn and related to the current understanding of low back pain.

The analysis and treatment of movement dysfunction is seen as a fundamental concept within physiotherapy (Carr and Shepherd 1987) and the primary notion that unites the different areas of physiotherapy (Carr and Shepherd 1987). If the treatment of movement dysfunction is seen as the fundamental tenet of the profession, it is worth asking if patients with acute low back pain are well served by such an approach. As was mentioned above, the idea of movement dysfunction is also central to the understanding of low back pain and is the elemental diagnostic classification of simple low back pain (Spitzer et al 1987, Waddell 1998). This parity between the fundamental philosophy of physiotherapy and the fundamental notion of simple low back pain theoretically places physiotherapy in an ideal position to manage simple low back pain.

This idea also sets the broad context of the physiotherapy treatment model. While it remains unclear if abnormal movement is an antecedent to pain or an emergent adaptive behaviour brought on by pain, there is an increasing sentiment among physiotherapy clinicians that changing motor performance is fundamental to the management of low back pain (McKenzie, 1981, Jull and Janda, 1987, Crosbie, 1993, Jull and Richardson,

1994, Norris, 1995, Po`terfield and DeRosa, 1998, Painting et al 1998, Vlayen and Crombez, 1999, O'Sullivan, 2000, Commerford and Mottram, 2001) These various approaches display quite different theoretical assumptions as to the reason for movement dysfunction, but illustrate the professions primary concern of promoting optimal physical function. To reflect the fundamental philosophy of physiotherapy, the central idea of a physiotherapy treatment model for acute low back pain should be the rehabilitation and optimisation of motor performance (Crosbie, 1993).

The epidemiological review illustrated the complex and multifaceted determinants of low back pain and low back pain related disability. In order to understand and therefore manage low back pain, clinicians need to appreciate this complexity. Integral to the philosophy of physiotherapy is this recognition of the multifaceted dimensions of health. Furthermore, as movement dysfunction provides the central role in the philosophy of physiotherapy, it is the movement sciences that inform clinical practice (Carr and Shepherd 1987). This area of science draws on information from a variety of biological, psychological and ecological fields. This demands of individual therapists that they consider beyond the biological domain when reflecting on the reasons for movement dysfunction, an imperative when trying to fully understand the low back pain experience (Waddell, 1998).

In practice, this requires that some form of diagnostic or classification process be undertaken with each patient to determine what the limits to effective performance are. Diagnosis features prominently in both the American and UK statements of philosophy. This again emphasises the appropriateness of physiotherapy in the management of low back pain and also suggests that diagnosis and individualisation of treatment should be an integral part of any physiotherapy treatment model.

Chapter Two highlighted the differences in the epidemiology of low back pain and low back pain related disability. This implies that to comprehensively manage low back pain attention needs to be given to both pain and function. Separation of disease/dysfunction and disability is implicit in both the American and UK statements. The suitability of physiotherapy in the management of simple low back pain is again verified. The treatment model must also be delivered within a framework that aims to

affect pain, dysfunction and disability. The clinical reasoning document acts to ensure that attention is paid to these three dimensions of the low back pain experience.

The final point to consider is the non-operational definitions of physiotherapy. This point is most easily understood if the opposite condition is considered. Some health care professions are very specific in their operational definition, such as an acupuncturist or masseur, or very closely associated with a particular intervention such as chiropractors and spinal manipulative therapy (Zusman 1997). In the case of low back pain, two problems arise with this approach. Firstly, as low back pain is a complex and multifaceted experience it is unlikely that one type of intervention will provide optimal care for all patients (Golby 1997). Secondly, results of clinical trials and changes in our understanding of low back pain mean that interventions need to evolve with these changes in scientific knowledge. A profession with a strong operational philosophy will be unable to adapt as theory progresses.

The philosophy of physiotherapy is not concerned with specific techniques and procedures, so is well positioned to offer comprehensive treatment algorithms supported by current theoretical and empirical research and to develop as the understanding of low back pain develops. A non-operational philosophy sets the context and the broad aims of treatment but does not stifle evolution and development of novel interventions.

This brief review of the philosophy of physiotherapy provides a number of important pieces of information to aid in development of the treatment model. Firstly, it demonstrates that on many levels, the physiotherapy profession is particularly well suited to the management of simple low back pain, maybe optimally so. Secondly, the philosophy also sets the broad framework for development of the treatment model. The essential tenet is the optimisation of functional motor performance within a model that attempts to identify the individual reasons both for pain and pain related disability and provides clinically proven interventions cognisant with these aims.

Scope of Practice

The discussion above has highlighted the suitability of physiotherapy in the management of acute low back pain, by underlining the compatibility between the philosophy of physiotherapy and the current theoretical understanding of acute low back pain. The philosophy of physiotherapy also needs to be related to the empirical axis, setting the boundaries for what physiotherapy intervention might involve and guiding the empirical review. This is generally referred to as the scope of practice.

The Chartered Society of Physiotherapy's document on scope of practice (Chartered society of Physiotherapy, 2000) reflects, to a certain extent, the non-operational philosophy of physiotherapy. The description of scope of practice offered is 'the services that members are educated, competent and insured to provide'. The American definition (APTA 2001) is similarly worded as 'the care and services provided by or under the direction and supervision of a physical therapist'. Both bodies, however, recognise that there are core skills within the profession, an area that has recently been extensively explored by the American Physical Therapy Association (APTA 2001).

The American Physical Therapy Association (2001) describes three major components of physiotherapy intervention. These are

- Coordination, communication and documentation
- Patient instruction
- Procedural interventions

The procedural interventions are further classified under a number of headings. Of primary concern in the management of acute low back pain are

- Therapeutic exercise and training
- Manual therapy
- Electrophysical and mechanical modalities

These last three procedures are also described in the UK document as the core skills of the physiotherapy profession (Chartered Society of Physiotherapy 2000).

These five components represent the agreed scope of practice of the physiotherapy profession and will form the boundaries for the treatment model. The next step is to see how these interventions fit in with the theoretical understanding of low back pain and

then review which of these interventions have been shown to improve outcomes in patients with acute low back pain.

Biological Axis

Introduction

Bogduk and Mercer (1995) refer to the biological axis as the theoretical mechanisms underlying a particular intervention. As the purpose of this review is to develop a treatment model rather than evaluate a particular intervention, the biological basis will be interpreted slightly differently in this chapter. If the elemental diagnosis for simple low back pain is activity-related pain (Spitzer et al 1987), the next logical step is to ask why does it hurt when patients with simple low back pain move?

The biological review will attempt to answer this question by reviewing the basic science underlying the low back pain experience. This information will help decide what the priorities of physiotherapy intervention should be and what changes physiotherapists should be trying to make in their patients to ensure good outcome.

There are three caveats to this section of the review. Firstly, while the model being developed is concerned with the management of acute low back pain, there is relatively little theoretical research into the nature of acute low back pain. Almost all of the vast body of literature is on chronic patients. While this may limit the usefulness of these findings it can be argued that a major part of the management of acute low back pain is to prevent the development of chronicity. Looking at what distinguishes chronic low back pain patients from the pain free population still gives insight into what the acute model should be trying to 'prevent'.

Secondly, while good quality research can establish a sound theoretical relationship between observed biological phenomena and symptoms in a group of patients, it does

not establish causality. This problem is partly circumvented by the integration of information from the empirical axis into the development of the treatment model. In addition, the applicability of theoretical research findings to a given individual patient is not always clear. This further emphasises the importance of a clinical reasoning process to help integrate relevant knowledge into the treatment plan and individualise the intervention.

Lastly, though the term 'biological' axis is used, this section is concerned with any theoretical data related to simple low back pain. Psychological contributors to the low back pain experience will also form part of this review.

The theoretical aspects of the low back pain experience will now be reviewed. The information has been grouped under the headings of structural, mechanical, neurophysiological, biochemical and psychological. It is believed that these five headings encapsulate much of the current research into the mechanics of low back pain and provide a comprehensive overview of what physiotherapists need to consider when managing low back pain.

Structural Basis of Simple Low back Pain

The first area to be considered is the relationship between anatomy or, more correctly, patho-anatomy and low back pain. A patho-anatomical approach to low back pain has dominated research for many years (Waddell 1998, Zusman 1997 1998), and has greatly increased the understanding of the structure of the lumbar spine. The applicability of this information to the understanding of simple low back pain however is open to question. While greater understanding of spinal anatomy and enhanced methods of imaging the spine have improved the care of patients with specific low back pain (Frymoyer 1997), it is less clear if this information has improved the treatment of simple low back pain (Boos and Lander 1996, Deyo 1998, Zusman 1997 1998). This section will consider the relationship between simple low back pain and structural problems within the spine.

Waddell (1998) points out that an important first step in determining if a structural anomaly is a causative agent in low back pain is to demonstrate that the particular finding is more common in people with low back pain than in those without. This relationship has been explored in two main ways, radiological examination and magnetic resonance imaging. Other forms of spinal imaging have received much less attention (Rowe 1997) and though in-vitro studies have provided important information about pathological processes operating on the spine (Twomey and Taylor 1995) and the effect of these changes on the mechanical behaviour of the spine (Krismer et al 2000, Fujiwara et al 2000), insufficient attempts have been made to link these findings with pain and disability.

Radiology

Radiological examination has been the mainstay of investigative procedures for low back pain for many years (Rowe 1997). The main purpose of radiographic examination is to exclude the presence of specific causes of low back pain such as malignancies, fractures, infections and inflammatory disease (Bigg-Wither and Kelly 1995, Rowe 1997). Subsequently, criteria have been proposed (red flags) for the selective use of radiographs in situations where the history and physical examination suggests specific low back pain (van Tulder et al 1997a, Koes et al 2001).

Despite the low prevalence of specific low back pain in primary care, there is substantial evidence that radiological examination remains a common part of the examination of low back pain (Clinical Standards Advisory Group 1994, Klaber-Moffett et al 1995, Mandiakis and Gray 1999). Owen et al (1990) found that eighty percent of general practitioners always or usually refer patients with recurrent low back pain for x-ray, and seventy percent always or usually refer patients with first episode low back pain lasting more than a month for x-ray. Furthermore, the primary reason doctors gave for ordering x-rays was to reassure the patient or to reassure themselves rather than as a method of identifying specific pathology. Whether x-ray findings provide any relevant information to guide treatment of simple low back pain will now be considered.

The list of structural anomalies identifiable from x-ray findings is quite impressive and includes a variety of degenerative, congenital and postural anomalies (Giles 1997, Waddell 1998). van Tulder et al (1997a) conducted a systematic review of relevant observational studies to explore whether there is a causal relationship between abnormal findings and simple low back pain. Thirty-five publications were identified, of these 18 studies were of good or acceptable methodological quality and were included in the analysis. The results did not show any relationship between low back pain and spina bifida occulta, transitional vertebrae, spondylosis, spondylolysis, spondylolisthesis or Scheuermann's disease. There was some association between degenerative changes and low back pain, though this relationship was weak and subject to a number of areas of bias. They concluded that they could not show a causal relationship between structural changes and clinical symptoms.

This review was recently updated to include data up to the end of 1998 (Nachemson and Vingard 2000). No information was found that changed the conclusion of the van Tulder et al (1997a) review. Furthermore, Nachemson and Vingard report on two earlier reviews that also discount any relationship between structural x-ray findings and low back pain (Andersson and Deyo 1996 Bigos et al 1992).

A more recent cross sectional study on lumbar spine degeneration (Peterson et al 2000) found no association between degeneration and low back pain related disability and only a weak association between degeneration and pain levels. Interestingly this study showed that patients who had been subject to previous trauma showed greater levels of facet degeneration than patients without a history of trauma. However, there was no difference in pain and disability between the trauma and non-trauma groups, again demonstrating a lack of clear association between back pain and structural changes. Similarly Clauw et al (1999) found no association between x-ray findings and pain or disability in a group of chronic low back pain patients.

There is little evidence to support any meaningful relationship between radiographically identifiable structural problems and the symptomology of simple low back pain. The obvious criticism of this conclusion is that x-ray provides only limited information on the structure of the lumbar spine and more detailed investigations of spinal morphology may provide different answers. This view will now be explored.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) uses the principles of nuclear magnetic resonance to obtain high quality imaging of spinal soft tissue (Bigg-Wither and Kelly 1995). It provides the best available images of spinal structure with the added advantage of being non-invasive and non-ionising (Rowe 1997). MRI has replaced most other investigative methods, such as myelogram and computed tomography (CT), as the investigation of choice in low back pain (Rowe 1997). MRI undoubtedly provides greater insight into the morphology of the lumbar spine. The next section attempts to establish if there is a causal relationship between abnormal anatomy as visualised by MRI and simple low back pain.

A number of studies using MRI have demonstrated a high incidence of abnormal findings in patients without back pain. Boden et al (1990) scanned sixty-seven subjects who had never had low back pain. Overall about a third demonstrated significant abnormalities. Amongst the subjects over 60 years old, fifty-seven percent had abnormal scans, thirty-six percent had herniated disc and twenty-one percent had spinal stenosis. Furthermore, in this age group all but one of the subjects demonstrated a disc bulge.

Jensen et al (1994) scanned ninety-eight asymptomatic individuals. Two thirds of this group demonstrated anatomical pathology. Fifty-two percent of subjects had a disc bulge, twenty-seven percent a protrusion and one subject an extrusion. Schmorl's nodes were found in nineteen percent of subjects, annular tears in fourteen percent and facet arthropathy in eight percent. They conclude that the presence of many forms of structural pathology in low back pain patients may be coincidental. They did however comment that the low incidence of disc extrusion might indicate this as being a relevant finding.

Boos et al (1995) scanned forty-six asymptomatic persons matched for age, sex and mechanical risk factors with a group of patients selected for disc surgery. In the pain free cohort, sixty-three percent demonstrated disc protrusions, thirteen percent extrusions and eighty-five percent some degree of disc degeneration. Twenty two

percent of the group demonstrated neural compromise. The quite high prevalence of disc extrusion is in contrast to the findings of Jensen et al (1994) and suggests reappraisal of their comment that disc extrusion may be a relevant finding in low back pain. The discrepancy between these results probably represents the greater methodological rigour employed by Boos et al (1995) in matching the groups they were comparing.

Boos et al (2000) followed up these same subjects five years later to determine the natural history of asymptomatic disc anomalies. They found no worsening of disc herniations or neural compromise, whereas degeneration progressed in seventeen individuals. However, changes in MRI findings were of no value in predicting the onset of low back pain and offered no explanation for the appearance of back pain.

Jarvik et al (2001) imaged the spine of 146 asymptomatic veterans affairs patients. Though all patients had been back pain free for four months prior to the scan, fifty-four percent of the cohort had experienced previous low back pain. Eighty-three percent of this group demonstrated desiccation of one or more discs, sixty-four percent had a disc bulge, thirty-two percent a disc protrusion and six percent demonstrated disc extrusion. Disc extrusion was the only finding that was significantly associated with a history of low back pain. There were no significant differences between those with and without a history of low back pain for stenosis, nerve root compression, disc degeneration, desiccation or bulging, loss of disc height, annular tears, endplate changes, facet joint degeneration or spondylolisthesis.

Savage et al (1997) scanned a group of 220 working men, a quarter of whom had never experienced low back pain. The scan was repeated twelve months later, and in this time thirteen of the subjects experienced low back pain for the first time. However, in these thirteen subjects there was no change in the MRI appearance of the lumbar spine to explain the onset of symptoms.

The high prevalence of structural anomalies identified in patients without low back pain offers little support for the idea of a structural basis for low back pain. This notion is further challenged by the results of studies on low back pain patients.

A number of papers have looked at the relationship between morphological changes in the disc and a positive discography. Lumbar discography involves the injection of contrast medium into the disc and is used to identify pain-generating discs (Rowe 1997). Itto et al (1998) found that structural anomalies apparent in the disc had limited validity in predicting discogenic lumbar pain. Saifuddin et al (1998) found that MRI evidence of annular tears was poorly related to positive discography.

Buirski et al (1993) scanned a group of low back pain patients who underwent discography as well as a control group of pain free subjects who underwent MRI only. While noting a high correlation between positive discography and the MRI finding of a degenerated disc with protrusion, there was no difference in the prevalence of disc degeneration or protuberance between the painful and control groups. They conclude that there is no morphological feature that would indicate if a disc is symptomatic. In contrast to these findings, Moneta et al (1994) noted a strong correlation between positive discography and the presence of a grade 3 radial fissure in the annulus on post discography imaging (CT), though these results have yet to be replicated.

There has been recent controversy about the relevance of a 'high intensity zone' on midsaggital and axial views of the lumbar intervertebral disc. Originally described by Aprill and Bogduk (1992), they produced data from low back pain patients indicating a high level of agreement between the presence of a high intensity zone and a positive discogram. A finding later supported by Schellhas et al (1996).

However, more recent studies have been unable to replicate these results. Stadnik et al (1998) and Ricketson et al (1996) both found little correlation between the presence of a high intensity zone and a positive discogram. Rankine et al (1999) investigated the relationship between the presence of a high intensity zone and a number of clinical features. They concluded that the presence of a high intensity zone does not define any patient group or relate to any particular clinical features. Carragee et al (2000e) reported the presence of high intensity zones in the discs of twenty four percent of asymptomatic subjects. While this is lower than the fifty-nine percent prevalence they noted in patients, they conclude that the prevalence of high intensity zones in asymptomatic population was too great for this to be a meaningful clinical finding.

Conclusion

It is possible to identify a myriad of structural anomalies in low back pain patients. In almost all cases these findings are frequently prevalent in pain free populations. When pain free groups have been followed up, there has been no relationship between morphological changes and the onset of low back pain. No single structural anomaly has consistently been shown to relate to the presence of pain and the degree of structural disturbance is not related to the severity of pain. Faced with these findings it is difficult to give credence to a structural basis for simple low back pain. It would seem that the explanation as to why people with low back pain experience pain when they move is not a structural one.

This conclusion should perhaps not be too surprising, as the data reviewed has related to simple low back pain. Patients who have identifiable, relevant structural problems are not included in this group. The lack of identifiable structural problems is almost part of the definition of simple low back pain. This idea further strengthens the notion that simple low back pain has little structural basis.

The implications of these findings to the development of a treatment model are two fold. Firstly, as the problem is unlikely to be primarily structural, this reinforces the view of the appropriateness of physiotherapy management of simple low back pain. Conservative physical treatment is unlikely to have much influence on a primarily structural problem.

Secondly, an important part of the scope of practice of physiotherapy is patient instruction (APTA 2001). The treatment model needs to involve an educational component that explains the insignificance of structural problems. This view has been recently reinforced by a controlled trial that randomised patients to an x-ray plus General Practitioner (GP) management group or GP management alone (Kendrick et al 2001). The x-ray group were more likely to report low back pain at three months, had a lower health status score and a higher rate of subsequent GP consultations. The authors hypothesised that the awareness that subjects in the x-ray group now have of (unrelated) radiographic anomalies reinforces the patient's belief that they are unwell. It

is worth noting that though the x-ray group demonstrated worse outcome, they were significantly more satisfied with their care.

Patients have been shown to place great importance on radiographic examination (Espeland et al 2001), and a structural basis for low back pain is the dominant belief among low back pain patients (Zusman 1997). These findings suggest that disavowing patients of a structural cause of simple low back pain might be difficult. The relevance of these findings are further emphasised by a significant body of research which shows that agreement on the underlying reason for a problem between patient and provider is an important determinant of outcome (Meade et al 1990, Zusman 1997, Cherkin et al 2001, Kalaokalani et al 2001). To overcome these issues, it is important that the educational component is intensive, in-depth and comprehensive as well as monitored to ensure that the information is understood.

The obvious caveat on these findings is that the structural problem might be one that is not accessible to current imaging techniques. This argument is not new and has been levelled at every form of imaging. However, no added value has been achieved in the understanding of simple low back pain despite a greatly increased sophistication in visualisation of anatomy. Furthermore, if a relevant structural problem were found in a sub-group of patients with simple low back pain, they would cease to have 'simple' low back pain. As mentioned above, the definition of simple low back pain almost precludes a structural explanation.

Mechanical Basis for Simple Low Back Pain

Mechanical models for acute low back pain are very pervasive, being particularly dominant in physiotherapy literature (for e.g. McKenzie 1981, Norris 1995, Edmonston and Elvey 1997, O'Sullivan 2000, Comerford and Mottram 2001). This is not surprising given the central role of movement dysfunction in physiotherapy. The central premise of the mechanical model is that people with low back pain experience pain when they move because they move abnormally. Pain results from abnormal function and can occur in the absence of structural damage (Waddell 1998).

Motor performance is a complex activity requiring the interaction of a number of different body systems (Lieber 1992). To simplify this complexity, the study of movement is usually categorized under one of three headings. Kinematics, the study of motion without reference to its cause; kinetics, the study of forces acting on a body; and muscle mechanics, the study of the body's organs of force production (Smith 1990). This taxonomy will be used to review the evidence for the relationship between abnormal movement and low back pain. It is important to note that this area of research is overwhelmingly on patients with chronic low back pain. Separating out whether movement dysfunction is causative or merely an adaptation to an ongoing pain state is particularly difficult. Caution needs to be taken in interpreting the significance of these findings to the management of acute low back pain.

Kinematics

The way subjects with low back pain move can be investigated in various ways. Most broadly, some researchers have studied kinematics by observing patients during the performance of functional tasks such as walking and lifting. This gives some indication of the way subjects choose to move and use their spine during their daily activities. A slightly more specific view is to look at the movement available in the lumbar spine in isolation. This may indicate the maximal amount of spinal movement available (potential) or the amount of movement the patient feels prepared to use at that time (performance). Finally it is possible to investigate motion at individual segments of the spine. It is commonly felt that this aspect of analysis is less dependent on subject motivation.

The major elements involved in appreciating the kinematics of human movement are the amount of movement and the pattern or quality of movement (Lee 1995). It is important to recognise that the parameters that describe movement quality will be different for functional tasks, whole spine movements and intersegmental motion.

Kinematics of Functional tasks

The most commonly studied functional activity is walking. In an early paper Keefe and Hill (1985) compared a group of chronic low back pain patients with a pain free control

group. They found that the patient group walked more slowly, took shorter steps and demonstrated greater asymmetry than healthy subjects.

Khodadadeh and Eisenstein (1993) performed a similar study on a much larger group of subjects. The back pain patients were measured prior to lumbar surgery, 6-months post surgery and 2-years post surgery. At baseline they found very similar results to Keefe and Hill (1985). The low back pain patients likewise walked slower, took smaller steps and were less symmetrical than healthy controls. Analysis 6-months after surgery revealed no significant change from the preoperative measurements. Two years post surgery walking speed had significantly improved, though not to levels seen in healthy subjects. None of the other walking parameters changed. This study shows differences in movement between patients and controls. These movement problems are long standing and persist despite surgical intervention. The authors further concluded that there appeared to be little relationship between gait parameters and patients' perception of treatment success.

Rowe and White (1996) observed the three-dimensional spinal mechanics during gait in a group of nurses after a mild bout of low back pain. They could not detect any influences of back pain on spinal movement during gait.

Vogt et al (2001) compared subjects with and without low back pain walking on a treadmill at 4.5 km/hour. Temporal parameters of gait were measured as well as three-dimensional movements of the lumbar spine. The low back pain group had significantly shorter stride times than controls, suggesting shorter steps. The phasic patterns and angular spinal displacements of patients with simple low back pain were within normal limits. However, the patients demonstrated significantly higher degrees of stride-to-stride variability. So while it seems that low back pain patients do not differ in the amount of spinal movement available during walking, there appears to be problems in the ability to optimally control the quality of motion.

In a small pilot study, Selles et al (2001) compared the walking performance of back pain patients and control subjects at a variety of speeds. Coordination of arm and leg movements as well as pelvic and thoracic rotations was analysed using a relative phase algorithm. Patients had a significantly slower self-selected walking speed. In addition,

patients demonstrated a greater asymmetry in phase-relations between left and right sides of the body and differences in coordination patterns between thorax and pelvis as speed increased.

A similar methodology to evaluate the amount and coordination of pelvic and thoracic rotations was used in a much larger study by Lamoth et al (2002). They found no difference in the amount of thoracic or pelvic rotation between patients and controls, but again demonstrated a slower self selected walking speed and a difference in coordination patterns. In normal subjects there is in-phase coordination between the pelvis and thorax at slow speeds. As speed increases this changes to a strategy of anti-phase coordination. Both Lamoth et al (2002) and Selles et al (2001) demonstrate that low back pain patients maintain in-phase coordination at all speeds.

The mechanics of walking is different between healthy subjects and patients with low back pain. The consistent findings are a decrease in speed, asymmetry of gait and differences in control and coordination strategies. There seems to be no difference in the amount of spinal motion during gait. These findings suggest increased guarding and more care with movement as well as problems with motor control.

Gait symmetry was used as an outcome measure in a study comparing manipulation to back school (Herzog, et al 1991). They found that symmetry could be normalised following manipulative treatment but not with back school. However normalisation of symmetry had no effect on pain or disability. In fact, patients in the back school group demonstrated significant improvement while the manipulation group did not. This finding, and the follow up results of Khodadadeh and Eisenstein (1993), questions the therapeutic utility regarding the observations of asymmetry and decreased speed of movement.

A research group based in Western Australia has investigated the relationship between low back pain and the kinematics of cricket fast bowling (Foster et al 1989, Elliot et al 1992). Their research suggests that it is possible to discriminate between bowlers with pain and bowlers without pain based on the mechanics of their bowling action. Subjects who demonstrate a mixed bowling action are more likely to develop back pain than those with either a pure front on action or a pure side-on action. The data indicates that

the key feature that discriminates between a painful and non-painful bowling action is the relative alignment between thorax and pelvis during delivery. If pelvic and thorax rotation is out of phase by more than 10 degrees there is a strong and significant chance of pain developing. This data supports the relationship between mechanics and pain but also suggests that the failure of low back pain patients to use an anti-phase co-ordination strategy during walking is likely to represent an adaptive response to pain rather than a causative mechanism.

A number of researchers have investigated the interaction between hip and lumbar spine movements during functional activities. Paquet, et al (1994) performed a small study to investigate differences in hip-spine movement interaction during forward and backward bending between low back pain patients and healthy controls. This study is particularly interesting as subjects were quite acute. Similar to the walking data, patients demonstrated a decreased velocity of movement. However, there was no difference in the amount of movement or the pattern of movement between subjects, though the data indicated a trend toward less lumbar movement contributing to the movement of forward flexion in patients.

Porter et al (1997) also compared the relative contribution of the hip and lumbar spine to forward bending in healthy subjects and low back pain patients. The subjects with chronic low back pain demonstrated a significant reduction in mean total range and in mean maximum lumbar flexion, though mean hip flexion was equivalent.

These results are partly supported by Lariviere, et al (2000). They looked at forward and backward bending and likewise demonstrated less lumbar movement in the patient group but with little change in total range of motion. They found that patients compensated for loss of lumbar movement by increasing the contribution of thoracic mobility to the total range of motion. A later study of lifting by the same authors (Lariviere et al 2002) found no difference in spinal mobility between groups. Finally, Dolan and Adams (1993) found that low back pain patients flexed their lumbar spines less during lifting than normals.

Together these studies suggest that when low back pain patients perform functional tasks they move their backs less than normal subjects. Furthermore, they may compensate for this lack of mobility by moving more elsewhere.

Esola et al (1996) analysed forward bending motion patterns in healthy subjects and in patients with a history of low back pain that were currently pain free. Though the total range of motion was not different, the patient group tended to use more lumbar motion than hip motion during the early phase of flexion.

These authors used the same methodology to look at hip-spine movement interaction during extension from a forward flexed position (McClue et al 1997). There was no difference between groups in the total available movement at either the hips or the back, but the pattern of movement interaction was different. The subjects with a previous history of low back pain again used more lumbar movement than hip movement in the early part of range.

These findings suggest little difference between patients and control subjects in the amount of movement available, but a consistent pattern of excessive lumbar movement relative to hip movement in patients with a history of low back pain.

This finding is in direct contrast to the studies mentioned above. The fact that the patients were back pain free at the time of testing may explain these discrepancies as well as offering some insight into the relationship between abnormal movement and pain. The pattern of excessive lumbar movement puts more load on spinal tissue so is unlikely to be a response to pain and could in fact be causative, an idea supported by the data on cricket fast bowlers and given further credence by a recent prospective cohort study of pain free subjects followed for 3 years (Sjolie and Ljunggren 2001). These authors found that a ratio of high lumbar mobility with low lumbar strength at baseline was predictive of future low back pain.

Continual use of a movement pattern that places more load on spinal tissue may eventually result in pain, at which time the individual will try and reduce load on the lumbar spine by restricting the lumbar spinal contribution to functional tasks. These findings suggest that part of the treatment model should be to consider how patients

control spinal motion during functional tasks and that attempts simply to increase spinal motion may not be optimal.

Kinematics of the Lumbar spine

The range of motion of the lumbar spine in low back pain patients has been extensively investigated. Early studies generally found that patients with low back pain had reduced range of spinal motion compared to healthy controls (Mayer et al 1984, Marras and Wongsam 1986, Triano and Shultz 1987, McIntyre et al 1991).

More recent studies using more accurate methods of motion analysis and more appropriate statistical models have begun to question this view. Burton et al (1989b) used a multivariate stepwise regression to explore the relationship between back mobility and low back pain. There was no evidence of a relationship between back pain and mobility in school age children. In adults there was some evidence that patients with present or past back pain were less mobile than healthy controls, but the strength of this association was very weak.

Using a stepwise discriminate analysis, Klein et al (1991) found that lumbar spine range of motion could not correctly identify subjects with or without low back pain. McGregor et al (1995) measured spinal range of motion with a computerised tri-axial potentiometric analysis system. They found no difference in range of motion between patients and controls for extension, side flexion or rotation, though low back pain patients did demonstrate less lumbar flexion. Masset, et al (1993) used an isometric dynamometer to measure spinal range. They were unable to demonstrate any significant difference in range of motion between patients and controls.

Adams and Dolan (1995) conclude that the variability found in healthy people precludes range of motion as a useful method of classifying low back pain. Moreover, McIntyre, et al (1993) demonstrated that there is a significant difference between preferred and maximal range of motion in low back pain patients, suggesting observable deficits in motion represent the amount patients are prepared to move rather than the available movement.

Lumbar range of motion has also been found to demonstrate poor concordance with the severity of low back pain. Spinal range of motion shows little relationship to levels of pain (Rainville et al 1992, McGregor et al 1998a, McGregor et al 1998b, Gronblad, et al 1997b) and disability (McGregor et al 1998b, Nattrass et al 1999, Sullivan, et al 2000, Waddell and Main 1984, Gronbald et al 1997b).

Furthermore, the clinical utility of range of motion has been questioned. Burton et al (1990) undertook a one-year prospective study of patients undergoing manipulative treatment to compare the relationship between saggital mobility and symptoms. While there was some association between improvement in pain and increased mobility in the first month, there was no relationship thereafter. Pain continued to improve with no concomitant change in mobility. Scatter plot analysis revealed that symptomatic improvement was as common in patients with unaltered or reduced mobility as it was in those whose mobility increased.

The literature on spinal range of motion offers little to the development of a treatment model other than emphasising that an approach that attempts to simply increase spinal mobility is unlikely to be successful.

In contrast to the lack of validity attributable to range of motion, quality of motion seems to be more strongly associated with low back pain. Movement velocities consistently and strongly discriminate between low back pain and non-low back pain groups (Marras and Wongsam 1986, Mcgregor et al 1995, McIntyre et al 1991, Marras et al 1993, 1995, 1999, Masset et al 1993) as well as demonstrating agreement with symptom severity (Marras et al 1993, 1999). Moreover Marras et al (1993, 1999) showed that while range of motion changed little with symptom resolution, improvements in both velocity and acceleration mirrored changes in clinical condition.

The consistent finding is that those with back pain move more slowly than normal subjects and have lower acceleration rates. Most authors attribute these findings to caution and fear of movement rather than indication of any fundamental mechanical problem. McIntyre et al (1993) showed that when encouraged, low back pain patients are able to greatly increase their movement velocity. So while these findings appear

consistent and valid they probably are an epiphenomenona rather than a causative factor, making it difficult to relate these findings to a particular management strategy.

More novel methods of measuring spinal motion may offer some clinically applicable information. Jayaraman et al (1994) monitored spinal side flexion using a computerised motion analysis system while subjects stood on a force plate. They independently measured upper (L1-L3) and lower (L3-S1) lumbar spine movements. The results showed decreased motion for low back pain patients compared to controls and these differences were greater for the lower lumbar spine. Furthermore, this decrease in range was more pronounced at higher speed. The speed dependency of the movement loss in low back pain patients again emphasises the view that the lack of movement is an issue of 'motivation' rather than indication of an underlying primary problem.

The inclusion of force plate data enabled a number of novel aspects of the quality of movement to be analysed. The authors conclude that the quality of lateral bending motion, rather than the maximum range of motion, is most affected in low back pain. Specifically they found that low back pain patients demonstrated anomalies in the way that stiffness was regulated through range. In the early part of range the stiffness of patients was less than for normal subjects. This pattern was reversed in outer range, with patients demonstrating an increase in stiffness.

These findings have concordance with the results of McClure et al (1997) and Esola et al (1996) in that patients seem to move excessively in their lumbar spines early in movement and this is independent of total available range. This mechanical behaviour may indicate failure of the active sub-system in controlling spinal stability (Panjabi 1992a). Early in range stiffness is dependant on active control, while later in range the passive sub-system is able to affect stability. While preliminary, these findings have some clinical applicability, indicating that it might be important to encourage the active control of spinal motion in the early part of range.

Intersegmental kinematics

The kinematics of the spine can also be studied by observing the mechanical behaviour of individual motion segments. There is considerable data on *in-vitro* measurements of intersegmental motion (Bogduk 1997) and there is a clear relationship between altered quality and quantity of intersegmental motion and degenerative changes in the spine (Gertzbeim et al 1986, Ogon et al 1997a, 1997b). However, the relationship of abnormal intersegmental mechanics to low back pain is less well investigated. Furthermore, the little data that is available is predominantly on specific back pain such as nerve root pain (Tibrewall et al 1996) or spondylolisthesis (Axelsson, et al 2000, Takayanagi 2001, Schneider, Pearcy and Bogduk 2001).

Stokes et al (1981) used bi-planar radiography to compare the intersegmental motion of low back pain patients and normal controls. The low back pain group included a sub-group of patients with nerve root pain as well as a group with non-specific low back pain. The results are difficult to interpret, but it appears that painful subjects display increased segmental motion and abnormal segmental coupling, particularly increased shear movement with saggital plane movements.

Pearcy, et al (1985) used a very similar methodology. They found decreased segmental mobility and abnormal coupling in low back pain patients at all lumbar levels. The variation in coupling seen related to increased side flexion and rotation during saggital movements in low back pain patients. This was explained as evidence of asymmetrical and poorly coordinated muscle action.

Sihvonen (1997) studied the intersegmental mobility in one hundred low back pain patients during the performance of saggital plane movements. Using previously established norms of intersegmental mobility they detected excessive anterior shearing in twenty-seven percent of the cohort and excessive posterior shearing in thirty-five percent.

Spratt et al (1993) likewise screened 612 patients with low back pain. They found that twenty-four percent had excessive posterior shear and seventeen percent excessive

anterior shear during the performance of flexion and extension movements. This is significantly higher than the rates of abnormal shear seen in healthy populations.

In contrast to these findings Okawa et al (1998) was unable to find any difference in segmental motion between patients and normals, though the numbers involved in this study were very small.

A recently developed way of evaluating the quality of segmental motion is mapping the instantaneous centre of rotation. The behaviour of the axis and the path it takes throughout movement can be plotted in a sequence to depict a locus known as a centrode (Bogduk 1997). In normal segments the centrode is tightly clustered and consistently located (Pearcy and Bogduk 1988), whereas in degenerated segments the pattern is much more erratic (Gertzbein et al 1986). Recently the centrodes of normal spines were compared with a group of simple low back pain patients (Schneider and Bogduk 2000). In each case, at one lumbar segment, the centrode was significantly different to the normal distribution. The authors surmised that the pattern of movement seen reflected excessive axial movement during extension.

There is some evidence that intersegmental motion is altered in low back pain. There is the suggestion of increased intersegmental mobility, though this finding is inconsistent and Dvorak et al (1991) comment that the large variation in rotational values between individuals in the normal population may limit the clinical usefulness of this finding. More interestingly, there is evidence of a disturbance in the pattern of segmental movement. The data consistently points to the presence of abnormal coupling, particularly excessive shearing and axial subluxation. From a clinical perspective this suggests poorly controlled segmental motion.

Summary

Numerous kinematic anomalies are apparent when studying the movement of low back pain patients. It is possible to identify two broad categories of movement anomaly. Firstly, there are a group of actions that would serve to decrease load on spinal tissue, such as limiting movement or decreasing the velocity of movement. Theoretically it

would be expected that these abnormalities are a response to pain rather than causative so have less clear clinical utility.

Secondly, there are some movement differences that would seem to increase the load on spinal tissue. These seem to consistently be related to poorly constrained or controlled spinal and intersegmental movement. Theoretically it would be expected that these abnormalities are causative or contribute to the maintenance of symptoms and are therefore important issues to integrate into a treatment model. Panjabi (1992a,b) would consider that these observed movement patterns are indicative of inadequate spinal stability. The primary therapeutic options for this problem relate to enhancing muscular and neurological control of the spine (Panjabi 1992a,b). For this reason the specific treatment implication of these findings will be considered below.

Kinetics-Spinal Loading

The mechanics of spinal loading has been extensively investigated using a variety of methodologies (Bogduk 1997, Adams and Dolan 1995, Pearcy 1997). There is an abundance of information on the magnitude of loads under various conditions and how spinal tissue attenuates and responds to these loads. The relationship of loading to tissue injury and degeneration is being increasingly understood (Lotz 1999). However, less attention is paid to how these loads relate to back pain (Lotz 1999). The purpose of this section is to discover if patients with low back pain load their back differently to pain free subjects. One possible reason why subjects with low back pain experience pain when they move is because they load their spine abnormally.

This question can be addressed in two ways. Firstly, do patients with low back pain participate more frequently in activities that load the spine excessively? Secondly, do low back pain patients move in such a way that their spine is loaded abnormally during the performance of normal daily activities?

The first question was addressed in Chapter two and will only be considered briefly here. Epidemiological evidence generally supports the view that exposure to high load

activities increases the likelihood of the development of low back pain. Heavy and repetitive lifting (especially in awkward postures), exposure to vibration and prolonged static loading have all been documented as risk factors for the development of low back pain (Shekelle 1997, Dionne 1999, Nachemson and Vingard 2000). Nachemson and Vingard (2000) note that there would appear to be a dose-response relationship between exposure to physical loads and the onset of low back pain, adding strength to the notion of an association. A similar pattern is seen with participation in sport. Moderate levels of sporting activity are not associated with an increased risk of back pain (Burton and Tillotson 1991), while more vigorous sporting activities are (Sward et al 1990).

To formally test the association between spinal loads and low back pain, Marras et al (1993a) assessed the dynamics of lifting requirements in high risk and low risk industries. Using a multiple logistic regression they found that lifting frequency, load size, trunk velocity and trunk saggital angle strongly distinguished between jobs that were high risk and jobs that were low risk for developing low back pain.

It does seem that patients with low back pain participate more often in activities that place high loads on the spine. While these findings have wide ergonomic applicability, their value in the development of this treatment model is limited. However, enquiring about the activities that the individual participates in during their working day will help with establishing a prognosis and planning the return to work. A section on work activities will be included in the assessment protocol to facilitate this process.

It is only very recently that researchers have started to consider whether patients with low back pain load their spine differently to healthy subjects during the performance of the same activity. Marras et al (2001) investigated this supposition by studying the loads on the lumbar spine of low back pain patients and pain free controls as they performed a number of low load lifts. Patients with low back pain experienced twenty-six percent greater spinal compression load and seventy-five percent greater lateral shear than the asymptomatic group. The increased spinal loads were due mainly to muscle coactivation, indicating problems of motor control.

Lariviere et al (2002) investigated the loading of the L5/S1 segment during lifting and lowering tasks. They found no difference in loading characteristics between patients

and controls. The differences in results might reflect different methodology. Lariviere et al (2002) placed the loads significantly closer to the body. The decreased load on the spine that this entails might not have been enough to cause the subjects to initiate a co-contraction strategy that Marras et al (2001) felt was responsible for the increased loading observed. An alternative explanation for the discrepancies seen may lie in the subjects recruited. Marras has published widely in the area of industrial low back pain and lifting injuries. It may be that he has recruited more subjects for whom lifting is a problem. Lariviere's group may have recruited fewer subjects for whom lifting is a painful task.

If the results of Marras et al (2001) are replicated, this provides some important insight into the mechanical basis of low back pain. It may be that inappropriately coordinated muscle activity and the resultant internal forces generated could be a causative agent in low back pain or the maintenance of chronic pain. This finding again supports the idea that addressing inappropriate muscle activity might be an important part of the treatment package.

Muscle performance

Muscles are the effector organs of the movement system, producing the desired movement (or stabilisation) of body segments required for a particular task. Muscles fulfil this role due to the fact that they are able to actively produce tension (Billeter and Hoppeler 1992). It is possible to describe four aspects of active force production necessary for the performance of functional tasks (Knuttgen and Komi 1992). These are: strength, the maximal force generating capacity of muscle; power, the rate of force production; endurance, the period over which a given force can be maintained; and timing, a term used synonymously with skill, dexterity, coordination or control (International Olympic Committee, 1991). Timing has been defined as the specification of the amount of stimulation for each muscle as a function of time (Bobbert and van Soest 1994).

The ability of muscles to perform these functions is related to their unique structure. Under the influence of the appropriate neural input, chemical energy is converted to mechanical energy by the activated muscle, a process well described elsewhere (for e.g.

Billeter and Hoppeler 1992). This intimate relationship between the structure and function of muscle is well documented (Herbert 1993, 1995, Bruton 2002) and has strongly influenced research into the role of muscle function in low back pain. Some researchers have concentrated on documenting morphological changes in the muscles of individuals with low back pain, while other groups have focused on aspects of muscle performance.

The following review of the role of muscle performance in low back pain will reflect this. Firstly, consideration will be given to morphological changes. While the reporting of morphological differences is interesting the clinical utility of these findings is not immediately obvious. This information might establish that the muscles of low back pain patients are different from healthy individuals but it is the understanding of performance deficiencies that will shape the development of a treatment model. To explore this, the literature on the performance of muscles in low back pain patients will then be reviewed. The overall aim of this section is to ascertain if problems of muscle performance explain why patients with low back pain experience pain when they move.

Morphological Changes in Muscles

Fibre composition

A number of authors have reviewed the literature on muscle fibre composition in low back pain (Ng et al 1998, O'Sullivan, et al 1997b, Richardson et al 1999). There is consistent evidence of selective atrophy of type II fibres in the muscles of low back pain patients, and some suggestion that there is an increase in the ratio of type IIx/IIa. Type I fibres seem to preserve their size but demonstrate some internal structural changes, namely a moth-eaten appearance (interruption of the intermyofibrillar network) and the appearance of central cores (an area devoid of mitochondria)(Mattila et al 1986). More recently published papers support these earlier findings (Ramsbacher et al 2001, Zhao et al 2000, Mannion et al 2000). In addition, Mannion et al (2000) demonstrated that type II atrophy is strongly dependant on symptom duration, while internal fibre structure changes were independent of duration, suggesting a different pathogenisis for these two findings. In fact, type I disruption was found to be strongly

related to age, prompting the authors to conclude that the changes in type I fibres might be non-specific to low back pain.

The clinical utility of these findings is supported by the work of Rantanen et al (1993). They biopsied the extensor muscles of low back pain patients 5 years apart. At five years, subjects were divided into either improved or not improved groups. The improved group demonstrated both fibre hypertrophy and reversal of type I degradation, while the non-improved group showed worsening of type I degradation and no type II fibre hypertrophy.

Further support can be found in the work of Mannion et al (2001). These authors showed an association between improved disability scores and an increased proportion of type II fibres at the expense of type I fibres in the multifidus muscle of chronic low back pains.

Muscle size

The force generating capacity of muscle is directly proportional to its cross sectional area, or more correctly the cross sectional area of the contractile elements (Herbert 1995). A decrease in the cross sectional area of spinal muscles of low back pain patients is well documented (Richardson et al 1999). Numerous studies have shown that low back pain patients have wasting of the paraspinal and psoas major muscles (Danneels et al 2000, Dangaria and Naesh 1998, Richardson et al 1999), though it appears the abdominal muscles are unaffected (Critchley and Coutts 2002). It also emerges that there is increased fat deposition and fibrosis in the paraspinal muscles of patients (Alaranta et al 1993, Parrkola, et al 1993, Lehto et al 1989, Mooney et al 1997) further compromising the muscles force generating capacity. Alaranta et al (1993) showed a moderate agreement between disability scores and the amount of fat deposition in the muscle. The important issue associated with these findings is whether these changes simply reflect disuse or if some other mechanism is operating.

Mannion et al (2000) used multivariate analysis to examine the relationship between muscle size and duration of symptoms. They found no relationship between degree of atrophy and symptom duration. Danneels et al (2000) showed that paraspinal wasting was only apparent at certain levels of the spine. Dangaria and Naesh (1998) noted

similar findings for the psoas major muscle. Unilateral wasting occurred at the level of symptom production but not necessarily at levels above and below. Finally Hides et al (1994) Danneels et al (2000) and Laasonen (1984) demonstrated that wasting can be apparent in one of the paraspinal muscles (in each case the lumbar multifidus) and not others. These findings are not in keeping with generalised disuse and suggest a more localised, specific phenomenon. It is important to consider that these observed muscle deficits might not respond to a general exercise approach.

The study of Hides et al (1994) offers some insight into the possible mechanism at play. The lumbar spines of first time low back pain patients were scanned soon after the onset of back pain. Localised, segmental multifidus wasting was demonstrated ipsilateral to the side of symptoms. The localisation of this phenomenon and its appearance so soon after the onset of symptoms suggests that it represents locally mediated painful inhibition rather than being related to disuse. In fact the authors noted significant atrophy in one subject only 24 hours after the onset of symptoms (Richardson et al 1999).

In a follow up study (Hides et al 1996) subjects were rescanned at 10 weeks. Those subjects who had been involved in a specific exercise programme for the multifidus demonstrated recovery of muscle wasting. Patients who had not been involved in this programme still demonstrated wasting despite being pain free and performing their normal functional activities. This again questions the view that disuse is the primary issue and further supports the view that generalised activities might not effectively resolve these observed deficits.

The muscles of low back pain patients are morphologically different to the muscles of healthy subjects. While it is possible that some of these findings might relate to disuse, the data generally points to a more specific localised phenomenon, possibly pain inhibition. There is some evidence that segmental atrophy does not spontaneously resolve in the first 10 weeks of low back pain despite the restoration of normal function. Additionally restoration of type II fibre morphology seems to be more closely associated with good outcome than restoration of type I fibre morphology.

Strength Changes

The maximal force generating capacity of muscles is the most investigated aspect of muscle performance in low back pain. This vast body of literature provides contradictory findings. Several investigators have found that the trunk muscles of patients with low back pain are weaker than those of healthy individuals (Mayer et al 1989, McNeil et al 1980, Kishino et al 1985, Mayer et al 1985, Nouwen 1987, Mooney et al 1997, Hultman et al 1993, Bayramoglu et al 2001,Cassisi et al 1993, Suzuiki and Endo 1983, Rantanen and Nykvist 2000, Brady et al 1994), whereas other researchers have noted no significant difference in trunk muscle strength between these groups (Masset, et al 1993, Klein et al 1991, Nicolsaisen and Jorgensen 1985, Holmstrom et al 1992, Newton et al 1993, Grabiner, et al 1992).

The discrepancies in the literature reflect the considerable methodological problems associated with measuring strength in low back pain subjects. Because pain can hinder maximal effort, the test might reflect more a measure of the patient's tolerance than a true measure of the maximal force generating capacity of muscles. Keller et al (1999) found that pain on exertion was a significant predictor of performance on an isokinetic strength test. McIntyre et al (1993) demonstrated considerable differences in strength between the preferred effort and the maximal effort of low back pain patients. Using an interpolated twitch technique, Verbunt et al (2002) showed that chronic low back pain patients demonstrate significantly increased levels of inhibition compared to controls.

Holm et al (2000) formally investigated the relationship between trunk muscle strength and pain. The isokinetic muscle strength of chronic low back pain patients was measured on three consecutive occasions, twice before a facet joint anaesthetic block and once afterwards. Patient's pain and fear levels were simultaneously monitored. Strength increased significantly between the first and second tests. At the same time pain had also increased, but fear was found to decrease. Strength was not significantly different between the second and third (post facet block) test, though pain and fear were dramatically less. Changes in reported pain seem to offer no explanation for muscle strength. Changes in fear might explain the differences between the first two tests, though there is also a learning component associated with the performance of isokinetic strength tests (Newton et al 1993). Together these studies indicate that the strength

testing on patient groups can produce results that do not only reflect maximal muscle strength.

Strength varies considerably in the normal population and is dependant on sex, age, build, activity levels and genetic factors (Bruton 2002). The wide variability seen raises the question of the sensitivity and specificity of strength measurements in discriminating between low back pain patients and normals. Studies that have used more powerful statistical models have found disappointing results. Newton et al (1993) reported that though mean strength values were different between patients and healthy controls, the ranges were wide and overlapping and that discrimination of individuals is limited.

Klein et al (1991) found poor discriminatory values for strength, with the correct classification of only fifty-seven percent of low back pain patients and sixty-three percent of non-low back pain patients. This study is further notable for the attempt to control for factors known to influence trunk strength. Unless these factors are controlled for, comparisons between normal and back pain groups are liable to be misinterpreted.

The contradictions in the literature make it difficult to determine the significance of lumbar muscle strength to low back pain. Epidemiological evidence has failed to find any relationship between back muscle strength and the onset of low back pain (Nachemson and Vingard 2000, Waddell 1998, Shekelle 1997) and the low sensitivity and specificity of strength changes in discriminating between painful and non-painful groups further decreases the importance of these findings to the development of a treatment model. Moreover, many of the activities that patients with low back pain describe as being painful are low force tasks (van dar Valk et al 1995) where trunk muscles would be operating at well below their maximal force generating capacity. It is doubtful that these tasks are strength limited. In many situations there is little likelihood that low back pain patients experience pain when they move due to lack of muscle strength. Obviously this point needs to be tempered by clinical reasoning as an individual patient may experience pain with a particular functional task that is strength limited.

The main unresolved issue in relation to these findings on strength is the discrepancy between strength findings and the observations of muscle size and composition. It seems clear that the low back muscles of low back pain patients have a decreased cross sectional area. The evidence relating cross sectional area to the intrinsic ability of muscle to generate tension is quite strong (Herbert 1993). Furthermore, studies on other parts of the body have shown that muscle atrophy is clearly related to strength decrements (Herbert 1993). In fact there are both clinical and laboratory data that observed strength reduction is usually of a higher order of magnitude than loss of muscle size (Herbert 1993).

One possible explanation is that as the observed atrophy appears to be quite localised the strength loss might be too small to be measurable with current techniques. Alternatively, loss of force generating capacity in one part of the erector spinae may simply be compensated for by increase in another part. Using MRI evaluation of lumbar muscles Flicker et al (1993) showed that different subjects recruited different paraspinal muscles during the performance of a standardised back extension exercise. Emphasising that though the load may be the same the way muscles are recruited is variable.

Endurance Changes

Endurance capacity can be measured by observing the ability of a muscle group to contract repetitively or sustain a single contraction over a prolonged period (Bruton 2002). In contrast to the findings on muscle strength, investigators have consistently demonstrated a reduced endurance capacity in the trunk muscles of patients with low back pain (Nicolaisen and Jorgensen 1985, Jorgensen and Nicolaisen 1987, Kankaanpaa et al 1998b, Suzuiki and Endo 1983, Holmstrom et al 1992, Hultman et al 1993 Simmonds et al 1998). Furthermore use of multivariate analysis has shown that the association appears to be quite strong (Roy et al 1995, 1990, Peach and McGill 1998).

While this literature has predominantly looked at the paraspinal muscles, some recent investigations have also demonstrated reduced endurance capacity in the abdominal muscles of low back pain patients (Evans and Oldreive 2000, Ng et al 2002).

It is also worth noting that in studies that have simultaneously measured strength and endurance, endurance has reliably been show to be a more significant determinant of low back pain (Nicolaisen and Jorgensen 1985, Jorgensen and Nicolaisen 1987, Suzuiki and Endo 1983, Holmstrom et al 1992).

Power spectral analysis of muscle activity during fatiguing tasks has also been used as a method of quantifying trunk muscle fatigability (Kankaanpaa et al 1999). The results of these studies provide further evidence that low back pain patients have poorer trunk muscle endurance than normals (Peach and McGill 1998, Capodaglio et al, 1995, Mayer et al 1989, Roy et al 1989).

One advantage of power spectral analysis is that it allows for the assessment of individual paraspinal muscles. Studies that have differentiated between paraspinal muscles during extension endurance tests have consistently shown that lumbar multifidus demonstrates the greatest fatigue rates (Biederman et al 1991, Thompson et al 1992, Roy et al 1989, 1990).

Endurance capacity shows some relationship with symptom severity. Holmstrom et al (1992) found that loss of muscular endurance was greater in patients with constant low back pain than those with intermittent pain. Kankaanpaa (1999), Thompson et al (1992) and Capodaglio, et al (1995) all established that improvements in pain and disability matched improvements in lumbar muscle fatigability, while both Luoto et al (1995) and Mannion et al (1997) provide evidence that reduced lumbar endurance predicts future low back pain.

The theoretical relationship between poor endurance and back pain is more robust. Many tasks that patients report problems with (van dar Valk et al 1995) are low force tasks that could be endurance limited. Some research has also identified that abnormal spinal movements can be induced by fatiguing the lumbar muscles (Kankaanpaa et al 1999). The data presented indicates that muscle endurance is a significant entity in low back pain and efforts to improve the trunk muscle endurance capacity of patients should form part of the treatment model.

The reason for the presence of the endurance deficit seen would also help shape the treatment model. Cooper et al (1993) found no difference in central drive between patients and controls during fatiguing contractions of the paraspinal muscles, decreasing the idea of a central problem. In support, Kovacs et al (2001) monitored blood flow and oxygen use in the erector spinae muscles of patients and control subjects during the performance of low load exercise. There was no difference in blood flow between groups, but low back pain patients used less oxygen, emphasising a peripheral dysfunction.

It is also worth considering if this peripheral dysfunction is general or specific to the back muscles. Wittnik et al (2000) compared the maximum oxygen uptake of low back pain patients with matched controls during the performance of a treadmill test. They found no difference in maximum oxygen uptake between these two groups. This further confirms that the endurance deficit is peripheral and that it is probably local to the spinal muscles. This findings adds support to the view developed in the morphological review that general exercises are unlikely to optimally influence the muscle deficits observed in low back pain patients.

The theoretical evidence consistently relates a deficit in trunk muscle endurance to the presence of low back pain. It is predictive of low back pain and improvements in muscle endurance mirror improvements in functional outcome. What's more, there is evidence that the problem is a peripheral one and largely specific to the trunk muscles, primarily multifidus. It seems reasonable to conclude that endurance training should be included in the treatment model and that to be affective in reversing these problems, it is necessary that the training is specific to the trunk muscles.

Timing Changes

The coordination of efficient muscle function is extremely complex and this is reflected in the multitude of ways researchers have used to evaluate the issue of coordination or motor control in low back pain patients. Three different areas of research have been identified and will be reviewed in a bid to discern if changes in neural control are related to low back pain.

Altered levels of activation:

One of the simplest and crudest ways to gain insight into the function of the neural control system is to record the level of activation of muscles. This is done through quantifying the electromyography (EMG) signals to working muscles. It is important to point out that EMG is difficult to accurately quantify, particularly for the purpose of comparisons between subjects (Smith 1990). Moreover differences in EMG amplitude may not represent a primary central processing problem. Increased EMG signal might represent a central response to a peripheral problem as, for example, a fatigued muscle will require more stimulation to produce the same force output as a non-fatigued muscle.

Decreased EMG signal amplitude of the lumbar paravertebral muscles relative to control subjects has been demonstrated by a number of authors (Cassisi et al 1993, Sihvonen et al 1991, Ahern et al 1988, Soderburgh and Barr 1983, Cooper et al 1993) whereas other authors have noted increased paraspinal muscle activity (Arendt-Nielson et al 1995, Kravitz, et al 1981). Other researchers report no difference in the EMG output of low back pain patients (Sihvonen et al 1997, Nouwen et al 1987)

The inconsistencies in these findings mean that muscle activation levels offer little insight into determining the presence or nature of motor control problems in low back pain patients.

Altered patterns of muscle activation and recruitment:

The most consistent EMG finding in low back pain is the disappearance of the flexion relaxation phenomena. During the activity of forward flexion from the standing position, there is normally a sudden cessation of muscle tension as the force required to maintain the position is transferred from the paraspinal muscles to the thoracolumbar fascia (Watson et al 1997). In contrast to normal subjects, low back pain patients have been shown to maintain muscle activation at the end of forward flexion (Watson et al 1997, Nouwen et al 1987, Ahern et al 1988, Sihvonen et al 1991, Haig et al 1993, Paquet et al 1994).

Findings from power spectral analysis also reveal differences in muscle recruitment. Biedermann et al (1991) evaluated the EMG power spectrum of low back pain patients and controls during the performance of a standardised isometric extension test. They demonstrated a difference in paraspinal muscle recruitment between groups. The back pain group fatigued their lumbar multifidus more than the iliocostalis lumborum, while controls showed no preferential fatigue.

Similarly Roy et al (1989) found that during a back extension task the medium frequency decay of multifidus and iliocostalis lumborum, but not longissimus thoracis, was higher than that of healthy subjects. These findings suggest that though the net extensor moment is the same low back pain patients produce this moment using different muscles. These findings have been supported by other researchers (Peach and McGill 1998, Thompson et al 1992, Roy et al 1990).

A similar response has been noted for rotation tasks. Ng et al (2002) compared muscle activation patterns of trunk muscles during an isometric trunk rotation task. Statistically significant differences were seen between patients and controls for muscles recruited. The back pain group demonstrated lower levels of activity in rectus abdominus and multifidus and higher levels of activity in the external obliques.

While these findings demonstrate that low back pain patients do recruit their muscle differently to normals, it is difficult to attribute this finding as evidence of a fundamental problem of motor control. The disappearance of the flexion relaxation phenomena probably represents a protective mechanism and has been shown to return to normal after symptoms have resolved (Haig et al 1993). The preferential differences in medium frequency shifts can be explained from a purely peripheral perspective. The findings may simply reflect a peripheral deficit in muscle morphology and function (see above). This view is supported by the work of Thompson et al (1992). They demonstrated reversal of these recruitment deficits following a back-care exercise programme aimed at improving lumbar muscle endurance.

Other studies provides more convincing evidence of fundamental recruitment problems in low back pain patients. Grabiner and El Ghazawi (1992) recorded bilateral paraspinal EMG during the performance of isometric trunk extension. In normal subjects there

was strong and consistent coupling of the EMG signal between the two sides. Furthermore, there was strong association between the EMG and trunk extensor moment. In contrast, the low back pain patients demonstrated both temporal and amplitude decoupling of the EMG signal between the two sides as well as decoupling between the EMG and extensor moment. For normal subjects, both sides contributed equally to the extensor moment throughout the period of force production. In low back pain patients, the extensor moment is produced first by muscles on one side then on the other. Force production is not smoothly coordinated between the two sides. It is difficult to envisage a plausible peripheral reason for this finding.

Some researchers have investigated the muscle recruitment patterns of patients when exposed to sudden loads. Radebold at al (2000) found that in contrast to healthy controls, patients demonstrate a significantly different muscle recruitment pattern in response to sudden trunk loading. The patients demonstrated greater rates of co-contraction and longer muscle reaction times both for switching off inappropriate muscles and switching on task appropriate muscles. The same group of researchers (Radebold et al 2001) demonstrated delayed muscle response time to quick force release in chronic low back pain patients.

Wilder et al (1996) found consistently delayed onset times in low back pain patients compared with healthy controls for a variety of sudden loading tasks. Leinonen et al (2001) found evidence of impaired feed forward control in subjects with low back pain. The short latency response to sudden unexpected loads was calculated for patients and controls. When subjected to sudden but expected upper limb loading, patients showed no shortening of the reflex latency in paraspinal muscles whereas normal subjects displayed significantly shortened latency. The authors conclude that this indicates impairment in the central processing of information.

O'Sullivan and co-workers (1997) compared muscle recruitment patterns during the abdominal drawing in exercise. In contrast to pain free controls, the low back pain group were unable to isolate the activity of internal obliques from that of rectus abdominus. The authors hypothesised that low back pain patients appear to substitute for dysfunction of the deep abdominal muscles by activating other synergists to stabilise the spine.

Hodges and Richardson (1996) analysed EMG start times of the abdominal muscles during rapid limb movement. They reported a delay in the timing of transverses abdominus relative to the onset of deltoid in patients with low back pain but not in controls. That is, in normal subjects transversus abdominus is active prior to the prime mover, but in low back pain it acts afterwards. This finding has since been replicated for lower limb movements (Hodges and Richardson 1998) and have led the authors to suggest that there might be a deficiency in preparatory spinal stabilisation in low back pain patients.

Further analysis of the data reveals a number of other differences in muscle recruitment between patients and controls (Richardson et al 1999). Firstly, these authors suggest that in the presence of low back pain the transversus abdominus activation becomes direction specific, that is, it no longer responds equally to all directions of movement but begins to be activated preferentially with particular movement directions. Secondly, there is a change in recruitment pattern. In normal subjects the transversus abdominus is seen to display tonic recruitment during the limb movement task, in patients, this recruitment becomes phasic.

Finally, there is some evidence of loss of independent control of transversus abdominus (Richardson et al 1999). Again the start times of abdominal muscles were assessed during arm movement. In this study subjects were given different preparatory cues about the direction of arm movement that they would be required to perform. The cues were either correct, neutral or incorrect with respect to the actual movement performed. Normal subjects displayed a delay in reaction time of deltoid with decreasing preparation, which was accompanied by a delay in all abdominal muscles except for transversus abdominus. In low back pain patients the transversus abdominus onset was also delayed. In other words the central nervous system of low back pain patients waited until it knew the direction of movement before activating transversus abdominus, yet in normal individuals it is preactivated regardless of direction or preparation time (Richardson et al 1999).

These latter studies reveal differences in muscle recruitment between controls and patients. Some of the differences in performance seen might best be explained by

differences in motor control. The general orthodoxy is that these neuromuscular deficits decrease the ability of an individual to adequately control the dynamic stability of the spine for unexpected external loads (Radebold at al 2000, Wilder et al 1996) and from internally generated loads (Richardson et al 1999, O'Sullivan 2000, Comerford and Mottram 2001). As the problem is related to skill and control a treatment approach aimed at ameliorating these problems needs to be informed by the skill acquisition literature (O'Sullivan 2000).

Changes in central processing, postural control and proprioception:

Insight into how the motor-control system of patients differs from that of normal subjects has been investigated by observing differences in central processing, proprioception and postural control.

Taimela et al (1993) compared the psychomotor reaction times of chronic low back pain patients and controls. Subjects had to press a button in response to a light stimulus. In this test, patients were significantly slower than controls. In a more complex reaction time task where subjects had a choice of buttons, back pain patients were again significantly slower. Luoto et al (1996, 1999) similarly investigated reaction times in chronic low back pain patients. Both studies again indicated that low back pain patients had increased reaction times compared to controls. These authors also noted that improvements in reaction time at follow up were related to improvements in disability. Luoto et al (1999) believe that these deficits represent problems with the processing of information within short-term memory.

Deficits in postural control in low back pain patients have also been observed. Nies et al (1991) found that compared to healthy controls, chronic low back pain patients demonstrated greater postural sway, orientated their centre of force more posteriorly and were less able to balance on one leg with eyes closed. Furthermore it appears that chronic low back pain patients used a different postural strategy, preferring to fulcrum about the hip and back to remain upright whereas normal subjects maintained their fulcrum for the centre of force about the ankle.

Luoto et al (1996) measured the postural sway of subjects in normal standing. They found that women, but not men, with chronic low back pain demonstrated more

postural sway. The same group of investigators (Luoto et al 1998) undertook a more detailed analysis of postural control. They looked at two footed standing, one footed standing and a postural disturbance task that involved muscle vibration. There was little difference in two footed postural stability or response to vibration. However, one-footed stability was significantly different between groups. Low back pain patients demonstrated significantly greater postural sway.

Mientjes and Frank (1999) compared the balance of chronic low back pain patients to healthy controls for seven different postural tasks of increasing complexity. Essentially there was little difference between groups for the simpler tasks, but the low back pain group demonstrated poorer balance as task complexity increased. This was particularly noticeable in tasks in which visual input was removed. Differences in sitting balance between patients and healthy controls were investigated by Radebold et al (2001). Patients with low back pain demonstrated poorer balance performance, especially at more difficult levels. In agreement with Mientjes and Frank (1999), balance was particularly affected in tasks in which visual input was removed.

The more noticeable deficits in performance of postural tasks when the eyes are closed suggest a possible proprioceptive cause. Some researchers have attempted to separately investigate spinal proprioception in low back pain patients. Parkhurst and Burnett (1994) found no difference between patients and controls for passive motion threshold, directional motion perception and repositioning accuracy.

Gill and Callaghan (1998) evaluated proprioception in standing and four point kneeling. Low back pain patients demonstrated greater error in performing a repositioning task in both positions. To further investigate the mechanisms underlying this difference, the researchers also looked at variation in repositioning error at the elbow joint in attempt to rule out differences in central processing. There was no difference in the elbow-positioning task leading the authors to conclude that the proprioceptive deficit is related to problems with peripheral spinal mechanoreceptors. This logic does not discount the possibility of a problem with central processing of spinal proprioceptive information in isolation from the central processing from other body parts.

Lam, et al (1999) found the repositioning error of low back pain patients was no different to the results of normal subjects from a previous study (Maffey-Ward et al 1996), though they did note that back pain patients overshot the reference position more frequently (79%) than controls (50%).

Newcomer and co-workers (2000a) investigated repositioning error for unrestricted lumbar spine/hip flexion and extension. They were unable to demonstrate any difference in positioning error between low back pain patients and controls for either flexion or extension. The same group (Newcomer et al 2000b) looked again at repositioning error, but this time with the hips and pelvis restrained to ensure only lumbar spine movement. They found that low back pain patients had greater repositioning error in flexion but performed better than controls in extension.

Taimela et al (1999) examined the ability of subjects to detect a change in lumbar position. Subjects were seated in a motorised trunk rotation unit and were asked to release a switch when they first perceived lumbar movement. Chronic low back pain patients had significantly poorer ability than controls to sense a change in lumbar position.

The small amount of research on proprioception in low back pain is inconsistent and offers little insight into the low back pain problem. In contrast, there is consistent evidence that low back pain patients have deficiencies in postural control and the speed of information processing. This information, combined with the observed increased latency times of trunk muscles to sudden loading and the delay in activation of transverses abdominus with limb movement, suggest low back pain patients may have a fundamental problem of controlling spinal motion.

Conclusion

Part of the reason why low back pain patients experience pain when they move may be related to the fact that they move abnormally. There seems to be agreement throughout the various parts of the mechanical review that control and consistency of movement is a problem. Quality of movement is more affected than the amount of movement both at

a whole spine and segmental level. The reasons for these observed deficits relate to the organisation of muscle recruitment and the endurance capacity of muscle rather than simply the strength of muscle. These findings suggest that a physiotherapy treatment model should include measures to improve spinal control through addressing the motor performance and endurance deficits present in trunk muscles. The nature of the deficits in muscle performance noted also support the notion that the exercise is specific to the back. Theoretical (Panjabi 1992a 1992b) and clinical (O'Sulivan 2000, Richardson et al 1999) models have been proposed that facilitate this process.

There are an enormous number of mechanical treatments advocated for low back pain. It is possible to categorise these interventions into two broad groups. Those manoeuvres that load the spine in an attempt to increase mobility and those that attempt to enhance stability and decrease loading on the spine. The available theoretical evidence would seem to support the latter approach.

Neurophysiological Basis for LBP

Pain is always a neurophysiological process as the nervous system is the final pathway for all pain problems regardless of the principal reason for nociceptive stimulation. However, recent research suggests the possibility that the nervous system can be the primary site of pain production in some cases of simple low back pain (Cervero and Laird 1996a 1996b, Codderre et al 1993, Wright 1999, Gifford 1998e, Zusman 1997, 1998, 2002, Lidbeck 2002). Lidbeck (2002) recently coined the term neurodysfunctional pain to describe this phenomenon.

An explosion of research into the neurobiology and molecular nature of neuroplasticity suggests that semi-permanent changes in the nervous system, which develop post peripheral nociceptive input, can contribute to and/or maintain pathological pain states after the original peripheral input has ceased (Codderre et al 1993, Wright 1999, 2002 Lidbeck 2002). The reason why some individuals experience low back pain when they move might relate to maladaptive changes in the nervous system. That is, the primary site of pain production has shifted from the periphery to the nervous system (Zusman 2002).

Pain mechanisms

The neurobiology of pain is highly complex and a full description of the mechanisms and workings of this system is beyond the scope of this thesis. It is however useful to have a conceptual model of the pain system to structure the proceeding discussion.

Gifford (1998c 1998e) has proposed a model of pain that helps facilitate this process, the 'mature organism model'. Fundamental to this model is placement of pain in the discipline of stress biology. In this model, the central nervous system is viewed as a central scrutinizing centre. It continually samples the outside environment, its own body and relevant past experiences. Outputs or responses are then made on what the organism finds to be to the best advantage for its body and the genes it contains (Gifford 1998c 1998a). This model recognises three types of mechanisms that are involved in the process of pain production:

1. **Input mechanisms:** This involves sampling of tissue health and transmission of this information along afferent peripheral nerve pathways.
2. **Processing mechanisms:** This involves the scrutinizing of incoming information at both a conscious and subconscious level. This process will be influenced by existing engrams of relevant past experiences to arrive at an appropriate response.
3. **Output mechanisms:** This involves the response that the organism makes. It can include motor, autonomic, neuroendocrine, neuroimmune and descending inhibitory responses

The maladaptive changes that can occur in each of these systems, leading to augmentation and central production of the pain response will be reviewed.

Input mechanisms

Up-regulation of the input mechanics is generally termed peripheral sensitisation. Under normal conditions peripheral nociceptors typically have a high stimulation threshold (Carlsson and Nachemson 2001). However, in the presence of tissue injury chemical mediators are released that greatly influence their sensitivity. Three main changes are witnessed. First, there is an increase in background activity, which is likely to cause spontaneous pain. Second, there is a lowering of the threshold of activation,

contributing to the phenomena of hyperalgesia (increased sensitivity to noxious stimulus) (Carlsson and Nachemson 2001, Zusman 1998, Stephenson 1999). Lastly, there appears to be inhibition of slow after-hyperpolarization leading to increased discharge rates, further increasing the nociceptive afferent input and contributing to hyperalgesia (Wright 1999).

Besides the action on active nociceptors there is substantial evidence that in many tissues there is a significant population of silent nociceptors that are activated with tissue injury (Wright 2002). Once activated these nociceptors exhibit marked sensitivity. Furthermore, recent evidence has demonstrated phenotype conversion of non-nociceptive afferents to nociceptive afferents. This greatly increased volume of active peripheral nociceptors further contributes to up-regulating the peripheral nociceptive input (Wright 2002, Lidbek 2002).

Occurring alongside, but distinct from these occurrences, is the process of neurogenic inflammation (Zusman 1998). Impulses arriving antidromically at the peripheral terminals of first order nociceptor afferent neurons trigger the release of chemical mediators into the surrounding tissue (Lynn 1996). These substances are powerfully pro-inflammatory, sustaining and spreading the inflammatory and sensitising responses. It is important to recognise that independent of tissue damage, the nervous system can produce the effects of inflammatory pain and sensitisation (Zusman 1998).

The purpose of these processes is to facilitate undisturbed tissue healing and repair (Zusman 1998). It is possible for these initially adaptive responses to persist beyond the acute healing phase and continue to contribute to the production of pain with minimal or absent peripheral nociceptive input (Zusman 1998, Lidbeck 2002).

Processing mechanisms

A number of dynamic changes are known to occur in the central nervous system following sustained nociceptive input, contributing to the phenomena of central sensitisation (Woolf 1983). Continued nociceptive afferent barrage on the dorsal horn of the spinal cord induces exaggerated and abnormal responses from post-synaptic neurons (Dubner and Busbaum 1994). This has four main functional repercussions

(Gifford 1998b). The sensitivity of dorsal horn cells to nociceptive input is increased, contributing to hyperalgesia. Dorsal horn second-order neurons alter their responsivity causing those neurons that previously only responded to nociceptive input to respond to input from other fibre types. This contributes to allodynia (pain due to stimulus which is normally innocuous) and secondary hyperalgesia. Dorsal horn cells increase their receptive fields leading to a spread of pain from the primary location. Finally, dorsal horn cells may become spontaneously active, causing the sensation of pain in the absence of peripheral input. In summary, these changes can cause widespread spontaneously arising or non-noxiously evoked pain in the area of injury and in distant normal tissue (Zusman 2002). There can be the perception of significant 'worsening' of the pain despite improvement or resolution of the original peripheral somatic problem.

As well as changes in the dorsal horn there is some evidence that continued nociceptive input can induce neuroplastic changes in higher centres (Lidbeck 2002). Gifford (1998b, 1998e) suggests that sustained nociceptive input may leave an imprint or central representation of a specific pain and its associated emotional content. Analogous to memories, this concept firmly places the pain source within the central nervous system. Like long-term memory, once 'imprinted' it is likely that this neural perceptual correlate of the pain would be very hard to remove (Gifford 1998b). In support of this view, Flor et al (1997) showed that the somatosensory cortical representation for the low back changed and reorganised in chronic low back pain patients.

Finally, mood and cognitions also play a role in central sensitisation (Gifford 1998b). Maladaptive thought and feelings such as stress, depression, anxiety and fear can influence the way that pain is perceived and interpreted (Main and Watson 1999). As well as leading to heightened pain perception these factors will strongly influence the output responses of the individual to pain (Gifford 1998b, Main and Watson 1999). Zusman (2002) recently reviewed a substantial body of evidence that identified dense connections between forebrain structures involved in movement, attention and emotions and brainstem pain nuclei associated with pain modulation. Furthermore, experimental evidence illustrates that attention can influence both the perception of pain as well as modify central neuroplastic changes (Zusman 2002). In summary, by shifting the balance in favour of facilitation, thoughts and emotions such as attention

and fear play a role in the magnification and maintenance of pain. Zusman (2002) concludes that somatization, catastrophyzing and hypervigilance can be thought of as 'forebrain' sensitisors and represent considerable barriers to the rehabilitation of some low back pain patients. Further consideration will be given to these issues in the psychological review.

Output Mechanisms

The outputs that occur in response to painful stimulus depend on multiple level central nervous system processing of peripheral inputs, previous experiences and current appraisal of the situation (Gifford 1998a 1998e). Possible maladaptive outputs include (Gifford 1998a):

1. **Neuroendocrine.** Chronic fluctuations in stress hormone levels may slow tissue healing and enhance pain sensitivity.
2. **Autonomic.** Increased noradrenalin can induce sympathetic dependant hyperalgesia (Wright 2002)
3. **Autoimmune.** Diminished immune responses can have adverse effects on mood, tissue healing and pain sensitivity.
4. **Motor responses.** Prolonged lack of use and avoidance of movement has significant detrimental effects on peripheral somatic tissue.
5. **Descending inhibition.** Ongoing attention and focus on pain may impede the function of the descending inhibitory system (Lidbeck 2002).

One criticism levelled at this body of evidence is that it is largely derived from animal models. The applicability of these findings to humans with low back pain is open to debate (Kumazawa 1998). Some recent evidence from human studies does however suggest greater applicability of these findings. Clauw et al (1999) used hierachical regression to investigate the contribution of pain sensitivity to self reported pain and disability. They found that pain sensitivity explained a significant amount of the variance in self-reported pain and disability in chronic low back pain patients. Flor et al (1997) provided evidence of enhanced cortical reactivity due to dorsal horn hyperexcitability in subjects with chronic low back pain. Moreover, the magnitude of

the cortical response to peripheral stimulation was positively correlated with symptom duration.

Carragee et al (2000a) performed provocative discography on a pain free group, a chronic neck pain group (a model for neurodysfunctional pain) and a group with somatization disorder (a model for psychological pain) all of whom were low back pain free. The response rate was very different between groups. Pain was reproduced in ten percent of the healthy group, forty percent of the chronic neck pain group and eighty-three percent of the somatization group. In a follow up study, Carragee et al (2000b) surveyed the same patients one year later. None of the pain free group experienced ongoing pain, yet forty percent of the chronic neck pain group and sixty-six percent of the somatization group still reported pain one year after injection. These two papers are a dramatic illustration of the mechanisms described above. Transient nociceptive stimulation of spinal tissue has no long-term consequences on a normal pain system, whereas the same stimulus acting on a sensitised system produces profoundly different outcomes.

Conclusion

This brief overview of the maladaptive events that can occur in a nervous system subject to ongoing nociceptive input raises several implications for the understanding of acute low back pain. The pain experienced by patients with acute low back pain reflects much more than the passive transmission of nociceptive impulses from peripheral receptor organs (Main and Watson 1999). It represents the complex interactions of a number of systems interpreting and processing information from multiple sources. Maladaptive alterations in the nervous system can account for on-going pain and enhanced sensitivity. The level of pain individual patients experience might not reflect the state of peripheral tissue but more reflect the sensitivity of the nervous system and the neuroplastic changes within (Gifford 1998e, Main and Watson 1999, Lidbeck 2002). To effectively manage back pain it is important that clinicians understand both the back and pain.

The primary implication of these findings for the development of a treatment model is recognition of non-peripheral causes of pain in simple low back pain. An important part

of understanding an individual's pain is to understand the mechanisms at play. Integration of the clinical analysis of mechanisms of pain into the formulation of a treatment plan has been proposed in a number of papers (Gifford and Butler 1997, Butler 1998, Jones et al 2002, Lidbeck 2002). The clinical reasoning process used to facilitate the treatment model will integrate this information to promote correct identification of underlying pain mechanics.

In situations where central mechanisms are felt to dominate, the management approach needs to reflect the underlying pain mechanics. Emphasis is on explanation of the pain experience and encouragement to engage in a programme of graded movement. Management needs to de-emphasise pain and encourage function and return to daily activities (Gifford 1998e Lidbeck 2002). Also important is endeavouring to understand the patient's beliefs about their problem and encouraging involvement of the patient in the rehabilitative process (Harding and Williams 1998). Other management strategies outlined in the psychological review will also be important. It is likely that overemphasis on specific musculoskeletal impairments will be detrimental in this patient group (Zusman 1998, Harding and Williams 1998).

In situations where it appears that maladaptive changes are not the dominant explanation for the patient's pain, these features of a treatment programme are still important as preventative measures. However it is likely that examining for musculoskeletal impairments will be a more relevant exercise and amelioration of these impairments is more likely to be associated with successful outcome (Lidbeck 2002). Also, as the pain is predominantly peripherally mediated, peripherally based methods of pain reduction may be indicated.

It is also important that the treatment model includes measures to minimise the development of centrally dominated pain. It seems that the neuroplastic changes described have as their root cause ongoing peripheral nociceptive input (Gracely et al 1992), so methods of reducing pain as soon as possible will have an important preventative role (Lidbeck 2002). Harding (1998) outlines some additional strategies that may minimise the risk of chronicity. These include the provision of non-threatening, positive advice, the encouragement of self-management, the incremental introduction of movement and encouragement to return to work as soon as possible.

Biochemical Basis for LBP

The chemical influences on the presentation and prognosis of low back pain has been investigated quite extensively with respect to nerve root pain (For review see Olmarker et al 1997). It has long been recognised that compression and traction on normal nerve roots and spinal nerves does not cause pain (Bogduk 1997). Pain is only produced with mechanical deformation in previously injured nerve roots (Bogduk 1997). While it has been traditionally thought that prolonged compression was the causative factor in sensitising the spinal nerve, the appearance of quite considerable compression in pain free subjects (see above) and the fact that previously symptomatic patients can still demonstrate compression on imaging despite being symptom free (Bogduk 1997) have challenged this idea. The current view is that chemical sensitisation of the nerve root is first required to produce nerve root pain (Olmarker et al 1997). Contemporary research suggests that the most likely sensitising chemicals are pro-inflammatory cytokines derived from the intervertebral disc (Aoki et al 2002, Olmarker and Rydevik 2001, Iwabuchi et al 2001).

The role of biochemical changes in simple low back pain is less investigated and less well understood. Nevertheless there is some evidence that suggests a chemical basis to this condition. Research has focused on two broad areas, the role of chemicals in pain production and sensitisation and the importance of chemicals in maintaining normal disc function.

The biochemical contribution to pain states has long been recognised. The majority of peripheral nociceptors are polymodal, that is they respond to chemical as well as mechanical and thermal stimulus (Wright 2002). Chemical mediators that are released with tissue damage or abnormal tissue loading can therefore directly stimulate nociceptive fibres (Wright 2002). Chemical mediators also play an important role in the up-regulation of the nociceptive system. A number of chemicals are implicated in the sensitisation of peripheral nociceptors, unmasking of silent nociceptors and, in

situations where inflammation has been present for some time, phenotype changes in some myelinated afferents such that these fibres acquire the properties of unmyelinated nociceptive fibres (Wright 2002).

The outer third of the annulus of normal lumbar discs is innervated (Gronbald and Viri 1997). Injury or abnormal loading of the disc could lead to the production of chemicals capable of stimulating these fibres. A number of studies have demonstrated the presence of inflammatory chemicals in painful human intervertebral discs (Gronbald et al 1994, Saal et al 1990, Jaffray and O'Brien 1986). Pain may be directly reproduced by chemical stimulation of nociceptors or the inflammatory chemicals may sensitise nerve endings rendering them activated to mechanical loads that would normally be pain free (Bogduk 1997). Furthermore, some researchers have noted a greater concentration of nerve fibres in painful degenerated discs, rendering them more sensitive to nociceptive input (Gronbald and Viri 1997). This process is likely to be attributable to the chemical environment of the disc (Wright 2002).

The primary functions of the intervertebral disc are to allow flexibility of the functional spinal unit and to absorb and transmit load from one vertebral body to the other (Bogduk 1997). Ultimately, the ability of the disc to perform these functions is dependant on the biochemical makeup of the disc (Poterfield and DeRosa 1998). The factors of primary importance for normal disc function are the water binding properties of proteoglycans within the nucleus and the tensile properties of the type I collagen in the annulus fibrosis (Bogduk 1997, Poterfield and DeRosa 1998). The concentration of these two critical substances is controlled by the balance between synthesis and degradation (Oegema 1993). Degradation is regulated primarily by a family of chemicals known as the matrix metalloproteinases (MMP's), while synthesis is chiefly under the control of the tissue inhibitors of metalloprotineinase (TIMP's) (Oegema 1993). Bogduk (1997) hypothesises that pain might be produced in situations where degradation predominates as normal functional loads would have to be attenuated by less tissue, giving rise to mechanical pain.

Hupli et al (1997) assayed serum samples from 41 chronic low back pain patients and sixteen age and sex matched healthy controls. They compared biochemical indicators of

type-I and type-III collagen synthesis. The low back pain patients demonstrated lower concentrations of all markers than the control group.

Kanemoto et al (1996) performed immunohistological staining of 100 discs from patients undergoing disc surgery and ten discs from fresh cadavers. They were interested in investigating the relationship between MMP-3 and TIMP-1. Their result demonstrated a disturbance in the equilibrium between MMP-3 and TIMP-1 in the patient's discs, favouring degradation.

Kang et al (1996) assayed the discs of fifteen subjects undergoing disc surgery and compared the results to eight normal discs. Besides noting an increase in inflammatory markers in the patients discs, they also noted a higher level of MMP activity. These three studies support the view that the spinal tissues of patients demonstrate an imbalance between degradation and synthesis, with degradation predominating.

In a follow up paper, Kang et al (1997) again assayed normal and painful discs, this time after the discs had been experimentally exposed to the cytokine interleukin-Ib, a chemical directly related to the production of pain. In normal discs the exposure to interleukin-Ib led to a substantial increase in MMPs. As remarked by Zusman (2000) it would be interesting to know if any normally occurring stimulus might release interleukin-Ib into the disc environment.

Okawa et al (1998) applied a small shear load to the spines of animals. One week later the discs were examined histologically and biochemically. The histological changes were minimal, suggesting minimal damage and little or no inflammatory reaction. However, unlike the control discs, the discs subject to loading demonstrated increased expression of interleukin-Ib and MMP-3. Interestingly, the appearance of these chemicals is well inside the time scale of acute low back pain used in this thesis.

Handa et al (1997) exposed human intervertebral discs to a number of different levels of hydrostatic load, (one, three or thirty atmospheres), for two hours. Three atmospheres was found to stimulate proteoglycan synthesis and demonstrate increased TIMP-1 production. Thirty atmospheres had a catabolic effect, with a reduction in proteglycan synthesis and an increase in MMP-3 production.

Similar findings have been demonstrated when cell apoptosis has been measured. Lotz and Chin (2000) loaded rat's spines *in-vivo* with a variety of external loads. Cell death was found to increase linearly with increased load. A similar study that applied a lower load and over a longer duration found that cell activity increased (Iatridis et al 1999).

Together these findings suggest that the biochemical make up of the disc is load dependant. Both high and low loads increase the activity of degradative enzymes, while moderate loads promote protein synthesis. Furthermore, at high loads at least, the production of pain-causing substances is increased. This has important implications for therapy. It is possible that controlled loading of the spine, with appropriate active or passive movements, could have a beneficial chemically mediated effect on certain clinical presentations (Zusman 2000).

Conclusion

In the parlance of this chapter, the reason some people experience back pain when they move may be chemically mediated. Pain-causing chemical substances have been found in the discs of low back pain patients and not in the discs of normal subjects. Also anti-inflammatory medications have been shown to be effective in the treatment of acute low back pain (van Tulder et al 1997b). Moreover, biochemical disturbances can detrimentally affect the ability of the intervertebral disc to function normally. The discs of patients demonstrate an increase in degradative enzymes relative to healthy discs. This could theoretically cause mechanical pain, as force attenuation would be less efficient, though this idea has not been formally tested. While this is a promising area of research it is insufficiently developed to provide definitive evidence concerning the biochemical role in simple low back pain. It is also limited by the lack of data on the biochemical influences on tissue other than disc and nerve.

One finding of particular interest in this area is the evidence of a (painful) chemical response to sudden loading. This is particularly germane in light of the findings from the mechanical review. There is further support for the ideas of seeking to improve spinal control and stability.

On a less specific level, these findings provide further support for the importance of movement in the treatment of simple low back pain and suggest that loading of the spine needs to be in an appropriate graduated manner, neither too little nor too great. Poterfield and DeRosa (1998) describe this as the 'optimal loading zone' for biological tissue. They suggest that the physiological capacity of tissue to accept load is altered by age, adaptive changes and injury. It is therefore likely that the optimal loading zone will vary considerably between individuals. This again highlights the importance of an individualised, reasoned treatment approach.

Finally, Hupli et al (1997) provide some evidence that biochemical problems are amenable to physical treatment. As was outlined above, they assayed the serum of chronic low back pain patients and healthy subjects for markers of collagen synthesis. The patients were found to have lower concentrations of markers for collagen synthesis than control subjects. The patients then participated in an 8-week active back restoration programme. At the completion of the programme the testing was repeated and the lowered rate of collagen synthesis was found to have normalised. Unfortunately, this study did not provide any data on associated improvements in pain and function or relate these findings to the change in collagen synthesis.

Psychological Basis for LBP

The review of neurophysiological models of low back pain touched on the role of psychosocial variables in influencing pain sensitivity. Psychological factors can influence back pain in many other ways and have received significant attention in recent years (Fordyce 1995, Linton 2000, Waddell 1998, Main and Watson 1999). The literature on the psychosocial influences on pain is vast, accommodating such issues as the perception of pain, the neurophysiology of pain, the identification of risk factors, prognostic factors and work related factors, the influence on motor performance and other output mechanisms, as well as the influences on health care utilisation (Main and Watson 1999). The psychological aspect of back pain is one area where there is a substantive body of knowledge on acute subjects. More significantly for this chapter, there are a number of investigations that address the question that is fundamental to the

development of a treatment model for acute low back pain. That is, what are the psychological variables present in an acute situation that contribute to the transition to chronicity. Identification of these factors would have strong implications for the development of the treatment model.

A major review of the primary literature on psychological factors in low back pain, as well as previous literature reviews on this topic, was undertaken by Linton (2000a, 2000b). Among the findings of this review, Linton concluded that there is substantial evidence that psychosocial variables are strongly linked to the transition from acute to chronic pain and disability. Furthermore there is strong evidence that psychosocial variables have more impact on low back pain than biomedical or biomechanical influences.

Pincus et al (2002) conducted a systematic review with the specific aim of determining the strength of evidence that psychosocial factors influence the transition to chronicity in low back pain. They identified eighteen independent investigations that addressed this issue. The final findings are based on the results from the six studies that were rated as high or moderate quality. However, the authors comment that the results from the remaining poor quality trials do not contradict the main findings of the review.

The most consistent finding was that distress/depression (a combination of depression, depressive symptoms and psychological distress) significantly predicted poor outcome. Moreover, the effect size for these findings was moderately strong, independent of baseline pain and function, and stronger than concurrently measured physical factors. Somatization, which is the propensity to experience and report somatic symptoms that have no pathophysiological basis, to misattribute them to disease, and seek medical help (Zusman 2002), was also consistently related to poor outcome. Finally catastrophization, an exaggerated orientation towards pain and pain experience (Pincus at al 2002), was significantly related to the development of chronicity. The effect size was particularly large in very acute subjects. Interestingly, fear avoidance beliefs were found not to significantly predict outcome despite their current high theoretical and research focus (Vlayen and Linton 2000).

Studies published since this paper do not substantially alter the conclusions of the review. Fransen et al (2002) used compensation status as a measure of chronicity. Psychosocial risk was calculated using a composite generic general health form. Locus of control was measured separately using the loci of control of behaviour scale. While 'psychosocial risk' predicted outcome the psychometric imprecision of the scale make meaningful interpretation impossible. Locus of control was found not to be predictive of a transition to chronicity.

van der Weide et al (1999) utilized return to work and the Roland and Morris Disability Questionnaire as measures of outcome. The psychological factors investigated were coping strategies and health locus of control. While there was some weak association of these factors with outcome on univariate analysis, neither contributed significantly to outcome in the final multivariate analysis. Other papers (Hurley et al 2001, Gaines et al 1999) have shown association between acute psychosocial status and outcome, but again the psychometric imprecision of the scales makes identification of the specific psychological parameter that requires attention difficult.

Conclusion

A major aim of any treatment model is to prevent chronic continuation of the problem. The available data clearly indicate that the psychological aspects of a patient's presentation impact on the development of ongoing low back pain and disability. That is, the reason why some people experience back pain when they move is due to what they think and feel. The neurophysiological review highlighted mechanisms of how this may occur, but many other influences are at play (Main and Watson 1999). It is imperative that the treatment model involves psychological screening as part of the examination process (Watson and Kendall 2000). Most importantly this process needs to identify the presence of significant distress, depression, somatization and catastrophization.

The format of psychosocial screening outlined by Watson and Kendall (2000) addresses these four issues comprehensively and the suggested questions have been integrated into the examination protocol. In addition, the treating therapist has access to the

baseline scores from the modified Zung depression scale and modified somatic perceptions questionnaire, validated scales of depressive symptoms and somatic distress respectively (see Chapter 4). Furthermore, the clinical reasoning document will guide the therapist into consideration of the outcome of the psychological screening and integration of this information into the treatment package.

Within the physiotherapy scope of practice, the treatment modality most likely to favourably affect the psychological components of the low back pain experience is education. The primary aim of the educational process should be to minimise aspects of distress, catastrophization and somatization. As part of the treatment model therapists will be encouraged to explain the nature of the patients symptoms, disavow the structural basis for low back pain, emphasis the self limiting nature and favourable outcome of the condition, encourage graded return to activity, emphasise the therapeutic benefit of movement and participation in normal work and leisure activities, decrease the focus on pain, explain the principles of sensitisation if appropriate and make clear that hurt does not equal harm (Kendall and Watson 2000, Gifford 1998a, Zusman 1997, Main and Watson 1999). These messages will further be reinforced by provision of 'The Back Book' (Roland et al 1996), a patient information booklet based on a biopsychosocial model of low back pain that addresses many of the issues outlined above.

Summary

The review of the biological basis of low back pain highlights a number of reasons why people with low back pain experience pain when they move. There are some consistent themes apparent throughout the various areas that help support the development of a treatment model. In the broadest sense there is clear agreement between the biological and philosophical axes. It has already been mentioned that the underlying philosophy of physiotherapy is closely related to the fundamental view of low back pain as activity related pain. Consistent within the biological review is the importance of movement in

the genesis of low back pain. This can be seen both from a purely mechanical perspective, as well as with reference to the effect of movement on pain mechanisms, biochemistry and psychology.

Furthermore, the core skills of education and specific active and passive movements are particularly suited to addressing a number of the problems seen in low back pain patients. There may also be a place for pain relieving modalities when pain limits the ability to participate in more active interventions and to minimise the impact peripheral nociceptive input has on central nervous system sensitivity.

The range of possibilities for activity related pain emphasise the importance of a thorough and well-reasoned examination and the formulation of an informed, individualised treatment package. It is likely that any education provided needs to be comprehensive, intensive and responsive to the individual's views and beliefs. Active and passive movements likewise need to be individualised, expertly applied and carefully monitored.

At a more specific level the biological review suggests that the educational aspects of a treatment need to explain the nature of the patients symptoms, disavow the structural basis for low back pain, emphasis the self limiting nature and favourable outcome of the condition, encourage graded return to activity, emphasise the therapeutic benefit of movement and participation in normal work and leisure activities, decrease the focus on pain, explain the principles of sensitisation if appropriate and make clear that hurt does not equal harm and encourage involvement of the patient in the rehabilitative process.

The active and passive movement aspects of the treatment model need to emphasise optimal spinal loading, improvements in control and quality of spinal motion, restoration of motor coordination, improvements in spinal stability and enhancement of the endurance capacity of trunk muscles. Interventions aimed merely at increasing spinal motion and treatments that focus on general endurance capacity might not be optimal.

Empirical Axis

Introduction

The final area of information to be considered in the development of the physiotherapy model of care is the empirical axis. This axis relates to valid clinical evidence obtained from controlled trials. Bogduk and Mercer (1995) regard this as the most relevant and important area of information, and the high profile that evidence-based practice now has within health care emphasises this importance. In a recent monologue on evidence-based practice in physiotherapy Herbert et al (2001) provide a number of interesting and convincing arguments as to the primacy of empirical data in informing clinical practice. Most importantly empirical evidence enables evaluation of an intervention regardless of inconsistencies or uncertainties in the theoretical constructs of the intervention, and well-conducted trials provide the only way of controlling for the considerable potential for bias when making observations of patient outcome.

The available empirical data on the management of acute low back pain has been appraised and synthesised in a number of ways, such as management guidelines (Koes, et al 2001) position statements (Manipulative Physiotherapy Association of Australia, 1999) systematic reviews (e.g. van Tulder, et al 1997b) and even reviews of reviews (Furlan et al 2001). In fact there are nearly as many reviews of clinical trials for acute low back pain as there are clinical trials (Physiotherapy Evidence Database (PEDro) 2001). Systematic reviews are seen as offering the strongest form of evidence in the evaluation of clinical interventions (Moore et al 1995). The methodology employed in systematic reviews ensures the most comprehensive and unbiased sampling of the literature and generally includes a rating of the methodological quality of each trial. There is however one aspect of all systematic reviews on acute low back pain that might not be ideal when trying to evaluate how to best manage acute low back pain. All reviews have categorised clinical trials by the type of intervention offered. This presents a number of problems when attempting to develop a model of care for acute low back pain.

Firstly, such an approach can oversimplify an intervention. This is most readily appreciated when considering exercise for acute low back pain. In their systematic

review of conservative treatment for low back pain van Tulder et al (1997b) included ten trials of exercise therapy for acute low back pain. These studies incorporated a variety of exercise approaches such as stretching, isometric strengthening exercises, general mobilising exercises and McKenzie exercises (McKenzie 1981). Seven of these studies were rated as negative and three as positive, and the authors conclude that there is strong evidence that exercise therapy is ineffective in the management of acute low back pain. However, the three trials that demonstrated some benefit were all McKenzie exercise trials. While some forms of exercise are certainly not effective it would seem that other forms of exercise are. To categorise all forms of exercise under a single heading oversimplifies the complexity of this intervention and leads to misrepresentation of the literature, in this case, on the efficacy of McKenzie exercises.

Related to this example is the possibility that operational classification leads to important information being missed. In most systematic reviews, individual clinical trials are generally classified as belonging to a particular treatment approach. The trial is then assigned either a positive or negative label depending on whether their results support or deny the effect of the treatment. The ability of clinical investigations to provide important insight to the understanding of the disease under investigation is lost. The example of exercise also illustrates this point. The three studies that supported the use of exercise in acute low back pain all used McKenzie type exercises. This treatment differs importantly from the other forms of exercise in that there is an attempt made to prescribe the exercise on an individual basis that is dependant on the patient's response to movement (McKenzie, 1981). None of the other exercise regimens studied allowed for individual variation in the type of exercise, or employed any diagnostic model to try and determine appropriate exercise for individual patients. It would seem that appreciation of these two points when using exercise to treat patients with acute low back pain improves outcome. By more closely scrutinising systematic reviews and the trials therein it is possible to more fully understand how to deliver effective treatment to patients with acute low back pain.

Categorisation can also lead to misrepresentation as to the specific content of treatment. Again this is most readily demonstrated by an example. Advice on staying active is one of the mainstays of conservative management of acute low back pain, widely advocated as the first line of treatment in National Low Back Pain Guidelines (Koes, et al, 2001).

This recommendation is largely based on the systematic review of bed rest and advice to stay active undertaken by Waddell et al (1997). Eight trials were included in the analysis of advice to stay active, with all but one of the trials providing evidence to support the active treatment. In almost all these trials the active intervention included much more than simply advice. In four of the trials this extended to formal exercise classes conducted by physiotherapists (Lindequist et al 1984, Fordyce et al 1986, Lindstrom et al 1992b, Philips and Grant 1991). Two of the studies without exercise classes still contained quite intensive treatment. Indahl, et al (1995) included an initial two-hour 'mini-back school' with a further one hour session two weeks later and further appointments at 3 months and 1 year. Linton et al (1993) permitted the administration of individual physiotherapy treatment for up to twelve weeks! To categorise these studies under the single heading of 'advice' misrepresents their true content and provides a false impression to clinicians on how to manage acute low back pain.

The purpose of this chapter is to help inform the content of a treatment package which can then be tested in a randomised controlled trial. With this aim in mind, and to avoid the problems mentioned above, trials were evaluated using categories that more readily facilitate this process. Studies were considered under the headings of: short-term benefit, long-term benefit and no benefit. Long-term was viewed as anything over 2 months. Within this framework studies were still looked at by intervention to help better understand the effects of that intervention, but the primary grouping was by effect. Also the epidemiological and biological reviews emphasise that managing acute low back pain requires consideration of both short-term pain relief and return to function as well as long term reduction in recurrences of pain and disability. The classification system described more easily identify what might constitute an effective treatment model and enabled consideration to be more readily given to both long and short term outcome. Separate consideration will also be given to clinical trials that have investigated timing of treatment.

The literature search was conducted in November 2001. To reflect the scope of practice of physiotherapy, studies on the three core procedural interventions of physiotherapy: manual therapy, exercise and electrotherapy were considered along with the co-ordination of care and education (APTA 2001). The use of medication, acupuncture or

injection therapy of any form was not included. While acupuncture and injection therapy are recognised within the scope of practice in the UK, they are not interventions used in the department where the model is to be tested.

Computer assisted searches were made of the Cochrane Registry of Clinical Trials, the Physiotherapy Evidence Database (PEDro), MEDLINE and CINHAL. The searches were limited to 1981 onwards. The first search used the keywords: low back pain, back pain, backache, spinal pain, lumbago and sciatica. For the second search the keywords were: physiotherapy, physical therapy, manual therapy, manipulation, chiropractic, osteopathy, exercise, electrotherapy, traction, back school and cognitive-behavioural therapy. These two searches were combined for the final outcome. PEDro uses a different search strategy and for this database the keywords chosen were pain and lumbar spine SIJ or pelvis, 'no selection' was chosen for 'Therapy' and 'Subdiscipline'. Hand searches were also made of the reference lists of all Cochrane library systematic reviews pertaining to acute low back pain. All abstracts were reviewed and those that were obviously on chronic low back pain were discarded. The remaining studies were obtained, read and reviewed using the criteria outlined by van Tulder et al (1997b), modified to include only simple acute low back pain (Waddell 1998). If papers contained a mix of acute and sub-acute patients they were still accepted if at least half the patients had experienced low back pain for less than 6 weeks. Trials in which it was not possible to determine duration of symptoms were not included. Finally, trials had to include at least one patient centred outcome and provide between group analyses.

A total of 48 trials met the inclusion criteria. To aid in judging the validity of the trials, each study was assigned a methodology score out of ten (se Appendix IX). Where possible this score was obtained from the PEDro database (www.fhs.usyd.edu.au/pedro). Those studies not on this database were scored using the PEDro system (www.fhs.usyd.edu.au/pedro) by the author. This represented only a very small number of studies (n=3) so is unlikely to affect the overall validity of the scoring system. High quality trials were those with a score of seven or above, moderate level trials were between four and six, while those with a score of three and below were deemed poor quality trials. These trials will now be considered in detail.

Studies demonstrating short-term benefit

High quality trials: Six of the trials were rated as high quality (PEDro score > 6). The earliest paper (Hadler, et al 1987) compared spinal manipulation with a spinal mobilisation manoeuvre, though the description of the mobilisation technique is very different to the way physiotherapists would mobilise the spine (Maitland, 1986) and is probably best thought of as a placebo intervention, controlling for the non-specific hands-on effects of manual treatment rather than giving a true comparison between low velocity and high velocity manual treatments. An *a priori* stratification was made of subjects into two groups. Those with back pain of less than 2 weeks and those who had suffered for 2-4 weeks. Disability was measured using the Roland and Morris Disability Questionnaire (RMDQ) (Roland and Morris 1983a) administered every three days for two weeks. In the group with the shorter duration of pain there was no difference in outcome at any time point. The second group demonstrated a significant and clinically meaningful (Stratford, et al 1996) difference in RMDQ scores at 3 days, with the results favouring the manipulation group. There was no significant difference between the groups at the other three time points. This study demonstrates that manipulation provides some short-term benefit in reducing low back pain related disability in a sub-group of acute low back pain patients. No data is available on long-term outcome.

A second high quality trial also investigated the effects of Manual therapy (Curtis, et al 2000). General Practitioners (GP) underwent training in the delivery of a limited package of manual therapy techniques. Subjects were randomised to receive standard GP care or GP care plus manipulation. Back pain related disability (RMDQ), pain (visual analogue scale) and time to functional recovery were the main outcome measures used. Disability and pain were measured at 2, 4 and 8 weeks. There was no difference between groups for pain or disability at any time point. However, the number of patients who reported functional recovery after the first treatment was significantly higher in the manual therapy group. This is the only outcome measure obtained in the same time scale as the Hadler et al (1987) study and reinforces the idea that manual therapy might hasten recovery in the short term but has little long term benefit. The finding also suggests there might be a sub-group of patients for whom manipulation is more effective.

A Swedish trial, which has been presented in a number of papers (Blomberg, et al 1992, Blomberg, et al 1993, Blomberg, et al 1994), is often included in reviews of manipulation, but contains a far more comprehensive treatment package than simply manipulation. Blomberg, et al (1992, 1993, 1994) compared usual medical and physiotherapy treatment, to treatment by a more experienced group of doctors and physiotherapists. The range of treatments provided by the more experienced clinicians included manipulation, stretches, autotraction and steroid injection of lumbrosacral soft tissues. It appears that the treatment by the less experienced physiotherapists used little manual therapy. There was no limit placed on the amount of treatment and recurrences were also treated. A large range of outcome measures was assessed at one, four and eight months, including range of motion (ROM), pain, drug use, disability and quality of life. At one month the experimental group showed significantly greater improvements in pain, work loss and disability. The long term results and implications of this study will be discussed below.

In one high quality trial looked education was the primary intervention considered. Little, et al (2001) compared an information booklet, advice to exercise, both or neither. Both the advice and the book contained similar information, designed to decrease fear about back pain, encourage normal activity and active self-management and emphasised the favourable self-limiting nature of low back pain. The study contained a mix of acute and sub-acute patients. The researchers measured outcome at 1 week and 3 weeks using a validated combined pain/function score and the Aberdeen Low Back Pain Disability Scale. Subjects in the booklet group and the advice group demonstrated significant improvement at 1 week. There was no difference between groups at 3 weeks. Interestingly the combined group was no different to the control at either time point. The authors cite other studies where this phenomenon has occurred and suggest a number of possible explanations, including information overload or confusion, as the written and oral information were not identical.

The remaining high quality study compared bed rest, exercise and ordinary activity (Malmivaara, et al, 1995). The same exercises were given to each patient and included back extension and lateral flexion movements. Short-term outcome was measured at 3 weeks using measures of pain, disability and return to work. The ordinary activity

group was significantly better on all measures. This study demonstrates the superiority of advice to stay active over bed rest or prescriptive exercises for acute low back pain.

Moderate Quality Trials: There were 13 trials of moderate quality that demonstrated short-term benefit (PEDro score 4-6). Eight of these trials featured manual therapy as a major part of the intervention. Hoehler, et al (1981) compared manipulation with soft tissue massage. This study contained a mix of acute and chronic patients, though the majority had been in pain for less than 1 month. Patients were assessed immediately after the first treatment, at the end of the course of treatment and 3 weeks after discharge. The only time significant differences were seen between the groups was immediately after the first treatment, when more patients in the manipulation group reported reduction in pain and improvement in function. This finding is consistent with the high quality trials on manipulation.

Farrell and Twomey (1982) compared manual therapy to a programme of general exercise and heat. Lumbar mobility, function, pain and time to pain free status were measured after the first treatment, after the third treatment and at 3 weeks. The only significant difference seen was in the time to pain free status. A significantly higher percentage of patients in the manipulation group reached symptom free status in less than 15 days. There were no important differences between the groups at 3 weeks.

Matthews et al (1988) report the results of two trials that compared manipulation with heat. The first study included patients with low back pain but no limitation of straight leg raise, while the second study involved patients with limited straight leg raise. Pain was measured (VAS) every second day for 8 days and again at 2 weeks. Outcome was presented as the percentage reaching pain free status. In the first study there were significantly more patients recovered at day 2 in the manipulation group but no difference at any other time point. The second study also demonstrated significantly better outcome for the manipulation group; in this case the difference was maintained for the entire 2 weeks.

MacDonald and Bell (1989) compared osteopathic manipulation to a programme of rest, education and graded return to activity. Outcomes of pain, disability and percent of

patients recovered were recorded twice weekly for 3 weeks and then weekly for 3 months. There was, however no reporting of the results from the pain scores. Analysis of the group as a whole revealed no difference in outcome. A subsequent *post hoc* sub-group analysis was performed. Subjects were divided into 3 groups depending on the duration of the present episode. No significant differences were seen in outcome in those with pain for less than 2 weeks duration, or over 4 weeks duration. However, in the 2-4 week duration group significantly more manipulation patients had recovered at the 2-week point, and at one week the manipulation patients also displayed significantly larger disability change scores. These last two studies again show that manipulation hastens short-term recovery, and suggests that its effect is different in different back pain presentations.

The next three studies differ importantly from most other trials on manipulation in that a pre-randomisation assessment was made of patients, and only those who met a defined criterion of mechanical dysfunction were recruited. Wreje, et al (1992) recruited patients who met their criteria for pelvic joint dysfunction and looked at the effect of a single session of manual therapy directed to the pelvic joints, compared to placebo massage. Number of analgesics, sick leave and numerical rating of pain for three functional tasks were the outcome measures used. Pain was measured at 3 weeks while analgesics and sick leave were monitored throughout the 3-week period. No significant difference was seen between the groups for the pain measurements, however the manipulation group took significantly less painkillers and had less sick leave.

In a small pilot study Delitto, et al (1993) screened a cohort of low back pain patients and identified a group they termed an extension-mobilisation category. These subjects were then randomised to receive either manipulation of the sacroiliac joints and mobilisation of the spine into an extension direction or flexion exercises. Disability (Oswestry Low Back Pain Questionnaire OLBPQ, Fairbank et al 1980) was measured at 3 and 5 days. The extension mobilisation group demonstrated significantly lower disability scores at both time points.

The same group then completed a similar study to further investigate these findings (Erhard, et al 1994). It is unclear from the first study if the results obtained were due to the sacroiliac joint manipulation or the extension mobilisation. In the second study

subjects were randomised to either an extension exercise group or a manipulation group. Outcome was measured as for the first study. At both time points the results significantly favoured the manipulation group. These studies provide some support for the short-term benefit of manipulation and suggest the diagnostic categories used may have some validity, though further testing to investigate the effects of this treatment approach on subjects who do not fit their extension-manipulation category is necessary.

The final manual therapy study compared spinal manipulation with lumbar spine stability exercises to lumbar spine stability exercises on their own (Morton, 1999). Spinal range of motion, pain and Disability (RMDQ) were measured at weekly intervals for the first month. Long-term outcome was also measured and will be considered in the next section. Pain scores favoured the treatment group at all time points. Disability scores were not significantly different during the first 3 weeks, but significantly favoured the manipulation group at 4 weeks. This study shows stronger effects in favour of manipulation than any of the other trials. The possible reasons will be considered in the next section.

Three moderate quality trials looked primarily at exercise. Stankovic and Johnell (1990) randomised patients to a McKenzie exercise programme or a mini back school. Pain and sick leave were the primary short-term outcomes measured. The duration of sick leave was considerably and significantly shorter in the McKenzie group. Pain levels at 3 weeks were also significantly less in the McKenzie group. There were also some significant long-term effects, which will be discussed in the next section.

Dettori, et al (1995) randomised subjects to a flexion exercise group an extension exercise group or a control group who had ice applied to their lumbar spine. Disability (RMDQ), pain (VAS) and return to work were measured at 1, 2, 4 and 8 weeks. Recurrence rate was also measured at 6 and 12 months. There was no difference in pain at any time point. At the one week assessment those patients in the exercise groups had significantly less disability and a greater return to work rate than the control subjects. There was no difference between groups at any other time point. It is interesting to contrast the findings of this study with the more pronounced effects seen in the Stankovic and Johnell (1990) paper. Both papers employed exercise regimens that

would appear to be very similar. However, they differ in that Stankovic and Johnell (1990) individualised the exercises on the basis of patient presentation.

Chok, et al (1999) investigated the effect of a progressive 6-week exercise programme designed to improve the endurance capacity of the back extensors. The content of this treatment was informed by a number of studies that demonstrate loss of extensor muscle endurance in low back pain patients (see above). The control group was given only posture and back care advice. Outcome was measured at 3 weeks and 6 weeks using a number of pain measures and the RMDQ. At 3 weeks the experimental group experienced significantly less pain and had lower disability scores. There was no difference between groups at 6 weeks. This study is the first to show meaningful improvements in a group of patients given a standardised exercise programme. This result might be reflected in the fact that experimental evidence from investigations of the function of patients with low back pain was used to inform practice.

Wilkinson (1995) compared 48 hours of bed rest with advice to remain active. At day 7 and day 28, disability was measured with the OLBPQ and the RMDQ. Time off work and lumbar range of motion were also recorded. The active group displayed significantly lower RMDQ scores at day 7. No other differences were noted. Though the effect is small this study provides further evidence that continuation of normal activities provides better outcomes than bed rest, though these findings may reflect the deleterious effects of bed rest rather than any benefit obtained from remaining active.

Low Quality trials: Two low quality studies were found (PEDro score < 4). In the first study, 160 low back pain subjects were randomised to receive manipulation, medication, electrotherapy, bed rest, education or a placebo treatment (Postacchini, et al 1988). This paper contained a sub-group of acute patients who were analysed separately. This group was further subdivided into low back pain alone or back and leg pain. Outcome was measured at 3 weeks, 2 months and 6 months using a non-validated score of global improvement of the authors' own devise. In patients with low back pain only, the change in this score was significantly greater in the manipulation group at 3 weeks. No other significant differences were found. For the back and leg pain group manipulation again demonstrated significant improvement at 3 weeks and this

superiority was also maintained at the 2 month follow up. These findings are similar to those found by Matthews, et al (1988), and again confirm the different response to manipulation of different groups of patients.

Phillips and Grant (1991) looked at the effect of one 45 minute counselling session, with follow up sessions scheduled for 3 and 6 months. The primary aim of the sessions was to emphasise return to activity and the importance of a rehabilitative approach to management. Pain, rate of recovery, and a number of measures of mood were used to gauge outcome. No differences were seen in pain or mood at any time point, however the rate of recovery was faster in the counselling group.

Conclusion

The synthesis of this literature allows for a number of conclusions to be drawn regarding the development of an effective treatment model. In general terms it would seem that it is better for patients to be active than to rest. Secondly it appears that it is important for patients to be assessed and treatment individualised to some extent. Physical treatments provided indiscriminately, at best have no effect and may worsen outcome, while tailoring treatment to a particular presentation may improve outcome.

To improve short term outcome an effective physiotherapy model of care should include education, encouragement to stay active and discouragement of bed rest, manipulation, individualised exercises that are dependant on the patients presentation as well as exercises to improve muscle function. These findings are in close agreement with the biological review presented above.

Studies demonstrating long-term benefit

High Quality studies: Only one study demonstrating long-term benefit was rated as high quality (PEDro score > 6). Rossignol, et al (2000) compared usual GP care with a programme of coordinated primary health care for compensated workers with acute

low back pain. The coordinated care approach involved a physical examination, explanation of the examination findings and an establishment of a plan of action with the patient and an explanation of conclusions and recommendations. The treating team also liased with the patients' GP and with the compensation body. Furthermore, patients were contacted weekly by telephone to discuss any problems. This contact continued until the patient returned to work. Outcome was measured at 3 and 6 months by return to work, pain (VAS) and a combination of disability scales. Return to work rates and pain were not significantly different at any point, but at the 6-month stage the coordinated care group demonstrated significant and clinically important lower disability scores. These findings support the idea of co-ordination between the parties involved in the care of patients with low back pain as well as the importance of formalised follow up procedures. However, it is unclear if the results of this study are transferable to populations not being compensated for their low back pain.

Moderate quality trials: Eight moderate quality trials established some long-term benefit (PEDro score 4-6). Indahl et al (1995) and Indahl, et al (1998) compared normal GP management with a treatment package that included an initial two-hour 'mini-back school' with an additional one hour session two weeks later and further appointments at 3 months and 1 year. The primary aims of the education sessions were to remove fear about low back pain and avoid sickness behaviours. Patients were encouraged to return to work, move as normal and to move and stretch if they felt any pain. The results were measured by length of sick leave and rate of return to work. At 200 days 60% of the control group were still off work compared to 30% in the intervention group. This significant difference in sick leave rates was also apparent at 1 year and 5 years. There was also a lower recurrence of sick leave in the intervention group over the five-year period.

In a similar study Linton et al (1993) looked at acute patients with no previous history of low back pain. Normal GP care was compared with an early activation approach. A physiotherapist assessed patients in the intervention group. Information was then provided about the examination findings, prognosis, treatment and training programmes. Patients were encouraged to remain active and to exercise. There was also provision for individual treatment and work place visits if the therapist thought it was

indicated. Follow up appointments to reinforce this message were made for 3 weeks and 12 weeks. Outcome was measured immediately post treatment, at 6 months and 12 months using a variety of pain measures, a disability questionnaire and questions about mood and general health, as well as work absenteeism. There were no differences between groups on any of the pain, disability, mood or general health questions. However in the 12-month period the active treatment group had significantly fewer days off work. There was also a significant difference in the number of patients who had become chronically incapacitated favouring the active group. It is interesting that these results were achieved without any differences in pain, disability or mood. The same interventions were also compared in a group of acute low back pain patients with a previous history of LBP. No effect was demonstrated in this group, suggesting that this approach is optimally effective in patients with first episode low back pain. Both these studies emphasis the importance of intensive patient education and maintenance of activity. It is worth noting that in similarity to Rossignol et al (2000) both these studies had a formalised follow-up as part of the package of care.

The study by Malmivaara et al (1995) mentioned above also demonstrated significant superiority of advice to stay active over bed rest and back mobilising exercises at 3 month follow up. The results of the Linton et al (1993) study suggest that these findings may be more a result of the detrimental effects of bed rest and general mobilising exercises than the beneficial effects of advice to stay active on the natural course of low back pain and disability. This study further emphasises that long-term sick leave and the development of chronicity can be minimised with the encouragement of activity.

Three studies have investigated primarily exercise-based approaches to treatment. The study of Stankovic and Johnell (1990, 1995) has been outlined above. As well as the short term improvements in pain and sick leave already noted, subjects in the McKenzie exercise group reported significantly less pain at 1 year. Patients in this group also had fewer recurrences of low back pain and less sick leave at one year and five year follow-up.

Fordyce, et al (1986) employed a very different type of exercise approach. Both groups received analgesics and a graded exercise programme. In the control group the intervention was on a pain behaviour contingent basis. Patients were told to let pain be

their guide and to continue the treatment as long as the pain continued, even the follow-up appointments to the clinic were on an 'as needed' basis. The experimental group received treatment on a time contingent basis. They were to complete a set number of exercises for a set time period and had one further clinic appointment at a set time. Outcome was measured at 6 weeks and 9 months using a complex group of measures that attempted to quantify vocational status, health care utilisation, disability and pain. No differences were seen at 6 weeks. At 9 months significant differences were seen favouring the experimental group. These patients were less disabled had less pain and used fewer health care resources. The beneficial outcomes seen in this study are usually explained from a behavioural context (Maher et al 1999), particularly with reference to decreasing fear avoidant behaviour, though it can not be discounted that the formalised follow up of the experimental group may in itself contribute to the superior outcomes seen in this group.

The final exercise study looked at specific training of the deep, stabilising muscles of the lumbar spine, particularly the lumbar multifidus (Hides, et al 1996, 2001). Subjects with first episode acute low back pain received either stability training or care from a medical practitioner. Short-term outcomes of pain and disability showed no difference between groups. Long-term outcome was evaluated by looking at 1 year and 3 year recurrence rates. At both time points the stability-training patients had lower recurrence rates than the control group. These last two trials, along with the study by Chok et al (1999) show some benefit from standardised exercises. It is important to note that each of these approaches used information from theoretical investigations on low back pain patients to inform treatment content rather than simply convention.

Finally, two manipulation trials demonstrate some long-term effects. The study by Blomberg et al (1992, 1993, 1994) has been outlined above. Long-term outcome was investigated at 3-4 months and 8 months. At 3-4 months patients in the enhanced care group had significantly less pain, disability and better outcomes on a comprehensive evaluation of quality of life. At eight months the patients receiving enhanced care had a significant reduction in sick leave. This is the first study to demonstrate long term effects of manual therapy on acute low back pain patients, and is also unique in that it significantly affected impairment, pain, disability, general health and work loss. There are several points worthy of discussion in this paper. Firstly, it shows that therapists and

doctors more expert in the care of low back pain delivered more effective treatment than those less expert. This finding has also been suggested based on the work of Koes et al (1992) on sub-acute low back pain. This idea was explicitly addressed in a recent trial on chronic low back pain (Levsen et al 2001). In this study patients were randomised to physiotherapists with different levels of training. The more experienced therapists achieved superior outcomes. Secondly, the intervention was individualised and varied in its approach. This would seem a sensible methodology given the heterogeneous nature of acute low back pain and may be why the treatment affected a number of different dimensions of outcome. Another possible explanation for the long-term benefits seen may have been the fact that the amount of treatment was not limited and recurrences were treated. Finally, the unique findings of this study may be due to the use of steroid injections, an idea that is worthy of further study.

Morton (1999) published the only other trial of manual therapy that has demonstrated long-term advantage. The short-term effects noted in pain and disability were still apparent at 2 and 3 month follow-up. Again this trial differs from other manipulation studies in that the manual treatment was combined with an exercise approach that has been shown to have long-term benefits (Hides et al 2001). The findings may represent the interaction of these two treatment approaches. Initial pain relief and return to function is obtained from the manipulation, while function is maintained through the action of the exercise programme. It is interesting that the combination of these two treatments offers far superior results to other studies where each treatment has been used in isolation.

These two studies add important information to the development of the treatment model. They suggest that manual therapy can have stronger and longer lasting benefit when delivered by experienced therapists, in a pragmatic fashion and when combined with other physical treatments.

Conclusion

This body of literature highlights a number of important features to consider in the development of a treatment model. A common thread in a number of these papers is the inclusion of follow up treatments. Six of the nine trials either scheduled review

appointments beyond the initial treatment phase or allowed for the treatment of recurrences. This approach will obviously increase the possibility of affecting outcome in the long term and may also decrease the development of the negative psychological sequelae of low back pain as patients continue to be supported.

Moreover, there is a strong cognitive component to these treatments. Six of the nine studies contained lengthy explanation of the problem, involved the patients in the treatment, emphasised the importance of activity and highlighted the positive natural history of low back pain. Related to this point, there is certainly the suggestion that better outcomes are achieved if the intervention is multifactorial, a point that the biological review strongly emphasised. Finally there is some indication that the therapeutic response depends on the outcome that is considered. This unsurprising result is sometimes overlooked in study design. To fully understand a treatment's effectiveness it is important that studies include outcome measures from a number of domains.

As far as treatment modalities are concerned there is certainly more support for educational and exercise based approaches than manual treatments. If manual treatments are to have any meaningful long-term benefit it seems that they need to be combined with other physical treatments

Studies demonstrating no benefit

In the comprehensive development of a treatment model it is also important to review the negative trials. Negative results provide two major sources of information. Firstly, if all trials of an intervention are negative then they clearly indicate the ineffectiveness of a particular intervention. Secondly, in the case where some trials show benefit and others not, comparing and contrasting the protocols used can give deeper insight into the mechanisms of action of a treatment and may indicate how to maximise the effect of a particular intervention. The negative trials will be considered under these two headings.

Interventions for which there is no support

The popularity of electrophysical agents in the management of low back pain has been highlighted in a number of studies (see for example, Foster, et al 1999). Though it would appear to be extensively used in the treatment of acute low back pain, there is no evidence supporting its use. TENS has been found to offer no benefit when used as an addition to an exercise programme (Herman, et al, 1994). A combination of analgesic currents and diathermy was no better than placebo (Postacchini, 1988) and Interferential was no better than an advice booklet (Hurley, et al 2001). One significant result was found in favour of the interferential group in this study. It is likely that this result is a methodological artefact as the interferential group had a baseline score almost twice that of the other groups and change scores were used to calculate outcome. Furthermore, three additional studies included electrotherapy in the control group (Farrell and Twomey, 1992; Matthews, et al, 1988; and Blomberg, et al 1992) In all three trials the electrotherapy group demonstrated poorer outcomes.

Bed rest is an intervention that has been extensively evaluated in the management of acute low back pain. Seven trials were found that evaluated this intervention. Five trials compared bed rest to a more active treatment (Malmivaara, et al, 1995; Rupert, et al 1985; Postacchini, et al 1988; Evans, et al 1987 and Wilkinson, 1995), and in all cases the active treatment produced superior results. The two remaining trials compared different durations of bed rest. One trial found slightly worse outcomes in the longer duration of rest (Deyo, et al 1986); the other reported no difference between 2 and 7 days of rest (Szpalski and Hayez, 1992). These results, coupled with the substantial body of theoretical knowledge illustrating the physiological harm caused by bed rest, should preclude bed rest from any treatment model for acute low back pain.

No randomised controlled trials were found that evaluated laser, ultrasound or electromagnetic treatment. There is no evidence to recommend these interventions from an empirical or biological perspective. Other systematic reviews (Beckerman et al 1993, van Tulder et al 1997b, van Tulder and Waddell 2001) have included studies on traction. All studies reviewed on traction contained substantial numbers of subjects with nerve root pain so were excluded from this review. The theoretical and convention axis related to traction also suggest that it be used primarily in nerve root pain, so will

not be recommended as part of the model for the management of simple low back pain. Information from the empirical axis provides enough evidence to exclude electrotherapy, bed rest and traction from the treatment model. Information from other parts of the review supports this decision.

Interventions for which there is conflicting evidence

The first part of this review demonstrated some benefit from education, exercise and manipulation in the management of acute low back pain. There are also trials denying the effect of these three treatments, which will now be reviewed.

Manipulation: Five trials were found that demonstrate no benefit from manipulation. In two trials the negative results may be because of the small number of patients randomised. Bronfort (1989) had 19 subjects in total, while Helliwell and Cunliffe (1987) recruited only 14 patients. The risk of committing a type II error is too high to make the results reliable.

Waterworth and Hunter (1985) devised a clinical trial that compared, GP treatment with anti-inflammatory medication, electrotherapy and general back mobilising exercises, and manipulative physiotherapy. Outcome was assessed at day 4 and day 12. Measurements were made of pain, ROM and treatment tolerance using non validated outcome measures. There was no significant difference between groups for any outcome. There are three major weaknesses in this trial. Firstly, the observers were not blind. In fact the assessments were undertaken by the same GP's who provided the first treatment arm of the trial. This introduces a considerable risk of observer bias. Secondly all outcome measure used lacked evidence of reliability and validity and used only a four-step scale (for e.g. no pain, mild pain, moderate pain and severe pain) so possibly lacked sensitivity to detect any difference in treatment outcome. Thirdly there were no functional outcome measures. These issues make interpretation of the results of this trial difficult.

Godfrey, et al (1984) present a slightly more methodologically sound trial of manipulative treatment. In their study patients were randomised to receive either a control treatment of massage and sham faradic current or spinal manipulation. The researchers used their own, non-validated outcome measures of pain, disability, ROM and spinal tenderness. No significant differences were seen at any time for any variable. For the purpose of analysis these measures were categorised into a three or a two-step scale (mild improvement, moderate improvement, and marked improvement; or, improvement, no improvement). No satisfactory explanation is offered for this decision. As mentioned above this might be too insensitive a measure to detect differences between groups. An alternative analysis in which means for the individual variables were calculated may have been more appropriate, especially given the substantial overall trend in favour of manipulation. A second possible explanation for the lack of effect involves patient selection. The inclusion criterion was pain for less than 14 days. In fact, the authors state that most patients had pain for less than a week. The results from the studies by Hadler, et al (1987) and MacDonald and Bell (1990) also failed to demonstrate the efficacy of manipulation in this very acute group, while still demonstrating benefit in groups of patients with symptoms of longer duration.

The lack of effect seen in these two trials may be due to their methodological shortcomings. It is worth noting that Godfrey, et al (1984) and Waterworth and Hunter (1985) scored the lowest methodological scores in the most recent Cochrane review of manipulation for acute low back pain (Koes, et al, 1996).

Cherkin, et al (1998) compared the efficacy of McKenzie exercises, manipulation and an education booklet in a high quality randomised controlled trial. Outcome was assessed using a 'bothersomeness' scale (0-10) and the RMDQ. Consideration was also given to recurrences and health care utilisation. Short-term outcome was measured at 4 weeks and 12 weeks, long-term outcome was measured at 1 year and 2 years. There was no difference between groups in terms of recurrence or additional health care utilisation at long-term follow up. Preliminary analysis demonstrated superiority of exercise and manipulation over the advice booklet on a number of variables. After adjustment for baseline variability and prognostic covariates, most of these differences were no longer significant, though there were still some significant differences in favour of the two active groups. After square-root transformation to adjust for non-

normal distribution, none of the differences remained significant. This statistical model has been criticised as inappropriate and invalid for this study (Giles 2001). However, the differences seen in raw outcome scores are minimal and are unlikely to be of clinical significance.

This is a very well performed study with high levels of external validity, though there are some issues that may partly explain the negative results. Firstly the power calculation was done for comparison between manipulation and exercise, and indicated about 120 subjects per group. However, the major analytical focus of the paper is the comparison between the control group (n=66) and the active groups, for which the power calculations do not hold (Giles 2001). Furthermore, 18% of patients in the control group sought additional treatment during the treatment phase, potentially biasing the results. Outcome was first recorded at 4 weeks. The previously mentioned studies on manipulation generally found significant differences at much earlier time points, with little differences apparent by 4 weeks. There is the possibility that earlier differences might have been missed. In addition this group was also very acute, with 60% having their back pain for less than 3 weeks. The speed of spontaneous recovery is likely to be higher in this group and detection of differences in outcome less likely (Hadler et al 1987, MacDonald and Bell 1990). Lastly, the intervention was limited to high velocity thrust, and only the exercise component of the McKenzie approach, with no other physical treatment permitted. The studies above highlighted that rigidly defined treatments are less likely to be of benefit.

The lack of effect of manipulation demonstrated in these trials is largely explicable for methodological reasons (2 trials scored 3/10, 2 scored 4/10 and 1 8/10). The one high quality trial that demonstrated no effect rigidly defined the delivery of treatment. This study provides some support for the view that manual treatment needs to be individualised and adaptable in order to achieve meaningful outcomes.

Exercise: Seven trials were found that fail to demonstrate any benefit of exercise in the management of acute low back pain. While there may be some methodological issues with these trials the overwhelming problem is one of clinical content. All but one of the trials uses a rigidly defined, fixed exercise regimen. Gilbert et al (1985) used isometric lumbar flexion; Underwood and Morgan (1998) used extension mobilising exercises.

Malmivaara et al (1995) employed back extension and lateral flexion mobility exercises. Faas, et al (1993), and Faas, et al (1995) had patients perform general mobility exercises and isometric flexion, and finally Buswell (1982) compared lumbar flexion with lumbar extension.

These studies provide strong evidence that general back exercises have no place in the management of acute low back pain patients, and in fact produce worse outcomes than simple advice to stay active (Malmivaara, et al 1995). They also provide strong evidence to help understand what the important aspects of an effective exercise programme might be. The exercises in these trials differ from those exercise regimens shown to be effective in three ways. Firstly, it would seem that it if back mobilising exercises are to be used then it is important to relate the exercises to the patient's presentation and problems. Better outcomes are demonstrated when exercise is prescribed in relation to the effect of movement on the patients problem (Stankovic and Johnell 1990). Secondly general whole body exercise seems to offer some benefit (Malmivaara, et al 1995). In addition, this effect is enhanced when the exercise is accompanied by intensive education to allay fears about back pain (Indahl et al 1995, Linton et al 1993) and is prescribed on a time contingent basis rather than a pain contingent basis (Fordyce et al 1986). These findings suggest that the context in which the exercises are prescribed is important. It seems that a rehabilitative context that emphasises education and patient involvement is important.

Finally, none of the negative studies employed a protocol that attempted to enhance performance of the back extensors. Two studies that involved intensive exercise of the lumbar spine extensors demonstrated some benefit (Chok et al, 1999; Hides et al 2001). It is important to again emphasise that treatment content in these two studies was strongly informed from theoretical data on patients with low back pain, something that was not apparent in any of the negative studies.

Education: The final area of investigation to be considered is patient education. Three studies on patient education detected no effect. For the negative manipulation studies the problem was mainly methodological and for the exercise papers it was treatment content that most characterized ineffective interventions. For patient education the primary issue would seem to be one of dose. Cherkin, et al (1996b) included only a

fifteen-minute education session with a practice nurse. Burton, et al (1999) simply compared two different advice booklets. Neither study demonstrated any meaningful benefit. The amount of patient contact involved in these two studies is far lower than that used in studies that demonstrated some benefit (see above). These results suggest that education needs to be intensive. The studies with the strongest effects tended to also involve a number of sessions and follow up appointments.

Leclaire, et al (1996) found no effect when a programme of daily physiotherapy was compared to daily physiotherapy and three 90 minute back school sessions. The dose of additional education sessions in this study is certainly contingent with that found in the positive trials. It may be that most of the relevant information and education was already provided by the daily physiotherapy sessions. The results of this study certainly do not preclude the inclusion of education in the management of acute low back pain.

Conclusion

The most important pieces of information obtained from review of the negative studies are:

1. The treatment model should not include bed rest, traction, electrotherapeutic modalities or indiscriminately applied back exercises.
2. The view already developed that manipulative treatment is less likely to be effective if delivered in isolation is further supported.
3. Exercise should be individualised and part of a broader rehabilitative framework.
4. Education needs to be intensive and is more likely to be successful if reinforced with follow up appointments.

Studies on the timing of intervention

The final issue to be considered in the empirical axis is the timing of the intervention. A number of pieces of information from the biological review suggested that early intervention is preferable. It is likely to reduce the amount of deconditioning and may

have implications for the neuropsychological, biochemical and psychological sequelae of low back pain.

The epidemiological literature also provides some support for this view. In a prospective cohort study, McIntosh et al (2000) found that lag time between injury and treatment was a significant predictor of time receiving disability benefit. Likewise, Ehrmann-Feldman et al (1996) found that patients referred to physiotherapy earlier after the onset of low back pain tended to return to work sooner.

The results of some non-randomised trials suggest that outcome can be improved if patients are comprehensively managed from an early stage (Miller 1995, Ryan et al 1995, van Doorn 1995, Zigenfus et al 2000), whereas other similarly designed studies have shown negligible effect (Haig, et al 1990, Cooper, et al 1996). In fact one study found that their early intervention programme actually increased time off work, while having no effect on pain and disability (Sinclair et al 1997). Moreover, the studies on manipulation provide some evidence that earlier intervention does not necessarily produce better outcomes (Hadler et al 1987, MacDonald and Bell 1990). It is obviously an area in urgent need of good quality randomised controlled studies.

Unfortunately, only one paper was found that utilised a truly randomised design to explicitly investigate the timing of intervention. Greenwood, et al (1990) studied coal miners who made a work related claim for back injury. Patients were either managed with an early intervention case management approach or usual care. The experimental group was seen within 8 days of injury while the average time for the control group was about 5 weeks. No clear description of the control treatment is given so it is unclear if time to intervention is the only independent variable. The primary outcome measures used were length of time off work, disability benefits paid and medical benefits paid.

There was no significant difference in the time off work between the two groups, whereas the average medical and disability costs were higher in the experimental group. The increased costs of medical care in the experimental group were completely accounted for by the added cost of the early intervention. The intervention proved neither to be clinically effective nor cost effective. Interestingly the authors conclude that the most probable reason for the negative results was that the early intervention

was performed too early. The very early intervention meant that the enhanced and costly package of care was utilised by all injured workers, even those with little risk of developing chronic problems. Delaying randomisation till a little longer after lodging of the original accident report might have ensured a more appropriate patient group on which to test this model.

The paucity of clinical trials evaluating timing of intervention and the inconsistent results from comparative cohort studies mean it is impossible to make definitive statements about the timing of intervention. A major focus of testing of the physiotherapy model will be trying to answer this important and under researched question.

A Physiotherapy Model of Care for Simple Low Back Pain

Information related to the management of acute low back pain was been reviewed from the biological, convention and empirical axis. The aim of this section is to outline a best practice model of care for acute low back pain.

Given the uncertainties and confusion related to the diagnosis and aetiology of simple low back pain the level of concordance that exists between the three areas reviewed is surprising. While there are some matters in which the understanding of low back pain is far from complete there is sufficient coherent evidence to develop a physiotherapy model of care.

The philosophical review suggests that the essential tenet of the model should be the optimisation of functional motor performance within a model that attempts to identify the individual reasons both for pain and pain related disability and provides clinically proven and expertly applied interventions cognisant with these aims. As was outlined above there is a reasonable case that, philosophically, physiotherapists are optimally placed to manage low back pain.

With respect to the scope of practice of physiotherapy there is support for education, exercise and manual therapy, but not electrotherapy. The heterogeneous nature of simple low back pain and the diagnostic uncertainties associated with it suggests that a pragmatically applied package of these effective interventions is likely to offer the best model of care. Close scrutiny and integration of the biological and empirical reviews affords a reasonably clear and coherent picture as to the specific elements of the model. The overview of the model and the make up of its component parts is set out below.

- A thorough clinical examination within a biopsychosocial framework (see Appendix III)
- Support of the examination process by a clinical reasoning protocol that encourages classification of patients and individualisation of the treatment programme (see Appendix VII)
- The clinical reasoning process is particularly concerned with identifying pain mechanics, relevant psychosocial features and various aspects of movement dysfunction.
- Education that aims to explain the nature of the patients symptoms, disavow the structural basis for low back pain, emphasis the self limiting nature and favourable outcome of the condition, encourage graded return to activity, emphasise the therapeutic benefit of movement and participation in normal work and leisure activities, decrease the focus on pain, explain the principles of sensitisation if appropriate and make clear that hurt does not equal harm and encourage involvement of the patient in the rehabilitative process.
- The education process should be individualised, intensive, comprehensive and continually reinforced.
- Manual treatment that is delivered within a rehabilitative framework. It should be individualised, monitored, and cognisant with the aims of decreasing pain and improving the quality of spinal movement. This may involve manual treatment to areas of the body other than the lumbar spine as a means of improving spinal loading. Care needs to be taken in situations when central pain mechanisms are felt to dominate.
- Exercise treatment that is delivered within a rehabilitative framework. It should be individualised and monitored. In situations where peripheral pain

mechanisms are felt to dominate the exercise should emphasise improvement in pain, spinal stability and control and address deficits in muscle recruitment and muscle endurance. In situations where central pain mechanisms are felt to dominate the exercise performed should be more general, based on clear quotas and have little emphasis on pain.

- Patients should be involved in the therapeutic process, including the broad content of treatment and the goals of intervention. The emphasis of goal setting will be in the attainment of functional milestones.
- Patients should be followed up after discharge. (Subjects were provided with the number of a contact person within the department to ring if they have any problems and recurrences were treated for up to a year after discharge).
- Traction or electrotherapies were not part of the treatment model.
- Dosage is a vitally important part of any treatment model. Though inadequate dosage has been cited as a possible reason for lack of treatment response (Manniche et al 1991, Jull and Moore 2002)), there is no clear evidence in the literature to guide recommendations regarding treatment dosage. This was circumvented somewhat by having the attainment of functional milestones as the exit point for treatment. There was no set minimum of treatment number or time. To minimise the risk of over treating, intervention could only be maintained for a maximum of 12 weeks.

The aim of the next part of this thesis is to test this model and the effects of timing of the intervention.

CHAPTER FOUR

TESTING THE MODEL

Introduction

The thesis so far has been about appreciating and gaining insight into the low back pain experience. This process has enabled the development of a physiotherapy model of care that best reflects the current understanding of low back pain and its management. The aims of this chapter are both to test the efficacy of this model as well as evaluate the effect of the timing of delivery of the intervention. An important first step is to revisit the clinical milieu that prompted this investigation.

In the introduction an overview was given of the development of clinical guidelines for the management of acute low back pain. The various national guidelines have been devised with reference to clinical trials of specific treatments for low back pain and unsurprisingly comparisons between the guidelines reveal similarities in the recommended interventions (Koes, et al 2001). Clear evidence is beginning to emerge that 'advice on staying active' and appropriate drug therapies are effective interventions for acute low back pain and that bed rest and general back exercises are not (Koes et al 2001).

However, there are some fundamental differences in the treatment algorithms of various countries. These differences concern primarily the use of physical therapy. The British, American and New Zealand guidelines all advocate the use of physiotherapy in the management of acute low back pain (Koes et al 2001). Alternatively the Dutch and Australian authorities surmise that the inconclusive evidence for physical therapy, the potential negative effect of treatment dependency, the cost of care and the sometimes-passive nature of the treatment preclude physical treatment from an algorithm of care

for the acute management of low back pain. The mainstay of acute management for low back pain in these guidelines is advice to stay active. This chapter intends to provide evidence to help resolve the conflicting views about the place of physiotherapy in acute low back pain management. This will be achieved by comparing a comprehensive physiotherapy model of care to advice to stay active.

The Dutch and Australian authors do not dismiss physicals therapy entirely (Koes et al 2001). Both authorities take a 'wait and see' approach to physical intervention. In these guidelines, patients with acute low back pain are assessed, reassured and advised to stay active. Physiotherapy is considered an option if symptoms have failed to resolve after six weeks (Koes, et al 2001). As was discussed in Chapter three there are good theoretical arguments to favour early intervention, but no empirical evidence to support this view. The second aim of this chapter is to explore the important issue of timing of intervention.

Finally, it is worth noting that the different guidelines suggest two distinct models of care for acute low back pain. In one system patients are assessed, advised to stay active and physical treatment is commenced early (assess/advise/treat). In the alternative model physical treatment is delayed (assess/advise/wait). Direct comparisons between these two models is lacking in the literature. Overall evaluation of the results of this study will help to ascertain which offers the best model of care for acute low back pain patients, as well as placing the physiotherapy model in the broader context of primary care management of acute low back pain.

The primary aim of the study is to comprehensively evaluate the role of physiotherapy in the management of acute low back pain. The study design will enable three essential questions to be answered these are;

1. At 6 weeks do patients treated with an active physiotherapy programme differ significantly from patients who have received advice on staying active only?
2. At long term follow up (3 months and 6 months) do patients who received physiotherapy early differ significantly from patients who were asked to wait six weeks for their treatment?

3. Do the overall results suggest any meaningful differences in outcome between an 'assess/advise/treat' model of care and an 'assess/advise/wait' model of care for acute low back pain?

Using outcomes of reported pain, functional disability, general health, social function and mood state, it is hypothesised that acute low back pain patients who receive active physiotherapy treatment will benefit more than those who are given advice on staying active only. It is also hypothesised that those patients treated early will benefit more than patients who are asked to wait six weeks for treatment. The final hypothesis is that assess/advise/treat will prove a more effective model of care than assess/advise/wait.

Advice on staying active

The literature review presented in Chapter Three provides some evidence that advice on staying active is an effective treatment strategy, leading to faster recovery and less chronic disability. Encouraging patients with simple low back pain to stay active and continue normal activities is included as first line treatment of all national guidelines reviewed (Koes et al 2001). Advice on staying active has been shown to be superior to usual medical care (Philips and Grant 1991), bed rest (Malmivarra et al 1995) and back mobilising exercises (Malmivarra, et al 1995). However whether advice on staying active is the optimal management for acute low back pain is still unclear.

Direct comparisons between advice on staying active and more active approaches to managing acute low back pain are lacking in the literature. There is some evidence from studies on sub-acute low back pain that more intensive treatments produce better outcomes. Torstensen, et al (1998) reported less pain and disability in patients treated with conventional physiotherapy or medical exercise therapy than those given advice on staying active. Maher et al (1999) present a comparison of effect size between two trials. They note a higher return to work rate in patients given advice on staying active and a graded exercise programme supervised by a physiotherapist (Lindstrom 1992a and 1992b) than those given advice on staying active only (Indahl, 1995).

Finally there would seem to be some discrepancy between the evidence base and the clinical guidelines as far as advice on staying active is concerned. As was mentioned in

Chapter Three the majority of studies included in the reviews on advice on staying active include far more than simply advice. This is not always explicit when reviewing the algorithms of care in management guidelines (see for e.g. Waddell, et al. 1999). It is important that more studies investigate advice on staying active in the way that it has been interpreted by clinical guidelines and applied in everyday practice, that is, as a one off intervention.

Physiotherapy.

Physiotherapy as a multifaceted philosophy of conservative care is rarely investigated in clinical trials (see Chapter three). Instead, investigations have focused on individual elements of physiotherapy practice, a situation that does not reflect the reality of clinical practice or the philosophical framework of physiotherapy (APTA 2001). Furthermore this is an approach particularly unsuited to the management of simple acute low back pain. The physiotherapy model of care developed in Chapter Three addresses these shortcomings and attempts to provide an individualised, optimal, biopsychosocial intervention for each patient. Specific modalities are delivered within a framework of rehabilitating and optimising physical function related to patient centred goals.

The literature reviews in Chapters Two and Three emphasised the importance of psychological and social factors in determining the clinical course of low back pain. All of the recently developed clinical guidelines recommend that assessment should address psychological, occupational and socio-economic factors (Koes et al 2001). Evidence indicates that these are more important risk factors for the development of chronicity than biomedical symptoms and signs (Linton 2000). Psychosocial assessment and behavioural management form an integrated part of assessment and treatment in this trial (Watson and Kendall, 2000).

Methods

The study is a randomised controlled trial, with the assessor independent and blind to the patient group allocation. Ethics approval was obtained from the local Research Ethics Committee. The study was designed to avoid or mitigate some of the methodological errors identified in a meta-analysis of previous research into the efficacy of physiotherapy for musculoskeletal disorders (Beckerman et al 1993). These included inadequacy of sample size, heterogeneity of study groups, clinically irrelevant outcome measures, an unstructured description of therapy and the lack of a follow up evaluation. A minimum of six months was recommended.

Power

The power of the study and required sample size was calculated using the method of Altman (1991). The primary outcome measure was the Roland and Morris Disability Questionnaire (RMDQ) (Roland and Morris 1983a). Previous work within the hospital (Frank et al, 2000) suggested that a standard deviation of approximately 6 points could be expected assuming similar characteristics between the samples of the two research studies. Therefore, 90 patients (45 in each group) would be sufficient to detect a clinically significant difference of 4 points on the RMDQ (Stratford, et al. 1996) (alpha = 0.05, beta = 0.95). To allow for a 10% attrition rate at each time point, the target for recruitment was set at 115 patients.

Subjects

Subjects were recruited from patients referred for physiotherapy by either their general practitioners or the hospital accident and emergency department (A&E) with acute low back pain. All new patients referred directly to the physiotherapy department with a complaint of low back pain were logged and considered possible candidates for the study. The intention was to include all patients with simple low back pain (Waddell 1998) of less than six weeks duration and therefore providing a more homogeneous group, as recommended by Beckerman et al (1993).

For the purposes of this study, simple low back pain was defined as pain of mechanical origin varying with physical activity and with time. The pain had to be between the shoulder blades and the folds of the buttocks, with or without leg pain and be reproducible on physical examination of lumbar musculoskeletal structures (Waddell 1998). Acute low back pain was defined as low back pain of 6 weeks or less duration (Long 1999). If there was a previous history of low back pain, the patient was still regarded as an acute low back pain patient if they reported a pain free period of at least 3 months immediately prior to the onset of the current episode (Bogduk 2000).

Local GP's and the hospital A&E department were visited by the author and provided with information about the trial and the referral procedure. A specific referral form, with a checklist of inclusion and exclusion criteria, was developed to facilitate the referral of appropriate patients (Appendix I). Recruitment began on the 31st of March 1998 and ended on the 21st of December 1999. This period was almost double the originally planned recruitment time. Furthermore The Department was about to be closed for two weeks over the Christmas period, and no subjects had been recruited in the preceding January. Data from the previous year suggested that it might require a further 5 months to recruit the 13 additional subjects. A decision was made to stop recruitment at this time as subject numbers were in excess of the numbers obtained from the power calculation.

Level 1 exclusion.

All referrals into the acute service of the physiotherapy department were screened by the author and logged onto a database. The database contained demographic information on the subjects as well as information related to inclusion and exclusion criteria. The following exclusion criteria were applied at this level:

- Back pain of more than 6 weeks duration
- Outside the age range of 20 – 55 years
- Pregnancy or within three months post partum
- Involved in litigation
- Already undergoing physiotherapy (or osteopathy, chiropractic)

- Being treated for a psychiatric problem
- Other physically disabling condition (e.g. neurological, diabetes)
- Pain in the neck, thoracic spine or hip etc.

Exclusion up to this point was only from information on the patients written referral. Of the 804 patients logged 539 were excluded. The remaining patients were contacted by telephone by the study secretary and screened using a standardised questionnaire (Appendix II). A further 30 patients were excluded as a result of the telephone interview. The remaining 235 patients were informed about the study by the secretary and invited to participate. Seven patients declined to partake in the study, 11 patients were unable to attend the department at the allotted time, and symptoms had resolved in 18 subjects. One hundred and ninety nine patients were given appointments to attend the physiotherapy department for assessment. Fifty-two patients did not attend their initial assessment and are included in the figures for level-one exclusion. Table 4.1 provides a summary of level-one exclusions.

Table 4.1. Level-one exclusions.

Reason for exclusion	**Number**	**Percentage**
LBP longer than 6 weeks	361	45
Outside age range	169	21
Pregnancy or within 3 months post partum	11	1
Litigation	4	0.5
Already receiving physical therapy	19	2
Psychiatric illness	1	0.1
Other physical condition	9	1
Co-existing musculoskeletal problems	2	0.2
Declined to participate	7	1
Unable to attend	11	1
Symptoms resolved	18	2
Did not attend	52	6
Total level one exclusions	664	83

Level 2 exclusions.

The remaining 147 patients attended the physiotherapy outpatient department and were seen initially by the research assistant. Patients were again checked for level 1-exclusion criteria and a further seven subjects were excluded. These subjects have been included in the figures for table 4.1. The research assistant gave an explanation of the study and it's aims and took subjects through the consent process. Subjects who provided signed consent entered a triage system which identified patients as having either simple acute low back pain, serious spinal pathology, nerve root pain (Waddell 1998) or pain not originating from the low back. This process was carried out by the treating physiotherapist, using a protocol developed by the research team (Appendix III). The level two exclusion criteria represent 6 broad categories:

- Fracture risk
- Inflammatory disorder
- Tumour risk
- Infection risk
- Positive neurological examination
- Neck, thoracic spine or hip etc. pain

38 patients were excluded at this level and were referred on as appropriate by the treating physiotherapist. The remaining 102 patients with simple acute low back pain entered the trial and were randomised. Table 4.2 provides a summary of level two exclusions.

Table 4.2. Level-two exclusion.

Reason for exclusion	**Numbe**	**Percentage**
Fracture risk	4	0.5
Inflammatory disorder	4	0.5
Tumour risk	2	0.2
Infection risk	0	0
Positive Neurological examination	15	2
Thoracic, cervical, hip pain	13	2
Total level two exclusions	**38**	**5**

Randomisation

Each patient entering the trial was randomised to either an 'assess/advice/treat' group or an 'assess/advise/wait' group using random number tables with odd/even number group allocation drawn by an independent person not involved in the study. Each random number was written on paper and sealed in an opaque envelope prior to the start of the trial. The research secretary held the envelopes and had no contact with the assessors. The treating physiotherapist received the sealed envelope from the secretary following the baseline assessments. Patient research numbers were written on the outside of the envelopes, which were then opened to reveal their group assignment. Information concerning the intervention and the outcome assessments were stored separately. The blind code was broken after the final patient completed his or her six-month follow up assessment. This study was a single blind design wherein the researchers administering the outcome measures were blind to subject group allocation. Respondents were not strictly blinded though the trial protocol enabled a measure of quasi-blindness (see below).

Protocol.

GP and A&E referrals were logged as described above. Those patients who fulfilled the level-one inclusion criteria were contacted by telephone by the research secretary. Subjects were told that there was currently a 6-week wait for physiotherapy treatment and that a study was underway to investigate the affect of early screening and advice on the outcome of acute low back pain. Those subjects willing to participate in the trial were booked for an assessment in the physiotherapy department. The research assistant and the treating physiotherapist undertook the initial patient assessment. A two-hour period was set aside for each initial assessment. An additional 15 minutes was set-aside in the therapists' diary for the filling in of the clinical reasoning document.

The research assistant informed patients again of the purpose of the study, obtained written consent (Appendix IV) and collected most of the baseline data set (Appendix VI). The baseline data set contained demographic, anthropometric, social and clinical

information as well as baseline values for all the dependant variables used in the study (see below).

The treating physiotherapist then undertook a full physical assessment using a locally developed biopsychosocial assessment form (Appendix III). Level-two exclusion and randomisation took place after the physical examination. All eligible patients were assessed, had the findings of their examination explained, were advised to stay active, received a programme of graded return to activity and a copy of the back book (Roland, et al. 1996). Subjects in the 'assess/advise/treat' group were told that their treatment could start immediately. 'Assess/advise/wait' patients were given an appointment to enter the treatment phase at 6 weeks from baseline, following their second outcome assessment. This produced a level of quasi-blindness in the participating subjects, as those in the assess/advise/wait group were unaware that there was the possibility of early treatment.

Treatment commenced after the initial assessment in patients in the early intervention group. Short and long term goals were formulated with the patient and an individualised treatment plan devised based on the model developed in Chapter Three. The study protocol enabled treatment to be continued for a period of up to three months, or until functional criteria indicating milestones in recovery had been met. As discussed in Chapter Three subjects were encouraged to contact their treating therapist after discharge if any further problems arose. Treatment of recurrences was permissible and was recorded in the patients' clinical notes and the study database.

Patients in the 'assess/advise/wait' group were given an appointment for 6-weeks after their initial assessment. The effectiveness of staying active and a graded return to activity was emphasised. Subjects were provided with a contact phone number and the name of their treating therapist in case of any problems. Patients in this group were given a written note of when their next appointment was and a further reminder of their follow up appointment at one month. Upon representation at 6 weeks, patients were given a second, briefer, physical examination and entered into the same physiotherapy treatment programme as those subjects in the early treatment group.

Treating therapists applied the treatment model as they saw appropriate for each patient. As outlined in Chapter Three this could include exercise, manual therapy and education. The clinical reasoning process and the setting of functional goals were an integral part of treatment selection and progression. The training sessions given to all participating staff and the piloting of the model for 1 month prior to commencement of the trial attempted to ensure compliance with the treatment model. Furthermore the author treated a large number of the study patients and was on hand for discussion of patient presentation and treatment planning. No formal assurance of compliance to the model was undertaken.

All patients were followed up by postal assessment at six weeks, three months and six months from baseline. These were sent and received by the research assistant and stored at a separate facility. Each postal assessment included an introduction/reminder letter, a freepost envelope for returning the questionnaire and the assessment booklet containing the self-report questionnaires (Appendix VI). If after 2 weeks from posting, the completed questionnaire was not returned, the assessment was re-sent and an attempt was made to contact the patient by telephone to determine any difficulties with completion. If the completed assessment was not received after a further 2 weeks an additional assessment was sent and a final telephone contact was attempted to encourage completion of the questionnaire. If the questionnaire was not received after the final telephone call, the patient was classified as a non-returnee for this follow up. Subsequent assessments were posted and the procedure started again at the next follow up period for all patients. The research assistant, who was unaware of group allocation, undertook this process.

The data presented in Chapter Three suggested that treatment by more experienced therapists' produces superior outcomes. It was decided that all treating therapists should be at least Senior I level. Furthermore, as high velocity thrust techniques were part of the treatment model all treating therapists needed to be using thrust techniques as part of their daily practice. Six physiotherapists, including the author, participated in the study with an average of 9 years in practice (range 5-15). Two therapists held masters' degrees in musculoskeletal physiotherapy, one had a postgraduate diploma in musculoskeletal physiotherapy and two therapists had completed the Manipulative Association of Chartered Physiotherapists (MACP) training programme. One therapists

had no formal musculoskeletal post graduate qualifications, but had been involved in treating back pain for five years and used high velocity manipulation as part of her regular clinical practice.

A number of teaching sessions were held prior to commencement of the trail to familiarise therapists with the study procedure, the examination protocol as well as the philosophy and content of the treatment model. All the therapists used the assessment protocol in their daily clinical practice in the month prior to commencement of the trial and attempted to integrate the treatment model into their daily practice during this time. To facilitate the application of the treatment model a clinical reasoning form was designed (Appendix VII and VIII). The forms differ slightly for the experimental and control groups. The forms were completed at the end of the initial assessment. The primary aims were to ensure goal setting and patient participation in the treatment planning, consideration of the biological, psychological and social influences on the patients problem and design a treatment programme within the study framework individualised to the patients needs. The treating therapists also used these documents for a month prior to the commencement of the trial. Debriefing sessions were held during this time to deal with any problems related to use of the model or the clinical reasoning documents.

In the early phase of the study 20 clinical reasoning documents were closely scrutinised by the research team. The current author and the physiotherapy consultant on the trial independently categorised subjects along a number of domains related to the treatment model. The documents all contained enough information to comprehensively categorise patient presentations and there was perfect agreement between the two assessors.

Baseline Variables

The full baseline data set is presented in Table 4.3. Besides baseline values for the dependent variables, a number of other important pieces of information are presented from the initial assessment. Information was obtained by questionnaire about the patient's age, marital status, occupational status and educational status.

Psychosocial risk was measured using the Acute Low Back Pain Screening Questionnaire (ALBPSQ) (Linton and Hallden 1998). This is a questionnaire developed specifically to screen for the risk of chronic problems developing. This questionnaire incorporates a number of evidence based psychosocial risk factors. The questionnaire is divided into five domains, namely, function, pain, psychological factors, fear avoidance and miscellaneous. The miscellaneous category covers a number of individual and work related issues. The document contains 21 items scored on a 0-10 scale, yielding a score range of 0-210, with the higher number indicating higher risk. The value of this tool in predicting return to work (Linton and Hallden 1998) and the number of treatment sessions (Hurley et al 2000) has been previously demonstrated, but its place in predicting other aspects of outcome have not been fully investigated. Patients were also measured and weighed by the research assistant and body mass index calculated.

From the physiotherapy examination, clinical information relating to source of referral, history of low back pain, type of onset, radiation of symptoms (Spitzer et al 1987), use of analgesia and duration of symptoms was also collected and used for analysis. Finally, patients straight leg raise was measured using an inclinometer and values for left and right leg recorded separately in degrees. An indication was also made if the test was comparable or not (Maitland 1986).

Table 4.3 Description of Study sample

Variable		n	%	Mean	sd
Subjects		102			
Sex	Male	50	49		
	Female	52	51		
Age (yrs)				34.8	8.5
Height (meters)				1.7	0.1
Weight (kgs)				75.5	14.9
Body Mass Index (BMI)				25.6	4.4
Marital Status					
	Married	44	43		
	Single	49	48		
	Divorced	9	9		
Occupational status					
	Employee		74		
	Self Employed	10	10		
	Unemployed	6	6		
	Housewife	5	5		
	Student	3	3		
	Other	3	3		
Educational Status					
	None	19	19		
	GCSE	11	11		
	A Levels	17	17		
	Diploma/Certificate/NVQ	22	21		
	Degree	21	20		
	Higher Degree	4	4		
	Other	8	8		

Working Status					
	Working	51	50		
	Off Work due to LBP	37	36		
	Off work for other reason	2	2		
	N/A	12	12		
Source of referral					
	GP	69	68		
	A&E	32	31		
	Consultant	1	1		
History					
	First episode	42	41		
	Recurrent	60	59		
Type of Onset					
	Gradual	29	28		
	Sudden	73	72		
Symptoms (QTF classification)					
	LBP only	57	56		
	Referral above knee	21	21		
	Referral below knee	24	24		
Analgesic use					
	No	45	44		
	Yes	57	56		
Duration of Pain (weeks)				2.9	1.5
Straight Leg raise-Right				67.4	17.2
Straight Leg raise-Left				68.0	16.3
ALBPSQ				91.9	28.6
VAS (usual pain intensity)				5.4	2.3
RMDQ				11.3	6.2
MZSRDS				21.9	10.8
MSPQ				7.4	5.2
STAIS				12.8	4.0
SF36 physical functioning				61.2	25.8
SF36 role physical				19.4	32.2
SF36 bodily pain				33.4	16.2
SF36 general health				84.0	16.2
SF36 vitality				52.1	12.7
SF36 social functioning				49.1	13.1
SF36 role-emotional				68.4	40.4
SF36 mental health				66.6	19.5
SF36 health transition				3.1	0.9
EuroQol Total Score				1.2	0.7
EuroQol Thermometer				66.8	20.2

Key for table 4.3

Dependant variable	Scale
ALBPSQ Acute low back pain screening questionnaire	0-210 higher scores = greater risk of chronicity
VAS visual analogue scale usual pain intensity	0-10 higher score = more pain
RMDQ Roland and Morris disability Questionnaire	0-24 higher score = more disability
MZSRDS modified Zung self rating depression scale	0-69 higher score = more depressive symptoms
MSPQ Modified somatic perception questionnaire	0-39 higher score = more distress
STAIS State anxiety index	6-24 higher score = more anxiety
SF36 physical functioning	0-100 higher score = better function
SF36 role physical	0-100 higher score = better health
SF36 bodily pain	0-100 higher score = less pain
SF36 general health	0-100 higher score = better health
SF36 vitality	0-100 higher score = more vitality
SF36 social functioning	0-100 higher score = better social function
SF36 role-emotional	0-100 higher score = better emotional health
SF36 mental health	0-100 higher score = better mental health
SF36 health transition	1-5 higher score = worse health
EuroQol Total Score	0-1.59 higher score = better health
EuroQol thermometer	0-100 higher score = better health

Dependent Variables

The literature review revealed a number of pieces of information relevant to the measurement of outcome in low back pain. Firstly, the lack of a clear disease process means that diagnosis and treatment plans are most often described in terms of impairments, functional limitations and disability (Delitto, 1994). Secondly, the interactions between these different factors are not always clear (Waddell 1998). Thirdly, the psychological dimensions of low back pain are vitally important. Finally different interventions impact differently on different factors (Chapter Three). To account for these complexities, it is important to measure a number of aspects of the low back pain experience when seeking to understand the effect of intervention on acute low back pain (Delitto, 1994).

Deyo et al (1994) in their monograph on outcome measurement in low back pain suggest that four dimensions should be measured in low back pain research. These include, symptoms, disease specific functional status, generic functional status and what they have termed role function, a factor that concerns primarily work issues. An outcome measure from the first three factors was chosen as well as measures of psychological status or mood as more recent work has emphasised the importance of psychological factors in the low back pain experience (Linton 2000). Measurement of return to work was part of the original study protocol but was dropped as only 36% of the study cohort was off work due to low back pain at Baseline. Details of the four dimensions of the acute low back pain experience that were assessed are set out below:

Pain.

Visual Analogue Scale (VAS). Usual Pain Intensity. Severity of usual or average pain experienced during the preceding week was assessed using an 11-point visual analogue scale (VAS) (Carlsson 1983). A horizontal line was marked at 1-centimetre intervals that were numbered from 0 to 10. 0 was defined as 'no pain' and 10 'worst pain'. The VAS is the most commonly used measurement of pain in acute low back pain research, so was chosen to help facilitate study comparisons and possible statistical pooling. Usual pain intensity was chosen for analysis as Bolton (1999) recently found that the

VAS Usual Pain Intensity was the best predictor of actual average pain intensity during the preceding week for patients with low back pain.

The McGill Pain Questionnaire (Melzak 1987) is also used in some acute low back pain research. It differs from the VAS primarily in that it also uses descriptors of pain and provides some measure of the affective properties of pain. A recent comparative study on the responsiveness of the McGill and the VAS (Scrimshaw and Maher 2001) found the VAS to be more responsive than the McGill and suggested the VAS is better suited for measuring pain in clinical trials. Furthermore, a number of more specific measures of mood and affect were included as separate dependant variables.

Functional disability.

Roland and Morris Disability Questionnaire (RMDQ) (Roland and Morris 1983a). Developed from items on the Sickness Impact Profile (Bergner et al 1976a,b 1981), the RMDQ is a 24-item questionnaire that measures restriction in everyday activities for patients with low back pain. There are a number of low back pain specific disability questionnaires available (Beattie and Maher 1997, Deyo et al, 1994). The RMDQ was chosen primarily as it has been tested for the minimal level of detectable change (Stratford et al, 1996), an important feature for the analysis to be carried out in Chapter Five. It has also been shown to demonstrate good levels of reliability (Kopec et al 1995) and sensitivity to change (Beurskens et al 1996).

Furthermore, a recent international task force on outcome measurements in low back pain recommended the RMDQ as the preferred disability measure (Bombardier, et al 2001). It is more sensitive to change than the Oswestry Low Back Pain Disability Questionnaire (Beurskens et al 1996) as well as being quicker to complete and more acceptable to patients (Roberts, 1991, cited in Waddell 1998).

General health status.

Deyo et al (1994) advocate the measurement of generic health status in low back pain research to help identify complications or side effects in areas of function not specifically spine related as well as providing a common measure to compare the impact of back problems on health with other disease processes. The non-specific and heterogeneous nature of simple low back pain also supports the use of generic measures

of health. Moreover, as the study compares two different, comprehensive models of care for acute low back pain and thus has implications for health care decision making it was felt that measuring overall health improvement was particularly important in this trial. Two measures of generic health status were chosen, one to measure different dimensions of general health and one to evaluate health overall.

i. Short Form 36 (SF36) (Ware et al 1992 1996). The SF 36 provides a comprehensive and psychometrically sound way to measure health by scoring standardised responses to standardised questions. The questionnaire comprises nine multi-item scales that assess dimensions of health from the patients' perspective. Scale scores are transformed to a 0-100 scale with higher scores indicating better health. The scales are: Physical Functioning (the extent to which current health limits physical activities, 10 items), Role Functioning-Physical (the extent to which current health interferes with work or other daily activities, 4 items), Bodily Pain (Pain intensity and effect of pain on work, 2 items), General Health (Personal evaluation of five health items), Vitality (feeling energetic and full of life versus tired and worn out, 4 items), Social Functioning (extent to which health problems interfere with social activities, 2 items), Role Functioning-Emotional (extent to which emotional problems interfered with work or other daily activities, 4 items), Mental Health (general mental, 5 items) and Reported Health Transition (evaluation of current health compared to one year ago, 1 item). The subscales are suitable for separate analysis (Anderson et al 1993)

ii. Euro Qol EQ5D (The EuroQol Group, 1990). The EuroQoL EQ-5D is a standardised non-disease-specific instrument for describing and measuring quality of life defined in terms of mobility, self-care, usual activity, pain or discomfort and anxiety or depression. Profiles scores are converted to a single score based on 'sets of values' derived from general population samples that represents overall 'Quality of Life' (EQtot). Scores range from 0 to 1.59 with the higher score indicating better health. Patient's self-perception of their general health is also measured on vertically orientated VAS, or 'health thermometer'. The score is derived by measuring along the 100 mm line. The higher the number the greater the self perceived health (EQht).

Both scales demonstrate good levels of reliability, sensitivity, validity and internal consistency when used on subjects with pain problems (Essink et al 1997, Bronfort and Bouter, 1999, Hurst et al 1994)

Mood.

The psychological features found to be most strongly associated with low back pain are depression, anxiety, increased bodily awareness, fear and anger (Main 1998). Most patients have a complex but variable mixture of these emotions (Main 1998). To help capture a comprehensive picture of the emotional state of subjects three of the psychological measurement tools most commonly used in low back pain research were chosen.

i. Modified Zung Self-Rating Depression Scale (MZSRDS) (Main and Waddell 1984). The MZSRDS is a measure of depressive symptoms, which have been shown to be important dimensions of the low back pain experience (Linton 2000) and a reasonable predictor of outcome (Main et al., 1992). The key feature of depression is a negative view of oneself, the world and the future (Main, 1998). The MZSRDS contains 23 questions that are scored on a 0-3 scale yielding a maximum score of 69. Scores of less than 17 are regarded as normal, between 17 and 33 is thought of as at risk, while scores over 33 indicate depression (Main 1998)

ii. Modified Somatic Perception Questionnaire (MSPQ) (Main 1983). The MSPQ is a uni-dimensional measure of heightened autonomic and somatic awareness, or 'somatic anxiety' and is usually thought of as a form of psychological distress. The MSPQ is scored on a 0 - 39 scale. Scores less than 12 are regarded as normal. The MSPQ has been shown to be an important predictor of outcome (Main et al., 1992) and particularly useful when used in conjunction with the MZSRDS (Burton et al 1995)

iii. Items from the State Anxiety Index – STATE (STAI-S) (Speilberger (1983). Six items were selected from the STAI–S (a measure of affect associated with current or state anxiety) to provide a simple and brief measure of anxiety. The items selected were; I feel calm, I feel tense, I feel upset, I feel relaxed, I feel content, I am worried. Item scores were summed (after reverse scoring 3 items) resulting in a scale ranging from 6 – 24 in which increasing scores represent increasing levels of current anxiety. Anxiety is regarded as one of the most basic emotions in illness (Main, 1998) and has been shown to have a major effect on health care utilisation and the decision to consult health care providers (Leigh and Reiser 1980 in Main 1998).

All three scales have the added advantage of being quick and easy to administer, easy to score and proven reliability and validity.

The questionnaires used for each of the dependant variables are presented in Appendix V.

Statistical Methods.

Standard descriptive statistics were employed to describe the baseline characteristics of the full data set. The baseline characteristics of the two randomised groups were compared using Fisher's exact test for categorical variables and two sample t-tests for continuous variables. Chi-square tests were used to compare the proportion of patients in the two groups who responded to the follow up assessments. Regression models investigated whether there was any interaction between group and responder status for each baseline variable. Between group differences in treatment number were analysed using independent sample t-tests.

The main hypotheses to be tested are concerned with group differences at short and long term follow up. All data analysed to answer these questions was interval data (Howell 1992), except for the independent variable of group allocation. This was scored as 0 or 1 for the analysis so can also be considered interval data. To test the main study hypotheses an *a priori* decision was made to use statistical tests that conformed to four major criteria.

1.Multivariate. The statistical test must be multivariate in that it must allow for the examination of relationships between multiple independent variables and a single quantitative dependent variable.
2.Statistical Control. It is desirable to examine the effect of group on outcome after removing the potential confounding effects of any baseline variability between the groups. With only fifty subjects per group there would be a high likelihood of non-equivalence at baseline so consideration needs to be given to the amount of change in a variable over time after taking into account any differences at baseline.
3.Interactions. The method of analysis must account for variability in baseline covariates. The complex and multifaceted nature of the low back pain experience necessitates using a statistical analysis that accounts for these interactions.

4.Effect Size. The methodology must give an easily interpretable idea of effect size. There has been a general reluctance in low back pain research to discuss and describe effect size. This is particularly true of trials on manipulation, probably because the effect sizes are so small. It is important that trials on low back pain begin to consider clinical significance, not just statistical significance (Herbert 2000). Furthermore, this concept is particularly important when attempting to answer the final study question about the relative merit of 'assess/advise/treat' or 'assess/advise/wait'.

Multiple regression is the statistical model that most closely conforms to these four criteria. The MANCOVA model fulfils the first three criteria but does not normally provide a ready measure of effect size. Therefore the main research questions were addressed by multiple regression techniques.

Covariates.

To adjust for baseline characteristics and the potential confounding effects of missing data at follow up six baseline co-variates were chosen. The decisions of what covariates to use were based upon the literature review of the main predictors of outcome (see Chapter Two) as well as an attempt to capture information from the various domains of the low back pain experience (Deyo et al 1994). The covariates relating to subjects symptoms were VAS usual pain intensity and QTF Classification. For function the RMDQ was used and for psychological status the MZSRDS and MSPQ scores were controlled for. Finally the ALBPSQ was also included. It contains questions that relate to a number of functional, psychological, work and pain related issues. The baseline value of the dependant variable being analysed was also included as a coefficient in each regression analysis.

Long-term follow up

After adjustments for baseline co-variates, regression co-efficients and their associated *p* values were calculated for each dependant variable at 6 weeks and at long-term follow up. Long-term follow up estimates were derived from all available data at 3 months and 6 months. If only one value was available from either time point this was used. If both values were available they were both integrated into the analysis. This was done primarily to account for the higher than expected drop out rate at long-term follow up. The suitability of this approach was tested by fitting a regression model for each outcome variable containing effects for group, assessment time (6 weeks, 3 months 6

months) and an interaction between assessment group and assessment time as independent variables. Data were analysed according to an intention to treat approach. The regression models used robust sandwich estimates of the standard errors of the regression co-efficients to take account of any correlation between the repeated assessments on the same subject. As repeated assessments on the same subject are likely to be correlated, each separate test reanalyses some of the variance and therefore increases the error rate. The robust sandwich estimates attempt to minimise this error.

Sensitivity analyses were performed by repeating the regression analyses using 'last value carried forward' for those patients who did not respond to follow up assessments.

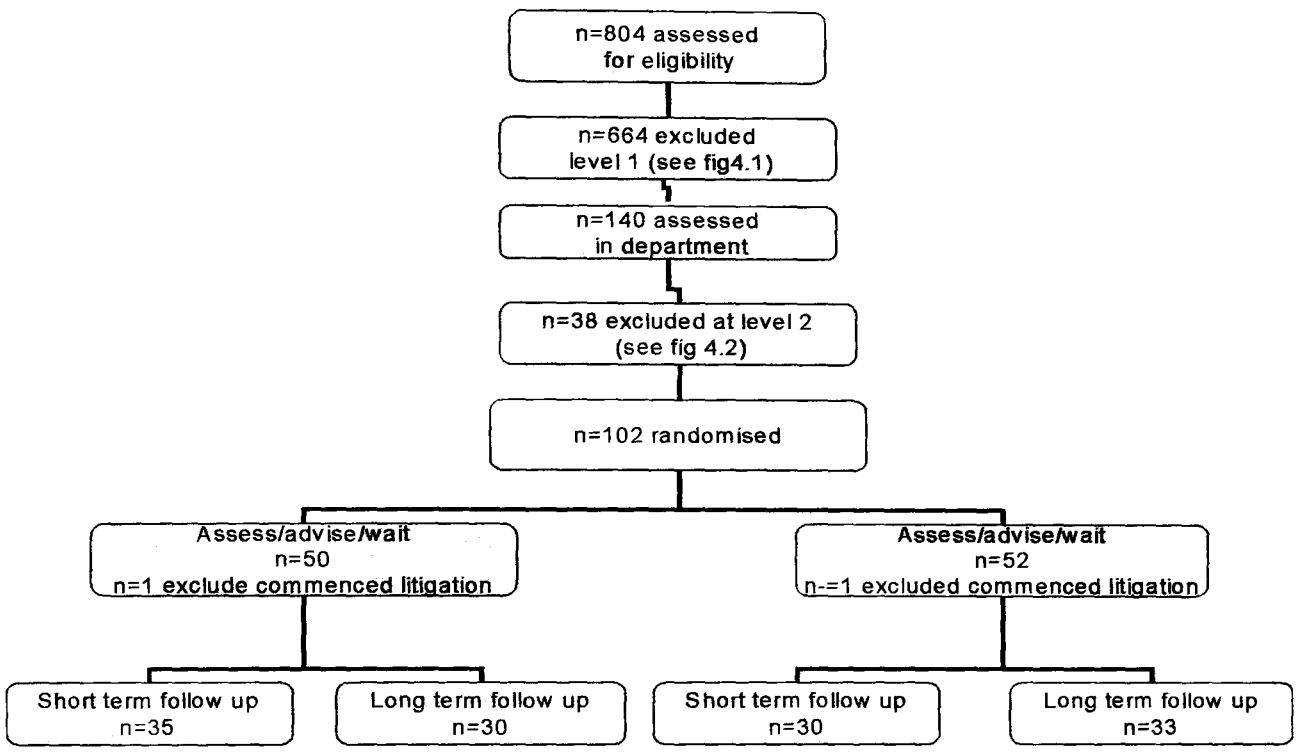
The statistical analysis was performed using Stata-Release 6 statistical software (stata statistical software 2000) and the Statistical Package for the Social Sciences version 7 (SPSS for windows 1996). Caroline Dore, senior statistician at the Medical Research Council Clinical Trials Unit, undertook the main analysis

Results.

Sample Derivation

804 patients were considered for eligibility in the study. Following the application of the exclusion criteria, 102 (13%) patients were randomised to either the 'assess/advise/treat' (n=50) or the 'assess/advise/wait' (n=52) group. 1 patient from each group was excluded after randomisation due to commencing litigation. The data presented is the analysis on the remaining 100 subjects. Figure 4.1 graphically represents the sample derivation.

Figure 4.1. Progression of participants through the trial



Baseline data.

Table 4.4 provides a summary of the baseline values for the intervention (assess/advise/treat) and control (assess/advise/wait) groups as well as the corresponding *p* values for group differences. No significant differences were found between the two groups for any of the baseline values measured, indicating success of the randomisation process.

Table 4.4 Baseline characteristics for assess/advise/wait and assess/advise/treat.

(See Table 4.3 for key)

	assess	advise group n=49	treat	assess	advise group n=51	wait	
							P value
Variable	**n**	**mean**	**sd**	**n**	**mean**	**sd**	
Sex (Female)	19			21			0.41
Age (yrs)		34.3	9		35.2	7.9	0.60
Body Mass Index (BMI)		25.9	3.7		25.3	5.0	0.52
Not working due to LBP	21			16			0.57
QTF Classification		1.81	0.85		1.53	0.83	0.11
Pain (VAS)		5.7	2.0		4.7	2.4	0.18
RMDQ		12.3	6.1		9.5	6.3	0.06
ZSRDS		19.8	9.7		23.7	11.4	0.08
MSPQ		6.6	4.8		8	5.4	0.17
STAIS		12.6	3.4		13	4	0.40
ALBP Screening Questionnaire		95.1	23.1		89.2	32.4	0.32
S36 bodily pain		33.6	17.7		33.1	15	0.88
SF36 role-physical		17.4	34.7		21.1	30.2	0.56
SF36 physical functioning		58.8	25.4		65.8	25.5	0.88
SF36 general health		86.9	12.1		81.5	18.8	0.11
SF36 vitality		49.1	13.7		54.6	11.4	0.74
SF36 social functioning		48.8	12.4		49.3	13.8	0.88
SF36 role-emotional		71.3	40.2		66	40.8	0.53
SF36 mental health		72.5	17.6		61.6	19.8	0.41
SF36 Reported health transition		3.1	0.9		3.1	0.8	0.94
Euroqol Total Score		1.4	0.7		1.1	0.7	0.33
Euroqol Health Thermometer		64.7	22.1		68.5	18.7	0.18

Number of Treatments.

The initial physiotherapy assessment for both groups included education and advice so is counted as a treatment session. Patients in the 'assess/advise/treat' group had a mean of 4.7 (sd 3.0 median 4) treatment sessions with the physiotherapist. Patients in the 'assess/advise/wait' group received a mean of 2.9 (sd 2.7 median 2) treatments. This difference is statistically significantly ($p<0.01$ *t*-test). However, the 'assess/advise/wait' group is comprised of two distinct sets of subjects. Firstly, patients who returned for treatment at 6-weeks (n=31) and received a mean of four treatments (sd 2.8 median 4.0). Secondly, those patients who did not return for treatment at 6 weeks (n=20) and had only one physiotherapy contact. There is no significant difference in treatment

number between those subjects in the treatment group as a whole and those in the control group who returned for treatment (p=0.832 *t*-test).

Outcome at six-weeks.

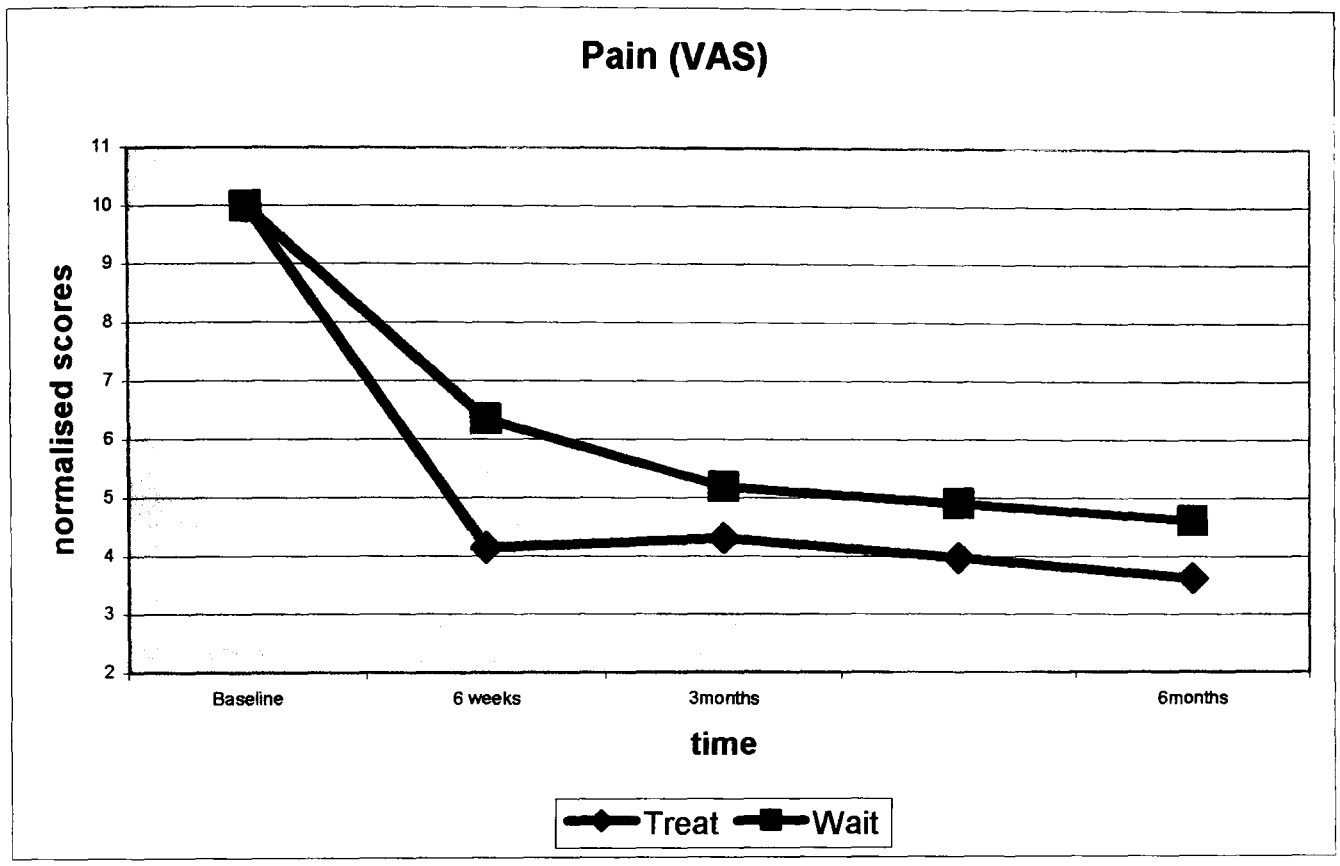
Table 4.5 summarises the comparisons between the intervention and control groups at six-weeks.

Table 4.5 Group differences at 6-weeks. (See table 4.3 for key)

	mean (sd)	mean (sd)	P value
Dependent Variable	***assess/advise/treat***	***assess/advise/wait***	
VAS (usual Pain)	2.4 (2.0)	3.3 (2.5)	0.22
STAIS	10.8 (4.2)	13.6 (4.5)	**0.01**
RMDQ	4.5 (4.5)	6.3 (5.9)	**0.02**
MZSRDS	14.4 (9.4)	22.8 (12.2)	**0.01**
MSPQ	3.9 (5.0)	4.9 (4.3)	0.67
EuroQol Total Score	0.8 (0.1)	0.7 (0.3)	**0.05**
EuroQol Health Them	80 (15)	69 (18)	**0.006**
SF36 Physical Function	78 (19)	75 (19)	0.96
SF36 Physical Role	61 (43)	50 (43)	0.13
SF36 Bodily Pain	65 (20)	54 (22)	0.06
SF36 General Health	89 (13)	77 (19)	0.12
SF36 Vitality	68 (19)	46 (21)	**<0.001**
SF36 Social Functioning	79 (21)	63 (25)	**0.004**
SF36 Emotional Role	82 (35)	63 (43)	0.11
SF36 Mental Health	80 (16)	58 (24)	**0.002**
SF36 Health Transition	2.9 (0.7)	3.1 (0.9)	0.15

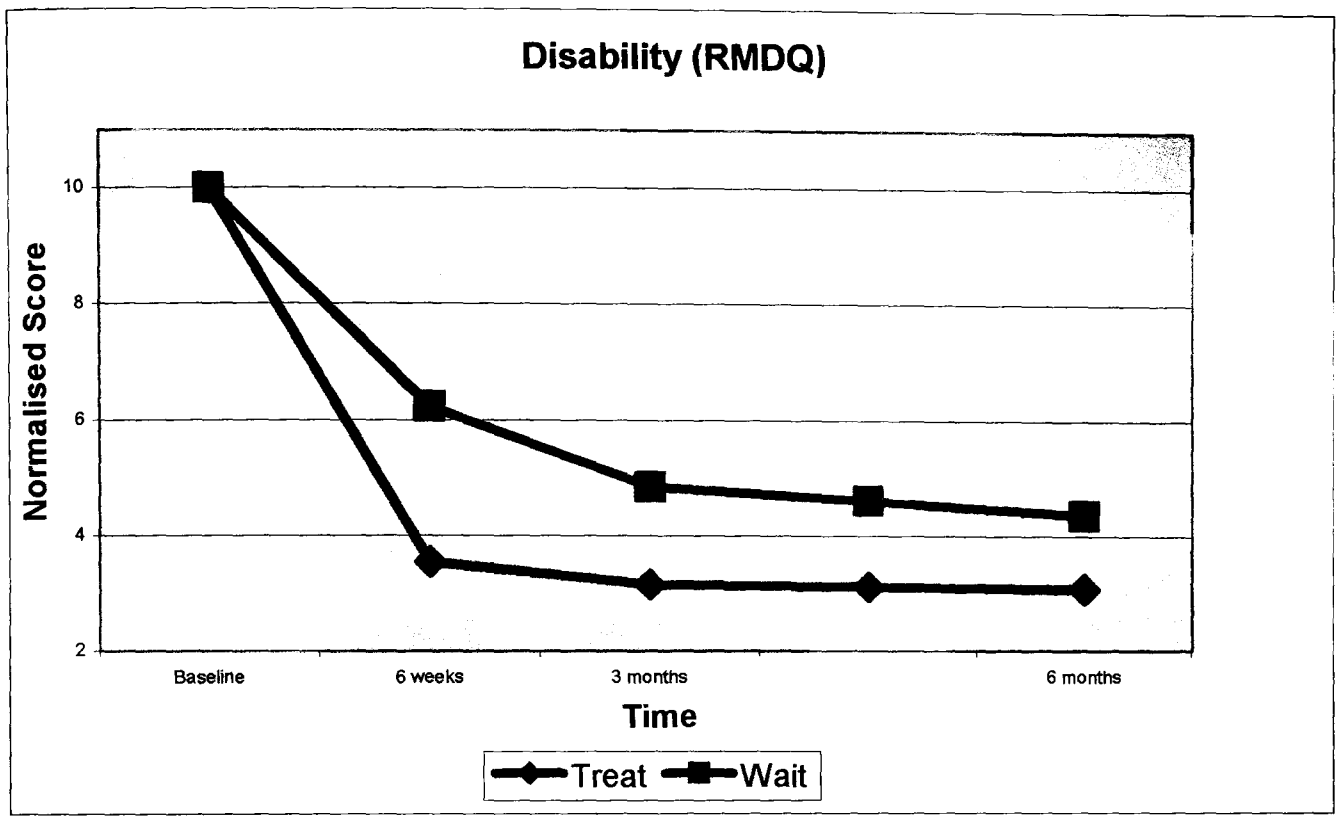
Pain. Both groups demonstrated significant reductions in pain during the initial 6 weeks. The early intervention group improved their pain scores from an average of 5.8 at baseline to 2.4 at the 6-week evaluation ($p<0.001$ *t*-test), a reduction of 59%. Pain scores for subjects in the advice group changed from 5.2 to 3.3 ($p=0.004$ *t*-test), a reduction of 37%. After controlling for baseline values the regression analysis demonstrated that group allocation did not significantly predict pain scores at 6 weeks ($p=0.22$ *t*-test). Pain improved equally in both groups during the first 6 weeks. This comparison is represented graphically in figure 4.2.

Figure 4.2. The mean change in pain (VAS) from baseline to 6-month follow-up for the two treatment groups. (The scores have been normalised to account for baseline variability)



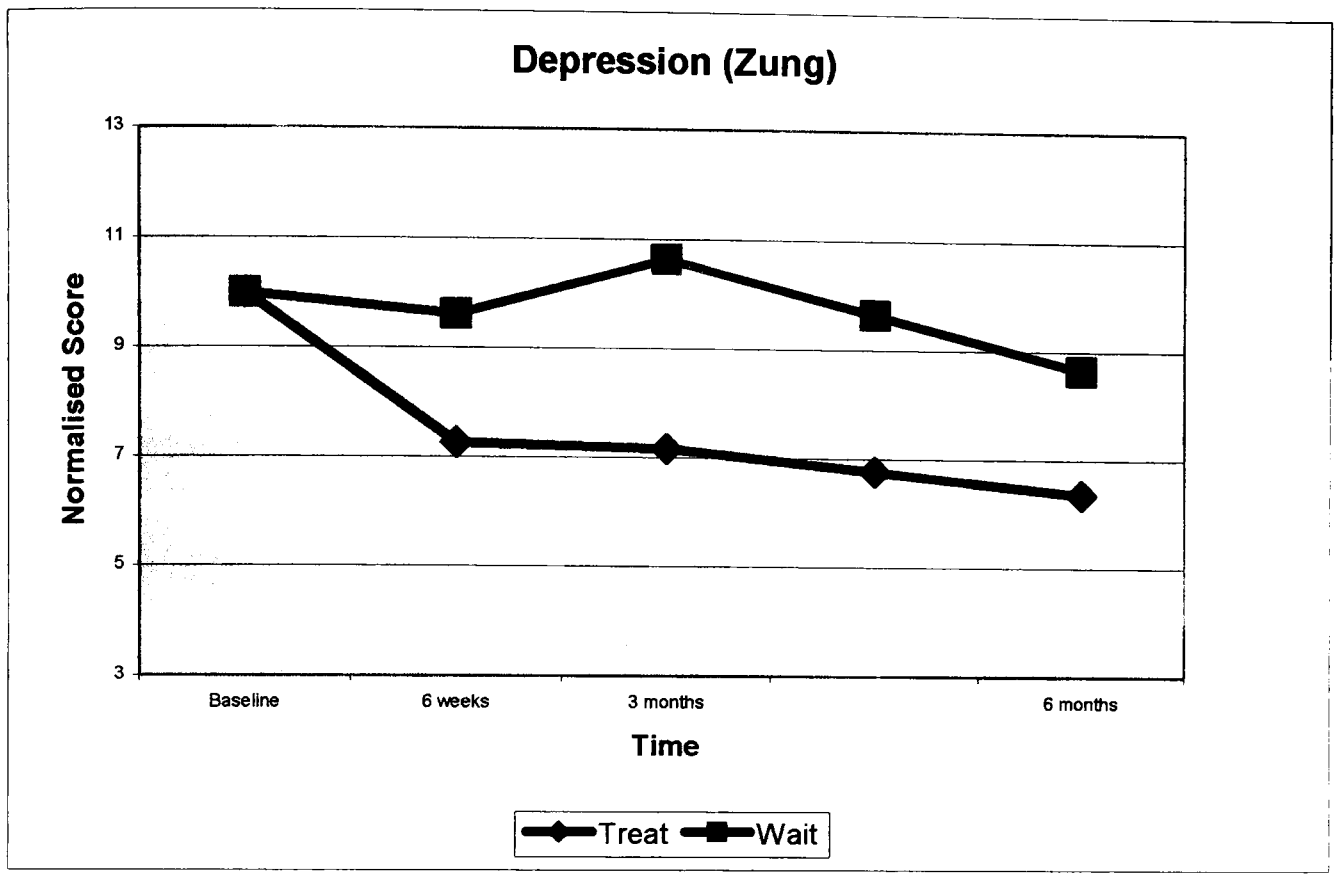
Disability. The treatment group demonstrated a statistically significant reduction in RMDQ score at 6 weeks ($p<0.001$ *t*-test). The mean score changed from 12.3 at baseline to 4.5 at 6-week follow up, a 7.8-point (62%) improvement. This represents almost double the minimal detectable change for the RMDQ (Stratford at al 1996). This change in disability scores is both statistically and clinically relevant. The difference in RMDQ scores for the control group was also statistically significant ($p=0.001$ *t*-test). However, the mean change was only 3.2 (34%), less than what would be considered clinically meaningful (Stratford et al 1996). The regression analysis demonstrated that group allocation significantly predicted RMDQ scores at 6 week follow up ($p=0.023$ *t*-test). Those subjects in the 'assess/advise/treat' group had significantly lower disability scores at 6 weeks. This comparison is represented graphically in figure 4.3

Figure 4.3. The mean change in disability (RMDQ) from baseline to 6-month follow-up for the two treatment groups. (The scores have been normalised to account for baseline variability).



Mood. There was a significant improvement in MZSRDS over the initial 6-week period in the treatment group (p=0.012 t-test) but not in the control group (p=0.959 t-test). While the mean MZSRDS in the 'assess/advise/treat' group improved by 4.1 (22%) the mean scores for the 'assess/advise/wait' group remained unchanged. The regression analysis demonstrated that group allocation significantly predicted MZSRDS scores at 6 week follow up (p=0.013 t-test). Those subjects in the 'assess/advise/treat' group had significantly less depressive symptoms at 6 weeks. This comparison is represented graphically in figure 4.4

Figure 4.4. The mean change in depressive symptoms (MZSRDS) from baseline to 6-month follow-up for the two treatment groups. (The scores have been normalised to account for baseline variability).



The mean STAIS scores for the 'assess/advise/treat' group improved from 12.4 to 10.8, representing a 13% decrease. The scores for the 'assess/advise/wait' group worsened slightly from 13.4 to 13.6. In neither group were these differences statistically significant (assess/advise/treat p=0.112, assess/advise/wait p=.534 *t*-test). The regression analysis demonstrated that group allocation significantly predicted STAIS scores at 6 week follow up (p=0.024 *t*-test). Those subjects in the 'assess/advise/treat' group had significantly lower anxiety scores at 6 weeks.

Both groups demonstrated improvements in somatic distress over the initial 6-week period. The mean MSPQ for the treatment group improved from a baseline value of 6.8 to 3.9 at 6 weeks (43%). This change was statistically significant (p<0.001 *t*-test). The control group changed from 7.5 at baseline to 4.9 at 6 weeks (35%); this change was also statistically significant (p=0.005 *t*-test). After controlling for baseline values the regression analysis demonstrated that group allocation did not significantly predict

somatic distress scores at 6 weeks (p=0.22 t-test). Somatic distress improved equally in both groups during the first 6 weeks.

General Health. For the intervention group the EuroQol composite score improved from 0.60 at baseline to 0.82 at 6 weeks, a change of 0.22 (37%). This change was statistically significant (p<0.001 t-test). The control group demonstrated a non-significant change in scores at short-term follow up (p=0.613 t-test). From a baseline value of 0.60 the mean score had improved to 0.69 by 6 weeks (15%). The regression analysis demonstrated that group allocation significantly predicted EuroQol composite scores at 6 week follow up (p=0.05 t-test). Those subjects in the 'assess/advise/treat' group had significantly better self-rated general health at 6 weeks.

The EuroQol health thermometer scores exhibited similar results. The intervention group demonstrated a 24% improvement in general health from 64.3 to 79.9. This difference was significant (p=0.001 t-test). The mean score in the control group remained stable over the initial 6 weeks (p=.464 t-test). The regression analysis demonstrated that group allocation significantly predicted EuroQol health thermometer scores at 6 week follow up (p=0.006 t-test). Those subjects in the 'assess/advise/treat' group had significantly better self-rated general health at 6 weeks.

The results from the SF36 represent nine different dimensions of general health status. The results from the regression analysis demonstrate that group allocation significantly predicted scores for vitality (p<0.001 t-test) social functioning (p=0.004 t-test) and mental health (p=0.002 t-test). Subjects in the 'assess/advise/treat' group had significantly better self rated vitality, social functioning and mental health. No significant differences were seen between groups for physical function (p=0.956 t-test), physical role (p=0.132 t-test), bodily pain (p=0.057 t-test) general health (p= 0.118 t-test) or emotional role (p=0.113 t-test)

Effect size at short term follow up.

The size of the treatment effect is represented by the change in the R square as well as by calculating the regression co-efficient. Table 4.5 summarises these results for the

variables that displayed statistical significance at short term follow up. Group allocation explained 20% of the variance in RMDQ, 23% of the variance in MZSRDQ, 21% for STAIS, 15% for EQtot, 23% for EQht and 25%, 26% and 14% for SF36 vitality, social function and mental health respectively.

Table 4.6. Size of treatment effect at short-term follow up

Dependent Variable	Change in the R square	Regression Co-efficient (SE) Treat-Advise
RMDQ	0.20	-2.9(1.3)
MZSRDQ	0.23	-7.0(2.7)
STAIS	0.21	-2.7(1.0)
EQ total score	0.15	0.13(0.06)
EQ health thermometer	0.23	12(4)
SF36 Vitality	0.25	18(5)
SF36 Social Function	0.26	16(5)
SF36 Mental Health	0.27	14(4)

The results indicate that involvement in an active physiotherapy programme leads to superior outcomes when compared to advice on staying active. Subjects involved in the physiotherapy programme had less low back pain related disability, fewer depressive symptoms, less anxiety and better general health. What is more, the effect sizes are moderate and the differences clinically meaningful.

Outcome at long-term follow up.

The analysis between group and time through out the follow up period indicated a significantly interaction for the SF 36 Vitality scale (p=0.05). Inspection of the data indicated that initial improvements in vitality for the 'assess/advise/treat' group and deterioration for the 'assess/advise/wait' group began to resolve during the follow up periods and by six months both groups exhibited similar mean scores. No other dependant variable demonstrated any significant interactions between group and time enabling the data from three and six months to be combined to form a single score. Table 4.6 summarises the comparisons between intervention and control at long-term follow up.

Table 4.7. Comparison between groups at long-term follow up. (see table 4.3 for key)

	Long term	mean (sd)	P value
Dependent Variable	***assess/advise/treat***	***assess/advise/wait***	
VAS (usual Pain)	2.4 (2.5)	2.6 (2.5)	0.61
STAIS	10 (3.8)	13.4 (5.5)	**0.01**
RMDQ	4.2 (5.2)	4.7(5.6)	0.94
MZSRDS	13.4 (10.4)	22.9 (14.9)	**0.001**
MSPQ	3.6 (3.5)	6.7 (8.4)	**0.004**
EuroQol Total Score	0.84 (0.22)	0.74 (0.26)	0.13
EuroQol Health Them	81 (16)	68 (19)	**0.009**
SF36 Physical Function	84 (18)	82 (19)	0.72
SF36 Physical Role	74 (43)	69 (40)	0.65
SF36 Bodily Pain	72 (18)	66 (20)	0.32
SF36 General Health	90(14)	78 (22)	0.11
SF36 Vitality	67(20)	60 (22)	0.09
SF36 Social Functioning	86 (20)	72 (28)	0.07
SF36 Emotional Role	87 (27)	71 (45)	**0.03**
SF36 Mental Health	81 (18)	64 (25)	**0.04**
SF36 Health Transition	2.5 (0.9)	2.8 (1.0)	**0.05**

Pain. Both groups demonstrated significant reductions in pain at long term follow up. The early intervention group improved their pain scores from an average of 5.7 at baseline to 2.4 ($p<0.001$ *t*-test), a reduction of 58%. Pain scores for subjects in the advice group changed from 5.2 to 2.6 ($p<0.001$ *t*-test), a reduction of 50%. After controlling for baseline values the regression analysis demonstrated that group allocation did not significantly predict long-term pain scores ($p=0.29$ *t*-test). Pain improved equally in both groups during the study period. (see figure 4.1).

Disability. The treatment group demonstrated a statistically significant reduction in RMDQ score at long term follow up ($p<0.001$ *t*-test). The mean score changed from 12.3 at baseline to 4.2 at long term follow up, an 8.1-point (66%) improvement. The difference in RMDQ scores for the control group was also statistically significant ($p<0.001$ *t*-test), with a mean change of 4.3 (45%). The regression analysis demonstrated that group allocation did not significantly predicted RMDQ scores at long term follow up ($p=0.32$ *t*-test). While RMDQ scores were significantly different at 6 weeks this difference had disappeared by 3 months. (see figure 4.3).

Mood. There was a significant long-term improvement in MZSRDS in the treatment group ($p=0.002$ *t*-test) but not in the control group ($p=0.190$ *t*-test). While the mean MZSRDS in the 'assess/advise/treat' group improved by 4.7 (25%) the mean scores for

the 'assess/advise/wait' group worsened slightly. The regression analysis demonstrated that group allocation significantly predicted MZSRDS scores at long term follow up (p=0.001 t-test). Those subjects in the 'assess/advise/treat' group had significantly less depressive symptoms. (see figure 4.4).

The mean STAIS scores for the 'assess/advise/treat' group improved from 12.4 to 10.3, representing a 17% decrease. The scores for the 'assess/advise/wait' group worsened slightly from 13.4 to 13.5. In neither group were these differences statistically significant ('assess/advise/treat' p=0.096, 'assess/advise/wait' p=0.252 t-test). The regression analysis demonstrated that group allocation significantly predicted STAIS scores at long term follow up (p=0.002 t-test). Those subjects in the 'assess/advise/treat' group had significantly lower anxiety scores.

The long-term results for somatic distress were different to those seen at 6 weeks. The mean MSPQ for the treatment group improved by 46%, from a baseline value of 6.8 to 3.7 at long term follow up. This change was statistically significant (p<0.001 t-test). The control group changed from 7.5 at baseline to 6.1 at long term follow up (19%). This change was also statistically significant (p=0.005 t-test). After controlling for baseline values the regression analysis demonstrated that group allocation did significantly predict long-term somatic distress scores (p=0.008 t-test). Somatic distress was significantly less in the 'assess/advise/treat' group.

General Health. For the intervention group the EuroQol composite score improved from 0.60 at baseline to 0.83 at long term, a change of 0.23 (38%). This change was statistically significant (p<0.001 t-test). The control group also demonstrated a significant change in scores at long-term follow up (p=0.016 t-test). From a baseline value of 0.60 the mean score had improved to 0.72 by the end of the study period (20%). The regression analysis demonstrated that group allocation did not significantly predict long-term EuroQol composite scores (p=0.10 t-test). The long-term improvements in general health scores were the same for both groups.

The EuroQol health thermometer scores behaved slightly differently. The intervention group demonstrated a 25% improvement in general health from 64.3 to 80.6. This difference was significant (p=0.004 t-test). The mean score in the control group

changed very little over the study period, from a baseline value of 69.2, to 67.9 at long term follow up. This difference was non significant (p=0.644 t-test). The regression analysis demonstrated that group allocation significantly predicted long-term EuroQol health thermometer scores (p=0.009 t-test). Those subjects in the 'assess/advise/treat' group had significantly better self-rated general health at long term follow up.

The results from the SF36 represent nine different dimensions of general health status. The results from the regression analysis demonstrate that group allocation significantly predicted scores for emotional role (p=0.03 t-test), mental health (p=0.04 t-test) and health transition (p=0.05 t-test). Subjects in the 'assess/advise/treat' group had significantly better self-rated mental health and emotional well-being and were more likely to perceive their general health to have improved in the last year. No significant differences were seen between groups for physical function (p=0. 72 t-test), physical role (p=0.65 t-test), bodily pain (p=0.32), general health (p= 0.11 t-test), vitality (p=0.09 t-test) or social function (p=0.07 t-test).

Effect size at long-term follow up.

The size of the treatment effect at long-term follow up is represented by the change in the R square as well as the by calculating the regression co-efficient. Table 4.6 summarises these results for the variables that displayed statistical significance at long term follow up. Group allocation explained 12% of the variance in MSPQ, 23% of the variance in MZSRDQ, 11% for STAIS, 11% for EuroQol health thermometer and 12%, 6% and 5% for SF36 Emotional Role, Mental Health and Health Transition respectively.

Table 4.8. Size of treatment effect at long-term follow up

Dependent Variable	Change in the R square	Regression Co-efficient (SE) Treat-Wait
MSPQ	0.12	-2.6(0.9)
MZSRDQ	0.23	-7.8(2.3)
STAIS	0.11	-2.4(0.9)
EQ health thermometer	0.11	13(5)
SF36 Emotional Role	0.12	17(8)
SF36 Mental Health	0.06	11(5)
SF36 Health Transition	0.05	-0.04(0.2)

Long-term outcome is affected by the timing of treatment. Patients who receive early treatment are less anxious, less distressed, display less depressive symptoms and rate their general health as better. The psychological features of general health particularly seem to be better in those patients receiving early treatment.

Patient Attrition

65 patients (64%) at 6 weeks and 63 patients (62%) at long-term follow up returned their assessments. There was no difference between the groups in the number of patients who returned questionnaires at either 6-week (chi square=1.75, p=0.19) or long-term follow up (chi-square=0.004, p=0.95). Regression analyses were used to explore the baseline characteristics of those who did respond and those who did not respond to follow up. None of the baseline variables were significantly different between responders and non-responders ($p>0.05$).

The potential effect of missing data was explored by re-fitting the regression models (for both short and long term effects of treatment) with missing data replaced by the last value carried forward (LVCF) (Howell 1992). Apart from VAS for usual pain intensity (short-term follow up was significantly lower in the 'assess/advise/treat' group (regression coefficient=-1.2, se=0.5, p=0.02)), there were no other differences between these models and the regression models from which cases with missing data had been deleted.

Discussion

Baseline

This study was undertaken in the physiotherapy department of a small metropolitan National Health Service Hospital. Patient baseline characteristics (table 4.4) indicated that on average patients fell within the normal range of distress or illness behaviour (Main et al., 1992). However 41% (n=38) of patients were assessed at baseline as either at Risk for Depression or Distressed – Depressive (Main et al., 1992). Similarly 31 patients (30%), demonstrated risk of long term work loss as assessed by the Yellow Flags Questionnaire (Linton and Hallden, 1998). These findings indicated that an important proportion of patients with acute low back pain referred for physiotherapy in a primary care setting exhibited psychosocial features associated with poor outcome (Main et al., 1992, Linton and Hallden 1998, Linton 2000). These findings further serve to highlight the importance of including psychosocial assessment and treatment as part of the treatment model.

The study was driven largely by the discrepancies that exist in recently published UK (CSAG 1994, Waddell et al 1999, Effective health Care 2000) and international low back pain guidelines (Koes et al 2001). The primary aim was to begin the search for what might constitute the optimal physiotherapy care for patients with simple acute low back pain. In this study the definition of simple low back pain offered by most National Guidelines (Koes et al 2001) was used as the inclusion criteria for the study, yet relatively few acute low back pain patients referred to the department fulfilled these criteria. The most conservative estimate from the data is that 73% of acute low back pain patients referred fell outside the criteria for simple acute low back pain (table 4.1).

Furthermore, the 804 patients logged only represent those referred into the department's acute service. During the study period a further 1800 low back pain patients were referred into the chronic services offered by the department. These findings have clear implications for the utility of these guidelines in primary care, as the population presenting for treatment might not represent the population from which the evidence base is derived. This was an unexpected finding and an unexpected

shortcoming of the available evidence based guidelines. It is imperative that physiotherapists and indeed all health care professionals become aware of the demographics of their client group and interpret and implement guidelines in keeping with these characteristics.

Six-week follow-up

Analysis at this time point enabled the first research question to be answered, that is, do patients treated with an active physiotherapy programme differ significantly from patients who have received advice on staying active only. Those patients participating in the treatment model demonstrate superior outcomes in measures of disability, general health, anxiety, depressive symptoms, mental health, social function and vitality. In the short term at least, it appears that the physiotherapy model is a superior intervention to advice on staying active for patients with acute low back pain. This is in keeping with findings on sub-acute low back pain (Torstensen, et al 1998). The treatment model was successful in positively influencing a number of different aspects of the low back pain experience and the effect sizes were reasonably large. The model favourably affected function, mood and general health but did not influence usual pain intensity. Both groups demonstrate significant reductions in pain over the first 6 weeks, and while this difference is considerably larger in the control group, the difference is not significant when baseline co-variates are included in the analysis.

The results that this trial demonstrates differ significantly from the majority of literature on the physical management of acute low back pain. Firstly, while 6 weeks is the short-term follow up period used in this study, it is considerably longer than the short-term follow up period used in the majority of trials reviewed in Chapter Three. Indeed in most trials demonstrating short-term benefit of physical treatment the effects were only seen at much earlier time points, having largely disappeared by 6 weeks (Little et al 2001, Dettori et al 1995, Chok et al 1999, Wilkinson et al 1995). This is particularly true of manipulation studies (Hadler et al 1987, Curtis et al 2000, Hoehler et al 1981, Matthews et al 1988, MacDonald and Bell 1990, Farrell and Twomey 1982). This finding suggests that it is doubtful that the effects seen are attributable solely to the manual therapy component of the treatment model. Furthermore, as both groups

received advice on staying active, this is unlikely to represent the major reason for the treatment effects seen, though it cannot be discounted that the 'assess/advise/treat' group had greater opportunity for this message to be reinforced during the initial 6 weeks.

Only the papers by Blomberg et al (1992, 1993, 1994) and Stankovic and Johnell (1990) provide results comparable with the present investigation. Interestingly, while the treatment content might not be exactly comparable, all three studies take a pragmatic, individualised approach to treatment and both include a number of modalities within the treatment package. Also explicit within all three studies is the idea of monitoring the effect of treatment and progressing or modifying the intervention if treatment goals are not met. The present study ensured this approach by the setting of long and short-term goals at the onset of treatment. Blomberg et al (1992) included a review process within their treatment algorithm and the philosophy of the treatment model investigated by Stankovic and Johnell (1990) strongly emphasises the importance of reassessment in determining the content and progression of treatment (McKenzie 1981).

However, the results of the present study differ from the results obtained by Blomberg (1992) and Stankovic and Johnell (1990) in one important aspect. In both of these studies the treatment model significantly improved pain. Review of the treatment content offers some explanation for this finding. Firstly, Blomberg et al (1992) employed steroid and anaesthetic injection of spinal soft tissue in his treatment model. This intervention was generally reserved for those patients who had local back or buttock pain that was not satisfactorily resolving with the package of manual therapy, exercise and traction. This study contained an additional treatment option that is specifically designed to relieve pain. Secondly, the McKenzie (1981) model of treatment employed by Stankovic and Johnell (1990) relies almost exclusively on patient's pain responses to dictate treatment content. Conceivably such an approach would be more efficacious in the treatment of pain.

The treatment model in the current study placed more emphasis on improving function and achieving functional goals and milestones, possibly to the detriment of optimally affecting pain. This finding is particularly germane given the results of two recent

studies that have explicitly sought to document the reasons for treatment success. Both Mannion et al (2001) and Woby et al (2001) found that pain reduction was one of the variables that most strongly predicted favourable functional outcome in patients with low back pain. Chapter five is concerned with further investigating these issues within the present study to help suggest refinements and improvements that could be made to the present model.

It is interesting to reflect on what approaches have been shown to be most successful in the management of acute low back pain. All guidelines offer support for medication and advice on staying active (Koes et al 2001). These two treatments could be thought of as non-specific interventions, that is, they have general effects and would offer the same benefit to patients regardless of the reason for the back pain. Secondly, studies that have taken a pragmatic approach to treatment provision, providing a diverse treatment model from which the most appropriate interventions for each individual are chosen, have demonstrated significant long and short-term benefit (Blomberg et al 1992, 1993, 1994, Stankovic and Johnell 1990, Linton et al 1993). These two approaches offer different solutions to the problems of diagnostic uncertainty and heterogeneity within simple low back pain and in effect offer two different paths by which the physiotherapy profession can proceed in the development of the management of acute low back pain. Some recent physiotherapy research has concerned itself with exploring general, less specific treatments for low back pain (for e.g. Klaber-Moffet et al 1999, Maher 2000a), while other researchers have investigated more specific treatment protocols based on various diagnostic approaches (for e.g. O'Sullivan et al 1999, Delitto et al 1995, van Dillen et al 1998).

A clear priority in physiotherapy research is to compare an optimal general treatment approach with an optimal individualised treatment approach. In the absence of comparative studies, calculating and comparing effect size between studies can provide some insight into which approach may offer the best results. Effect size can be calculated as (postmean –premean)/pre-SD (Mannion, et al 1999). Computations based on this formula represent the effects of treatment in standard deviation units (Hildebrandt et al 1997). For example an effect size of 0.50 represents an effect of one half the standard deviation. The study by Klaber-Moffett et al (1999) offers a useful comparison with the present study. The treatment investigated was a physiotherapy

supervised exercise classes where the content of the classes was strongly informed by available research, the patients had sub-acute low back pain and the treatment took place in a UK primary care setting. Moreover, the outcome measures and time scales are comparable.

In the present study the formula yields the value of 1.7 for the RMDQ at 6-weeks. As the standard deviation is 4.5 this represents an effect size of 7.7. The corresponding result in the Klaber-Moffett et al (1999) study is 0.71. The standard deviation is 4.02 producing an effect size of 2.9. The other comparable measure is the EuroQol total score. The value for the present study is 1.1. The standard deviation for this variable is 0.1, yielding an effect size of 0.11. For the Klaber-Moffett et al (1999) study the corresponding value is 0.69. As the standard deviation is 0.16 the effect size is also 0.11.

For disability, these figures suggest an individualised treatment programme is more efficacious than a general treatment approach. Conversely, Both approaches seem to effect general health equally. This result, however, may simply reflect the poorer responsiveness of the EuroQol to change (Garratt et al 2001). It is also worth noting that the number of treatments in the Klaber-Moffett study was almost double that of the present study. Further research is needed to formally evaluate the relative merits of these two approaches.

One common criticism for the use of specific treatments in the management of acute low back pain is the level of diagnostic uncertainty associated with low back pain. The discussion in Chapter Three suggested that a structured comprehensive clinical reasoning process might offer the best solution to this problem. The results from this trial and other pragmatic trials, which demonstrate superior results to trials with more strictly defined treatment protocols, indicate that individual therapists are making valid diagnostic decisions, an idea further supported by Levsen et al (2001). In this paper subjects were randomised to either a normal physiotherapy group or to treatment by therapists who had advanced training in clinical reasoning. An efficiency score was calculated that combined functional outcome and treatment number. Those therapists with enhanced clinical reasoning skills were significantly more efficient in the management of chronic low back pain patients than those with less training in clinical

reasoning. It is an imperative of the physiotherapy profession that this reasoning process is explored and dissected to begin the search for physiotherapy specific, valid, relevant and reliable diagnostic categories for low back pain.

Long- term follow-up

Analysis of data at three and six months enables the second research question to be answered, namely, do patients who received physiotherapy early differ significantly from patients who were asked to wait six weeks for their treatment. This provides insight into the importance of the timing of the intervention. Neither pain nor disability was significantly different between the groups during the course of the long-term follow up. It seems that these two aspects of the acute low back pain experience are unaffected by the timing of intervention. Delaying treatment led to a delay in improvement of disability, but with no long-term consequences for this variable.

A number of other important outcome variables, however, were adversely affected by delaying treatment. Patients seen promptly had significantly less anxiety, depressive symptoms and distress. They also had better general health, mental and emotional health, and quality of life. The psychosocial aspects of the low back pain experience would seem to be strongly influenced by the timing of treatment. It is interesting to note that these significant differences in the psychosocial aspects of well being occur despite there being no significant differences in the physical aspects of health. Not only are there no long-term differences in pain and disability but all the physical sub-scales of the SF36 (physical function, physical role, bodily pain and vitality) are equivalent between the two groups at long-term follow up. The treatment model seems to be able to affect the physical dimensions of low back pain regardless of the timing, but it only favourably affects the psychosocial aspects of low back pain when delivered early.

Related to this is the effect of the treatment model on the patient's general health. The EuroQol calculates two different general health scores. One score represents patients self perception of their general health based on the score given on the health thermometer, the second score is calculated from the combination of a number of subscales representing various dimensions of general health. The total health score is no

different between groups, while the self perceived health is significantly poorer in the control group. It would seem that even though both groups have similar levels of physical function and pain, the patients in the control group rate their general health as considerably worse, emphasising the importance that psychological well being has on perceptions of general health.

This is the first physiotherapy study to formally include time to treatment as an independent variable, and only the second controlled trial on acute low back pain to explicitly address this issue (see Chapter Three). The study has shown timing of intervention to have an important influence on outcome for an episode of acute low back pain, with prompt intervention producing better results. Early physiotherapy treatment can improve psychosocial outcomes but delaying the onset of treatment did not provide the opportunity for physiotherapy intervention to have this favourable effect.

There is a clear distinction in the responsiveness of physical and psychosocial parameters to intervention. Even though the treatment model was primarily a physical treatment model there was a strong emphasis in the assessment, clinical reasoning and treatment plan on identifying and addressing psychosocial barriers to recovery. The model seemed to achieve these goals when applied early, but was unable to favourably affect the psychosocial aspects when treatment was delayed.

Review of the data set suggests that rate of change of pain and disability might offer some explanation for these findings. The change in disability and pain scores between baseline and 6 weeks is a measure of the rate of change of these variables in the short term. The rate of change of disability and pain in the 'assess/advise/treat' group was nearly 2 ½ times greater than the control group. The mean change in disability for the control group was 3.2 and for the intervention group 7.8. This difference is statistically significant ($p<0.001$ *t*-test). The mean change for pain in the early treatment group was 3.3 and only 1.4 in the control group. This difference was also significant ($p=0.01$ *t*-test). So while both groups attained similar levels of pain and disability in the long term, the rate at which these were achieved differed between the two groups.

Both groups were equivalent at baseline on all psychosocial variables and displayed moderate levels of psychosocial dysfunction (see above). Also the differences seen at long term are essentially due to the intervention group improving and the control group remaining relatively static with respect to mood, rather than the control group worsening. It might be that as long as patients perceive an improvement and progression in their physical condition then the distress, anxiety and concern about their health is diminished. If on the other hand the changes in pain and disability are slower than expected the patient might remain concerned, anxious and distressed. Furthermore a quick resolution of symptoms may lead subjects to view low back pain as a trivial problem, which can be easily and quickly 'fixed'. Whereas delay in the resolution of pain and disability leads to a more catastrophic and distressing view of low back pain. Chapter Five will attempt to provide more understanding of the interrelationships between the physical and psychological aspects of the low back pain experience and what factors are responsible for producing favourable outcomes.

Alternatively, the differential effects seen might simply be due to the difference in treatment number. It was anticipated that a relatively large number of subjects in the 'assess/advise/wait' group would not require further treatment at 6 weeks. The data on the natural history of low back pain would suggest a high degree of natural recovery. Indeed, this is one of the main reasons for the suggestion of a wait and see approach. However, it was also hypothesised that the patients who did return for treatment at 6 weeks would require more intervention, as their problems would be more chronic. Though patients were given a written appointment and reminded of their appointment, it was felt that any further attempts to return patients to formal care was not in keeping with the philosophy of a wait and see approach.

The more favourable outcome seen in the 'assess/advise/treat' model of care did entail more treatments then the 'assess/advise/wait' group. This difference is totally accounted for by the 20 subjects in the 'assess/advise/wait' group who did not re-attend for treatment, as there is no difference in treatment number between the 'assess/advise/treat' group and those within the 'assess/advise/wait' group who did re-attend. Delaying treatment by 6 weeks does not seem to influence the number of sessions required to meet the functional goals of those who undertook formal treatment.

The exit point for treatment was dependant on attaining functional goals, which were without exception physical goals. The equivalence in physical outcomes between groups supports that this model was adhered to and suggests that acute and sub-acute patients respond equally favourably and equally quickly in attaining functional goals. The failure of treatment to favourably effect psychosocial outcomes in the sub-acute stage might have been in part due to the treatment termination being overly dependant on physical milestones. Other researchers have noted that the interaction of biological, psychological and social variables changes with the stage of the disorder (Waddell 1998), so while such an approach was effective in dealing with all dimensions of the low back pain experience in the acute stage, it might be that over reliance on physical milestones is less than optimal in the sub-acute stage.

The data presented in Chapters Two and Three emphasised the importance of psychosocial variables in predicting chronicity in low back pain. The present findings suggest that early intervention may significantly reduce the chance of chronic problems developing. A longer follow up period would be necessary to fully test this hypothesis. These are unique and unexpected findings, though it is unclear if the results are a feature of the treatment model or are apparent simply because previous studies on acute low back pain have not generally included psychological outcome measures. Regardless it is important to recognise that these findings demonstrate the importance of considering a number of aspects of the low back pain experience to fully evaluate the effect of any intervention.

Models of Care

The third research question asked was do the overall results suggest any meaningful differences in outcome between an 'assess/advise/treat' model and an 'assess/advise/wait' model. Overall evaluation of the results suggests that patients derive considerably more benefit from being involved in an 'assess/advise/treat' model of care. Within this model subjects experience short-term benefits in disability, general health and mood as well as long-term benefits in mood and general health. Furthermore, as delaying treatment significantly delays the resolution of depression,

anxiety and distress, there is an increased risk of chronic problems developing when treatment is delayed.

A formal cost analysis was not included in the study plan, though in the short term the 'assess/advise/treat' model of care would be more expensive given the significantly larger number of treatments. This extra cost does, however, lead to large and significant improvements in short and long term outcome, and may lead to less chronic problems developing. A judgement on the cost/benefits ratio would need to consider both the more favourable outcome and the possibility that less future health care resources will be utilised by patients in the 'assess/advise/treat' model of care. The lack of longer-term follow up data precludes any definitive statements on the comparative costs of the two treatment models.

The CSAG report (1994) called for a change in NHS services for patients with low back pain. The report concluded that although there is a high probability that an acute attack will settle, the current statistics show that this should not be taken as grounds for complacency, inactivity or a policy of 'wait and see' on the part of health professionals. The CSAG (1994) report was criticised (Feder and Hemingway, 1995) for basing this recommendation on anecdotal evidence and on making a bold claim that the provision of 'NHS services at the acute stage...will prevent chronic pain and disability'. More recent evidence based guidelines (Faas et al 1996, Bogduk 2000) have added support to this view and have advocated that a 'wait and see' approach to physical intervention is more representative of the current evidence base. This study directly compared these two models of care. The results indicate that the CSAG recommendation of early intervention does produce superior outcomes in both the short and long term. These findings are in favour of an "early intervention" model of care.

ALTERNATIVE INTERPRETATIONS OF THE RESULTS

MISSING DATA

The greatest threat to the validity of these results is presented by the missing data. A number of steps have been taken to minimise this threat. Firstly the statistical model used was the most efficient available to control for the possible confounding effects of

missing data, as baseline values were controlled for in both long and short term analysis (Tabachnik and Fidell 1996). Secondly, combining 3 and 6-month scores into a single long-term outcome measure increased the available data for long-term analysis.

In addition a number of statistical checks were undertaken to investigate the possible biasing effect of non-response. There was no difference between the groups in the number of patients who returned questionnaires at either 6 week or long term follow up. There were no significant interactions between group and responder status for any baseline variable, and the sensitivity analysis with missing data replaced by the last value carried forward (Howell 1992) produced equivalent results to the regression models from which cases with missing data had been deleted.

The data set was also interrogated in a number of other ways to assess potential for bias. The results would be biased by the missing data if the non-responding subjects in the 'assess/advise/wait' had better than average outcomes and the non-responders in the 'asses/advise/treat' group had worse than average outcomes. The data available suggests that this is not the case. At 6-weeks, 5 non-responders from the 'assess/advise/wait' group attended for physiotherapy treatment. Four of the 5 still had significant levels of pain and had not achieved their agreed functional goals at this time point. Review of the case notes of the 8 non-responders in the early treatment group show that by 6-weeks, 5 of the subjects were pain free, had achieved their functional goals and completed their physiotherapy treatment. Non-responders at 6-weeks for whom long-term follow up data is available do not differ significantly from the rest of the cohort at long term follow up. Similarly, non-responders at long-term follow up for whom there is 6-week data available are not significantly different from the rest of the cohort at 6-weeks.

A final piece of evidence that the non-responders did not bias the results can be seen when the reasons for non-response are reviewed. Sixteen of the 29 non-responders (55%) did so because they were non-contactable due to a change of address. Such a high number of subjects being non-contactable may partly be explained by the demographics of the local area, an inner city borough with considerable economic and social deprivation. Whatever the explanation, the reason for non-response in a

significant proportion of the subjects was due to a factor that is unlikely to introduce bias to the results.

The higher than expected level of patient attrition certainly decreases the confidence in the study findings. Every attempt made to investigate the possible bias introduced by the missing data suggests that the patients who responded were representative of those who did not respond. Also, the numbers analysed were lower than the power calculation indicated. Replication of these results in a study with lower attrition rates is necessary before final judgement on these findings can be made.

CHANCE

The large number of outcome measures used in this study enabled various aspects of the low back pain experience to be investigated. However, by using so many variables there is an increased possibility that some of the significant results have occurred by chance (Howell 1992). Close scrutiny of the data does not support this interpretation of the results. While at short-term follow up there are significant treatment effects for a variety of factors, the significant long-term outcomes are quite banded. None of the measures of symptoms or physical function display any significant treatment effect, while significant group differences are seen only in the psychosocial measures. This clustering of outcome diminishes the likelihood that these findings are a product of chance.

GENERALISABILITY

There are a number of factors that may limit the generalisability of the study findings. The review of studies on early intervention in Chapter 3 highlighted a number of studies were early treatment was ineffective or even detrimental (Haig et al 1990, Cooper et al 1996, Sinclair et al 1997) and 2 manipulation studies clearly demonstrated that earlier intervention does not produce better outcomes (Hadler et al 1987, MacDonald and Bell 1990). It became apparent from talking to GP's prior to the study and during subsequent study days that their normal referral practice was not to send patients to physiotherapy on first consultation for acute low back pain. Patients were generally only referred on their second or even third consultation. This is reflected in the study sample where the average duration of symptoms for the cohort at initial

presentation was 3 weeks. This referral practice meant that a filtering mechanism for spontaneous responders was in operation. Less impressive results might be seen in a setting where this filtering mechanism is not in place.

A second threat to the generalisability lies in the level of expertise of the staff. The level of experience was considerable, and all but one of the treating therapists had postgraduate qualifications in musculoskeletal physiotherapy. This represents a level of proficiency that is probably not the norm in most NHS outpatient departments.

A final issue of generalisability lies in the local nature of the model used. The model was developed primarily by the author with some consultation with the other members of the research team. The model lacks consensus from the wider physiotherapy community and this is seen as a priority for further development and use of the model.

PLACEBO

Finally it is possible that non-specific treatment effects were responsible for the results seen. It was not possible to blind the therapists to treatment allocation. This introduces potential for bias, though this is likely to be of minimal significance as the only therapist contact was during the performance of active treatment. Of more significance is the blinding of subjects to treatment allocation. While subjects were obviously aware that they were in an early or a late intervention group, the study design tried to minimise the biasing effect of this by introducing a form of quasi-blindness. Subjects in the assess/advise/wait group were not aware that there was a possibility of them receiving early treatment. While this might not discount a placebo effect on those receiving early treatment, it decreases the nocebo effect on those who waited for treatment. A more satisfactory way of dealing with this problem would have been to involve the assess/advise/wait group in an inactive intervention during the initial 6-week period. However, this solution reintroduces the problem of therapist blindness and is less in keeping with a 'wait and see' philosophy of care.

Other areas of possible bias were well controlled. Volunteer bias was minimal as only 7 of 265 patients contacted failed to give consent. Selection bias caused by patients who changed treatment did not occur. Detection bias was minimised by collecting data at the

set time points of 6 weeks 3 months and 6 months. Finally confounding bias seems unlikely as randomisation was successful and multivariate analyses were used.

CONCLUSION

The evidence based physiotherapy model developed in Chapter Three was tested in a randomised controlled trial that enabled a number of important research questions to be answered. Firstly, the model appears to be efficacious, producing significantly better short-term outcomes than advice on staying active. Secondly, the effectiveness of the model is dependant on the timing of intervention. Delaying physiotherapy treatment decreases its effectiveness and increases the risk of chronic problems developing. Finally, The overall results support the role of physiotherapy in the management of acute low back pain, emphasise the importance of early intervention and suggest that an 'assess/advise/treat' model of care is superior to an 'assess/advise/wait' model of care.

Discussion of these findings provides interesting insight into the possible mechanisms of action of the intervention as well as some of the possible shortcomings of the model. It is the purpose of the next Chapter to formally explore these issues to develop improvements and refinements to the current model.

CHAPTER FIVE

REFINING THE MODEL

Introduction

The final aim of this thesis is to try to understand more fully the mode of action of the treatment model. The purpose of clinical research should be to add to the evidence base, inform clinical practice and extend the theoretical appreciation of the clinical condition. The current understanding of low back pain precludes the application of research designs that fully address all these issues. Further analysis needs to be undertaken to understand the mode of action of the intervention, to help further refine the treatment model and gain greater insight into the clinical course of acute low back pain.

Information from a number of sources was used to develop the model. This has included case controlled studies, prospective longitudinal cohort studies and clinical trials. All these forms of data collection provide important information and answer different clinical questions. What is less often investigated is the process that mediates change in patients undergoing therapy. A lot is known about what baseline variables predict successful outcome (Watson 2000, Waddell 1998, Linton 2000), but the important question of what happens during the therapeutic process receives scant attention. Investigating the interactions between variables during treatment provides insight into how favourable outcome is mediated. This process will enable further refinement of physical treatment for acute low back pain patients.

The increased awareness of the importance of the patient's perspective in shaping health care (Deyo et al 1994) and the difficulties associated with diagnosis in acute low back pain (Chapter Three) have led to an emphasis on patient centred measurement of outcome in acute low back pain (Delitto 1994). In most recent studies the aims to

improve patients' functional abilities and decrease pain are generally explicit within the therapeutic process and are seen as the primary outcome measures. However, Chapter Three highlighted the diversity in opinion of how this is best achieved. Clinicians and researchers attest to a variety of assumptions associating various aspects of patients' performance with meaningful, patient centred outcome.

Numerous studies have documented change in assorted aspects of performance with therapy. Both Gatchel et al (1999) and Leggett et al (1999) report improvements in patient self-perceived general health with functional restoration programmes. Spinal range of motion has been shown to increase with manual treatment (Blomberg et al 1994, Morton 1999), graded exercise programmes (Lindstrom et al 1992a) and functional restoration programmes (Rainville et al 1992, Magnusson et al 1998). Subjects participating in exercise programmes also demonstrate improvements in trunk muscle strength and endurance (Rainville et al 1992, Lindstrom et al 1992a, Brady et al 1994, Manniche et al 1993, 1991, Kankaanpaa et al 1999 and Chok et al 1999).

The use of more sophisticated measuring tools has highlighted a number of other performance parameters that are changed by therapy. Lindgren et al (1993) reported improvements in multifidus activation with exercise therapy. O'Sullivan et al (1998) noted changes in the automatic pattern of activation of trunk muscles with spinal stabilizing exercises. Similarly, Hides et al (1996) demonstrated restoration of muscle cross sectional area in subjects performing a similar exercise regimen. Tawfik (2001) showed improvements in symmetry of trunk function with functional rehabilitation, likewise Herzog et al (1991) noted improvements in gait symmetry with manual treatment. Finally Magnusson et al (1998) found an increased velocity of trunk motion as subjects progressed through a functional rehabilitation programme.

The relationship of these changes to successful patient centred outcome in the management of acute low back pain is not always clear. Various controlled studies have demonstrated that the changes in performance observed in patients undergoing therapy are no different to changes observed in control groups (Serfelis et al 1998, Pope et al 1994 Farrell and Twomey 1982), or bear little relationship to the changes observed in patient centred outcome (Farrell and Twomey 1982, Klaber-Moffett et al 1999, Herzog et al 1991). Whereas other investigations have noted similar behaviour between various

aspects of performance and patient centred outcome (Dettori et al 1995, Blomberg et al 1994, Morton 1999, Lindstrom et al 1992a). This uncertainty in the literature results from the multifaceted nature of the low back pain experience and the absence of unequivocal methods of assessing physical and psychological impairment (Mannion et al 2001). Other ways of exploring these relationships have begun to appear in the literature and start to offer greater insight into the understanding and treatment of low back pain.

One study, while not offering formal statistically evaluation of the determinants of outcome, provides interesting and unique insight into the acute low back pain experience. Hides et al (1994, 1996, 2001) investigated the effect of a spinal stabilisation exercise programme for patients with first time acute low back pain. Localised multifidus wasting was demonstrated on ultrasound, ipsilateral to the side of symptoms. The localisation of this phenomenon, its appearance so soon after the onset of symptoms and the fact that all patients were first time low back pain patients led the researchers to conclude that these findings were induced by the onset of low back pain, rather than an antecedent to the pain. Subjects were then randomised to receive either medical treatment or spinal stabilisation exercises. Subjects who undertook stability training demonstrated restoration of their multifidus cross sectional area, while the control did not. Interestingly, subjects in the exercise group had large and significant reductions in the rate of low back pain recurrence. This study provides evidence of clear links between therapy, change in a physiological variable and improvement in patient centred outcome. There is a strong suggestion from these data that the mechanism of action of successful outcome in this study was due to improvement in back muscle performance.

A handful of studies have assessed low back pain patients before and after rehabilitative programmes and explicitly investigated the relationship between various measured variables and favourable patient centred outcome. Hildebrandt et al (1997) followed chronic low back pain (CLBP) patients through an 8-week functional restoration programme. The primary treatment outcome was return to work. Patients were categorised into two mutually exclusive groups of either back to work or not working. They found that physical parameters such as mobility, flexibility, strength, endurance and lifting capacity demonstrated little correlation with return to work. However,

changes in disability scores, decrease in depression and not requiring individual treatment predicted return to work with 85% accuracy. The secondary outcome measure of pain intensity was similarly investigated. The results were comparable to return to work, with reduction in disability and depression most strongly predicting pain reduction, though trunk flexion and lower limb leg press strength also demonstrated some weak association.

Vendrig (1999) also categorised patients into two groups of returned to work and not returned to work and looked at the relationship between changes in outcome and assignment to group. Only changes in self rated disability and pain predicted return to work status. None of the physical and psychological measures used were related to return to work.

Mannion et al (2001) used a similar methodology to investigate the factors influencing changes in low back pain related disability (RMDQ). Subjects were pooled from a clinical trial on three different forms of exercise therapy for chronic low back pain. A range of physical, psychological and pain related factors were included in the analysis. Only changes in pain and psychological stress (MSPQ+ZUNG) were significantly related to disability. Multivariate analysis confirmed change in pain (16%) and distress (4.1%) as the most important determinants of good outcome.

Woby et al (2001) assessed patients undergoing a chronic low back pain rehabilitation programme. Subjects were divided into two mutually exclusive groups of 'clinically improved' and 'not clinically improved' based on changes in their pre and post RMDQ scores. Changes in pain scores and a number of psychological measures were calculated and used to determine if mean changes in these scores differed between the clinically improved and not improved groups. Mean change scores were significantly different between the two groups for six factors. These include pain, anxiety, depression, catastrophizing, pain-related fear and functional self-efficacy. These were further analysed using a stepwise discriminate analysis. Reduction in perceived pain and decrease in pain related fear were the most significant predictors of change in disability. The dominance of pain in explaining treatment success in these last two papers is particularly important in light of the non-significant effects that the treatment model tested in this thesis had on pain.

This chapter aims to add to this emerging evidence base by seeking to explain both short and long-term changes in low back pain related disability (RMDQ). Back pain related disability is a good clinical assessment of the severity of a low back disorder (Waddell 1987) as well as a good predictor of return to work (Nordin et al 1997). It is regarded as a fundamental outcome measure in low back pain research (Delitto 1994, Deyo et al 1994) and restoration of patients' functional performance was central to the treatment model tested. For these reasons patients self reported low back pain related disability was chosen as the primary outcome measure to be investigated. It also has the added advantage of allowing ease of comparison with the results of Mannion et al (2001) and Woby et al (2001).

Exploration of the factors that mediate good functional outcome will enable three research questions will be addressed. These are:

1.What are the processes that mediate clinically important short-term change in disability in patients undergoing an active physiotherapy programme?

2.Are the processes that mediate clinically important short-term change in disability in patients undergoing an active physiotherapy programme different to the processes that mediate change in a group receiving advice to stay active?

3.What are the processes that mediate clinically important long-term change in disability in patients undergoing an active physiotherapy programme?

This information will then be used to refine and suggest changes that could be made to the treatment model developed in Chapter Three.

Methods

Subjects.

Details of the recruitment methods employed and the inclusion and exclusion criteria for participants in the study are described in Chapter Four. Baseline characteristics of the study sample can be found in table 4.1.

Subjects were divided into two groups based on the criteria of Stratford et al (1996). Stratford et al (1996) investigated the sensitivity to change of the RMDQ and calculated that the minimum level of detectable change was a difference of four points for patients with a baseline score less than 11 or a difference of five for patients with a baseline score over 11. Based on their findings the change scores for the RMDQ were classified as either clinically improve or not clinically improved (Woby et al 2001). This process was completed separately for 6-week and long-term follow up. Patients with a baseline RMDQ score of less than 2 were excluded from this analysis unless their baseline scores worsened in which case they were classified as unchanged. Subjects with a baseline score of 3 were excluded unless their baseline score remained unchanged or worsened, in which case they were classified as unchanged.

Interventions.

Subjects were randomly assigned to an assess/advise/treat group or an assess/advise/wait group. At the six-week period half the subjects had been involved in an active physiotherapy treatment programme, while the remainder had received advice to remain active. At this time point both groups will be considered separately to highlight differences in the mode of action of the interventions. After six weeks both groups had the opportunity to participate in an active physiotherapy programme. Analysis of long-term changes in disability will consider both the total cohort as well as the two study groups individually to ascertain if delaying treatment has any effect on the way long-term outcome is mediated.

Assessments.

Assessments were made at baseline, six weeks, three months and six months. Data from the three and six month assessments was combined to provide a single measure of long-term outcome. Full details of the dependant variables used and the method of data collection can be found in Chapter Four.

Data Reduction.

For each of the dependant variables change scores were calculated from baseline to six week follow up and from baseline to long term follow up. Differences in RMDQ scores were categorised as outlined above. For all other dependant variables the raw difference score was used for further analysis.

The fifteen secondary outcome measures represent five main factors. These are:

- Symptoms: VAS usual pain intensity and SF36 Bodily Pain
- Physical Function: SF36 physical function, SF36 physical role and SF36 vitality.
- Psychological: MZSRDS, STAIS, MSPQ, SF36 mental health and SF36 emotional role.
- Social: SF36 social function.
- General Health: EuroQol total score, EuroQol health thermometer, SF36 general health and SF36 health transition.

The large number of variables increases the possibility of finding significant effects by chance. While it is felt that it is important for at least one item to be included from each factor, to decrease the number of variables two criteria will be applied. Firstly, only variables that have clear clinical correlates will be included. For example it is difficult to see how information from the health transition or emotional role SF36 sub-scales would inform or modify a treatment model. Secondly, if a number of outcome measures contain very similar information as far as informing the treatment model is concerned, only one of these variables will be considered. The application of these criteria produced a set of seven variables. These are, VAS usual pain intensity, SF36 physical

function, MZSRDS, STAIS, MSPQ, SF36 social function and EuroQol health thermometer. This set achieves the best compromise between efficiency and comprehensiveness. This covers all factors with minimal replication and includes the variables that are most able to inform clinical practice.

Of the exclude variables SF36 bodily pain was felt to offer little extra information and was less precise than the VAS. Likewise SF36 physical role and vitality offered little additional information to the understanding of physical function and were much less precise measures than the SF36 physical function. The MZSRDS, STAIS and MSPQ all represented discrete facets of psychological functioning and offered more exact and clinically informative measures of psychological function than the two SF36 measures. Finally it was felt that the EuroQol health thermometer offered the most unique insight into general health. The other two scales constructed a score for general health based on a number of items already well represented in other dependant variables.

Statistics.

Levene's Test for Equality of Variance was used to check for homogeneity-of-variance between groups for each dependant variable. For each case this test computes the absolute difference between the value of that case and its cell mean and performs a one-way analysis of variance on those differences (Tabachnick and Fidell 1996). Independent sample 2 tailed *t*-Tests were used to determine whether mean change scores in the dependant variables differed between the clinically improved and non-clinically improved groups. This analysis was undertaken separately for the assess/advise/treat and assess/advise/wait patients for 6-week and long-term change scores and for the group as a whole for long-term change scores.

Because the nature of the study was exploratory, testing a wide range of outcome measures, an adjustment of the alpha error, as would be necessary in a confirmative study, was not performed.

The results were further analysed to determine which of the factors contributed most to the prediction of clinically important changes in disability. The factors found to be

significantly related were entered individually as independent variables into a univariate linear regression with change in disability as the dependent variable. This provided a clearer indication of the strength of association of each variable and helped determine the significance of each variable in modification of the treatment model.

The strength of association for each variable was further explored through multivariate analysis. Each factor found to be significantly related was entered as an independent variable into a stepwise linear regression with change in disability as the dependent variable. This provided further insight into the relative importance of each factor in determining good outcome. With stepwise regression, statistics computed from sample data control order of entry and it is therefore thought of as a better model-building procedure than hierarchical regression (Tabachnik and Fidell 1996).

RESULTS

Explaining Changes in Disability at Short Term Follow Up

Active Physiotherapy

In the active physiotherapy group twenty-four subjects demonstrated clinically meaningful changes in disability, while eleven subjects remained unchanged.

Pain. There was a significant difference in change scores for pain between the clinically improved and clinically not improved groups (p=0.025). The mean change in pain for the clinically improved group was 4.0 (SD=2.07) and 1.9 (SD=2.07) for the clinically not improved group.

Physical Function. There was a significant difference in change scores for physical function between the clinically improved and clinically not improved groups (p=0.012).

The mean change in physical function for the clinically improved group was –33.12 (SD=26.2) while the clinically not improved group demonstrated an average change of only –0.50 (SD=31.7).

Depressive Symptoms. There was no significant difference in change scores for depressive symptoms between the clinically improved and clinically not improved groups (p=0.127). The mean change in scores on the MZSRDS for the clinically improved group was 5.41 (SD=10.35) while the clinically not improved group demonstrated an average change of 1.27 (SD=5.29).

Distress. There was no significant difference in change scores for the MSPQ between the clinically improved and clinically not improved groups (p=0.331). The mean change in scores on the MSPQ for the clinically improved group was 2.46 (SD=4.3) while the clinically not improved group demonstrated an average change of 3.72 (SD=3.1).

Anxiety. There was no significant difference in change scores for anxiety between the clinically improved and clinically not improved groups (p=0.394). The mean change in scores on the STAIS for the clinically improved group was 2.21 (SD=5.57) while the clinically not improved group demonstrated an average change of 0.2 (SD=6.28).

Social Function. There was no significant difference in change scores for social function between the clinically improved and clinically not improved groups (p=0.429). The mean change in social function scores for the clinically improved group was –27.60 (SD=23.60) while the clinically not improved group demonstrated an average change of –33.75 (SD=18.68).

General Health. There was no significant difference in change scores for self reported general health between the clinically improved and clinically not improved groups (p=0.982). The mean change in general health scores for the clinically improved group was –15.64 (SD=22.31) while the clinically not improved group demonstrated an average change of –15.45 (SD=21.83).

Table 5.1 provides a summary of these results

Table 5.1 Differences in short-term change scores for clinically improved and clinically not improved groups for subjects receiving active physiotherapy. See figure 4.1 for key.

Variable	Improved (n=24) Average Change (SD)	Not Improved (n=11) Average Change (SD)	*p* value
VAS pain	4.0 (3.06)	1.9 (2.07)	**0.025**
Physical Function	-33.12 (26.2)	-0.5 (31.7)	**0.012**
MZSRDS	5.41 (10.35)	1.27 (5.29)	0.127
MSPQ	2.46 (4.3)	3.72 (3.1)	0.331
STAIS	0.2 (6.28)	2.21 (5.57)	0.394
Social Function	-33.75 (18.68)	-27.60 (23.60)	0.429
General Health	-15.45 (21.83)	-15.64 (22.31)	0.982

Regression

Univariate analysis indicated that change in pain contributed to 11% of the variance in disability while change in physical function contributed 23%.

Stepwise regression analysis included change in pain and change in physical function scores as independent variables. This analysis showed that only change in physical function still contributed significantly to explaining the variance in disability, accounting for 23% of the variance. Change in pain was excluded from the multivariate model.

Advice on Staying Active

In the advice on staying active group eleven subjects demonstrated clinically meaningful changes in disability, while nineteen subjects remained unchanged.

Pain. There was a significant difference in change scores for pain between the clinically improved and clinically not improved groups (p=0.020). The mean change in pain for the clinically improved group was 2.72 (SD=1.95) and 0.68 (SD=2.5) for the clinically not improved group.

Physical Function. There was no significant difference in change scores for physical function between the clinically improved and clinically not improved groups (p=0.384). The mean change in physical function for the clinically improved group was –13.63 (SD=25.99) while the clinically not improved group demonstrated an average change of –5.52 (SD=19.85).

Depressive Symptoms. There was no significant difference in change scores for depressive symptoms between the clinically improved and clinically not improved groups (p=0.639). The mean change in scores on the MZSRDS for the clinically improved group was 1.0 (SD=8.21) while the clinically not improved group demonstrated an average change of –0.7 (SD=11.77).

Distress. The difference in change scores for the MSPQ between the clinically improved and clinically not improved groups just failed to reach significance (p=0.059). The mean change in scores on the MSPQ for the clinically improved group was 4.63 (SD=6.07) while the clinically not improved group demonstrated an average change of 1.36 (SD=3.08).

Anxiety. There was no significant difference in change scores for anxiety between the clinically improved and clinically not improved groups (p=0.891). The mean change in scores on the STAIS for the clinically improved group was –0.33 (SD=2.97) while the clinically not improved group demonstrated an average change of –0.55 (SD=4.48).

Social Function. There was no significant difference in change scores for social function between the clinically improved and clinically not improved groups (p=0.449). The mean change in social function scores for the clinically improved group was –20.45 (SD=30.76) while the clinically not improved group demonstrated an average change of –11.87 (SD=26.51).

General Health. There was no significant difference in change scores for self reported general health between the clinically improved and clinically not improved groups (p=0.718). The mean change in general health scores for the clinically improved group

was –0.125 (SD=23.63) while the clinically not improved group demonstrated an average change of –3.7 (SD=26.56). Table 5.2 provides a summary of these results.

Table 5.2 Differences in short-term change scores for clinically improved and clinically not improved groups for subjects receiving advice to stay active. See figure 4.1 for key.

Variable	Improved (n=11) Average Change (SD)	Not Improved (n=19) Average Change (SD)	*p* value
VAS pain	2.72 (1.95)	0.68 (2.5)	**0.020**
Physical Function	-13.63 (25.99)	-5.52 (19.85)	0.384
MZSRDS	1.0 (8.21)	-0.7 (11.77)	0.639
MSPQ	4.63 (6.07)	1.36 (3.08)	0.059
STAIS	-0.33 (2.97)	-0.55 (4.48)	0.891
Social Function	-20.45 (30.76)	-11.87 (26.51)	0.449
General Health	-0.125 (23.63)	3.7 (26.56)	0.718

Performance of the same tests on the whole cohort at short-term follow up did not change the results. Only changes in pain (p<0.001.) and change in physical function (p=0.001) were different between the clinically improved and clinically non-improved groups.

Regression

As only one variable was significantly related only the univariate analysis was used. The regression analysis included change in pain score as an independent variable. This analysis showed that change in pain accounted for 16% of the variance in disability.

Explaining Changes in Disability at Long-Term Follow Up

Analysis By Group

Active Physiotherapy

In the active physiotherapy group twenty-five subjects demonstrated clinically meaningful changes in disability at long term follow up, while six subjects remained unchanged.

Pain. There was a significant difference in change scores for pain between the clinically improved and clinically not improved groups (p<0.001). The mean change in pain for the clinically improved group was 4.0 (SD=2.5) and –0.667 (SD=1.63) for the clinically not improved group.

Physical Function. Contrary to the findings at short-term follow up, there was no significant difference in change scores for physical function between the clinically improved and clinically not improved groups (p=0.397). The mean change in physical function for the clinically improved group was –27.5 (SD=36.8) while the clinically not improved group demonstrated an average change of –13.0 (SD=14.8).

Depressive Symptoms. There was no significant difference in change scores for depressive symptoms between the clinically improved and clinically not improved groups (p=0.235). The mean change in scores on the MZSRDS for the clinically improved group was 6.1 (SD=10.4) while the clinically not improved group demonstrated an average change of –1.2 (SD=10.6).

Distress. There was a significant difference in change scores for the MSPQ between the clinically improved and clinically not improved groups (p=0.029). The mean

change in scores on the MSPQ for the clinically improved group was 3.9 (SD=4.3) while the clinically not improved group demonstrated an average change of –1.0 (SD=6.3).

Anxiety. There was no significant difference in change scores for anxiety between the clinically improved and clinically not improved groups (p=0.243). The mean change in scores on the STAIS for the clinically improved group was 2.6 (SD=4.6) while the clinically not improved group demonstrated an average change of 0.0 (SD=3.5).

Social Function. There was a significant difference in change scores for social function between the clinically improved and clinically not improved groups (p=0.040). The mean change in social function scores for the clinically improved group was –39.5 (SD=15.2) while the clinically not improved group demonstrated an average change of –7.5 (SD=24.4).

General Health. There was a significant difference in change scores for self reported general health between the clinically improved and clinically not improved groups (p=0.029). The mean change in general health scores for the clinically improved group was –21.21 (SD=21.0) while the clinically not improved group demonstrated an average change of –2.2 (SD=13.1).

Table 5.3 provides a summary of these results

Table 5.3 Differences in long term change scores for clinically improved and clinically not improved groups for assess/advise/treat subjects. See figure 4.1 for key.

Variable	Improved (n=25) Average Change (SD)	Not Improved (n=6) Average Change (SD)	*p* value
VAS pain	4.0 (2.5)	-0.7 (1.6)	**<0.001**
Physical Function	-27.5 (36.8)	-13.0 (14.8)	0.397
MZSRDS	6.1 (10.42)	-1.2 (10.6)	0.235
MSPQ	3.9 (4.3)	-1.0 (6.3)	**0.029**
STAIS	2.6 (4.6)	0.0 (3.5)	0.243
Social Function	-39.5 (15.2)	-7.5 (24.4)	**0.040**
General Health	-21.2 (20.1)	-2.2 (13.1)	**0.029**

Regression

Univariate analysis for each significant variable indicated that pain explained 39% of the variance in disability, social function 35%, general health 20% and distress (MSPQ) 15%.

The multivariate stepwise regression analysis included pain, MSPQ, social function and general health change scores as independent variables. This analysis showed that only change in pain still contributed significantly to explaining the variance in disability, accounting for 40% of the variance. Change in MSPQ, Social function, and general health were excluded from the model.

Advice on Staying Active

In the advice on staying active group twenty-three subjects demonstrated clinically meaningful changes in disability, while nine subjects remained unchanged.

Pain. There was no significant difference in change scores for pain between the clinically improved and clinically not improved groups (p=0.284). The mean change in pain for the clinically improved group was 2.83 (SD=2.8) and 1.7 (SD=2.4) for the clinically not improved group.

Physical Function. There was a significant difference in change scores for physical function between the clinically improved and clinically not improved groups (p=0.009). The mean change in physical function for the clinically improved group was –22.6 (SD=18.6) while the clinically not improved group demonstrated an average change of –1.1 (SD=22.2).

Depressive Symptoms. There was no significant difference in change scores for depressive symptoms between the clinically improved and clinically not improved groups (p=0.1.38). The mean change in scores on the MZSRDS for the clinically

improved group was 1.3 (SD=12.6) while the clinically not improved group demonstrated an average change of –4.4 (SD=10.2).

Distress. There was no significant difference in change scores for the MSPQ between the clinically improved and clinically not improved groups (p=0.958). The mean change in scores on the MSPQ for the clinically improved group was 0.52 (SD=7.5) while the clinically not improved group demonstrated an average change of 0.67 (SD=4.7).

Anxiety. There was no significant difference in change scores for anxiety between the clinically improved and clinically not improved groups (p=0.113). The mean change in scores on the STAIS for the clinically improved group was –0.35 (SD=5.2) while the clinically not improved group demonstrated an average change of –3.0 (SD=4.1).

Social Function. There was no significant difference in change scores for social function between the clinically improved and clinically not improved groups (p=0.385). The mean change in social function scores for the clinically improved group was –26.6 (SD=28.8) while the clinically not improved group demonstrated an average change of –16.7 (SD=28.6).

General Health. There was no significant difference in change scores for self reported general health between the clinically improved and clinically not improved groups (p=0.392). The mean change in general health scores for the clinically improved group was 1.47 (SD=22.9) while the clinically not improved group demonstrated an average change of 9.88 (SD=22.93).

Table 5.4 provides a summary of these results.

Table 5.4 Differences in long-term change scores for clinically improved and clinically not improved groups for asses/advise/wait subjects. See figure 4.1 for key.

Variable	Improved (n=23) Average Change (SD)	Not Improved (n=9) Average Change (SD)	*p* value
VAS pain	2.8 (2.8)	1.7 (2.4)	0.284
Physical Function	-22.6 (18.6)	-1.1 (22.2)	**0.009**
MZSRDS	1.26 (12.6)	-4.4 (10.2)	0.138
MSPQ	0.52 (7.5)	0.67 (4.7)	0.958
STAIS	0.35 (5.2)	-3.0 (4.1)	0.113
Social Function	-26.6 (28.8)	-16.7 (28.6)	0.385
General Health	1.47 (22.9)	9.9 (22.9)	0.392

Regression

As only one variable was significantly related only the univariate analysis was used. The regression analysis included change in physical function score as an independent variable. This analysis showed that change in physical function accounted for 21% of the variance in disability.

Total Cohort

At long-term follow up forty-eight subjects demonstrated clinically meaningful changes in disability, while fifteen subjects remained unchanged.

Pain. There was a significant difference in change scores for pain between the clinically improved and clinically not improved groups (p=0.001). The mean change in pain for the clinically improved group was 3.44 (SD=2.7) and 0.73 (SD=2.4) for the clinically not improved group.

Physical Function. There was a significant difference in change scores for physical function between the clinically improved and clinically not improved groups (p=0.007). The mean change in physical function for the clinically improved group was –25.22

(SD=29.5) while the clinically not improved group demonstrated an average change of only –5.36 (SD=20.14).

Depressive Symptoms. There was a significant difference in change scores for depressive symptoms between the clinically improved and clinically not improved groups (p=0.035). The mean change in scores on the MZSRDS for the clinically improved group was 3.77 (SD=11.63) while the clinically not improved group demonstrated an average change of –3.13 (SD=10.09).

Distress. There was no significant difference in change scores for the MSPQ between the clinically improved and clinically not improved groups (p=0.174). The mean change in scores on the MSPQ for the clinically improved group was 2.27 (SD=6.22) while the clinically not improved group demonstrated an average change of 0.00 (SD=5.25).

Anxiety. There was a significant difference in change scores for anxiety between the clinically improved and clinically not improved groups (p=0.018). The mean change in scores on the STAIS for the clinically improved group was 1.52 (SD=4.99) while the clinically not improved group demonstrated an average change of –1.85 (SD=3.99).

Social Function. There was a significant difference in change scores for social function between the clinically improved and clinically not improved groups (p=0.020). The mean change in social function scores for the clinically improved group was –33.3 (SD=23.40) while the clinically not improved group demonstrated an average change of –13.39 (SD=26.61).

General Health. There was a significant difference in change scores for self reported general health between the clinically improved and clinically not improved groups (p=0.022). The mean change in general health scores for the clinically improved group was –9.54 (SD=24.5) while the clinically not improved group demonstrated an average change of 6.9 (SD=19.48).

Table 5.5 provides a summary of these results.

Table 5.5 Differences in change scores for clinically improved and clinically not improved groups at long-term follow up for all subjects. See figure 4.1 for key

Variable	Improved (n=48) Average Change (SD)	Not Improved (n=15) Average Change (SD)	*p* value
VAS pain	3.44 (2.7)	0.73 (2.4)	**0.001**
Physical Function	-25.22 (29.5)	-5.36 (20.1)	**0.007**
MZSRDS	3.77 (11.6)	-3.13 (10.1)	**0.025**
MSPQ	2.27 (6.2)	0.00 (5.3)	0.174
STAIS	1.52 (5.0)	-1.85 (4.0)	**0.018**
Social Function	-33.3 (23.4)	-13.39 (26.6)	**0.020**
General Health	-9.54 (24.5)	6.9 (19.48)	**0.022**

Regression

The results from the univariate analysis demonstrate that individually physical function explains 8% of the variance in disability change score, social function 8%, anxiety 8%, depressive symptoms 7%, pain 17% and general health 9%.

Stepwise regression analyses of the results at long term follow up included change scores for pain, physical function, depressive symptoms, anxiety, social function and general health as independent variables. This analysis showed that only changes in physical function and social function still contributed significantly to explaining the variance in disability. Physical function accounted for 14% of the variance and social function 8%. Pain, depressive symptoms, anxiety and general health were all excluded from the model

DISCUSSION

SHORT-TERM OUTCOME

The results of these analyses provide insight into how favourable outcome is achieved. A notable finding is that there appears to be a difference in the mechanism of action between the two groups. The only variable that explains meaningful change in disability for the advice group is change in pain, while both change in pain and physical function are significantly related to change in disability for the physiotherapy group. The results of the regression analyses further confirm this difference, as only change in physical function was included in the multivariate model to explain the variance in disability for the physiotherapy group and only change in pain for the advice group. This implies that the mechanism of action of the physiotherapy treatment model was not just through advice. Improvement in outcome was also achieved by involvement in an active physical treatment that explicitly sought to improve physical function.

The importance of change in pain in explaining short-term outcome in chronic low back pain patients noted by Woby et al (2001) is also reflected in the results of the current investigation. It appears to be a significant determinant of outcome in both groups of acute low back pain patients. This suggests that regardless of the treatment protocol and regardless of chronicity, changing pain is an important determinant of outcome. Recent trends in the management of low back pain have suggested a move away from an emphasis on pain towards models of care that stress the importance of improving function (for e.g. Klaber-Moffet et al 1999, Maher 2000a, Indahl et al 1995 see also Chapter Three). These findings do not wholly support this view. Both improvements in function and improvements in pain are important. The challenge is to develop interventions that effectively target both pain and function.

It is also noteworthy that none of the psychological variables were significantly related to good (or bad) outcome. There was a strong emphasis in the treatment model on identifying and addressing the psychological aspects of the patient's presentation.

While this was effective in decreasing short-term depression and anxiety, this is unlikely to be the mechanism by which good functional outcome was achieved. Whether the intervention is advice to stay active or active physiotherapy, changes in psychological variables are not strongly related to changes in patient-centred functional outcome. This is contrary to the findings on chronic low back pain (Woby et al 2001). In this study changes in psychological variables were significantly related to improvements in disability. It is important for clinicians working in back pain to recognise that good short-term outcome appears to be mediated differently in acute and chronic patients. Clinical implications drawn from the literature on one patient group might not be applicable to the other group. A large part of the biological review used to develop this treatment model was based on research from chronic low back pain patients. These findings suggest that this might not be the optimal source of information to develop a treatment model for acute low back pain. It is important that future research begins to target acute low back pain expressly to answer questions and provide information specific to this group.

This lack of complexity in explaining short-term outcome was an unexpected finding. It is particularly notable as the base line characteristics of the cohort indicated an appreciable level of psychological dysfunction. The time scale over which psychosocial factors affect outcome would seem to be greater than 6-weeks. It seems good functional short-term outcome in acute low back pain is predominantly a 'biological' entity. This is not a feature of chronic low back pain (Woby et al 2001), nor is it a feature of long-term outcome. This is particularly interesting given the findings presented in Chapter Four. Here it was shown that improvements in psychological variables were achievable only if the treatment was delivered early. So while psychology does not seem to be affecting outcome in the short term, it needs to be addressed at this stage, as it is not easily amenable to intervention that is delivered later.

LONG-TERM OUTCOME

The determinants of good long-term outcome were investigated for the total cohort as well as for early and late intervention separately. This produced three sets of long-term

results. There is substantial dissimilarity in the results from these three analyses. The disparities in these findings present a difficulty in determining what is associated with good long-term outcome. There are a number of arguments that suggest the long-term analysis from the early intervention group best represents the determinants of long-term outcome and provides the most useful data set for suggesting modifications to the treatment model. These are

1.Long-term outcome includes data collected at 3 months from baseline. For patients who waited to receive treatment this time point is too soon after active treatment commenced to be considered long-term outcome. The results may have simply captured another short-term outcome effect.
2.Twenty patients in the assess/advise/wait group did not participate in the physiotherapy treatment model. Though all subjects were encouraged to return for physiotherapy treatment a substantial number did not re-attend for treatment at 6 weeks.
3.The results from Chapter Four already suggest that the model should be delivered early. As the primary aim of this chapter is to investigate how physiotherapy treatment can be improved and optimised it seems most appropriate that the early intervention data set is used.

The first point is reinforced by review of the results of the long-term analysis by group. Only physical function explains changes in disability in the group in which treatment is delayed. This is very similar to the results of the early physiotherapy group at short-term follow up. It appears that the results have simply captured another short-term outcome effect. It is worth pointing out that these results offer further support for the effectiveness of the treatment model. It is only after involvement in an active physical treatment approach that change in physical function begins to affect outcome in the late intervention group. The results further emphasise the importance of improving physical activity in determining good outcome.

The discussion will centre on the long-term results from the early intervention group, and in theorising about modifications to the treatment model only results from the early intervention group will be considered.

The most striking aspect of the results on long-term follow up is how different they are from the short-term results. These findings provide strong evidence that long-term outcome is mediated differently to short-term outcome. Good long-term outcome is influenced by many more factors. Pain, distress, social function and general health are all significantly related to good outcome. Furthermore the results of the regression analysis show that pain rather than improvement in physical function is the most important determinant of disability.

It is particularly intriguing that long-term change in physical function does not relate to good disability outcome. The two scales explore similar dimensions though the RMDQ asks specifically about back pain, while the physical function sub-scale of the SF36 relates level of physical function to general health. It may be that as time passes patients make clearer distinctions between their back pain and their general health. However the results from the general health scale do not back this up. Self perceived general health and back pain related disability appear to be more closely associated at long term follow up than at short-term follow up.

Another explanation may relate to the contents of the RMDQ. While most of the items relate to physical function, a number of questions are less clearly activity related. Particularly questions relating to getting other people to do things, help with dressing, the constancy of the pain, sleep, appetite and irritability and bad temperedness are outside the purely physical domain. It may be that at different time points the factors that make up a particular disability score are different. It would be an interesting area of further research to investigate how the disability scores change on an item-by-item basis and what aspects of the scale are most resilient to change.

The disparity between long and short-term outcome is further emphasised by the relationship between short-term and long-term disability scores. Though they are significantly correlated (p=0.008, r=0.48), a univariate regression analysis showed that only 23% of the variance in long-term disability is explained by short-term changes in disability, less than changes in pain or social function. Furthermore when short-term disability changes are included with the other significantly related variables in the multivariate analysis of long-term outcome, it is excluded from the model.

Mannion et al (2001) evaluated outcome at a comparable time for chronic low back pain patients and found similar results. In their study pain, distress and fear avoidance were the variables most strongly related to favourable outcome. These findings suggest that the way good long-term outcome is achieved is similar for both acute and chronic low back pain.

There was a strong suggestion from both the biological and empirical reviews that single interventions are unlikely to provide long term benefits for patients with acute low back pain. The findings of this chapter support this view and further emphasise the importance of a comprehensive and multifaceted treatment approach. Efforts to make improvements in pain, distress, general health and social function all seem likely to enhance the treatment effect. Furthermore the results of the regression analysis demonstrated quite strong associations for all these variables. Though the multivariate results again emphasised the importance of pain in mediating good outcome.

Given the differences in outcome between groups seen in Chapter Four it would be interesting to know if good outcome is mediated differently with early or late interventions. It is likely that the treatment priorities, emphasis and therapeutic processes would be different between groups as the clinical presentations would be different at the commencement of treatment. Though both groups had similar pain levels at baseline the results from Chapter Four show that the average pain levels and amount of disability at the commencement of active treatment would have been significantly lower for the assess/advise/wait group. Pain relief might be the clinical priority in the early intervention group, while the late intervention group would have more emphasis on improving function and physical activity. Results from longer-term follow up would be needed to confidently answer this question.

Finally, it is important to note that none of the regression models explained a large part of the variance in outcome. Though a number of outcome variables explaining a number of different factors were used, the primary reasons for good outcome were not identifiable from this data set.

STUDY LIMITATIONS

Statistical Issues

The methodology of allocating subjects into changed and not changed groups certainly decreases the amount of variance available for investigation. It would have been possible to use raw RMDQ change scores in a linear regression and investigate how much of the variance in raw mean difference scores are explained by changes in the other variables. This method was rejected for two reasons. Firstly, the method used enabled ease of comparison with the results of Woby et al (2001). A particularly interesting comparison, as the subjects in this study were chronic low back pain patients being managed by physiotherapists in a UK hospital. Secondly, it was felt that it was important to look at what explained clinically meaningful change in disability not simply the variance in raw difference scores.

The exploratory nature of the study prompted the decision to use 2-tailed *t*-tests when testing for group differences. Future confirmatory studies may wish to use 1-tailed tests, particularly as some between group differences approached significance.

It would also have been possible to use hierarchical regression to explore the strength of association between variables. As was mention above, stepwise regression uses statistical criteria to determining order of entry into the model, so probably represent a less biased and more robust method for model building (Tabachnik and Fidell 1996). Hierarchical analysis might serve as an interesting next step in gaining further insight into the interaction of factors that effect good outcome.

Generalisability

It is not known if these findings are applicable to other groups with acute low back pain. The fact that pain is consistently related to good outcome in this cohort and other low back pain groups suggest that this cohort is representative of other populations. However, the strong relationship of general health and social function to good long term outcome may be a reflection of local demographics, and may prove to be less important in other samples.

Finally the study is limited in that only a moderate amount of the variance in RMDQ score could be explained from the available data. A large proportion of what explains good outcome in acute low back pain patients was not identifiable from the set of outcome measures used in this study.

CHANGING THE MODEL

Change in pain was the one variable significantly related to good outcome at both short and long term follow up. This finding is in agreement with the work of Woby et al (2001) and Mannion et al (2001) on chronic low back pain patients. The results presented in Chapter Four indicated that the physiotherapy treatment model did not affect pain any more than advice to stay active. The importance of pain in determining outcome and the failure of the treatment model to significantly change pain provide strong evidence for modification to the treatment model. There needs to be a greater emphasis on pain reduction.

Two randomised controlled trials on the management of acute low back pain provide some insight into possible modifications to the treatment model. Blomberg et al (1992) used steroid injection of locally painful lumbo-pelvic soft tissues as an adjunct to a treatment model that was similar to the one presented here. The injections were generally only administered to those patients who had failed to obtain significant pain relief from the package of specific exercises, manual therapy and traction. They reported improvements in short-term pain relief that were significantly greater than that of the control group (electrotherapy, massage and general exercises). Assuming that the results of this control intervention are no worse than the advice to stay active group in the present study, these findings suggest that steroid injections can lead to improvements in short-term pain relief. Injection therapy is now within the scope of practice of physiotherapy (CSP 2001) and may be a useful addition to the current treatment model.

Stankovic and Johnell (1990) reported a significant difference in short-term pain relief between McKenzie treatment and advice. In the McKenzie approach treatment content is strongly influenced by determining which direction(s) of movement decrease and centralise pain (McKenzie 1981). The clinical implications drawn from this trial were more related to the individualisation of treatment rather than strictly applying the concepts of centralisation (McKenzie 1981). It is possible that the benefits of this treatment model could be enhanced if more emphasis were placed on determining the effects of repeated movements on pain intensity and distribution and integrating this information into the treatment plan. This approach has the added benefit of fitting easily within the active philosophy of the model.

The discussion in Chapter Four also touched on the issue of how to better affect pain. Here the suggestion was made that the functional emphasis of the treatment model may have been less than optimal for the treatment of pain. The results of the regression analyses provide an interesting perspective on this view. At short-term follow up improvement in physical function was found to be more strongly associated with good outcome than changes in pain, in fact change in pain was excluded from the multivariate model. These findings support the importance of emphasising activity and function. However in the long term change in pain is overwhelmingly more important in determining good outcome than changes in physical function. Obviously both pain and function need to be targeted for good outcome. An emphasis on functional restoration still should be a central part of the model but the full benefit of the treatment model might not be realised if exit from treatment is functionally determined. An interesting study would be to see if a pain dependant exit or a functional dependant exit offer meaningfully different outcomes. In the absence of definitive data, a reasonable modification to the treatment model would be to include both functional and pain related goals and milestones.

It should not be overlooked that the main factor determining good short-term outcome was improvement in physical function. Promoting physical activity and improving physical function were fundamental to the treatment model. This was obviously not achieved in all subjects. Broadly speaking this can occur for two main reasons. Firstly the treatment model may have been adequate but the incorrect treatment decision was

made for a particular patient. Secondly the treatment model was not adequate to address the individual's reason for problems with physical function.

To address the first issue, a review process should be part of the treatment model. This was included in the treatment algorithm of the Blomberg et al (1992) study and formalised follow up appointments were a common feature of those clinical trials demonstrating benefit (Chapter Three). The current treatment model integrated this information by permitting the treatment of recurrences. In retrospect this probably served as an inadequate review process as only patients who had had a successful outcome the first time would be likely to re-present. The review process needs to be concurrent with treatment to ensure that it is inclusive of all patients.

The second reason presents more of a problem to address. One study provides some information on how chronic low back pain patients may be encouraged to participate in physical activity. Keen et al (1999) conducted a qualitative analysis of low back pain subjects involved in an exercise trial. The study found that to encourage subjects to take up physical activity, issues of fear of pain and avoidance of physical activity need to be addressed. Fear avoidance was not strongly associated with the development of chronicity in acute low back pain patients (Pincus et al 2002), so was not a strong feature of the current model. A stronger emphasis on fear avoidance might be a useful addition to the model. The results from this chapter also suggest that more attention to resolving pain might be a valuable solution. Intervention aimed at fear of pain and activity avoidance is likely to be more useful when combined with effective pain relieving measures.

Another reason is that inadequate support was provided for subjects to participate in physical activity. The results from a recent unpublished audit in the department where the study was performed indicated that patients felt that there were insufficient local facilities for the performance of exercise. A pilot scheme whereby patients are provided with an exercise programme and a 'membership' to the physiotherapy department gym has proved successful in increasing patient satisfaction and improving outcomes. The social environment and the level of social deprivation present in the area where the study was conducted were not considered adequately in the development of the model.

Improvement in access to environments were patients might be able to exercise should be part of a comprehensive model of care for low back pain.

This could also be thought of as an issue of patient compliance. Studies have shown that patient compliance is an important determinant of outcome (Hartigan et al 2000 McAuley et al 1993). No attempts were made to monitor compliance or explore possible reasons for non-compliance. It may be that the current treatment model would benefit from more explicitly exploring reasons that patients feel that they are unable to participate in exercise and working towards solving this aspect of the problem. Furthermore, Schneiders et al (1998) demonstrated that exercise compliance rates in low back pain patients could be significantly enhanced by the provision of written instructions and illustrations. This may also serve as a useful adjunct to the treatment model.

Successful long-term outcome was associated with changes in pain, distress, social function and general health. Regression analyses confirm that pain is the most important determinant of good outcome. The modifications suggested above are all relevant to long-term outcome, but one other issue deserves exploration. Long-term improvements in pain may entail equipping patients with the means to manage their pain beyond the end of formal treatment. Exercises based on the McKenzie approach are designed to have this effect, and the long-term results of Stankovic and Johnell (1995) support this view. This provides further evidence for the inclusion of a Mckenzie based form of exercise prescription and education about how to self manage pain.

Distress was measured using the MSPQ. This questionnaire is thought to evaluate awareness of bodily symptoms and function (Main and Waddell 1998). Main and Waddell (1998) outline a treatment approach for distress in low back pain patients. The three main aspects of their approach are understanding, reassurance and support. The understanding and reassurance aspects of their methods were firmly integrated into the treatment model. While support was also offered by the treating therapist it may be that peer support and support beyond the end of formal treatment might more effectively manage distress. The modification mentioned above to involve patients in group exercise classes and enrolment in the department gym may fulfil this role. A further

advantage is that it is likely that enrolment in maintenance exercise classes or long-term participation in physical activity could have favourable effect on social function and general health as well.

This chapter has provided evidence as to how good outcome was achieved in patients undergoing physiotherapy treatment. An important use for this information is to change and refine the treatment model developed in Chapter Three.

The analysis revealed that both improvements in physical activity and reduction in pain were significantly related to short-term improvements in disability. The regression analysis confirmed that enhancement of physical function was the most important feature. To ensure maximisation of improvements in physical function the following modifications to the treatment model are suggested

- The inclusion of a formalised revision process concurrent with the treatment model to ensure treatment goals are being achieved
- Monitoring of patient compliance with the activity and exercise aspects of the model
- Attempts to identify and address reasons for non compliance
- Provision of written information on any prescribed therapeutic exercises
- Attempts to address fear of pain and avoidance of physical activities.
- Provision of a suitable and free environment for participation in exercise outside of formal physiotherapy sessions.

The treatment model would also benefit from more effective measures to deal with pain. It is suggested that these include

- Provision of injection therapy for appropriately selected patients (Blomberg et al 1992).
- Closer attention to pain modification when prescribing exercises and education regarding the self-management of pain (McKenzie 1981).
- Exit from the treatment should be determined by both pain and function

The mediators of long-term outcome are more diverse. Attention to pain relief remains a key determinant to achieving long-term success. It may be that involvement in group exercise classes and use of the department gym after the end of formal treatment will

have favourable effects on social function and general health as well as providing peer support to help deal with distress.

CONCLUSION

This chapter highlighted the ways in which good short and long-term outcome are mediated. It is noteworthy that physiotherapy has a different mode of action to advice on staying active and that good short-term outcome is moderated quite differently to good long-term outcome.

Elucidation of the factors that promote successful outcome in subjects undergoing physiotherapy treatment enabled a number of modifications to be made to the treatment model. These include primarily improvements in pain management strategies and tactics to enhance physical activity. It would appear that it is also important to take steps to enhance social support and general health beyond the end of formal treatment.

A number of areas of further research are suggested by these findings. Firstly, there needs to be greater investigation into how to significantly effect pain in acute low back pain. Consideration needs to be given to both understanding more about why pain is present as well as investigating the mechanism of action and efficacy of various treatments.

Exercise classes have been shown to be effective in the management of sub-acute low back pain (Klaber-Moffat et al 1999, Torstensen et al 1998). This study suggests that they could also fulfil an important function beyond the end of an individualised treatment programme. It may be that a period of individualised treatment followed by a exercise class represents an optimal solution to low back pain management, and is certainly a protocol that is worthy of further study.

CHAPTER SIX

Conclusions and Suggestions for Future Research

In the introduction to this thesis an outline was given of the discrepancies that exist in various national guidelines on the management of acute low back. Despite the existence of a common evidence base, expert opinion is divided on the role physiotherapy has in the acute management of low back pain (Koes et al 2001). A major aim of this thesis was to try and help resolve the discrepancies that exist between guidelines through the development and testing of a comprehensive model of care for acute low back pain.

A deliberately comprehensive approach was taken in development of the model. Information was drawn from epidemiological data, basic science pertaining to low back pain, the empirical literature on management of the problem as well as the relevant philosophical aspects of the physiotherapy profession. While the empirical and epidemiological data on acute low back pain is quite extensive, the scientific literature on low back pain is dominated by work on chronic low back pain patients. There certainly exists a need for good research on the mechanisms underpinning the acute low back pain experience.

There is a good deal of empirical research available on the physiotherapy management of acute low back pain. Unfortunately, little of this research addresses the major pitfalls associated with studying low back pain (Foster 1999), or reflects the philosophical framework of physiotherapy. The quality of research into low back pain is certainly improving (Maher 2000b), but there is further need for good quality research that more closely attends to the unique features of simple low back pain. One major gap in the evidence base on the management of acute low back pain, relates to the dose of intervention. While this has received some attention in the data on chronic low back

pain (Manniche et al 1990), there was little information about dose available to guide development of the treatment model. It should be seen as a matter of some priority in physiotherapy to attend to this important issue in future research.

The heterogeneity of simple low back pain is often considered a major barrier to the development of effective algorithms of care (Foster 1999). The literature is replete with suggestions for sub-group classifications within simple low back pain (Riddle 1998, Peterson et al 1999). The reliability and validity of these classification systems is less than ideal (Riddle 1999, Peterson et al 1999) and none have demonstrated clinical utility. Moreover, it is the author's opinion that the classification systems have been developed more as an extension of the clinicians' biases and preconceptions rather than on fundamental differences in low back pain presentations.

The development of meaningful and useful classification systems is certainly an imperative, but this process needs to be guided by good science. A useful starting point may be to closely scrutinise the clinical reasoning process employed by therapists managing actual patients. The clinical reasoning process is essentially one of classification and the results of this and other trials demonstrate that something useful is happening when expert clinicians clinically reason (Koes et al 1992, Blomberg et al 1992, Levson et al 2001). The comprehensive clinical reasoning document used in this study would serve as a good starting place for this process. Added value may be obtained by relating aspects of the clinical reasoning process to good or bad outcome.

An alternative approach to the development of a classification system would be to utilise factor analysis of clinical parameters. Insufficient attention has been paid to systematically observing patterns of clinical presentation. Interrogation of the successful clinical reasoning process may identify what the important clinical parameters are. The next step would be to see if patterns emerge from unbiased observations of the variants of these parameters.

The treatment model developed attempted to offer as complete a package of physiotherapy treatment as possible. While individual elements of the model had been tested, no studies have evaluated so comprehensive a multimodal treatment algorithm.

Furthermore, the model was unique in its explicit use of a clinical reasoning process to guide the individualisation of treatment.

The efficacy of the model was tested in a randomised controlled trial. Evaluation of outcome at six weeks enabled comparison to be made between active physiotherapy and advice to stay active. At this time point the results demonstrated significant benefit of the treatment model over advice. Significant differences were noted in disability, mood, general health and well-being.

Evaluation of long-term outcome enabled comparison to be made between early and late application of the treatment model. At this time point the results favoured the early intervention group. While the physical aspects of the low back pain experience did not seem to be adversely affected by delaying intervention, a number of psychosocial parameters were significantly better in the early intervention group. Delaying the onset of treatment did not provide the opportunity for physiotherapy to favourably affect the psychosocial variables.

These findings suggest two primary areas for further study. Firstly, the effect sizes seen in this study are quite large and the treatment effects certainly more long lived than many other investigations into the management of acute low back pain. Further exploration of the individual treatments used and the clinical reasoning process employed may offer some interesting insight into these findings. Secondly, the difference in treatment response of the psychosocial variables to early and late intervention is a particularly fascinating finding and the process by which this was mediated would make for interesting further analysis.

Physiotherapy can also be looked at in the broader context of primary care management of acute low back pain. The two groups in the study represent two different models of care for acute low back pain. In one approach subjects are assessed, given advice and treated promptly (assess/advise/treat). In the other group treatment is delayed (assess/advise/wait). The overall study results support an assess/advise/treat model of care due to the superior short and long term outcomes. Furthermore, it is recognised that psychosocial variables are predictive of chronicity in low back pain (Linton 2000). Early physiotherapy treatment may therefore reduce the risk of chronic problems

developing. A priority of further research should be to explore the long-term consequences of this difference in psychosocial outcomes.

Closer scrutiny of the data set provided information to help refine the treatment model. Changing physical function and pain are the things that lead to good short-term outcome, while changes in pain, distress, social function and self perceived general health lead to good long-term outcome. A number of modifications were suggested to help facilitate improvement in these parameters and possibly increase the treatment models effectiveness.

The priority for future research from this study should be to formally test the refinements suggested in Chapter Five to evaluate their worth. Also, this study particularly highlighted the necessity for greater consideration to be given to the understanding and management of pain in low back pain patients.

This cyclical process of developing, testing and refining treatment of acute low back pain needs to continue. This thesis suggests that physiotherapy does have an important role to play in the management of acute low back pain, but this represents only the start. It is important that clinical questions continue to be addressed to move management of low back pain from, simply, **effective** towards identifying what is **optimal**. This process should not be based on serendipity, clinical fads or the desire to prove a pet theory. It needs to be driven by a logical, structured and informed approach that serves the needs of patients. This thesis provides a model of how that may be achieved.

REFERENCES

Abenhaim L, Rossignol M, Gobeille D, Bonvalot Y, Fines P, Scott S. The prognostic consequences in the making of the initial medical diagnosis of work-related back injuries. Spine. 1995; 20: 791-795

Abenhaim L, Rossignol M, Valat J-P, et al. The role of activity in the therapeutic management of back pain. Report of the International Paris Task Force on Back Pain. Spine 2000; 25 (Suppl4S): 1S-33S

Abenhaim L, Suissa S. Importance and economic burden of occupational back pain: A study of 2500 cases representative of Quebec. J Occup Med 1987; 29: 670-4

Accident Rehabilitation and Compensation Insurance Corporation and the National Health Committee. New Zealand acute low back pain guide. Wellington, NZ: Ministry of Health, 1997:1-13.

Adams M and Dolan P. Recent advances in lumbar spinal mechanics and their clinical significance. Clinical Biomechanics. 1995;10(1):3-19.

Adams MA, Freeman BJ, Morrison HP, et al. Mechanical initiation of intervertebral disc degeneration. Spine 2000; 25: 1625-36.

Adams MA, Mannion AF, Dolan P. Personal risk factors for first-time low back pain. Spine 1999; 24: 2497-505

Ahern DK, Follick MJ, Council JR, et al. Comparison of lumbar paravertebral EMG patterns in chronic low back pain patients and nonpatient controls. Pain 1988; 34: 153-60

Ahern DK, Hannon DJ, Goreczny AJ, et al. Correlation of chronic low back pain behavior and muscle function examination of the flexion-relaxation response. Spine 1990; 15: 92-5.

Alaranta H, Tallroth K, Soukka A, et al. Fat content of lumbar muscles and low back disability: a radiographic and clinical comparison. J Spinal Disord 1993; 6: 137-40

Altman DG. Practical statistics for Medical Research. Chapman and Hall, London: 1991.

American Physical Thrapy Association. Guide to physical therapy practice. Physical Therapy. 2001; 81(1).

Anderson RT, Aaronson NK, Wilkin D Critical review of the international assessments of health-related quality of life. Quality of Life Research, 1993. 2, 369-395.

Anderson RT. An Orthopedic Ethnography in Rural Nepal. Medical Anthropology 1984;Winter:46-59.

Andersson GB. Deyo RA. History and physical examination in patients with herniated lumbar discs. Spine. 1996. 21(24 Suppl):10S-18S, Dec 15.

Andersson G and Weinstein J. Introduction: Health outcomes related to low back pain. Spine. 1994, 19: 2026S-2027S

Aoki Y, Rydevik B, Kikuchi S et al. Local application of Disc- Related Cytokines on Spinal Nerve Roots. Spine 2002: August 1:27(15): 1614-1617.

Aprill C, Bogduk N. High-intensity zone: a diagnostic sign of painful lumbar disc on magnetic resonance imaging. Br J Radiol 1992; 65: 361-9

Arendt-Nielsen L, Graven- Nielsen T, Svarrer H et al. The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. Pain 1995; 64: 231- 240.

Axelsson P, Johnsson R, Stromquist B. Is there increased intervertebral Mobility in Isthmic Adult Spondylolisthesis? Spine 2000 July: 25 (13) 1701-1703

Balague F. Skovron M. Nordin M. Dutoit G. Pol LR. Waldburger M. Low back pain in school children: a study of familial and psychological factors. Spine, 1995 20(11):1265-70.

Battie MC, Cherkin DC, Dunn R, et al. Managing low back pain: attitudes and treatment preferences of physical therapists. Phys Ther 1994; 74: 219-26

Bayramoglu M, Akman M, Kilinc S et al. Isokinetic measurement of trunk muscles strength in women with chronic low back pain. American Journal of Medicine and Rehabilitation 2001. Volume 80,no 9: 650-655.

Beattie P, Maher C. The role of functional status questionnaires for low back pain. Australian Journal of Physiotherapy, 1997, 43, 29-38.

Beattie PF.Meyers SP. Magnetic Resonance Imaging in Low Back Pain: General Principles and Clinical Issues. Physical Therapy, 1998;7:738-53.

Beckerman H, Bouter LM, Van Der Heijden GJMG, De Bie RA, Koes BW. Efficacy of physiotherapy for musculoskeletal disorders: what can we learn from research? British Journal of General Practice, 1993,43, 73-77.

Bergenudd H. Nilsson B. Back pain in middle age; occupational workload and psychologic factors: an epidemiologic survey Spine. 1988, 13(1):58-60.

Bergenudd H. Nilsson B. The prevalence of locomotor complaints in middle age and their relationship to health and socioeconomic factors. Clinical Orthopaedics & Related Research, 1994, 308:264-70.

Bergner M. Bobbitt RA. Carter WB. Gilson BS. The Sickness Impact Profile: development and final revision of a health status measure. Medical Care. 1981,19(8):787-805.

Bergner M. Bobbitt RA. Kressel S. Pollard WE. Gilson BS. Morris JR. The sickness impact profile: conceptual formulation and methodology for the development of a health status measure International Journal of Health Services. 1976a, 6(3):393-415.

Bergner M. Bobbitt RA. Pollard WE. Martin DP. Gilson BS. The sickness impact profile: validation of a health status measure. Medical Care. 1976b, 14(1): 57-67.

Beurskens AJHM, de Vet HCW, Koke AJA. Responsiveness of functional status in low back pain: a comparison of different instruments. Pain. 1996; 65:71-6.

Biedermann HJ, Shanks GL, Forrest WJ, et al. Power spectrum analyses of electromyographic activity discriminators in the differential assessment of patients with chronic low back pain. Spine. 1991;16:1179-1185.

Bigg-Wither G and Kelly P. Diagnostic Imaging in Musculoskeletal Physiotherapy In: Refshauge K and Gass E (eds) Musculoskeletal Physiotherapy. Clinical Science and Practice. Oxford: Butterworth-Heinemann 1995.

Bigos S, Bowyer O, Braen G, et al. Acute low back problems in adults. Clinical Practice Guideline No. 14, AHCPR Publication No. 95-0642, Rockville, MD: Agency for Health Care Policy and Research, Public Health Service, US Dept of Health and Human Services 1994:1-160.

Bigos SJ, Battie MC, Fisher LD, Hansson TH, Spengler DM, Nachemson AL. A prospective evaluation of preemployment screening methods for acute industrial back pain. Spine 1992; 17:922-6.

Bigos SJ, Battie MC, Spengler DM, et al. A longitudinal, prospective study of industrial back injury reporting. Clin Orthop Rel Res 1992;279:21-34.

Bigos SJ, Battie MC, Spengler DM, et al. A prospective study of work perceptions and psychosocial factors affecting the report of back injury. Spine 1991; 16:1-6

Billeter, R. & Hoppeler, H. Muscular basis of strength. In Komi, P. (ed) Strength and Power in Sport. Oxford: Blackwell 1992.

Bjordal J.M, Greve G. What may alter the conclusions of reviews? Physical Therapy Reviews. 1998;3:121-132.

Bland J. Mechanisms of Adaptation in the Joint In: Crosbie J and McConnell J (eds) Key Issues in Musculoskeletal Physiotherapy. Oxford: Butterworth-Heinemann. 1993.

Blomberg S, Hallin G, Grann K, Berg E, Sennerby U. Manual therapy with steroid injections - a new approach to treatment of low back pain. A controlled multicentre trial with an evaluation by orthopaedic surgeons. Spine 1994;19:569-577.

Blomberg S, Svardsudd K, Mildenberger F. A controlled, multicentre trial of manual therapy in low back pain: initial status, sick leave and pain score during follow-up. Scand J Prim Care 1992;10:170-178.

Blomberg S, Svardsudd K, Tibblin G. Manual therapy with steroid injections in low-back pain. Scand J Prim Health Care 1993;11:83-90

Bobbert S and van Soest J. Effects of muscle strengthening on vertical jump height: a simulation study. Med Sci Sports and Exerc. 1994;8:1012-20.

Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. J Bone J Surg 1990; 72-A:403-8.

Bogduk N Low Back Pain. Clinical Anatomy of the Lumbar Spine and Sacrum, New York: Churchill Livingstone, 1997.

Bogduk N National Musculoskeletal Health Initiative. Evidence-based practice guidelines for the management of acute low back pain. Newcastle, Australia: University of Newcastle, 1997.

Bogduk N. Draft evidence based clinical guidelines for the management of acute low back pain. National Health and Medical Research Council, Australia, 2000

Bogduk N. The sources of low back pain. In: Jayson MIV, ed. The Lumbar Spine and Back Pain. Edinburgh: Churchill Livingstone, 1992: 61-88.

Bogduk, N. and Mercer, S. Selection and application of treatment. In Refshauge, K. and Gass, E. (eds) Musculoskeletal Physiotherapy. Clinical Science and Practice. Butterworth Heinemann: Oxford. 1995.

Bolton JE. Accuracy of recall of usual pain intensity. Pain 1999; 83: 533-539.

Bombardier C, Hayden J, Beaton DE. Minimal clinically important difference. Low back pain: outcome measures. Journal of Rheumatology 2001. Feb;28 (2):431-438.

Bongers PM, de Winter CR, Kompier MA, Hildebrandt VH. Psychosocial factors at work and musculoskeletal disease. Scand J Work Environ Health 1993; 19:297-312

Boos N, Rieder R, Schade V, Spratt KF, Semmer N, Aebi M. 1995 Volvo Award in clinical sciences: the diagnostic accuracy of magnetic resonance imaging, work perception, and psychosocial factors in identifying symptomatic disc herniations. Spine 1995; 20: 2613-25

Boos N, Semmer N, Elferin A et al. Natural History of Individuals with Asymptomatic Disc Abnormalities in Magnetic Resonance Imaging. Spine 2000; 25(12):1484-1492.

Boos N. Lander PH. Clinical efficacy of imaging modalities in the diagnosis of low-back pain disorders. European Spine Journal. 1996.5(1):2-22.

Borkan JM, Cherkin DC. An agenda for primary care research on low back pain. Spine 1996;24:2880-4

Brady S, Mayer T and Gatchel R. Physical progress and residual Impairment Quantification after Functional restoration. Spine 1994; 19,(4):395—400.

Brattberg G, Thorsland M, Wikman A. The prevalence of pain in a general population. The results of a postal survey in a county of Sweden. Pain. 1989;37:215-22.

Bronfort G and Bouter L. Responsiveness of general health status in chronic low back pain: a comparison of the COOP Charts and the SF-36. Pain 1999; 83:201-209.

Bronfort GDC. Chiropractic versus general medical treatment of low-back pain: A small scale controlled clinical trial. Am J Chiropract Med 1989;2:145-50.

Bronfort, G. Spinal Manipulation. Current state of research and its indications. Neuro clin North Am. 1999. 17(1):91-111

Bruton A. Muscle Plasticity. Response to training and detraining. Physiotherapy 2002; 88,7, 398-408.

Buirski G, Silberstein M. The symptomatic lumbar disc in patients with low-back pain: magnetic resonance imaging appearances in both a symptomatic and control population. Spine 1993; 18: 1808-11

Buirski G. Magnetic resonance signal patterns of lumbar discs in patients with low back pain. A prospective study with discographic correlation. Spine 1992; 17:1199-204

Burton AK, Tillotson KM, Troup JDG. Prediction of lowback trouble frequency in a working population. Spine 1989a;14:517-22

Burton AK, Tillotson KM, Troup JDG. Variation in lumbar saggital mobility with low back trouble. Spine, 1989b;14:584-590

Burton AK, Tillotson K, Edwards V et al. Lumbar saggital mobility and low back symptoms in patients treated with manipulation. Journal of Spinal disorders. 1990;3:262-268

Burton AK, Clarke RD, McClune TD, Tillotson KM. The natural history of low back pain in adolescents. Spine 1996;21:2323-8

Burton AK, Tillotson KM, Main CJ, Hollis S. Psychosocial predictors of outcome in acute and subchronic low back trouble. Spine, 1995, 20(6), 722-728.

Burton AK, Tillotson KM. Does leisure sports activity influence lumbar mobility or the risk of low back trouble? J Spinal Disord 1991; 4:329-36.

Burton AK, Tillotson KM. Prediction of the clinical course of low-back trouble using multivariate models. Spine 1991;16:7-13

Burton AK, Waddell G, Tillotson KM, et al. Information and advice to patients with back pain can have a positive effect. A randomized controlled trial of a novel educational booklet in primary care. Spine 1999; 24: 2484-91

Buswell J. Low back pain: a comparison of two treatment programmes. NZ J Physiotherapy 1982;10:13-7

Butler D. Integrating Pain Awareness into Physiotherapy- Wise Action for the Future In: Gifford L (ed) Topical issues in Pain. Falmouth: NOI Press. 1998.

Butler, D. S. Mobilisation of the Nervous System. Melbourne: Churchill Livingstone. 1991.

Byl NN, Sinnott PL. Variations in balance and body sway in middle-aged adults: subjects with healthy backs compared with subjects with low back dyfunction. Spine 1991;16:325-30

Cady LD, Bischoff DP, O'Connell ER. Strength and fitness and subsequent back injuries in fire fighters. J Occup Med 1979;21:269-72

Capodaglio P, Nilsson J, Jurisic DH. Changes in paravertebral EMG spectrum parallel to strength increases after rehabilitation in chronic low back pain patients. Clin Rehab 1995; 9: 354-62

Carlsson AM. Assessment of chronic pain. I. Aspects of the reliability and validity of the visual analogue scale. Pain. 1983,16(1):87-101.

Carlsson C and Nachemson A. Neurophysiology of Back Pain: In Nachemson A, Jonsson E, eds. Neck and Back Pain: The Scientific Evidence for Causes, Diagnosis and Treatment. Philadelphia: Lippincott, Williams and Wilkins, 2000.

Carr J and Shepherd R.Series Editors' Preface In: Crosbie J and McConnell J (eds) Key Issues in Musculoskeletal Physiotherapy. Oxford: Butterworth-Heinemann. 1993.

Carr, J. & Shepherd, R. Movement Science: Foundations for Physical Therapy in Rehabilitation. Aspen: Rockville. 1987.

Carragee E, Chen Y, Cary M et al. Provocative Discography in Patients after Limited Lumbar Disectomy. Spine 2000a;25:3065-3071.

Carragee E, Cary M, Tanner MD et al. The Rates of False Positive Lumbar Discography in Select patients Without Low Back Symptoms. Spine 2000b;25:1373-1381.

Carragee E, Chen Y, Cary M et al. Can discography cause long-term back symptoms in previously asymptomatic subjects? Spine 2000c;25:1803-1808.

Carragee E, Paragioudakis S and Khurana S. Lumbar high Intensity Zone and Discography in Subjects without Low Back Problems. Spine 2000d. December;25(23):2987-2992.

Cassidy JD, Carroll LJ, Cote; P. The Saskatchewan Health and Back Pain Survey: The prevalence of low back pain and related disability in Saskatchewan adults. Spine 1998; 23: 1860-7

Cassisi, J. E. M. E. Robinson, P. O'Connor, and M. Macmillan. Trunk strength and lumbar paraspinal muscle activity during isometric exercise in chronic low back pain patients and controls. Spine 1993.18: 245-251.

Cervero F. Laird JM. From acute to chronic pain: mechanisms and hypotheses. Progress in Brain Research. 1996a, 110:3-15.

Cervero F. Laird JM. Mechanisms of allodynia: interactions between sensitive mechanoreceptors and nociceptors. NeuroReport. 1996b, 7(2):526-8,

Cervero F. Laird JM. Mechanisms of touch-evoked pain (allodynia): a new model. Pain. 1996c, 68(1):13-23.

Charterd Society of Physiotherapy. Standards of Physiotherapy Practice. Chartered Society of Physiotherapists, London. 1993.

Chartered Society of Physiotherapy. Scope of practice of physiotherapy. Information paper No. PA44. 2000.

Cherkin DC, Deyo RA, Battie M, Street J, Barlow W. A comparison of physical therpay, chiropractic manipulation, and provision of an educational booklet for the treatment of patients with low back pain. N Engl J Med 1998;339:1021-9

Cherkin DC, Deyo RA, Loeser JD, et al. An international comparison of back surgery rates. Spine 1994; 19: 1201-6

Cherkin DC, Deyo RA, Street JH, Barlow W. Predicting poor outcomes for back pain seen in primary care using patients' own criteria. Spine. 1996a; 21: 2900-2907

Cherkin DC, Deyo RA, Street JH, Hunt M, Barlow W. Pitfalls of patient education: Limited success of a program for back pain in primary care. Spine 1996b;21:345-55

Cherkin DC, Eisenberg D, Sherman KJ, Barlow W, Kaptchuk TJ, Street J, Deyo RA. Randomized trial comparing traditional Chinese medical acupuncture, therapeutic massage, and self-care education for chronic low back pain. Arch Intern Med 2001;161(8):1081-8

Cherkin DC. Primary care research on low back pain. Spine 1998; 23: 1997-02

Chiou W, Lee Y-H, Chen W-J. Use of surface EMG coactivational pattern for functional evaluation of trunk muscles in subjects with and without low back pain. International Journal of Industrial Ergonomics 1999; 23: 51-60.

Chiou W, Wong M, and Lee Y. international Jouirnal of Nursing Studies. 1994; 31(4), 361-369.

Chok B, Lee R, Latimer J, et al. Endurance training of the trunk extensor muscles in people with subacute pain. Phys Ther 1999; 79: 1032-42

Clauw D, Williams D, Lauerman W et al. Pain Sensitivity as a Correlate of Clinical Status in Individuals with Chronic Low Back Pain. Spine 1999. 24(19):2035-2041.

Clinical Standards Advisory Group. Back Pain: Report of a Clinical Standards Advisory Group Committee on Back Pain. London: Her Majesty's Stationery Office, 1994

Coderre TJ, Katz J, Vaccarino AL, Melzack R. Contribution of central neuroplasticity to pathological pain: Review of clinical and experimental evidence. Pain 1993;52:259-85.

Commerford M.J. and Mottram S.L. Movement and Stability Dysfunction-Contemporary Developments. Manual Therapy 2001;6(1):15-26.

Cooper J E, Tate R B, Yassi A et al. Effect of an early Intervention Program on the Relationship between Subjective Pain and Disability Measures in Nurses with Low Back Injury. Spine 1996;21:2329-2336.

Cooper RG, Stokes MJ, Sweet C, Taylor RJ, Jayson MIV. Increased central drive during fatiguing contractions of the paraspinal muscles in patients with chronic low back pain. Spine 1993;18:610-6

Critchley D and Coutts F. Abdominal Muscle Function in Chronic Low back Pain Patients.Physiotherapy 2002. June,vol 88,no6, 322-331.

Croft P, Papageorgiou A, Ferry S et al. Psychologic Distress and Low Back Pain. Evidence from a prospective study in the general population. Spine 1996. 20(24),2731-2737.

Croft PR, Macfarlane J, Papgeorgiou AC, Thomas E, Silman AJ: Outcome of low back pain in general practice: a prospective study. Br Med J 1998, 316: 1356-1359.

Croft PR, Rigby AS. Socioeconomic influences on back problems in the community in Britain. J Epidemiol Community Health. 1994;48:166-170

Croft, P. Papageorgiou, A Thomas, E. Macfarlane, G. Silman, A. Short-Term Physical Risk Factors for New Episodes of Low Back Pain: Prospective Evidence From the South Manchester Back Pain Study. Spine. 1999. 24(15):1556,

Crosbie, J. Optimization and musculoskeletal physiotherapy. In Crosbie, J. and McConnell, J. (eds) Key Issues In Musculoskeletal Physiotherapy. Oxford: Butterworth Heinemann. 1993.

Curtis, Peter Carey, Timothy S. Evans, Paul Rowane, Michael P. Mills Garrett, Joanne. Jackman, Anne Training Primary Care Physicians to Give Limited Manual Therapy For Low Back Pain: Patient Outcomes. Spine. 2000, 25(22):2954-2961.

Dangaria T and Naesh O. Changes in Cross-Sectional Area of Psoas Major Muscle in Unilateral Sciatica caused by Disc Herniation. Spine 1998;23,8,928-931.

Danneels LA. Coorevits PL. Cools AM. et al. Differences in electromyographic activity in the multifidus muscle and the iliocostalis lumborum between healthy subjects and patients with sub-acute and chronic low back pain. European Spine Journal. 2002,11(1) (pp 13-19).

Danneels LA. Vanderstraeten GG. Cambier DC. et al. CT imaging of trunk muscles in chronic low back pain patients and healthy controls. European Spine Journal. 2000;9(4):266-72.

Danneels LA. Vanderstraeten GG. Cambier DC. et al. Effects of three different training modalities on the cross sectional area of the lumbar multifidus muscle in patients with chronic low back pain. British Journal of Sports Medicine. 2001, 35(3) 186-191.

De Luca CJ. Use of the surface EMG signal for performance evaluation of back muscles. Muscle Nerve 1993; 16: 210-6.

Delitto A. Are measures of function and disability important in low back care? Physical Therapy, 1994,74(5), 452-462.

Delitto A, Cibulka MT, Erhard RE, Bowling RW, Tenhula JA. Evidence for use of an extension-mobilization category in acute low back syndrome: a prescriptive validation pilot study. Phys Ther 1993;73:216-228

Delitto A, Erhard R E, Bowling R W. A treatment-based classification approach to low back syndrome: Identifying and staging patients for conservative treatment. Physical Therapy, 1995, 75 (6) 471-485.

Dettori LCJR, Bullock CSH, Sutlive CTG, et al. The effects of spinal flexion and extension exercises and their associated postures in patients with acute low back pain. Spine 1995; 20:2303-12.

Deyo R, Andersson G, Bombardier C et al. Outcome Measures for Studying Patients with Low Back Pain. Spine 1994;19(18s),2032s-2036s.

Deyo R, Diehll A and Rosenthal M. How many days of bedrest for Acute Low Back Pain? The New England Journal of Medicine 1986;1064-1070.

Deyo R. Low-Back Pain. Scientific American 1998;29-33.1

Deyo RA, Battie M, Beurskens AJHM, et al. Outcome measures for low back pain research: A proposal for standardized use. Spine 1998; 23: 2003-13.

Deyo RA, Phillips WR. Low back pain. A primary care challenge. Spine,1996, 21(24), 2826-2832.

Deyo RA. Rainville J. Kent DL. What can the history and physical examination tell us about low back pain? JAMA. 1992 268(6):760-5.

Dionne CE. Low Back Pain. In Crombie IK, ed. Epidemiology of Pain, Seattle: IASP Press, 1999.

Dolan P. Adams MA, Recent advances in lumbar spinal mechanics and their clinical significance. Clin Biomech 1995;10:3-19

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

Donelson R, Aprill CN, Medcalf R, et al. A prospective study of centralization of lumbar and referred pain: A predictor of symptomatic discs and anular competence. Spine 1997; 22: 1115-22

Donelson RG, McKenzie RA. Mechanical assessment and treatment of spinal pain. In: Frymoyer JW, ed. The Adult Spine, Principles and Practice. 2nd ed. Philadelphia: Lippincott-Raven, 1996: 1821-35.

Doody C and McAteer M. Clinical Reasoning of Expert and Novice Physiotherapists in an Outpatient Setting. Physiotherapy 2002,88,5,258-268.

Dubner R and Basbaum A. Spinal Dorsal Horn Plasticity Following Tissue or Nerve Injury In: Wall P and Melzak R (eds) Textbook of Pain. London: Churchill Livingstone. 1994.

Dvorak J, Panjabi MM, Novotny JE, Chang DG, Grob D. Clinical validation of functional flexion-extension roentgenograms of the lumbar spine. Spine 1991;16:943-50

Ebrall PS. Some anthropometric dimensions of male adolescents with idiopathic low back pain. Journal of Manipulative & Physiological Therapeutics. 1994 ,17(5):296-301.

Ebrall PS. The epidemiology of male adolescent low back pain in a north suburban population of Melbourne, Australia. Journal of Manipulative & Physiological Therapeutics. 1994, 17(7):447-53,

Edmonston S and Elvey R. Physiotherapy Management of Low Back Pain of Mechanical Origin. In: Giles L and Singer K (eds) The Clinical Anatomy and Management of Back Pain. Volume One. Oxford: Butterworth- Heinemann. 1997.

Effective Health Care. Acute and chronic low back pain. York: University of York 2000;6:5.

Ehrmann-Feldman D, Rossignol M, Abenhaim L, Gobeille D. Physician referral to physical therapy in a cohort of workers compensated for low back pain. Physical Therapy, 1996, 76(2), 150-157.

Elliot, B. Hardcastle, P. Burnett, A. and Foster, D. The influence of fast bowling and physical factors on radiological features in high performance young fast bowlers. Sports. Med. Training and Rehab. 1992, 3, 113-130.

Erhard RE, Delitto A, Cibulka MT. Relative effectiveness of an extension program and a combined program of manipulation and flexion and extension exercises in patients with acute low back syndrome. Phys Ther 1994; 74: 1093-100

Esola, M. McClure, P. Fitzgerald, K. Siegler, S. Analysis of lumbar spine and hip motion during forward bending in subjects with and without a history of low back pain. Spine, 1996, 21, 71-78.

Espland A, Baerheim A, Albrektsen. Patients' Views on Importance and Usefulness of Plain Radiography for Low Back Pain. Spine 2001;26(12):1356-1363.

Essink-Bok M-L, Krabbe PFM, Bonsel GJ, Aaronson NK. An empirical comparison of four generic health status measures. The Nottingham Health Profile, the Medical Outcomes Study 36-item Short-Form Health Survey, the COOP/WONCA Charts, and The EuroQol Instrument. Medical Care, 1997,35(5), 522-537.

EuroQol Group, The. EuroQol - a new facility for the measurement of health-related quality of life. Health Policy, 1990,16, 199-208.

Evans C and Oldreive W. A Study to Investigate Whether Golfers with a History of Low Back Pain Show a Reduced Endurance of Transverses Abdominis. The Journal of Manipulative Therapy 2000;8(4):162-174.

Evans C, Gilbert JR, Taylor W, Hildebrand A. A randomized controlled trial of flexion exercises, education, and bed rest for patients with acute low-back pain. Physiotherapy Canada 1987;39:96-101

Evans G, Richards S. Low Back Pain: An Evaluation of Therapeutic Interventions. Bristol (UK): Health Care Evaluation Unit, University of Bristol, 1996.

Faas A, Chavannes A, van Eijk JTM, Gubbels J. A randomised placebo-controlled trial of exercise therapy in patients with acute low back pain. Spine 1993;18:1388-1395.

Faas A, Chavannes AW, Koes BW, van den Hoogen JMM, Mens JMA, Smeele LJM, Romeijnders ACM, van der Laan JR. Practice Guideline 'Low Back Pain'. Dutch College of General Practitioners, 1996.

Faas A, van Eijk JThM, Chavannes AW, Gubbels JW. A randomized trial of exercise therapy in patients with acute low back pain. Spine 1995;20:941-947

Faas A. Exercises: which ones are worth trying, for which patients, and when? Spine 1996; 21: 2874-9

Fairbank JC, Couper J, Davies JB, O'Brien JP. The Oswestry low back pain disability questionaire. Physiotherapy 1980;66:271-273

Farrell JP, Twomey LT. Acute low-back pain: Comparison of two conservative treatment approaches. Med J Aust 1982;1:160-4.

Feder G and Hemingway H. Bad backs, good policy? British Journal of General Practice.1995;456-57.

Flicker P , Fleckenstein J, Ferry K et al. Lumbar Muscle Usage in Chronic Low Back Pain. Magnetic Resonance Image Evaluation. Spine 1993;18(5),582-586.

Flor H. Braun C. Elbert T. Birbaumer N. Extensive reorganization of primary somatosensory cortex in chronic back pain patients. Neuroscience Letters. 224(1):5-8, 1997 Mar 7.

Fordyce W, Brockway J, Bergman J, Spengler D. Acute back pain: a control-group comparison of behavioural vs traditional management methods. J of Behav Med 1986;9:127-140.

Fordyce WE (ed). Back pain in the workplace: Management of disability in non-specific conditions. Seattle: IASP Press, 1995:1-75.

Foster N. Researching low back pain? An overview of the pitfalls. Physical Therapy Reviews 1998;3:9-17.

Foster NE, Thompson KA, Baxter GD, et al. Management of nonspecific low back pain by physiotherapists in Britain and Ireland: A descriptive questionnaire of current clinical practice. Spine 1999; 24: 1332-42

Foster, D. John, D. Elliott, B. Ackland, T. and Fiche, K. Back injuries to fast bowlers in cricket: A prospective study. British Journal of Sports Medicine, 1989,23, 150-154.

Frank AO, De Souza LH, McAuley JH, Sharma V, Main CJ. A cross-sectional survey of the clinical and psychological features of low back pain and consequent work handicap: use of the Quebec Task Force Classification. International Journal of Clinical Practice 2000;54:639-44.

Frank JW, Brooker AS, Demaio S, et al. Disability resulting from occupational low back pain. Part I. What do we know about primary prevention? A review of the scientific evidence on prevention before disability begins. Spine 1996a; 21:2908-17

Frank JW, Brooker AS, DeMaio SE, et al. Disability resulting from occupational LBP: Part II. What do we know about secondary prevention? A review of the scientific evidence on prevention after disability begins. Spine 1996b;21:2918-29

Fransen M, Woodward M, Norton R et al. Risk factors Associated With the Transition from Acute to Chronic Occupational Back Pain. Spine 2002;27(1):92-98.

Fritz J, and Stevens M. The use of a classification approach to identify subgroups of patients with acute low back pain: Interrater reliability and short-term treatment outcomes. Spine 2000; 25: 106-121.

Fritz J. Use of a Classification Approach to the Treatment of 3 Patients with Low Back Syndrome. Physical Therapy 1998;78(7):767-777.

Frymoyer JW, Durett CL. The economics of spinal disorders. In: Frymoyer JW,ed. The Adult Spine: Principles and practice. Philadelphia: Lippincott-Raven; 1997;143-50

Frymoyer JW. Back pain and sciatica. N Engl J Med 1988;318:291-300.

Fujiwara A, Lim T, Tanaka N et al. The effect of Disc Degeneration and Facet Joint Osteoarthritis on the Segmental Flexibility of the Lumbar Spine. Spine 2000;25(23):3036-3044.

Furlan A, Clarke J, Rosmin E. A Critical review on the Treatment of Chronic Low Back Pain. Spine 2001;26(7):E155-E162.

Gaines W and Hegman K. Effectiveness of Waddell's Nonorganic Signs in Predicting a delayed Return to regular Work in Patients experiencing Acute Occupational Low Back Pain. Spine 1999;24(4):396-401.

Garratt, A, Klaber-Moffett J, and farrin A. Responsiveness of generic and specific measures of health outcome in low back pain. Spine, 2001,26(1): 71-77.

Gatchel R, Mayer T, Jeffrey BA et al. The Association of the SF-36 Health Status Survey with 1 Year Socio-economic Outcomes in a Chronically Disabled Spinal disorder Population. Spine 1999;24(20):2162-2180.

Gertzbein S, Seligman J, Holtby M et al. Centrode Characteristics of the Lumbar Spine as a Function of Segmental Instability. Clinical Orthopaedics and Related Research. 1986;208:48-51.

Gifford L. Output Mechanism In: Gifford (ed) Topical Issues in Pain. UK: NOI Press. 1998a.

Gifford L. The 'Central' Mechanism in Topical Issues In: Gifford L (ed) Pain. UK: NOI Press. 1998b.

Gifford L. The Mature Organism Model in Topical Issues In: Gifford L (ed) Pain. UK: NOI Press. 1998c.

Gifford L. Tissue and input related mechanisms: Gifford (ed) Topical Issues in Pain. UK: NOI Press. 1998d.

Gifford L. Pain, the tissues and the nervous system: a conceptual model. Physiotherapy, 84(1):27-36, 1998e Jan

Gifford LS. Butler DS. The integration of pain sciences into clinical practice. Journal of Hand Therapy, 1997,10(2):86-95.

Gilbert J, Taylor D, Hildebrand A, Evans C. Clinical trial of common treatments for low back pain in family practice. Br Med J 1985;291:789-792.

Giles L. Miscellaneous pathological and Developmental (anomalous) Conditions In: Giles L and Singer K (eds). The Clinical Anatomy and Management of Back Pain. Volume One. Oxford: Butterworth- Heinemann. 1997.

Giles L and Singer KP. The Clinical Anatomy and Management of Back Pain Series. Volume 1: Clinical Anatomy and Management of Low Back Pain. Oxford. Butterworth-Heinemann. 1997.

Giles L. Evidence based guidelines submitted to the Australian national health and Medical research Council for the management of acute low back pain: a critical review. Journal of manipulative and Physiological therapeutics. 2001;24(2);131-9.

Gill K and Callaghan M. The Measurement of Lumbar Proprioception in Individuals with and without Low Back Pain. Spine 1998;23(3):371-377.

Godfrey C, Morgan P, Schatzker J. A randomized trial of manipulation for low back pain in a medical setting. Spine 1984;9:301-304.

Goldby L. Low back Pain: The Evidence for Physiotherapy. Physical Therapy Review 1997;2:7-11.

Grabiner MD. Koh TJ. el Ghazawi A. Decoupling of bilateral paraspinal excitation in subjects with low back pain. Spine. 1992,17(10):1219-23.

Gracely RH, Lynch.S.A. Bennett GJ. Painful neuropathy-altered central processing maintained dynamically by peripheral input. Pain 1992;51:175-94.

Greenberg R, Daniels S, Flanders D, Williams J and Boring J. Medical Epidemiology. Prentice Hall. New York. 1999.

Greenwood J, Harvey H, Pearson J, et al. Early intervention in low back disability among coal miners in West Virginia: negative findings. Journal of Occupational medicine. 1990;32:1047-52.

Gronblad M and Virri J. Nerves, Neuropeptides and Inflammation in Spinal Tissues: Mechanisms of Back Pain In: Giles L and Singer K (eds) The Clinical Anatomy and Management of Back Pain Series. Volume One. Oxford: Butterworth- Heinemann. 1997a.

Gronblad M, Hurri, Kouri JP. Relationships between spinal mobility, physical performance tests, pain intensity and disability assessments in chronic low back pain patients. Scand J Rehabil Med 1997b; 29: 17-24.

Gronblad M, Virri J, Tolonen J et al. A Controlled Immunohistochemical study of Inflammatory cells in the Herniation Tissue. Spine 1994;19:2744-2751.

Guo HR. Tanaka S. Cameron LL. Seligman PJ. Behrens VJ. Ger J. Wild DK. Putz-Anderson V. Back pain among workers in the United States: national estimates and workers at high risk. American Journal of Industrial Medicine. 1995,28(5):591-602.

Hadler NM, Curtis P, Gillings DB, et al. A benefit of spinal manipulation as adjunctive therapy for low back pain: a stratified controlled trial. Spine 1987; 12: 703-6

Haig A, Weismann G, Haugh L et al. Prospective evidence for Change in paraspinal Muscle Activity after Herniated Nucleus Pulposus. Spine 1993;18(7):926-930.

Haig A, Linton P, McIntosh M, et al. Aggressive early medical management by a specialist in physical medicine and rehabilitation: effect on time lost due to injuries in hospital employees. Journal of Occupational medicine. 1990;32:241-44.

Handa T. Ishihara H. Ohshima H. Osada R. Tsuji H. Obata K. Effects of hydrostatic pressure on matrix synthesis and matrix metalloproteinase production in the human lumbar intervertebral disc. Spine. 1997,22(10):1085-91.

Harding V. Minimising chronicity after whiplash injury. In: Gifford (ed) Topical Issues in Pain. UK: NOI Press. 1998.

Harding VR. de C Williams A. Activities training: integrating behavioral and cognitive methods with physiotherapy in pain management. Journal of Occupational Rehabilitation, 1998, 8(1):47-60.

Hartigan C. Rainville J. Sobel JB. Hipona M. Long-term exercise adherence after intensive rehabilitation for chronic low back pain. Medicine & Science in Sports & Exercise, 2000,32(3):551-7.

Heliovaara M. Risk factors for low back pain and sciatica. Ann Med. 1989; 21:257-64

Helliwell PS, Cunliffe G. Manipulation in low-back pain. The Physician 1987;April:187-8

Hennekens CH, Buring JE, Mayrent SL. Measures of disease frequency and associations. In: Mayrent SL (ed) Epidemiology in Medicine. 1st ed. Boston: Little, Brown and Company, 1987: 54-66

Hennekens CH, Buring JE. Epidemiology in Medicine. In Mayrent SL, ed. Epidemiology in Medicine, Boston: Little, Brown and Company, 1987.

Herbert, R. Human strength adaptations - Implications for therapy. In Crosbie, J. and McConnell, J. (eds). Key Issues in Musculoskeletal Physiotherapy. Oxford: Butterworth Heinemann. .

Herbert, R. Adaptations of muscle and connective tissue. In Refshauge, K. and Gass, E. (eds) Musculoskeletal Physiotherapy. Clinical Science and Practice. Oxford: Butterworth Heinemann. 1995.

Herbert RD. Critical appraisal of clinical trials. I: estimating the magnitude of treatment effects when outcomes are measured on a continuous scale. Australian Journal of Physiotherapy. 2000;46:229-35.

Herbert R, Sherrington C, Maher C, et al. Evidence based practice – imperfect but necessary. Physiotherapy Theory and Practice. 2001;17(3):201-211.

Herman E, Williams R, Stratford P, Fargas-Babjak A, Trott M. A randomised controlled trial of Transcutaneous Electrical Nerve Stimulation (CODETRON) to determine its benefits in a rehabilitation program for acute occupational low back pain. Spine 1994;19:561-568.

Herzog W, Conway PJW, Willcox BJ. Effects of different treatment modalities on gait symmetry and clinical measures for sacroiliac joint patients. J Manipulative Physiol Ther 1991;14:104-9

Hickman M, Mason V. The prevalence of back pain. A report prepared for the Dept of Health by the Office of Population Censuses and Surveys, Social Survey Division, based on the Omnibus Survey, March, April, June 1993. London: HMSO, 1993:1-24.

Hides JA, Jull GA, Richardson CA. Long-term effects of specific stabilizing exercises for first-episode low back pain. Spine. 2001;26(11):E243-8.

Hides JA, Richardson CA, Jull GA. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. Spine 1996; 21 (23): 2763-9

Hides, J. J. M. Stokes, M. Saide, G. Jull, and D. H. Cooper. Evidence of lumbar Multifidus muscle wasting ipsilateral to symptoms with acute/subacute low back pain. Spine 19: 165-172, 1994

Hildebrandt J, Pfingsten M, Saur P et al. Prediction from a Multidisciplinary Treatment Program for Chronic Low Back Pain. Spine 1997;22(9): 990-1001.

Hillman M, Wright A, Rajaratnam G, et al. Prevalence of low back pain in the community: Implications for service provision in Bradford, UK. J Epidemiol Community Health 1996; 50: 347-52

Hodges, P. W. and C. A. Richardson. Delayed postural contraction of transversus abdominis in low back pain associated with movement of the lower limb. J. Spinal Disord. 19981,1: 46-56.

Hodges, P. W. and C. A. Richardson. Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. Spine 1996,22: 2640-2650.

Hoeler F, Tobis J, Buerger A. Spinal manipulation for low back pain. J Am Med Assoc 1981;245:1835-1838.

Holm I. Friis A. Brox JI. Gunderson R. Steen H. Minimal influence of facet joint anesthesia on isokinetic muscle performance in patients with chronic degenerative low back disorders. Spine. 2000,25(16):2091-4.

Holmstrom E, Moritz U, Andersson M. Trunk muscle strength and back muscle endurance construction workers with and without low back disorders. Scand J Rehabil Med 1992; 24: 3-10

Honeyman PT, Jacobs EA. Effects of culture on back pain in Australian aboriginals. Spine. 1996; 21:841-3.

Howell D. Statistical Methods for Psychology. Boston: PWS Kent Publishing Company. 1992.

Hultman G, Nordin M, Saraste H, Ohlsen H. Body composition, endurance, strength, cross-sectional area, and density of MM erector spinae in men with and without low back pain. J Spinal Disord 1993; 6: 114-23

Hupli M, Hurri H, Luoto S, et al. Isokinetic performance capacity of trunk muscles: Part I. The effect of repetition on measurement of isokinetic performance capacity of trunk muscles among healthy controls and two different groups of low-back pain patients. Scand J Rehab Med 1996; 28: 201-6

Hupli M, Hurri H, Luoto S, et al. Low synthesis rates of type I procollagen is normalised during active back rehabilitation. Spine.1997;22(8):850-4.

Hurley D, Dusoir T, Moore A. How effective is the Acute Low Back pain Screening Questionnaire for Predicting 1 Year Follow-Up in Patients With Low Back Pain? Clinical Journal of Pain 2001; 17(3): 256-263.

Hurley DA. Dusoir TE. McDonough SM. Moore AP. Linton SJ. Baxter GD. Biopsychosocial screening questionnaire for patients with low back pain: preliminary report of utility in physiotherapy practice in Northern Ireland. Clinical Journal of Pain. 2000,16(3):214-28.

Hurley DA. Minder PM. McDonough SM. Walsh DM. Moore AP. Baxter DG. Interferential therapy electrode placement technique in acute low back pain: a preliminary investigation. Archives of Physical Medicine & Rehabilitation. 2001,82(4):485-93.

Iatridis JC. Kumar S. Foster RJ. Weidenbaum M. Mow VC. Shear mechanical properties of human lumbar annulus fibrosus Journal of Orthopaedic Research. 1999,17(5):732-7.

Iatridis JC. Mente PL. Stokes IA. Aronsson DD. Alini M. Compression-induced changes in intervertebral disc properties in a rat tail model. Spine. 1999, 24(10):996-1002.

Indahl A, Haldorsen EH, Holm S, et al. Five-year follow-up study of a controlled clinical trial using light mobilization and an informative approach to low back pain. Spine 1998; 23: 2625-30

Indahl A, Velund L, Reikeraas O. Good prognosis for low back pain when left untampered: a randomized clinical trial. Spine 1995;20:473-477.

International Olympic Committee (IOC). Terminology and units of measurement for the description of exercise and sport. Journal of Applied Sport Science Research. 1991,2, 108.

Itto M, Incorvaia K, Yu S et al. Predictive Signs of Discogenic Lumbar Pain on Magnetic Resonance Imaging with Discography Correlation. Spine 1998; 23(11): 1252-1260.

Iwabuchi M, Rydevik B, Kikuchi S et al. Effects of Anulus Fibrosus and Experimentally Degenerated Nucleus Pulposus on nerve Root Conduction Velocity. Spine 2001;26(15):1651-1655.

Jackson D. How is low back pain managed? Physiotherapy 2001;87(11):573-581.

Jaffray D, O'Brien J. Isolated intervertebral disc resorption: a source of mechanical and inflammatory back pain? Spine. 1986;11:397-401.

Jarvik J, Hollingworth W, Heagerty P et al. The longitudinal Assessment of Imaging and Disability of the Back (LAIDBack) Study. Spine 2001;26(10):1158-1166.

Jaryaraman, G. Nazre, A. McCann, V. Redford, J. A computerized technique for analysing lateral bending behaviour of subjects with normal and impaired lumbar spine. Spine. 1994,19, 824-832.

Jensen M, Brant-Zawadzki M, Obuchowski N et al. Magnetic Resonance Imaging of the Lumbar Spine in People without Back pain. The New England Journal of Medicine 1994;331(2): 69-73.

Jette A, Smith K, Haley S et al. Physical Therapy Episodes of Care for Patients with Low back Pain. Physical Therapy 1994;74(2):101-115.

Jones M. Clinical reasoning and pain. Manual Therapy 1995;1:17-24.

Jones M. Clinical Reasoning in Manual Therapy. Physical Therapy 1992;72(12):875-884.

Jones M. Clinical Reasoning: The Foundation of Clinical Practice. Part 1. Australian Physiotherapy 1997;43(3):167-170.

Jones M. Edwards I. Gifford L. Masterclass. Conceptual models for implementing biopsychosocial theory in clinical practice. Manual Therapy, 2002,7(1):2-9.

Jones MA. Clinical reasoning process in manipulative therapy. In: Boyling JD, Palastanga N (eds.) Grieve' Modern Manual Therapy. The Vertebral Column. Churchill Livingstone: Edinburgh. 1994, 471-489.

Jorgensen K and Nicolaisen. Trunk extensor endurance: determination and relation to low- back trouble. Ergonomics 1987;30(2): 259-267.

Jull GA and Moore A. What is a suitable dosage of physical therapy treatment? Manual Therapy. 7(4) 2002 181-182.

Jull GA, Richardson CA. Rehabilitation of active stabilisation of the lumbar spine. In: Twomey LT, Taylor JR, eds. Physical Therapy of the Low Back. New York: Churchill Livingstone, 1994:251-73.

Jull, G. and Janda, V. Muscles and motor control in low back pain: Assessment and management. In Twomey, L. and Taylor, J. (eds) Physical Therapy of the Low Back. New York: Churchill Livingston. 1987.

Kalaoukalani D, Cherkin DC, Sherman KJ, Koepsell TD, Deyo RA. Lessons from a trial of acupuncture and massage for low back pain. Spine 2001;26(13):1418-1424

Kanemoto M, Hukuda S, Komiya Y et al. Immunohistochemical Study of Matrix Metalloproteinase-3 and Tissue Inhibitor of Metalloproteinase-1 in Human Intervertebral Discs. Spine 1996; 21(1):1-8.

Kang J, Georgescu H, McIntyre-Larkin L, et al. Herniated lumbar intervertebral disc spontaneously produces matrix metalloprotinases, nitric oxide, interleukin S and prostaglandin E2. Spine. 1996;21(3):271-7.

Kang J, Stefanovic-Racic M, McIntyre L et al. Toward a Biomechanical Understanding of Human Intervertebral Disc Degeneration and Herniation: Contributions of Nitric Oxide, Interleukins, Prostaglandin E2, and Matrix Metalloproteinases. Spine 1997; 22(10): 1065-1073.

Kankaanpaa M, Taimela S, Airaksinen O, Hanninen O. The efficacy of active rehabilitation in chronic low back pain: Effect on pain intensity, self-experienced disability, and lumbar fatigability. Spine 1999; 24: 1034-42

Kankaanpaa M, Taimela S, Airaksinen O. Reference change limits of the paraspinal spectral EMG in evaluation of low back rehabilitation. Pathophysiology 1998a; 5: 217-24

Kankaanpaa M, Taimela S, Laaksonen D, et al. Back and hip extensor fatigability in chronic low back pain patients and controls. Arch Phys Med Rehabil 1998b; 79: 412-7

Keefe FJ, Hill RW. An objective approach to quantifying pain behaviour and gait patterns in low back pain patients. Pain 1985; 21: 153-61

Keen S, Dowell A, Hurst K et al. Individuals with low back pain: How do they view physical activity? Family Practice. 1999,16(1):39-45.

Keller A, Johansen JG, Hellesnes J, et al. Predictors of isokinetic back muscle strength in patients with low back pain. Spine 1999; 24: 275-80

Kelsey JL, White III AA. Epidemiology and impact of low back pain. Spine 1980; 5: 133-42

Kendall N and Watson P. Identifying Psychosocial Yellow Flags and Modifying Management In: Gifford L (ed) Topical Pain Issues 2. UK: CNS Press. 2000.

Kendall NA. Psychosocial approaches to the prevention of chronic pain: the low back paradigm. Best Practice & Research in Clinical Rheumatology. 1999,13(3):545-54.

Kendall NAS, Linton SJ and Main CJ. Guide to Assessing Psychological Yellow Flags in Acute Low Back Pain: Risk Factors for Long-Term Disability and Work Loss. Wellington, NZ: Accident Rehabilitation & Compensation Insurance Corporation of New Zealand and the National Health Committee, 1997: 1-22

Kendrick D, Fielding K, Bently E et al. Radiography of the Lumbar Spine in Primary Care Patients with Low Back Pain. BMJ 2001;322: 400-405.

Khodadadeh S, Eisenstein S, Summers B, et al. Gait asymmetry in patients with low back pain. Neuroorthopedics 1988; 6: 24-7

Khodadadeh S, Eisenstein SM. Gait analysis of patients with low back pain before and after surgery. Spine 1993; 18: 1415-55

Kishino ND, Mayer TG, Gatchel RJ, et al. Quantification of lumbar function: Part 4. Isometric and isokinetic lifting simulation in normal subjects and low back dysfunction patients. Spine 1985; 10: 921-7.

Klaber Moffett J, Torgerson D, Bell-Syer S, et al. Randomised controlled trial of exercise for low back pain: clinical outcomes, costs, and preferences. Br Med J 1999; 319: 279-83

Klaber-Moffett J, Richardson G, Sheldon TA, Maynard A. Back Pain: It's management and cost to society. University of York: Centre for Health Economics. 1995.

Klein AB, Snyder-Mackler L, Roy SH, et al. Comparison of spinal mobility and isometric trunk extensor forces with electromyographic spectral analysis in identifying low back pain. Phys Ther 1991; 71: 445-54.

Knuttgen, H. & Komi, P. Basic definitions for exercise. In Komi, P. (ed) Strength and Power in Sport. Oxford: Blackwell. 1992.

Koes B, Bouter L, van Mameren H, et al. Randomised clinical trial of manipulative therapy and physiotherapy for persistent back and neck complaints: results of one year follow-up. Br Med J 1992;304:601-605.

Koes BW, Assendelft WJJ, van der Heijden GJMG, Bouter LM. Spinal Manipulation for Low Back Pain: An Updated Systematic Review of Randomized Clinical Trials. Spine 1996;21:2860-2873.

Koes BW. van Tulder MW. Ostelo R. Kim Burton A. Waddell G. Clinical guidelines for the management of low back pain in primary care: an international comparison. Spine. 2001, 26(22):2504-13.

Kopec JA, Esdaile JM. Functional disability scales for back pain. Spine. 1995;20:1943-9.

Kovacs K, Marras W, Litsky A et al. Localised Oxygen Use of healthy and Low Back Pain Individuals during controlled trunk Movements. Journal of Spinal Disorders 2001;14(2): 150-158.

Kravitz E, Moore M, Glaros A. Paralumbar Muscle Activity in Chronic Low Back Pain. Archives of Physical and Medical Rehabilitation 1981; 62: 172-175.

Krismer M. Haid C. Behensky H. Kapfinger P. Landauer F. Rachbauer F. Motion in lumbar functional spine units during side bending and axial rotation moments depending on the degree of degeneration. Spine. 2000,25(16):2020-7.

KumazawaT. Primitivism and plasticity of pain--implication of polymodal receptors. Neuroscience Research. 1998;32:9-31.

Laasonen E.M. Atrophy of sacrospinal muscle groups in patients with chronic, diffusely radiating lumbar back pain. Neuroradiology 1984;26:9-13.

Lam SS. Jull G. Treleaven J. Lumbar spine kinesthesia in patients with low back pain. Journal of Orthopaedic & Sports Physical Therapy, 1999,29(5):294-9.

Lamoth C, Meijer O, Wuisman P et al. Pelvis- Thorax Coordination in the Transverse Plane during Walking in Persons with Non-specific Low Back Pain. Spine 2002;27(4): E92-E99.

Lariviere C, Gagnon D, Loisel P. The effect of load on the coordination of the trunk for subjects with and without chronic low back pain during flexion-extension and lateral bending tasks. Clin Biomech 2000; 15: 407-16.

Larivière C. Gagnon D. Loisel P. A biomechanical comparison of lifting techniques between subjects with and without chronic low back pain during freestyle lifting and lowering tasks. Clinical Biomechanics, 2002,17(2):89-98.

Leboeuf-Yde C, Lauritsen JM. The prevalence of low back pain in the literature: A structured review of 26 Nordic studies from 1954-1993. Spine 1995; 20: 2112-18

Leclaire R, Esdaile JM, Suissa S, et al. Back school in a first episode of compensated acute low back pain: a clinical trial to assess efficacy and prevent relapse. Arch Phys Med Rehabil 1996; 77: 673-9

Lee JH, Ooi Y, Nakamura K. Measurement of muscle strength of the trunk and the lower extremities in subjects with history of low back pain. Spine 1995; 20: 1994-6

Lee M. Biomechanics of Joint Movement. In Refshauge, K and Gass E (eds) Musculoskeletal Physiotherapy: Clinical Science and practice. Butterworth-Heinemann. Oxford. 1995.

Leggett S. Mooney V. Matheson LN. Nelson B. Dreisinger T. Van Zytveld J. Vie L. Restorative exercise for clinical low back pain: a prospective two-center study with 1-year follow-up. Spine, 1999,24(9):889-98.

Lehto M. Hurme M. Alaranta H. Einola S. Falck B. Jarvinen M. Kalimo H. Mattila M. Paljarvi L. Connective tissue changes of the multifidus muscle in patients with lumbar disc herniation. An immunohistologic study of collagen types I and III and fibronectin. Spine. 1989,14(3):302-9.

Leigh H and Reisner M (1980). The patient: Biological, psychological and social dimensions of medical practice. Plenum, New York. Cited in Main C and Waddell G. Psychologic Distress In: Waddell G The Back Pain Revolution. UK: Churchill Livingstone. 1998.

Leino P, Aro S, Hasan J. Trunk muscle function and low back disorders: A ten-year follow-up study. J Chronic Dis 1987; 40:289-96

Leinonen V, Kankaanpaa, Luukkonen O et al. Disc herniation-Related Back Pain Impairs Feed-Forward Control of Paraspinal Muscles. Spine 2001;26(16): E367-E372.

Levsen M, Hansen M, Kent A et al. Effects of Physical Therapist Training on Outcomes of Patients with Chronic Low Back Pain or Chronic Shoulder Pain. The Journal of Manual & Manipulative Therapy 2001;9(2):84-91.

Li L and Bombardier C. Physical Therapy management of Low Back Pain: An Exploratory Survey of Therapist Approaches. Physical therapy 2001;81(4):1018-1027.

Lidbeck J. Central Hyperexcitability in Chronic Musculoskeletal Pain: A Conceptual Breakthrough with Multiple Clinical Implications. Pain research and Management 2002;7(2):81-92.

Lieber, R. Skeletal Muscle Structure and Function. Baltimore: Williams and Warwick. 1992.

Lindequist SL, Lundberg B, Wikmark R, Bergstad B, Loof G, Ottermark AC. Information and regime at low-back pain. Scand J Rehab Med 1984;16:113-6

Lindgren K, Sivhonen T, Leino E et al. Exercise Therapy Effects on Functional Radiographic Findings and Segmental Electromyographic Activity in Lumbar Spine Instability. Archives of Physical Medicine and Rehabilitation 1993;74: 933-939.

Lindstrom I, Ohlund C, Eek C, Wallin L, Peterson L, Nachemson A. Mobility, strength, and fitness after a graded activity program for patients with subacute low back pain. A randomised prospective clinical study with a behavioural therapy approach. Spine 1992a;17:641-649.

Lindstrom I, Ohlund C, Eek C, Wallin L, Peterson LE, Fordyce WE, Nachemson AL. The effect of graded activity on patients with subacute low back pain: A randomized prospective clinical study with an operant-conditioning behavioral approach. Physical Therapy 1992b;72(4):279-290

Linton S and Andersson T. Can Chronic Disability be Prevented? Spine 2000;25(21):2825-2831.

Linton SJ, Buer N, Vlaeyen J, et al. Are fear-avoidance beliefs related to a new episode of back pain? A prospective study. Psychol Health 2000; 14: 1051-9.

Linton SJ, Hallden K. Can we screen for problematic back pain? A screening questionnaire for predicting outcome in acute and subacute back pain. Clin J Pain 1998; 14: 209-15.

Linton SJ, Hellsing A-L, Andersson D. A controlled study on the effects of an early intervention on acute musculoskeletal pain problems. Pain 1993;54:353-359

Linton SJ, Hellsing AL, Hallden K. A population-based study of spinal pain among 35- to 45-year-olds: Prevalence, sick leave, and health-care utilization. Spine 1998; 23: 1457-63.

Linton SJ. Psychological risk factors for neck and back pain. In Nachemson A, Jonsson E, eds. Neck and Back Pain: the Scientific Evidence. Philadelphia: Lippincott Williams and Wilkins, 2000a.

Linton SJ. Review of Psychological Risk Factors in Back and Neck Pain. Spine 2000b; 25:1148-56.

Little P, Roberts L, Blowers H et al. Should we give detailed advice and information booklets to Patients with Back Pain? Spine 2001;26(19):2065-2072.

Loisel P, Abenhaim L, Durand P, Esdaile J, Suissa S, Gosselin L, Simard R, Turcotte J, Lemaire J. A population-based, randomized clinical trial on back pain management. Spine 1997;22:2911-8

Long D. Chronic Back Pain. In: Wall P and Melzak R (eds) Textbook of Pain. London: Churchill Livingstone. 1999.

Lotz J and Chin J. Intervertebral Disc Cell Death is Dependent on the Magnitude and Duration of Spinal Loading. Spine 2000;25(12):1477-1483.

Lotz JC. The biomechanics of prevention and treatment for low back pain: 2nd international workshop. Clinical Biomechanics. 1999,14(3):220-3.

Luoto S, Hurri H, Alaranta H. Reaction time in patients with chronic low-back pain. Eur J Phys Med Rehabil 1995;5:47-50.

Luoto S, Taimela S, Alaranta H, Hurri H. Psychomotor speed in chronic low-back pain patients and healthy controls: Construct validity and clinical significance of the measure. Percept Mot Skills 1998;87:1283-96.

Luoto S, Taimela S, Hurri H, Aalto H, Pyykko I, Alaranta H. Psychomotor speed and postural control in chronic low-back pain patients: A controlled follow-up study. Spine 1996;21:2621-7.

Luoto S, Taimela S, Hurri H, et al. Mechanisms explaining the association between low back trouble and deficits in information processing. A controlled study with follow-up. Spine 1999; 24 (3): 255-61.

Luoto, S. H. Aalto, S. Taimela, H. Hurri, I. Pyykko, and H. Alaranta. One-footed and externally disturbed two-footed postural control in chronic low back pain patients and healthy controls: a controlled follow-up study. Spine 1998,23: 2081-2089.

Luoto, S. M. Heliovaara, H. Hurri, and H. Alaranta. Static back endurance and the risk of low back pain. Clin. Biomech. 1995,10: 323-324.

Lynn B. Neurogenic inflammation caused by cutaneous polymodal receptors. Progress in Brain Research. 1996,113:361-8.

MacDonald RS, Bell CMJ. An open controlled assessment of osteopathic manipulation in nonspecific low-back pain. Spine 1990;15:364-70

Macfarlane GJ, Thomas E, Croft PR, et al. Predictors of early improvement in low back pain amongst consulters to general practice: the influence of pre-morbid and episode-related factors. Pain 1999; 80: 113-9

Macfarlane GJ. Thomas E. Papageorgiou AC. Croft PR. Jayson MI. Silman AJ. Employment and physical work activities as predictors of future low back pain. Spine. 1997,22(10):1143-9.

Maffey-Ward L, Jull G, Wellington L. Toward a clinical test of lumbar spine kinesthesia. Journal of Sport and Physical Therapy, 1996,24 (6), 354-358.

Magnusson M, Bishop J, Hasselquist L. Range of Motion and Motion Patterns in patients with Low Back Pain Before and after Rehabilitation. Spine.1998,23(23): 2631-2639.

Maher C. The Australasian low back pain study. In: Proceedings of the 7th scientific conference of the IFOMT. 2000a Perth.

Maher C. The quality of clinical evidence in musculoskeletal physiotherapy. In: Proceedings of the 7th scientific conference of the IFOMT. 2000b Perth.

Maher, C. et al. Prescription of Activity for Low Back Pain: What Works? Aust J of Phys. 1999, 45:121-132.

Main C and Waddell G. Psychologic Distress In: Waddell G The Back Pain Revolution. UK: Churchill Livingstone. 1998.

Main C and Watson P. Psychological Aspects of Pain. Manual Therapy 1999;4(4):203-215.

Main C, Wood P, Hollis S, Spanswick C, Waddell G. The distress and risk assessment method. A simple patient classification to identify distress and evaluate the risk of poor outcome. Spine 1992;17:42-52.

Main CJ and Waddell G. The Detection of Psychological Abnormality in Chronic Low Back Pain Using Four Simple Scales. Current Concepts in Pain. 1984,2: 10-15.

Main CJ, Wood PL, Hollis S, et al. The Distress and Risk Assessment Method. A simple patient classification to identify distress and evaluate the risk of poor outcome. Spine 1992; 17 (1): 42-52

Main CJ. The modified somatic perception questionnaire (MSPQ). J Psychosom Res 1983; 27 (6): 503-14

Maitland, G. D. Vertebral manipulation. (5th ed.) London: Butterworths. 1986.

Malmivaara A, Hakkinen U, Aro T, Heinrichs ML, Koskenniemi L, Kuosma E, et al. The treatment of acute low back pain - bed rest, exercises or ordinary activity? N Engl J Med 1995;332:351-355

Maluf KS. Sahrmann SA. Van Dillen LR. Use of a classification system to guide nonsurgical management of a patient with chronic low back pain. Physical Therapy. 2000,80(11):1097-111.

Maniadakis N.Gray A. The economic burden of back pain in the UK. Pain 2000;84:95-103.

Manipulative Physiotherapy Association of Australia. Position statement on the efficacy of physiotherapy for the treatment of low back pain. 1999.

Manniche C, Lundberg E, Christensen I, Bentzen L, Hesselsoe G. Intensive dynamic back exercises for chronic low back pain: a clinical trial. Pain 1991;47:53-63

Manniche C, Skall H, Braendholt L, et al. Clinical trial of postoperative dynamic back exercises after first lumbar discectomy. Spine 1993;18:92-97.

Mannion A, Dolan P, Adams M. Psychological questionnaires: Do abnormal scores precede or follow first time low back pain? Spine. 1996:21:2603-2611.

Mannion AF, Junge A, Taimela S, et al. Active therapy for chronic low back pain. Part 3. Factors influencing self-rated disability and its change following therapy. Spine 2001; 26: 920-9

Mannion AF, Kaser L, Weber E, et al. Influence of age and duration of symptoms on fiber type distribution and size of the back muscles in chronic low back pain patients. Eur Spine J 2000; 9: 273-81

Mannion AF, Muntener M, Taimela S, et al. A randomised clinical trial of three active therapies for chronic low back pain. Spine 1999; 24 (23): 2435-48

Mannion, A. F. B. Connolly, K. Wood, and P. Dolan. The use of surface EMG power spectral analysis in the evaluation of back muscle function. J. Rehabil. Res. Dev. 1997,34: 427-39.

Marras WS, Ferguson SA, Gupta P, et al. The quantification of low back disorder using motion measures. Methodology and validation. Spine 1999; 24 (20): 2091-100

Marras WS, Lavender SA, Leurgans SE, Fatallah FA, Fergusson SA, Allread WG, Rajulu SL. Biomechanical risk factors for occupationally related low back disorders. Ergonomics 1995; 38:377-410.

Marras WS, Lavender SA, Leurgens SE, et al. The role of dynamic three-dimensional trunk motion in occupationally related low back disorders: The effects of workplace factors trunk position and trunk motion characteristics on risk of injury. Spine 1993a;18:617-28

Marras WS, Parnianpour M, Ferguson SA, et al. Quantification and classification of low back disorders based on trunk motion. Eur J Phys Med 1993b;3:218-35

Marras WS, Parnianpour M, Ferguson SA, Kim J, Crowell RR, Bose S, Simon SR. The classification of anatomic and symptom-based low back disorders using motion measure models. Spine, 1995,20 (23), 2531-2546.

Marras WS, Wongsam PE. Flexibility and velocity of the normal and impaired lumbar spine. Arch Phys Med Rehabil 1986; 67: 213-7.

Marras WS. Davis KG. Ferguson SA. Lucas BR. Gupta P. Spine loading characteristics of patients with low back pain compared with asymptomatic individuals Spine, 2001,26(23):2566-74.

Mason V. The Prevalence of Back Pain in Great Britain. A Report on OPCS Omnibus Survey Data Produced on Behalf of the Department of Health. London, England: Her Majesty's Stationery Office; 1994

Masset D, Malchaire J, Lemoine A. Static and dynamic characteristics of the trunk and history of low back pain. International Journal of Industrial Ergonomics 1993;11:279-90

Mathews W, Morkel M, Mathews J. Manipulation and traction for lumbago and sciatica: Physiotherapeutic techniques used in two controlled trials. Physiotherapy Practice 1988;4:201-6.

Mattila M, Hurme M, Alaranta H, et al. The multifidus muscle in patients with lumbar disc herniation. A histochemical and morphometric analysis of intraoperative biopsies. Spine 1986; 11 (7): 732-8

Mayer TG, Kondraske G, Mooney V, et al. Lumbar myoelectric spectral analysis for endurance assessment. A comparison of normals with deconditioned patients. Spine 1989; 14 (9): 986-91

Mayer TG, Smith SS, Keeley J, et al. Quantification of lumbar function: Part 2. Sagittal plane trunk strength in chronic low back pain patients. Spine 1985; 10: 765-72

Mayer TG, Tencer AF, Kristoferson S, et al: Use of noninvasive techniques for quantification of spinal range-of-motion in normal subjects and chronic low-back dysfunction patients. Spine 1984; 9: 588-95

McAuley E.Courneya KS. Adherence to exercise and physical activity as health-promoting behaviors: Attitudinal and self-efficacy influences. Applied and Preventive Psychology 1993;2:65-77.

McClure P, Esola M, Schreier R et al. Kinematic Analysis of Lumbar and Hip Motion while Rising from a Forward, Flexed Position in Patients With and Without a History of Low Back Pain. Spine 1997;22(5):552-558.

McGregor A, Cattermole H and Hughes S. Spinal motion in Lumbar Degenerative Disc disease. Journal of Bone Joint Surgery 1998a;80-B:1009-1013.

McGregor AH. Dore CJ. McCarthy ID. Hughes SP. Are subjective clinical findings and objective clinical tests related to the motion characteristics of low back pain subjects? Journal of Orthopaedic & Sports Physical Therapy, 1998b,28(6):370-7.

McGregor A, Mccarthy I, Hughes S. Motion characteristics of normal subjects and people with low back pain. 1995;81(10):632-7

McIntosh, G. et al Prognostic Factors for Time Receiving Workers' Compensation Benefits in a Cohort of Patients With Low Back Pain. Spine. 2000,25:147-157.

McIntyre DR, Glover LH, Conino MC, et al. A comparison of the characteristics of preferred low-back motion of normal subjects and low-back patients. J Spinal Disord 1991;3:90-5

McIntyre DR, Glover LH, Reynolds DC. Relationship between preferred and maximum effort low back motion. Clin Biomech 1993;8:203-9

McKenzie R. The Lumbar Spine Mechanical Diagnosis and Therapy. Waikanae, New Zealand: Spinal Publications Ltd. 1981.

McNeill T, Warwick D Andersson G et al. Trunk Strengths in Attempted Flexion, Extension, and Lateral Bending in Healthy Subjects and Patients with Low-Back Disorders. Spine 1980;5(6): 529-537.

Meade TW, Dyer S, Browne W, et al. Low back pain of mechanical origin: randomised comparison of chiropractic and hospital outpatient treatment. Br Med J 1990; 300: 1431-7

Melzack R. The Short Form McGill Pain Questionnaire. Pain 1987; 30: 191-7

Mientjes M.I.V, and Frank. Balance in chronic low back pain patients compared to healthy people under various conditions in upright standing. Clinical Biomechanics 1999;14:710-716.

Miller M, McAndrews B, Wiesel S, et al. Health care for the injured worker. A management challenge. Orthopaedic Physical Therapy Clinics of North America. 1995;4:367-373.

Moneta GB, Videman T, Kaivanto K, Aprill C, Spivey M, Vanharanta H et al. Reported pain during lumbar discography as a function of annular ruptures and disc degeneration. Spine 1994;19:1968-74.

Mooney V, Gulick J, Perlman M, et al. Relationship between myoelectric activity, strength, and MRI of lumbar extensor muscles in back pain patients and normal subjects. J Spinal Disord 1997; 10 (4): 348-56

Moore A, McQuay H and Gray J Evidence-based everything. Bandolier 1995,1(12):1.

Morton J and Hilth M. Manipulation in the Treatment of Acute Low Back Pain. The Journal of Manual & Manipulative Therapy 1999;7(4):182-189.

Muller CF. Monrad T. Biering-Sorensen F. Darre E. Deis A. Kryger P. The influence of previous low back trouble, general health, and working conditions on future sick-listing because of low back trouble. A 15-year follow-up study of risk indicators for self-reported sick listing caused by low back trouble. Spine. 1999,24(15):1562-70.

Muller-Berger S. Karbach D. Kang D. Aranibar N. Wood PG. Ruterjans H. Passow H. Roles of histidine 752 and glutamate 699 in the pH dependence of mouse band 3 protein-mediated anion transport. Biochemistry. 1995,34(29):9325-32.

Murphy PL, Volinn E. Is occupational low back pain on the rise? Spine 1999; 247: 691-7

Nachemson A, Jonsson E, (Eds). Neck and back pain: the scientific evidence of causes, diagnosis and treatment. Philadelphia: Lippincott, Williams & Wilkins 2000.

Nachemson A, Vingard E. Influences of individual factors and smoking on neck and low back pain. In Nachemson A, Jonsson E, eds. Neck and Back Pain: The Scientific Evidence of Causes, Diagnosis and Treatment, Philadelphia: Lippincott Williams and Wilkins, 2000.

Nachemson A, Waddell G, Norlund AI. Epidemiology of neck and low back pain. In Nachemson A, Jonsson E, eds. Neck and Back Pain: The Scientific Evidence for Causes, Diagnosis and Treatment. Philadelphia: Lippincott, Williams and Wilkins, 2000.

Nagi SZ, Riley LE, Newby LG. A social epidemiology of back pain in a general population. J Chron Dis 1973; 26: 769-79

Nattrass CL, Nitschke JE, Disler PB, et al. Lumbar spine range of motion as a measure of physical and functional impairment: an investigation of validity. Clin Rehabil 1999; 13 (3): 211-8.

Newcomer K, Laskowski E, Bing Y et al. Differences in Repositioning Error among Patients with Low Back Pain Compared with Controlled Subjects. Spine 2000b;25(19):2488-2493.

Newcomer K. Laskowski ER. Yu B. Larson DR. An KN. Repositioning error in low back pain. Comparing trunk repositioning error in subjects with chronic low back pain and control subjects. Spine. 2000a ,25(2):245-50.

Newton M, Thow M, Somerville D, et al. Trunk strength testing with isomachines: Part 2. Experimental evaluation of the Cybex II Back Testing System in normal subjects and patients with chronic low back pain. Spine 1993; 18: 812-24

Newton M, Thow M, Somerville D, Henderson I, Waddell G. Trunk strength testing with iso-machines. Spine 1993; 18:812-24

Ng J, Richardson C, Kippers V et al. Relationship between Muscle Fibre Composition and Functional Capacity of Back Pain Muscles in Healthy Subjects and Patients with Low Back Pain. Journal of Orthopaedic & Sports Physical Therapy 1998;27(6):389-402.

Ng J, Richardson C, Parnianpour M et al. Fatigue-Related Changes in Torque Output and Electromyographic Parameters of Trunk Muscles During Isometric Axial Rotation Exertion. Spine 2002;27(6):637-646.

NHS Centre for Reviews and Dissemination University of York. Effectiveness Matters: Acute and Chronic Low Back Pain. York: NHS Centre for Reviews and Dissemination University of York, 2000:1-8.

Nicolaisen T, Jorgensen K. Trunk strength, back muscle endurance and low back trouble. Scand J Rehabil Med 1985; 17: 121-7

Nies N, Sinnott PL. Variations in balance and body sway in middle-aged adults: Subjects with healthy backs compared with subjects with low-back dysfunction. Spine 1991; 16: 325-30

Nordin M, Skovron ML, Hiebert R, et al. Early predictors of delayed return to work in patients with low back pain. J Musculoskel Pain 1997; 5 (2): 5-27

Norlund A and Waddell G. Cost of Back Pain in some OECD Countries. In Nachemson A, Jonsson E, eds. Neck and Back Pain: The Scientific Evidence for Causes, Diagnosis and Treatment. Philadelphia: Lippincott, Williams and Wilkins, 2000.

Norris, M. Spinal stabilisation. 1. Active lumbar stabilisation: Concepts. Physiotherapy, 1995,81, 61-78.

Nouwen A, van Akkerveeken PF, Versloot JM. Patterns of muscular activity during movement in patients with chronic low-back pain. Spine 1987; 12: 777-82

O'Sullivan PB, Allison GT, Twomey LT, et al: Evaluation of specific stabilizing exercise in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. Spine 1997a; 22: 2959-67

O'Sullivan PB, Beales D, Beetham J et al. Altered Motor Control Strategies in Subjects with Sacroiliac Joint Pain during the Active Straight-Leg-Raise Test. Spine 2002;27(1):E1-E8.

O'Sullivan PB, Twomey L and Allison. Dysfunction of the Neuro-Muscular System in the Presence of Low Back Pain- Implications for Physical Therapy Management. The Journal of Manual & Manipulative Therapy. 1997b;5(1):20-26.

O'Sullivan PB. Masterclass. Lumbar segmental 'instability': clinical presentation and specific stabilizing exercise management. Manual Therapy, 2000,5(1):2-12.

O'Sullivan PB. Twomey L. Allison GT. Altered abdominal muscle recruitment in patients with chronic back pain following a specific exercise intervention. Journal of Orthopaedic & Sports Physical Therapy. 1998,27(2):114-24.

Oegema T. Biochemistry of the intervertebral disc. Clinics in Sports Medicine. 1993;12(3):419-39.

Ogon M, Bender B, Hooper D et al. A Dynamic Approach to Spinal Instability. Part One. Spine 1997a;22(24):2841-2858.

Ogon M, Bender B, Hooper D et al. A Dynamic Approach to Spinal Instability. Part Two. Spine 1997b;22(24):2859-2866.

Okawa A, Shinomiya K, Komori H et al. Dynamic Motion Study of the Whole Lumbar Spine by Videofluroscopy. Spine 1998;23(16):1743-1749.

Okawa A, Shinomiya K, Komori H, et al. Dynamic motion studies of the whole lumbar spine by videofluoroscopy. Spine 1998; 23: 1743-9

Olmarker K and Rydevik B. Selective Inhibiition of Tumour Necrosis Factor Prevents Nucleus Pulposus- Induced Thrombus Formation, Intraneural Oedema and Reduction of Nerve Conduction Velocity. Spine 2001;26(8): 863-869.

Olmarker K, Kikuchi S and Rydevik B. Anatomy and Physiology of Spinal Nerve Roots and the Results of Compression and Irritation In: Giles L and Singer K (eds) The Clinical Anatomy and Management of Back Pain. Volume One. Oxford: Butterworth-Heinemann. 1997.

Owen JP, Rutt G, Keir MJ, Spencer H, Richardson D, Richardson A, et al. Survey of general practitioners' opinions on the role of radiology in patients with low back pain. Br J Gen Pract 1990; 40: 98-101

Painting S, Favarin I, Swales J. The management of acute industrial low back pain. Physiotherapy, 1998,84(3), 110-117.

Palmer KT, Walsh K, Bendall H, Cooper C, Coggon D. Back pain in Britain: comprision of two prevalence surveys at an interval of 10 years. British Medical Journal 2000;320:1577-8.

Panjabi MM. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. Journal of Spinal Disorders 1992a;5:383-9.

Panjabi MM. The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis. Journal of Spinal Disorders 1992b 2;5:390-6.

Papageorgiou AC, Croft PR, Ferry S, Jayson MIV, Silman AJ. Estimating the prevalence of low back pain in the general population: Evidence from the South Manchester Back Pain Survey. Spine 1995;20:1889-94

Papageorgiou AC, Croft PR, Thomas E, Ferry S, Jayson MI, Silman AJ. Influence of previous pain experience on the episode incidence of low back pain: Results from the South Manchester Back Pain Study. Pain 1996; 66:181-5

Papageorgiou AC. Macfarlane GJ. Thomas E. Croft PR. Jayson MI. Silman AJ. Psychosocial factors in the workplace--do they predict new episodes of low back pain? Evidence from the South Manchester Back Pain Study. Spine. 1997,22(10):1137-42.

Paquet N, Malouin F, Richards CL. Hip-spine movement interaction and muscle activation patterns during sagittal trunk movements in low back pain patients. Spine 1994; 19: 596-603

Parkhurst TM. Burnett CN. Injury and proprioception in the lower back. Journal of Orthopaedic & Sports Physical Therapy. 1994,19(5):282-95.

Parkkola R, Rytokoski U, Kormano M. Magnetic resonance imaging of the discs and trunk muscles in patients with chronic low back pain and healthy control subjects. Spine 1993; 18: 830-6

Peach JP, McGill SM. Classification of low back pain with the use of spectral electromyogram parameters. Spine 1998; 23: 1117-23

Pearcy M. Biomechanics of the Lumbosacral Spine. In: Giles L and Singer K (eds) The Clinical Anatomy and Management of Back Pain. Volume One. Oxford: Butterworth-Heinemann. 1997.

Pearcy M and Bogduk N. Instantaneous Axes of Rotation of the Lumbar Intervertebral Joints. Spine 1988;13(9):1033-1041.

Pearcy M, Portek I, Shepherd J. The effect of low-back pain on lumbar spinal movements measured by three-dimensional x-ray analysis. Spine 1985;10:150-3

Peterson C, Bolton J and Wood A. A Cross-Sectional Study Correlating Lumbar Spine Degeneration With Disability and Pain. Spine 2000;25(2):218-223.

Peterson T, Thorson H, Manniche C et al. Classification of Non-Specific Low Back Pain: A Review of the Literature on Classifications Systems relevant to Physiotherapy. Physical Therapy Reviews 1999;4:265-281

Philips HC, Grant L. The evolution of chronic back pain problems: A longitudinal study. Behav Res Ther 1991;29:435-41

Physiotherapy Evidence Database. www.fhs.usyd.edu.au/pedro.

Pincus T, Burton A, Vogel S et al. A Systematic Review of Psychological Factors as Predictors of Chronicity/Disability in Prospective Cohorts of Low Back Pain. Spine 2002;27(5):E109-E120.

Pope MH, Philips RB, Haugh LD, Hsieh CY, MacDonald L, Haldeman S. A prospective randomized three-week trial of spinal manipulation, transcutaneous muscle stimulation, massage and corset in the treatment of subacute low back pain. Spine 1994;19:2571-7

Porter J, App M, Wilkinson A et al. Lumbar-Hip Flexion Motion. A Comparative Study Between Asymptomatic and Chronic Low Back Pain in 18-to 36-year-old Men. Spine 1997;22(13):1508-1513.

Postacchini F, Facchini M, Palieri P. Efficacy of various forms of conservative treatment in low back pain: a comparative study. Neuroorthopedics 1988; 6: 28-35

Poterfield J and De Rosa. Mechanical Low Back Pain. Perspectives in Functional Anatomy. Philadelphia: W.B. Saunders. 1998.

Radebold A, Cholewicki J, Panjabi MM, et al. Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. Spine 2000; 25 (8): 947-54

Radebold A, Cholewicki J, Polzhofer G et al. Impaired Posrueal Control of the Lumbar Spine is Associated with Delayed Muscle Response Times in Patients with Chronic Idiopathic Low Back Pain. Spine 2001;26(7):724-730.

Rainville J, Ahern D, Phalen L et al. The Association of Pain with Physical Activities in Chronic Low Back Pain. Spine 1992;17(9):1060-1064.

Rainville J, Carlson N, Polatin P et al. Exploration of Physicians' Recommendations for Activities in Chronic Low Back Pain. Spine 2000;25(17):2210-2220.

Ramsbacher J, Theallier-Janko A, Stoltenburg-Didinger G. Ultrasoniocal Changes in Paravertebral Muscles Associated with Degenerative Spondylolisthesis. Spine 2001;26(20):2180-2185.

Rankine J, Gill P, Hutchinson C et al. The Clinical Significance of the High Intensity Zone on Lumbar Spine Magnetic Resonance Imaging. Spine 1999;24(18):1913-1920.

Rantanen P and Nykvist F. Optimal sagittal motion axis for trunk extension and flexion tests in chronic low back trouble. Clinical Biomechanics 2000;15:665-671.

Rantanen, J. M. Hurme, B. Falck, et al. The lumbar multifidus muscle five years after surgery for a lumbar intervertebral disc herniation. Spine 1993,18: 568-574.

Ready AE, Boreskie SL, Law SA, Russell R. Fitness and lifestyle parameters fail to predict back injuries in nurses. Can J Appl Physiol 1993; 18:80-90

Richardson C, Jull G, Hodges P, et al: Therapeutic Exercises for Spinal Segmental Stabilization in Low Back Pain. London, Churchill Livingstone, 1999

Ricketson R. Simmons JW. Hauser BO. The prolapsed intervertebral disc. The high-intensity zone with discography correlation. Spine. 1996,21(23):2758-62.

Riddle D, Stratford P and Binkley J. Sensitivity to Change of the Roland-Morris Back Pain Questionnaire: Part 2. Physical Therapy 1998;78(11):1197-1207.

Riddle DL. Classification and Low Back Pain: A Review of the Literature and Critical Analysis of selected Systems. Physical Therapy 1998;78(8):708-37.

Roberts A. 1991. The Conservative Treatment of Low Back Pain. MD Thesis, University of Nottingham. Cited in Waddell G The Back Pain Revolution. UK: Churchill Livingstone. 1998.

Roland M, Waddell G, Klaber-Moffett J, et al. The Back Book. London: The Stationery Office, 1996.

Roland M, Morris R. A study of the natural history of back pain. Part 1: Development of a reliable and sensitive measure of disability in low-back pain Spine 1983a ,8(2), 141-144.

Roland M, Morris R. A study of the natural history of low-back pain Part II: Development of guidelines for trials of treatment in primary care. Spine 1983b;8:145-50.

Rossignol M, Abenhaim L, Seguin P, et al. Coordination of primary health care for back pain: A randomized controlled trial. Spine 2000; 25:251-9.

Rowe L. Imaging of Mechanical and Degenerative Syndromes of the Lumbar Spine In: Giles L and Singer K (eds) The Clinical Anatomy and Management of Back Pain. Volume One. Oxford: Butterworth- - Heinemann. 1997.

Rowe PJ, White M. Three-dimensional, lumbar spinal kinematics during gait following mild musculoskeletal low back pain in nurses. Gait Posture 1996; 4: 242-51.

Roy SH, DeLuca CJ, Casavant DA. Lumbar muscle fatigue and chronic low back pain. Spine 1989;14:992-1001.

Roy SH, DeLuca CJ, Snyder-Mackler L, Emley MS, Crenshan RL, Lyons JP. Fatigue, recovery and low back pain in varsity rowers. Med Sci Sports Exerc 1990;22:463-9

Roy, S. H. C. J. Deluca, M. Emley, and R. J. Buijs. Spectral electromyographic assessment of back muscles in patients with low back pain undergoing rehabilitation. Spine 1995,20: 38-48.

Rupert R, Wagnon R, Thompson P and Ezzeldin M. Chiropractic adjustments. Results of a controlled clinical trial in Egypt. ICA International review of chiropractic. 1985; winter: 58-60.

Ryan WE, Krishna MK, Swanson CE. A prospective study evaluating early rehabilitation in preventing back pain chronicity in mine workers. Spine 1995;20:489-91.

Saal J, Franson R, Dobrow R et al. High Levels of Inflammatory Phospholipase A2 Activity in Lumbar Disc Herniations. Spine 1990;15(7):674-678.

Saifuddin A, Braithwaite I, White J, Taylor BA, Renton P. The value of lumbar spine magnetic resonance imaging in the demonstration of anular tears. Spine 1998; 23: 453-7

Savage RA, Whitehouse GH, Roberts N. The relationship between the magnetic resonance imaging appearance of the lumbar spine and low back pain, age and occupation in males. Eur Spine J 1997; 6: 106-14

Schellhas KP. Pollei SR. Gundry CR. Heithoff KB. Lumbar disc high-intensity zone. Correlation of magnetic resonance imaging and discography. Spine. 1996,21(1):79-86.

Schiottz-Christensen B, Nielsen GL, Hansen VK et al. Long-term Prognosis of Acute Low Back Pain in Patients see in general practice: A 1 Year prospective follow-up study. Family Practice 1999,16(3):223-233.

Schneider G and Bodguk N. Abnormal Quality of Segment Motion, without Excess Horizontal Displacement, found in cases of Lumbar Instability. Journal of Bone and Joint Surgery 2000;82B Supplement 3:280-281.

Schneider G, Pearcy M, Bogduk N. The biomechanical basis of instability in spondylolitic spondylolisthesis is not excessive translation, but rather, segments operating around an abnormal point of axial compression. Joirnal of Bone and Joint Surgery B. 2001;83-B(Suppl III):360-361.

Schneiders A, Zusman M and Singer K. Exercise therapy compliance in acute low back pain patients. Manual therapy, 1998,3(3): 147-152.

Scrimshaw SV, Maher C. Responsiveness of visual analogue and McGill Pain Scale Measures. Journal of Manipulative & Physiological Therapeutics 2001;24(8):501-504.

Seferlis T, Nemeth G, Carlsson AM, Gillstrom P. Conservative treatment in patients sick-listed for acute low-back pain: a prospective randomised study with 12 months' follow-up. Eur Spine J 1998;7: 461:70

Selles R, Wagenaar R, Smidt T. Disorders in Trunk Rotation during Walking in Patients with Low Back Pain: A Systematical Approach. Clinical Biomechanics 2001;16:175-181.

Shekelle P. The epidemiology of low back pain. In: Giles LGF, Singer KP, eds. Clinical Anatomy and Management of Low Back Pain. Oxford, UK: Butterworth-Heinemann, 1997, pp 18-31

Sihvonen T, Herno A, Paljarvi L, Airaksinen O, Partanen J, Tapaninaho A. Local denervation atrophy of paraspinal muscles in postoperative failed back syndrome. Spine 1993;18:575-81

Sihvonen T. Lindgren K. Airaksinen O. Manninen H. Movement disturbances of the lumbar spine and abnormal back muscle electromyographic findings in recurrent low back pain. Spine, 1997,22(3):289-95.

Sihvonen T. Partanen J. Hanninen O. Soimakallio S. Electric behavior of low back muscles during lumbar pelvic rhythm in low back pain patients and healthy controls. Archives of Physical Medicine & Rehabilitation, 1991,72(13):1080-7.

Simmonds M, Olson S, Jones S. Psychometric Characteristics and Clinical Usefulness of Physical Performance Tests in Patients with Low Back Pain. Spine 1998;23(22):2412-2421.

Sinclair SJ, Hogg-Johnson SH, Mondloch MV, et al. The effectiveness of an early active intervention program for workers with soft-tissue injuries. The Early Claimant Cohort Study. Spine 1997; 22: 2919-31

Sjölie AN. Ljunggren AE. The significance of high lumbar mobility and low lumbar strength for current and future low back pain in adolescents. Spine, 2001,26(23):2629-36.

Skovron ML, Szpalski M, Nordin M, Melot C, Cukier D. Sociocultural factors and back pain: A population-based study in Belgian adults. Spine 1994;19:129-37

Smedley J, Egger P, Cooper C, Coggon D. Prospective cohort study of predictors of incident low back pain in nurses. BMJ 1997;314:1225-8

Smith A. The Measurement of Human Motor Performance in Ada L and Canning C (eds) Key Issues in Neurological Physiotherapy. Oxford: Butterworth- Heinemann. 1990.

Snook SH, Webster BS, McGorry RW, Fogleman MT, McCann B. The reduction of chronic non-specific low back pain through the control of early morning lumbar flexion. A randomised controlled trial. Spine 1998; 23:2601-7

Soderberg GL. Barr JO. Muscular function in chronic low-back dysfunction. Spine. 1983,8(1):79-85.

Spielberger G, Jacobs G, Russell S, Crane RS. Assessment of Anger: The State-Trait Anger Scale. In Butcher JN, Speilberger CD, eds. Advances in Personality Assessment, pp 161-89. Hillsdale, N.J.: Erlbaum, 1983.

Spitzer WO, LeBlanc FE, Dupuis M, Abenhaim L, Bloch R, Bomardier C, et al. Scientific Approach to the Assessment and Management of Activity-related Spinal Disorders: A Monograph for Clinicians. Report of the Quebec Task Force on Spinal Disorders. Spine 1987;12:S4-S59.

Spratt KF, Weinstein JN, Lehmann TR, Woody J, Sayre H. Efficacy of flexion and extension treatments incorporating braces for low back pain patients with retrodisplacement, spondylolisthesis, or normal sagittal translation. Spine 1993;18:1839-49

SPSS Base 7.0 for Windows User's Guide [computer program]. SPSS Inc. 7. Chicago: SPSS, 1996

Stadnik TW, Lee RR, Coen HL, Neirynck EC, Buisseret TS, Osteaux MJ. Annular tears and disk herniation: prevalence and contrast enhancement on MR images in the absence of low back pain or sciatica. Radiology 1998; 206: 49-55

Stankovic R, Johnell O. Conservative treatment of acute low back pain. A prospective randomized trial: McKenzie method of treatment versus patient education in 'Mini Back School'. Spine, 1990,15(2),120-123.

Stankovic R, Johnell O. Conservative treatment of acute low back pain. A 5-year follow-up study of two methods of treatment. Spine 1995;20:469-72

Stata Statistical Software, version 6.0. College Station, TX: Stata Corporation, 2000

Stephenson R. The Complexity of Pain: Part 1. No Pain without Gain: The Augmentation of nociception in the CNS. Physical Therapy Review 1999;4:105 116.

Stewart AL, Hays RD, Ware JE. The MOS Short-Form General Health Survey: Reliability and validity in a patient population. Med Care 1988; 26: 724-35

Stokes I.A.F, Wilder D.G, Frymoyer J.W et al. Assessment of Patients with Low Back Pain by Biplanar Radiographic Measurement of Intervertebral Motion. Spine 1981;6(3):233-239.

Stratford PW, Binkley J, Solomon P, et al. Defining the minimum level of detectable change for the Roland-Morris questionnaire. Phys Ther 1996; 76: 359-65

Sullivan MS, Kues JM Mayhew TP. Treatment categories for low back pain: A methodological approach. Journal of Sport and Physical Therapy, 1996,24(6), 359-364.

Sullivan S, Donegan Shoaf L and Riddle D. The Relationship of Lumbar Flexion to Disability in Patients with Low Back Pain. Physical Therapy 2000;80(3):240-250.

Suzuki N and Endo S. A Quantitative Study of Trunk Muscle Strength and Fatigability in the Low-Back-Pain Syndrome. Spine 1983;8(1):69-73.

Sward L. Hellstrom M. Jacobsson B. Peterson L. Back pain and radiologic changes in the thoraco-lumbar spine of athletes. Spine. 1990,15(2):124-9.

Szpalski M, Hayez JP. How many days of bed rest for acute low back pain? Objective assessment of trunk function. Eur Spine J. 1992; 1:29-31

Szpalski M. Nordin M. Skovron ML. Melot C. Cukier D. Health care utilization for low back pain in Belgium. Influence of sociocultural factors and health beliefs. Spine. 1995,20(4):431-42.

Tabachnick BG, Fidell LS. Using multivariate statistics. 3rd ed. New York: Harper Collins, 1996.

Taimela S, Kankaanpaa M, Luoto S. The effect of lumbar fatigue on the ability to sense a change in lumbar position. A controlled study. Spine 1999; 24 (13): 1322-7.

Taimela S, Osterman K, Alaranta H, Soukka A, Kujala UM. Long psychomotor reaction time in patients with chronic low-back pain: Preliminary report. Arch Phys Med Rehabil 1993;74:1161-4.

Takayanagi K, Takahashi K, Yamagata M et al. Using Cineradiography for Continious Dynamic-Motion Analysis of the Lumbar Spine. Spine 2001;26(17):1858-1865.

Tawfik B. Symmetry and linearity of trunk function in subjects with non-specific low back pain. Clinical Biomechanics 2001;16:114—120.

Taylor H, Curran N M. The Nuprin Pain Report. Louis Harris and Associates, New York. 1985, 1-233.

Theorell T. Karasek RA. Eneroth P. Job strain variations in relation to plasma testosterone fluctuations in working men--a longitudinal study. Journal of Internal Medicine. 1990,227(1):31-6.

Thompson DA, Biedermann H-J, Stevenson JM, et al. Changes in paraspinal electromyographic spectral analysis with exercise: Two studies. J EMG Kinesiol 1992; 2: 179-86

Tibrewal SB, Pearcy MJ, Portek I, Spivey J. A prospective study of lumbar spinal movements before and after discectomy using biplanar radiography: Correlation of clinical and radiographic findings. Spine 1996;10:455-60

Torstensen TA, Ljunggren AE, Meen HD, Odland E, Mowinckel P, Geijerstam S. Efficiency and costs of medical exercise therapy, conventional physiotherapy, and self-

exercise in patients with chronic low back pain: a pragmatic, randomized, single-blinded, controlled trial with 1-year follow-up. Spine 1998;23:2616-24

Triano JJ, Schultz AB. Correlation of objective measure of trunk motion and muscle function with low back disability ratings. Spine 1987; 12: 561-5.

Tsui-Wu Y-J Deyo RA. Functional disability due to back pain. Arthritis Rheum 1987;30:1247-53.

Twomey L, Taylor J. Exercise and spinal manipulation in the treatment of low back pain. Spine, 1995,20(5), 615-619.

Underwood MR, Morgan J. The use of a back class teaching extension exercises in the treatment of acute low back pain in primary care. Fam Pract 1998;15:9-15

van dar Valk RWA, Dekker J, Van Baar ME. Physical therapy for patients with back pain. Physiotherapy, 1995,81(6), 345-351.

van den Hoogen H. Low Back Pain in General Practice. Diagnosis and Prognosis. The Netherlands: Ponson&Looijen BV. 1996.

van den Hoogen H, Koes B, Van Eijik J et al. Pain and Health Status of Primary Care Patients with Low Back Pain. The Journal of Family Practice 1997;44(2):187-192.

van den Hoogen HJ, Koes BW, Deville W, et al. The prognosis of low back pain in general practice. Spine 1997; 22: 1515-21

van der Weide WE, Verbeek JH, Salle HJ, van Dijk FJ. Prognostic factors for chronic disability from acute low-back pain in occupational health care. Scand J Work Environ Health. 1999; 25: 50-56

van Dillen LR. Sahrmann SA. Norton BJ. Caldwell CA. Fleming DA. McDonnell MK. Woolsey NB. Reliability of physical examination items used for classification of patients with low back pain. Physical Therapy. 1998, 78(9):979-88.

van Doorn JW. Low back disability among self-employed dentists, veterinarians, physicians and physical therapists in The Netherlands. A retrospective study over a 13-year period and an early intervention program with 1-year follow-up. Acta Orthopaedica Scandinavica. Supplementum. 1995,263:1-64.

van Tulder M and Waddell G. Conservative Treatment of Acute and Subacute Low Back Pain. In Nachemson A, Jonsson E, eds. Neck and Back Pain: The Scientific Evidence for Causes, Diagnosis and Treatment. Philadelphia: Lippincott, Williams and Wilkins, 2000.

van Tulder M, Malmaviarra A, Esmail R et al. Exercise Therapy for Low Back Pain. A Systematic review within the framework of the Cochran collaboration back review group. Spine 2000;25(21):2784-2796.

van Tulder MW, Assendelft WJJ, Koes BW, Bouter LM: Spinal radiographic findings and nonspecific low back pain: a systematic review of observational studies. Spine 1997a, 22:427-434

van Tulder MW, Koes BW, Bouter LM. Conservative treatment of acute and chronic nonspecific low back pain. A systematic review of randomized controlled trials of the most common interventions. Spine 1997b;22:2128-56.

Venning P, Walter S and Stitt L. personal and job related factors as determinants of incidence of back injuries among nursing personnel. Journal of Occupational Medicine. 1987,29(10),820-825.

Vendrig A. Prognostic factors and treatment related changes associated with return to work in the multimodal treatment of chronic back pain. Journal of Behavioural Med. 1999,22 (3) 217-232.

Verbunt J, Seelen H, Bousema E et al. The Interpolated Twitch Technique as a Measure of Muscular Inhibition in Chronic Low Back Pain. An Experimental Investigation. In: Proceedings of the International association of for the study of pain conference. 2002. San Diego: 1014

Vlaeyen J. Crombez G. Fear of Movement/(re)injury, Avoidance and Pain Disability in Chronic Low Back Pain. Manual Therapy 1999; 4(4) 187-195.

Vlaeyen JWS, Linton SJ. Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. Pain 2000; 85: 317-32

Vogt L, Pfeifer K, Prtscher M et al. Influences of Non-specific Low Back Pain on Three-Dimensional Lumbar Spine Kinematics in Locomotion. Spine 2001;26(17):1910-1919.

Volinn E, Mayer J, Diehr P et al. Small area analysis of surgery for low back pain. Spine 1992,17(5), 575-579.

Volinn E. The Epidemiology of Low Back Pain in the Rest of the World. A review of surveys in low- and middle- income countries. Spine 1997;22:1747-54.

von Korff M, Dworkin SF, Le Resche L, et al. An epidemiologic comparison of pain complaints. Pain 1988; 32: 173-83

Von Korff M, Saunders K, Roland M, Van de Hoogan HMM. The course of back pain in primary care. Spine 1996;21:2833-9.

Von Korff M. Studying the natural history of back pain. Spine 1994 19(Suppl):S2041-46

Waddell G. Low back pain: a twentieth century health care enigma. Spine. 1996,21(24), 2820-2825.

Waddell G, Feder G, Lewis M. Systematic reviews of bed rest and advice to stay active for acute low back pain. Br J Gen Practice. 1997; 47: 647-52

Waddell G, Feder G, McIntosh A, Lewis M, Hutchinson A. Low back pain evidence review. London: Royal College of General Practitioners, 1999.

Waddell G, Main CJ. Assessment of severity in low back disorders. Spine, 1984,9(2), 204-208.

Waddell G, Waddell H. A review of social influences on neck and back pain and disability. In Nachemson A, Jonssson E, eds. Neck and Back Pain: In Nachemson A, Jonsson E, eds. Neck and Back Pain: The Scientific Evidence for Causes, Diagnosis and Treatment. Philadelphia: Lippincott, Williams and Wilkins, 2000.

Waddell G. A new clinical model for the treatment of low back pain. 1987 Volvo award in clinical sciences. Spine 1987;12:632-644.

Waddell G. The Back Pain Revolution. Edinburgh: Churchill Livingstone, 1998.

Walsh K, Cruddas M, Coggon D. Low back pain in eight areas of Britain. Journal of Epidemiology and Community Health 1992; 46:227-30.

Ware JE Jr, Sherbourne CD. The MOS 36-Item Short-Form Health Survey (SF-36): I. Conceptual framework and item selection. Med Care 1992; 30: 473-83

Ware JE, Kosinski M, Keller SD. A 12-item short-form health survey. Med Care 1996; 34: 220-33

Waterworth RF, Hunter IA. An open study of diflunisal, conservative and manipulative therapy in the management of acute mechanical low back pain. N Z Med J 1985;95:372-375.

Watson P and Kendall N. Assessing Psychological Yellow Flags In: Gifford L (ed) Topical Pain Issues 2. UK: CNS Press. 2000.

Watson P, Booker C and Main C. Evidence for the Role of Psychological Factors in Abnormal Paraspinal Activity in Patients with Chronic Low Back Pain. Journal of Musculoskeletal Pain 1997;5(4):41-56.

Watson P. Psychosocial Predictors of Outcome from Low Back Pain In: Gifford L (ed) Topical Issues in Pain 2. UK: CNS Press Ltd. 2000.

Waxman R, Tennant A, Helliwell P. A prospective follow-up study of low back pain in the community. Spine 2000; 25: 2085-90.

Webster BS, Snook SH. The cost of 1989 workers' compensation low back pain claims. Spine 1994;19:1111-16.

Wilder D, Aleksiev A, Magnusson M, et al. Muscular response to sudden load - A tool to evaluate fatigue and rehabilitation. Spine 1996; 21 (22): 2628-39.

Wilkinson MJB. Does 48 hours' bed rest influence the outcome of acute low back pain? Br J Gen Pract 1995;45:481-4

Wilson L, Hall H, McIntosh G, Melles T. Intertester Reliability of a Low Back Pain Clasification System. Spine 1999;24:248-54.

Wittink H, Hoskins M, Wagner et al. Deconditioning in Patients with Chronic Low Back Pain: Fact or Fiction? Spine 2000;25(17):2221-2228.

Woby S, Watson P, Roach N and Birch K. What factors explain why chronic pain patients improve following rehabilitation? PPA news. 2001; December: 29-30.

Woolf CJ. Evidence for a central component of post-injury pain hypersensitivity. Nature 1983;306:686-8.

Wreje U, Nordgren B, Aberg H. Treatment of pelvic joint dysfunction in primary care: A controlled study. Scand J Prim Health Care 1992;10:310-5.

Wright A. Neurophysiology of pain and pain modulation. In: Strong J, Unruh A, Wright A and David-Baxter G (eds). Pain: A textbook for therapists. Churchill Livingston. London. 2002.

Wright A. Recent Concepts in the Neurophysiology of Pain. Manual Therapy 1999;4(4):196-202.

Zhao Wei-Ping, Kawaguchi Y, Matsui MD et al. Histochemistry and Morphology of the Multifidus Muscle in Lumbar Disc Herniation. Comparative Study between Diseased and Normal Sides. Spine 2000;25(17):2191-2199.

Zigenfus GC, Jiahong Y, Giang G, et al. Effectiveness of early physical therapy in the treatment of acute low back musculoskeletal disorders. J Occup Environ Med. 2000; 42: 35-39

Zusman M. Instigators of activity intolerance. Manual Therapy, 1997,2(2):75-86.

Zusman M. Irritability. Manual Therapy, 1998, 3(4):195-202.

Zusman M. Structure-oriented beliefs and disability due to back pain. Australian Journal of Physiotherapy, 1998,44(1):13-20.

Zusman M. The biochemical basis of back pain. In: Proceedings of the 7th scientific conference of the IFOMT. Perth. 2000,518-523.

Zusmann M. Forebrain-mediated sensitisation of central pain pathways: 'non-specific' pain and a new image for manual therapy. Manual Therapy2002;7(2):80-88.

Appendix

Appendix I: Referral Form

Central Middlesex Hospital NHS Trust, in conjunction with Brunel University, has obtained joint funding from the NHS Research and Development Directorate to conduct a clinical trial evaluating physiotherapy for low back pain.

The project is running from March 1998 for two years. It is a single-blind randomised clinical trial to evaluate the effect of research-based physiotherapy intervention on patients with simple low back pain. Ethical approval (BEC 313) has been obtained from the Brent Medical Ethics Committee.

The □Clinical Guidelines for the Management of Acute Low Back Pain□, published by the Royal College of General Practitioners in September 1996, recommend physiotherapy (manipulation) within the first six weeks of onset of simple low back pain. With both limited access to physiotherapy and the majority of sufferers recovering within the first six weeks, the question of the optimal time for physiotherapy intervention remains unanswered. One of the principal aims of the trial will be to answer this question.

The success of the trial relies on recruiting suitable subjects with simple acute low back pain. The diagnostic triage recommended by the RCGP is used to ensure that only patients with simple low back pain are included. Exclusions include nerve root pain, serious spinal pathology and cauda equina syndrome.

Consent from the patient is necessary. Following consent patients are randomly allocated into one of two groups: those for immediate treatment, and those who will return to the waiting list to await treatment in due course. All patients should be seen sooner than the routine waiting times.

Your patient has met the study criteria and is happy to participate in the trial. I would be grateful if you would consent to their inclusion by completing the attached form.

It is anticipated that during the recruitment phase of the trial there will be improved access for patients with simple acute back pain. If you wish to refer other suitable patients please send their details to the department. The attached sheet details the study criteria.

Yours sincerely,

Benedict Wand BAppSc, GradDipAppSc, MAppSc
MCSP MMACP
Research Physiotherapist

To: Ben Wand, Research Physiotherapist
Physiotherapy Department,
Central Middx Hospital
Acton Lane, Park Royal
London NW10 7NS

LOW BACK PAIN CLINICAL TRIAL

RE:

Surname.....Mr/Mrs/Miss

First Name.....DOB: .....

Address.....

.....

.....

Tel.....

This patient meets the study criteria (see below) and is happy to participate in the clinical trial

Dr.....

Signed.....

Date.....

INCLUSION CRITERIA

- Age 20-55 years
- Simple low back pain **< 6 weeks duration** (mechanical pain in lumbrosacral region, buttock or thigh)
- Patients with recurrent LBP should be symptom free for 3/12 prior to this episode.

EXCLUSION CRITERIA

- Pregnancy, or within 3/12 post-partum
- Undergoing psychiatric treatment
- Currently receiving physiotherapy, osteopathy or chiropractic treatment
- Presence of another physically disabling condition
- Systemic disease (diabetes, etc)
- Involved in litigation for LBP

LOW BACK PAIN CLINICAL TRIAL

RE; .....

.....

.....

☐ I consent for my patient to participate in the clinical trial evaluating physiotherapy for low back pain.

☐ I do not consent for my patient to participate in the clinical trial evaluating physiotherapy for low back pain for the following reason/s

.....

.....

.....

Dr.....

Signed.....

Date.....

Appendix II: Telephone Questionnaire

BACK PAIN STUDY

Brunel University & Central Middlesex Hospital

Exclusion Checklist (1)

- ❑ Aged between 20 and 55
- ❑ Back pain < 6 weeks (if acute flare up of chronic - then must be stable/ clear or pain free for previous 3 months)
- ❑ Pregnant
- ❑ Within 3 months post partum
- ❑ Involved in litigation
- ❑ Already receiving physiotherapy (or osteopathy, chiropractic)
- ❑ Other ongoing medical problem
- ❑ Other physically disabling condition

Appendix III: Examination Protocol

CENTRAL MIDDLESEX NHS TRUST

LOW BACK PAIN PROTOCOL

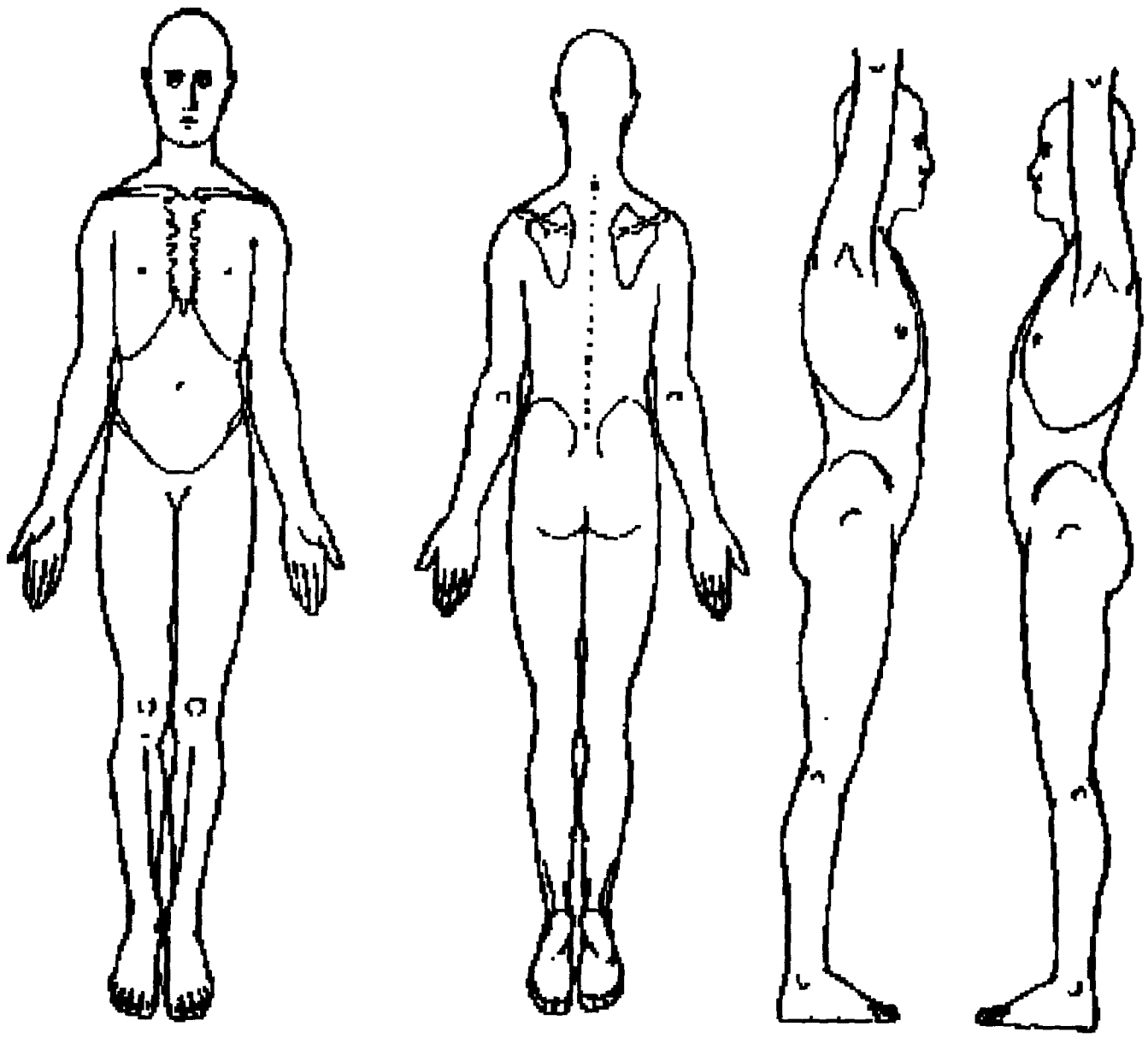
Name: Address: Tel: Home: Work: DOB: First Language: Interpreter Required?	GP: Address: Tel: Date of referral: Treating Therapist:
Type of work (e.g. manual/shift): Is patient working? Yes ☐ No ☐ If no, is this due to current complaint? Yes ☐ No ☐ Time off work? ____years ____months ____weeks ____days Does the patient think they will return to work? Yes ☐ No ☐ If yes, when? _____	Social History (support/response from family): Hobbies/Recreation: Sports/Exercises:

Height (m)	Weight (Kg)	BMI

☐ SIMPLE LOW BACK PAIN ☐ NERVE ROOT PAIN ☐ CONSULTANT REFERRAL OR RED FLAGS

Therapist: Date:

SUBJECTIVE ASSESSMENT



Overall severity: /10 at the moment /10 usual /10 least
of symptoms (1-10)

Aggravating factors	**Activities limited/stopped**	**Easing factors/coping strategies**

24 HOUR BEHAVIOUR:
Start of day:

During day:

Sleep: **Change in sleep pattern**
Frequency of disturbance
Best position
Worst position

Irritability Non ☐ Mild ☐ Moderate ☐ Severe ☐

HISTORY OF PRESENTING COMPLAINT
Onset

Previous History of LBP (including time off work)

Previous treatment for LBP (helpful?)

Subjective cause of LBP

Overall how has the back pain affected patient's life?

What are the patient's expectations of treatment

What is the patient's view of their problem

GENERAL HEALTH

WOMEN ONLY	YES	NO
Post menopause	☐	☐
Hysterectomy	☐	☐
HRT	☐	☐
Calcium supplements	☐	☐
History of back pain related to menstrual cycle	☐	☐

Age at time of menopause

PMH / SURGERY

MEDICATIONS (incl. Steroids/anticoagulants)

	YES	NO
Unexplained weight loss	☐	☐
Smoker	☐	☐
Diabetes	☐	☐
RA	☐	☐

	YES	NO
Bowel/Bladder dysfunction	☐	☐
Saddle area parasthesia	☐	☐
Cough/Sneeze	☐	☐
Gait disorder	☐	☐
Glove & stocking parasthesiae	☐	☐

INVESTIGATIONS (Type and Results/Comments)

PHYSICAL EXAMINATION

Observation

ACTIVE MOVEMENTS
Lumbar spine

Thoracic spine

PPIVM's

SIJ

HIP

KNEE

NEURAL TISSUE PROVOCATION TESTS

	R	L	Comparable sign:	Y ☐	N ☐
P1					
P2/R2					
PNF					
SLUMP					
PKB					

MOVEMENT CONTROL

Flexion control

	Abnormal	Normal
	☐	☐

Extension control

	Abnormal	Normal
	☐	☐

Rotation dissociation (bent knee fall out in supine)

	Abnormal	Normal
R	☐	☐
L	☐	☐

Multifidus setting

	R=L	Decreased
R	☐	☐
L	☐	☐

TrA setting

ADDITIONAL TESTS

NEUROLOGICAL TESTS

Reflexes	R=L Decreased	Absent R L	Increased R L	R L
L3 Quadriceps	☐	☐ ☐	☐ ☐	☐ ☐
S1 Gastrocnemius	☐	☐ ☐	☐ ☐	☐ ☐

Myotomes	Level	R=L Decreased		Sensation
Hip flexion	L2	☐	☐	Light Touch
Knee ext	L3,4	☐	☐	
Dorsi/Inv	L4	☐	☐	
Great toe ext	L5	☐	☐	Sharp/Blunt
Plantar flex	S1	☐	☐	
Toe flexion	S2	☐	☐	

Cord signs	Negative R L	Positive R L
Clonus	☐ ☐	☐ ☐
S2 Babinski	☐ ☐	☐ ☐

THERAPIST IDENTIFIED PROBLEMS

Main subjective findings

Main objective findings

CLASSIFICATION OF BACK PAIN

SYMPTOMS

1. Back pain without radiation ☐
2. Back pain with referral to leg, above the knee ☐
3. Back pain with referral to leg, below the knee ☐
4. Leg pain greater than back pain ☐

HISTORY

5. Acute injury - first episode ☐
6. Reinjury/exacerbation of previous problem ☐

DURATION OF SYMPTOMS SINCE ONSET

7. < 1/52 ☐

8. 1/52 - 6/52 ☐

9. >6/52 ☐

FUNCTIONAL MILESTONES because of this episode of LBP:

		Y	N	N/A
10. (→Q11)	Is the patient off work	☐	☐	☐
11	Are normal activities affected	☐	☐	
12.	Are usual leisure and social activities affected	☐	☐	
13.	Is the patients usual sleeping pattern disturbed	☐	☐	

Appendix IV: Patient Consent Form

AGREEMENT TO PARTICIPATE IN RESEARCH PROJECT
Brent Medical Ethics Committee (BEC 313)

I (Name of participant) _____

of (Address) _____

_____ Postcode _____

agree to take part in the research project.

An Evaluation Of Physiotherapy For Back Pain Patients (Research Study NHS PCD2/A1/288)

I confirm that the nature and demands of the research have been explained to me and that I understand and accept them. I also confirm that I have seen the Patient Information Sheet and understand it. I understand that I may withdraw from the research project if I find I am unable to continue for any reason.

Signed _____ Date _____

Witness _____

I have explained the nature, demands and foreseeable risks of the above research to the participant.

Signature _____ Date _____

Principal Investigator
Professor Lorraine De Souza, PhD FCSP Director of Postgraduate Physiotherapy Studies
Brunel University, Osterley Campus, Borough Road, Isleworth, Middlesex TW7 5DU

Research Co-ordinator
Ben Wand, MSc MCSP

Physiotherapy Department, Central Middlesex Hospital NHS Trust, Acton Lane, Park Royal, London NW10 7NS Telephone: 0181 453 2292

BACK PAIN STUDY

Brunel University & Central Middlesex Hospital

Participant Information Sheet

Title of Research Study An evaluation of physiotherapy for back pain patients.

What is the research about?

Back pain is a very common problem and often GPs refer their patients with back pain for physiotherapy. This research aims to find out what is the best way to treat back problems with physiotherapy, and will also examine how your back problem changes over time. The results from this research will help to improve treatment for back pain patients.

Who is running the research?

Researchers from Central Middlesex Hospital and Brunel University are undertaking this study jointly. The National Health Service Research and Development Programme is funding this research and the study has obtained ethical approval from the hospital ethics committee.

Who can take part?

If you are between 20 and 55 years of age, and have had your back trouble for less than six weeks, we would like to invite you to take part in this study. The overall outcome of the research will not be clear until the research study has finished. Your contribution to this work will be very helpful for improving physiotherapy for back pain patients in the future.

What happens if I agree to take part?

At your first appointment at the Physiotherapy Department at the Central Middlesex Hospital, a researcher will explain the project to you and ask you to read and sign a form in which you give your consent to be part of the research. You will then be given a questionnaire to complete. This should take about 30 minutes, and a research assistant will be there to answer any questions you might have. The physiotherapist will then see you to assess your back problem, and so take a little longer than normal because this will be a detailed assessment. If the physiotherapist decides that you need to see a consultant or another hospital service, he or she will refer you on as appropriate. The physiotherapist will make sure that low back pain is your main problem and you will have the same examination and tests as all back patients have. We will give you a copy of *The Back Book*, and the date and time of your next appointment for treatment. The physiotherapist will let your doctor know that you are taking part in the study.

What treatment will I get, and when?

You will get the same physiotherapy treatment as other back pain patients receive, from experienced senior staff at the Physiotherapy Department. No one who is part of the research study will have to wait any longer for treatment than low back pain patients who are not part of the study.

What happens then?

The physiotherapist treating you will monitor your progress throughout your treatment and follow you up after the physiotherapy is finished in order to study the longer term effects of the treatment. You will be sent follow-up questionnaires by post, along with a prepaid return

envelope, at six weeks, three months, six months and twelve months after your first appointment.

How long will it take?

The amount of time you will spend attending physiotherapy will be decided by the physiotherapist treating you, depending on what sort of back trouble you have. Apart from the physiotherapy you will receive, each set of questionnaires should take approximately 30 minutes to complete. You should receive the last questionnaire for completion 12 months after we first see you. Your contribution to the research will then be completed.

What will happen to the information I provide for the research project?

All information you provide as a research volunteer will be analysed and used to examine the effects of physiotherapy. Your personal information will remain entirely confidential and safe. When the results of this research are written up, the report will refer only to groups of patients with back pain, not to individual people.

Can I change my mind?

You are free to decide not to participate in this study or to withdraw at any time. If you do withdraw, you will still receive all of the appropriate physiotherapy treatment for your back problem. If you have any problems or feel you may wish to withdraw please contact your physiotherapist or the Research Co-ordinator as soon as possible to see if we can help.

Who can I contact?

If you have any queries about the overall study and your involvement in it, please feel free to contact Ben Wand, the Research Co-ordinator at the Central Middlesex Hospital on 0181 453 2292.

Thank you for taking the time to read this information sheet. If you decide to join this research project your contribution witll be very valuable in helping us to improve phsyiotherapy for people with back pain.

Research Co-ordinator:

Ben Wand, MSc MCSP
Chartered Physiotherapist
Physiotherapy Department
Central Middlesex Hospital NHS Trust
Acton Lane, Park Royal
London NW10 7NS
Telephone: 0181 453 2292

Research Assistant:

JamesMcCauley
Department of Health Studies
Brunel University, Osterley Campus
Borough Road, Isleworth
Middlesex TW7 5DU

Telephone: 0181 891 0121 x 2553

Appendix V: Baseline Questionnaire

BACK PAIN STUDY

Brunel University & Central Middlesex Hospital

Date _____

Thank you for agreeing to participate in this study.

This questionnaire is in several sections, asking about your health, your back problem, and about yourself in general. This is the first of five questionnaires we will ask you to complete over the next 12 months. It is also the longest and should take about 30 minutes to complete.

Please read each question carefully, and give the best answer you can. Do not take too long to answer any one question. However, it is important that you answer every question. There is always a response for your particular situation.

If you have any difficulties or queries about the questionnaire, or the study, the Research Assistant who has given you this questionnaire will be happy to help in any way possible.

Section 1: About Your General Health

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

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

1. In general would you say your health is:

(Circle one)

Excellent	1
Very good	2
Good	3
Fair	4
Poor	5

2. Compared with one year ago, how would you rate your health in general now?

(Circle one)

Much better than one year ago _____ 1
Somewhat better now than one year ago ____ 2
About the same as one year ago _____ 3
Somewhat worse than one year ago _____ 4
Much worse than one year ago _____ 5

3. The following items are about activities you might be doing during a typical day. Does your health now limit you in these activities? If so, how much?

(circle one number on each line)

ACTIVITIES	YES, LIMITED A LOT	YES, LIMITED A LITTLE	NO, NOT LIMITED AT ALL
a. Vigorous activities, such as running, lifting heavy objects, participating in strenuous sports	1	2	3
b. Moderate activities, such as moving a table, pushing a vacuum cleaner, bowling, or playing golf	1	2	3
c. Lifting or carrying groceries	1	2	3
d. Climbing several flights of stairs	1	2	3
e. Climbing one flight of stairs	1	2	3
f. Bending, kneeling, or stooping	1	2	3
g. Walking more than a mile	1	2	3
h. Walking half a mile	1	2	3
i. Walking one hundred yards	1	2	3
j. Bathing or dressing yourself	1	2	3

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

(circle one number on each line)

	YES	NO
a. Cut down on the amount of time you spent on work or other activities	1	2
b. Accomplished less than you would like	1	2
c. Were limited in the kind of work or other activities	1	2
d. Had difficulty performing the work or other activities (for example, it took extra effort)	1	2

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

(circle one number on each line)

	YES	NO
a. Cut down on the amount of time you spent on work or other activities	1	2
b. Accomplished less than you would like	1	2
c. Didn't do work or other activities as carefully as usual	1	2

6. During the past four weeks, to what extent has your physical health or emotional problems interfered with your normal social activities with family, friends, neighbours, or groups?

(Circle one)

Not at all _____ 1
Slightly _____ 2
Moderately _____ 3
Quite a bit _____ 4
Extremely _____ 5

7. How much bodily pain have you had during the past four weeks?

(Circle one)

None _____ 1
Very mild _____ 2
Mild _____ 3
Moderate _____ 4
Severe _____ 5
Very severe _____ 6

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

(Circle one)

Not at all _____ 1
A little bit _____ 2
Moderately _____ 3
Quite a bit _____ 4
Extremely _____ 5

9. These questions are about how you feel and how things have been with you during the past four weeks. For each question, please give the one answer that comes closest to how you have been feeling.

How much of the time during the past four weeks -

(circle one number on each line)

			A good		A	None

	All of the time	Most of the time	bit of the time	Some of the time	little of the time	of the time
a. Did you feel full of life?	1	2	3	4	5	6
b. Have you been a very nervous person?	1	2	3	4	5	6
c. Have you felt so down in the dumps that nothing could cheer you up?	1	2	3	4	5	6
d. Have you felt calm and peaceful?	1	2	3	4	5	6
e. Did you have a lot of energy?	1	2	3	4	5	6
f. Have you felt downhearted and low?	1	2	3	4	5	6
g. Did you feel worn out?	1	2	3	4	5	6
h. Have you been a happy person?	1	2	3	4	5	6
i. Did you feel tired?	1	2	3	4	5	6

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

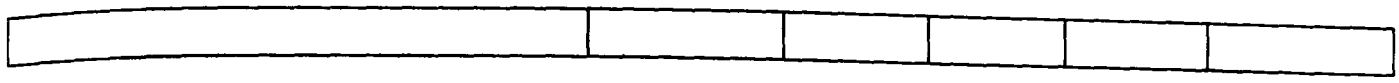
(Circle one)

All of the time _____ 1
Most of the time _____ 2
Some of the time _____ 3
A little of the time _____ 4
None of the time _____ 5

11. How TRUE or FALSE is each of the following statements for you?

(circle one number on each line)

	Definitely True	Mostly True	Don't Know	Mostly False	Definitely False
a. I seem to get ill more easily than other people	1	2	3	4	5
b. I am as healthy as anybody I know	1	2	3	4	5
c. I expect my health to get worse	1	2	3	4	5
d. My health is excellent	1	2	3	4	5



By placing a tick in one box in each group below, please indicate which statements best describe YOUR OWN HEALTH STATE TODAY.
Do not tick more than one box in each group.

Mobility

I have no problems in walking about	θ
I have some problems in walking about	θ
I am confined to bed	θ

Self-Care

I have no problems with self-care	θ
I have some problems washing or dressing myself	θ
I am unable to wash or dress myself	θ

Usual Activities *(e.g. work, study, housework, family or leisure activities)*

I have no problems with performing my usual activities	θ
I have some problems with performing my usual activities	θ
I am unable to perform my usual activities	θ

Pain/Discomfort

I have no pain or discomfort	θ
I have moderate pain or discomfort	θ
I have extreme pain or discomfort	θ

Anxiety/Depression

I am not anxious or depressed	θ
I am moderately anxious or depressed	θ
I am extremely anxious or depressed	θ

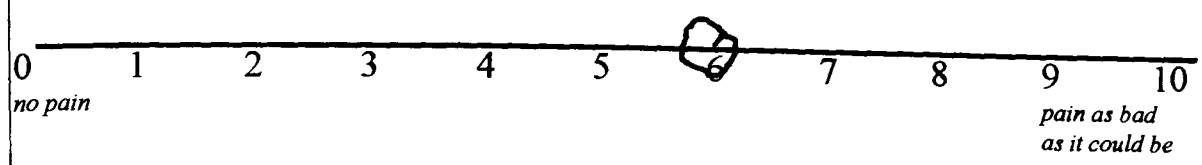
Compared with my general level of health over the past 12 months, my health state today is:

Better	θ	PLEASE TICK
Much the same	θ	ONE BOX
Worse	θ	ONLY

Section 2: Your Back Problem

The following questions assess the level (or severity) of any pain you might be experiencing with your back problem. Please answer each of the questions below by circling the appropriate number on the accompanying scale (see example).

Example: this person rated the severity of their usual pain as 5 out of 10:
1. How would you rate your usual pain, on average over the past week, on a 0 to 10 scale?



For each scale 0 is equal to no pain at all, and 10 equal to pain as bad as it could be.

1. How would you rate your usual pain, on average over the past week, on a 0 to 10 scale?

0 1 2 3 4 5 6 7 8 9 10
no pain *pain as bad as it could be*

2. How would you rate your pain at its least, on average over the past week, on a 0 to 10 scale?

0 1 2 3 4 5 6 7 8 9 10
no pain *pain as bad as it could be*

3. How would you rate your pain at this moment in time, on a 0 to 10 scale?

0 1 2 3 4 5 6 7 8 9 10
no pain *pain as bad as it could be*

The following questions and statements apply if you have aches or pains, such as back, shoulder or neck pain. Please read and answer each question carefully. Do not take too long to answer the questions. However, it is important that you answer every question. There is always a response for your particular situation.

1. Where do you have pain? Place a ✓ for all the appropriate sites.

θ neck θ shoulders θ upper back θ lower back θ leg

2. How many days of work have you missed because of pain during the past 18 months? Tick (✓)one.

θ 0 days θ 1 - 2 days θ 3 - 7 days θ 8 - 14 days θ 15 - 30 days
θ 1 month θ 2 months θ 3 - 6 months θ 6 - 12 months θ over 1 year

3. How long have you had your current pain problem? Tick (✓) one.

θ 0 - 1 week θ 1 - 2 weeks θ 3 - 4 weeks θ 4 - 5 weeks θ 6 - 8 weeks

θ 9 - 11 weeks θ 3 - 6 months θ 6 - 9 months θ 9 - 12 months θ over 1 year

4. Is your work heavy or monotonous? Circle the best alternative.

0 1 2 3 4 5 6 7 8 9 10
Not at all *Extremely*

5. How would you rate the pain that you have had during the past week? Circle one.

0 1 2 3 4 5 6 7 8 9 10
No pain *Pain as bad as it Could be*

6. In the past three months, on average, how bad was your pain? Circle one.

0 1 2 3 4 5 6 7 8 9 10
No pain *Pain as bad as it could be*

7. How often would you say that you have experienced pain episodes, on average, during the past 3 months? Circle one.

0 1 2 3 4 5 6 7 8 9 10
Never *Always*

8. Based on all the things you do to cope, or deal with your pain, on an average day, how much are you able to decrease it? Circle one.

0 1 2 3 4 5 6 7 8 9 10
can't decrease it at all *Can decrease it completely*

9. How tense or anxious have you felt in the past week? Circle one.

0 1 2 3 4 5 6 7 8 9 10
Absolutely calm and relaxed *As tense and anxious as I've ever felt*

10. How much have you been bothered by feeling depressed in the past week? Circle one.

0 1 2 3 4 5 6 7 8 9 10
Not at all *Extremely*

11. In your view, how large is the risk that your current pain may become persistent? Circle one.

0 1 2 3 4 5 6 7 8 9 10
No risk *Very large risk*

12. In your estimation, what are the chances that you will be working in 6 months? Circle one.

0 1 2 3 4 5 6 7 8 9 10
No chance *Very large chance*

13. If you take into consideration your work routines, management, salary, promotion possibilities and work mates, how satisfied are you with your job? Circle one.

0 1 2 3 4 5 6 7 8 9 10
Not at all satisfied *Completely satisfied*

Here are some of the things which other people have told us about their back pain. For each statement please circle one number from 0 to 10 to say how much physical activities, such as bending, lifting, walking or driving would affect your back.

14. Physical activity makes my pain worse.

0 1 2 3 4 5 6 7 8 9 10
Completely disagree *Completely agree*

15. An increase in pain is an indication that I should stop what I am doing until the pain decreases.

0 1 2 3 4 5 6 7 8 9 10
Completely disagree *Completely agree*

16. I should not do my normal work with my present pain.

0 1 2 3 4 5 6 7 8 9 10
Completely disagree *Completely agree*

Here is a list of 5 activities. Please circle the one number which best describes your current ability to participate in each of these activities.

17. I can do light work for an hour.

0 1 2 3 4 5 6 7 8 9 10
Can't do it because of pain problem *Can do it without pain being a problem*

18. I can walk for an hour.

0 1 2 3 4 5 6 7 8 9 10
Can't do it because of pain problem *Can do it without pain being a problem*

19. I can do ordinary household chores.

0 1 2 3 4 5 6 7 8 9 10
Can't do it because of pain problem *Can do it without pain being a problem*

20. I can go shopping.

0 1 2 3 4 5 6 7 8 9 10

Can't do it because of pain problem

Can do it without pain being a problem

21. I can sleep at night.

0 1 2 3 4 5 6 7 8 9 10

Can't do it because of pain problem

Can do it without pain being a problem

When your back hurts, you may find it difficult to do some of the things that you normally do. The list below contains some sentences that people have used to describe themselves when they have back pain. When you read them you may find that some of them stand out because they describe you *today*. As you read the list, think of yourself *today*. When you read a statement that describes you today, put a tick in the 'Yes' column. If the sentence does not describe you, tick the 'No' column.

REMEMBER, ONLY MARK THE BOX IF YOU ARE SURE THAT THE SENTENCE DESCRIBES YOU TODAY.

	YES	**NO**
1. I stay at home most of the time because of my back.	θ	θ
2. I change position frequently to try and get my back comfortable.	θ	θ
3. I walk more slowly than usual because of my back.	θ	θ
4. Because of my back, I am not doing any of the jobs that I usually do around the house.	θ	θ
5. Because of my back, I use a handrail to get upstairs.	θ	θ
6. Because of my back, I lie down to rest more often.	θ	θ
7. Because of my back, I have to hold on to something to get out of an easy chair.	θ	θ
8. Because of my back, I try to get other people to do things for me.	θ	θ
9. I get dressed more slowly than usual because of my back.	θ	θ
10. I only stand up for short periods of time because of my back.	θ	θ
11. Because of my back, I try not to bend or kneel down.	θ	θ
12. I find it difficult to get out of an easy chair because of my back.	θ	θ
13. My back is painful almost all of the time.	θ	θ
14. I find it difficult to turn over in bed because of my back.	θ	θ
15. My appetite is not very good because of my back pain.	θ	θ

16. I have trouble putting on my socks (or stockings/tights) because of the pain in my back.	θ	θ
17. I only walk short distances because of my back pain.	θ	θ
18. I sleep less well because of my back.	θ	θ
19. Because of my back pain, I get dressed with help from someone else.	θ	θ
20. I sit down for most of the day because of my back.	θ	θ
21. I avoid heavy jobs around the house because of my back.	θ	θ
22. Because of my back pain, I am more irritable and bad tempered with people than usual.	θ	θ
23. Because of my back, I go upstairs more slowly than usual.	θ	θ
24. I stay in bed most of the time because of my back.	θ	θ

A number of statements which people use to describe themselves are given below. Read each statement and then circle the most appropriate number to the right of the statement to indicate how you feel right at this moment.

There are no right or wrong answers. Do not spend too much time on any one statement.

	NOT AT ALL	SOMEWHAT	MODERATELY	VERY MUCH
I feel calm	1	2	3	4
I feel tense	1	2	3	4
I feel upset	1	2	3	4
I feel relaxed	1	2	3	4
I feel content	1	2	3	4
I am worried	1	2	3	4

Please indicate for each of these questions which answer best describes how you have been feeling recently by marking X in the appropriate box.

	Rarely or none of the time (less than	Some or little of the time (1-2	A moderate amount of time (3-4 days per	Most of the time (5-7 days per

	1 day per week)	days per week)	week)	week)
1) I feel downhearted and sad				
2) Morning is when I feel best				
3) I have crying spells or feel like it.				
4) I have trouble getting to sleep at night.				
5) I feel that nobody cares.				
6) I eat as much as I used to.				
7) I still enjoy sex.				
8) I notice I am losing weight.				
9) I have trouble with constipation.				
10) My heart beats faster than usual.				
11) I get tired for no reason.				
12) My mind is as clear as it used to be.				
13) I tend to wake up too early.				
14) I find it easy to do the things I used to.				
15) I am restless and can't keep still.				
16) I feel hopeful about the future.				
17) I am more irritable than usual.				
18) I find it easy to make a decision.				
19) I feel quite guilty.				
20) I feel that I am useful and needed.				
21) My life is pretty full.				
22) I feel that others would be better off if I were dead.				
23) I am still able to enjoy things I used to.				

Please describe how you have felt in the PAST WEEK by making a tick (✓) in the appropriate box. Please answer all questions. Do not think too long before answering.

	Not at all	A little, slightly	A great deal, quite a bit	Extremely. could not have been worse
Heart rate increase				
Feeling hot all over				
Sweating all over				
Sweating in a particular part of the body				
Pulse in neck				
Pounding in head				
Dizziness				
Blurring of vision				
Feeling faint				
Everything appearing unreal				
Nausea				
Butterflies				
Pain or ache in stomach				
Stomach churning				
Desire to pass water				
Mouth becoming dry				
Difficulty swallowing				
Muscles in neck aching				
Legs feeling weak				
Muscles twitching or jumping				
Tense feeling across forehead				
Tense feeling in jaw muscles				

We are interested in your own personal views of how you see your back problem.

In your opinion, what caused your present problem? _____
_____
_____

What are your main symptoms?_____
_____
_____

Do you feel that any of the following contributed to your back problem?

θ stress
θ an awkward movement (lifting/turning)
θ poor medical care in the past months
θ chance
θ hereditary (runs in the family)
θ other people
θ germ/virus
θ pollution/environment
θ my own state of mind

θ diet θ workplace factors θ it just happened

What do you expect of physiotherapy for your back problem?_____
_____
_____

Thinking about your back problem, please indicate how much you agree or disagree with each of the following statements below. Tick (✓) one for each:

	STRONGLY AGREE	AGREE	NEITHER AGREE NOR DISAGREE	DISAGREE	STRONGLY DISAGREE
a) My back problem will last a short time					
b) What I do can determine whether my back problem gets better or worse					
c) My back problem will last for a long time					
d) My treatment will be effective in curing my back problem					
e) My back problem has had major consequences on my life					
f) There is a lot which I can do to control my symptoms					
g) My back problem has not had much effect on my life					
h) My back problem has strongly affected the way others see me					
i) My back problem has become easier to live with					
j) My back problem has strongly affected the way I see myself as a person					
k) My back problem will improve in time					
l) My back problem has serious economic and financial consequences					
m) There is very little that can be done to improve my back problem					
n) My back problem is a serious condition					
o) Recovery from my back problem is largely dependent on chance or fate					
p) My back problem is likely to be permanent rather than temporary					

Thank you for completing this section.

Please check that you have completed all of the questions before continuing.

Section 3: About You

Please could you fill in the following details about yourself.

Surname: _____ **Other Names**:_____

_____

Address: _____

_____

_____

Postcode: _____ **Telephone** (day): _____

_____

Sex: _____ Male/Female

θ_1

θ_2

Date Of Birth. _____ ddmmyy

θ_3

θ_4

Marital Status:

Single

Married/cohabitee

Divorced/separated

Widow/widower

Occupational Status:

Please tick only one **What is your occupation**? _____

θ_1 employee
θ_2 self-employed
θ_3 unemployed
θ_4 housewife
θ_5 student
θ_6 retired
θ_7 other

If you are retired, unemployed or unable to work, what was your last occupation?

_____

θ8 unable to work because of disability

What is your ethnic background or cultural origin? _____

Your religion? _____ Country of birth? _____

How long have you lived in this country? _____

What languages do you speak? _____

Education Status

What is your highest level of training or education? Please tick only one box.

θ1	none	θ5	NVQs/HNVQs level ___
θ2	GCSEs	θ6	Primary Degree (e.g. BA, BSc)
θ3	O/A Levels	θ7	Second/Higher Degree (e.g. MA, MSc, PhD)
θ4	Diploma/Certificate	θ8	other _____

Ethnic Background

Please tick one of the following boxes that best describes your ethnic or cultural background:

θ1	White	θ6	Indian
θ2	Irish	θ7	Pakistani
θ3	Black - African	θ8	Bangladeshi
θ4	Black - Caribbean	θ9	Chinese
θ5	Black - Other _____	θ10	Other (please specify) _____

Thank you for your time and assistance.
If you have any further questions please feel free to ask.

Appendix VI: Follow-up Questionnaire

BACK PAIN STUDY

Brunel University & Central Middlesex Hospital

Questionnaire Two

Thank you once again for agreeing to participate in this study. This is the second of five questionnaires we will ask you to complete over the next 12 months. It should take no more than 30 minutes to complete.

This questionnaire is in several sections, the first asking about your health, the second about your back problem, and the third section concerns yourself in general.

Please read each question carefully, and give the best answer you can. Do not take too long to answer any one question. However, it is important that you answer every question. There is always a response for your particular situation.

If you have any difficulties or queries about this questionnaire, or the study, please contact me at Brunel University (Tel. 0181 891 0121 extension 2553, or 0181 453 2292), and I will be happy to help in any way possible.

Todays date _____

Section 1: About Your General Health

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

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

1. In general would you say your health is:
(Circle one)

Excellent _____ 1
Very good _____ 2
Good _____ 3
Fair _____ 4
Poor _____ 5

2. Compared with one year ago, how would you rate your health in general now?
(Circle one)

Much better than one year ago _____ 1
Somewhat better now than one year ago ____ 2
About the same as one year ago _____ 3
Somewhat worse than one year ago _____ 4
Much worse than one year ago _____ 5

3. The following items are about activities you might be doing during a typical day. Does your health now limit you in these activities? If so, how much?

(circle one number on each line)

ACTIVITIES	**YES, LIMITED A LOT**	**YES, LIMITED A LITTLE**	**NO, NOT LIMITED AT ALL**
a. Vigorous activities, such as running, lifting heavy objects, participating in strenuous sports	1	2	3
b. Moderate activities, such as moving a table, pushing a vacuum cleaner, bowling, or playing golf	1	2	3
c. Lifting or carrying groceries	1	2	3
d. Climbing several flights of stairs	1	2	3
e. Climbing one flight of stairs	1	2	3
f. Bending, kneeling, or stooping	1	2	3
g. Walking more than a mile	1	2	3
h. Walking half a mile	1	2	3
I. Walking one hundred yards	1	2	3
j. Bathing or dressing yourself	1	2	3

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

(circle one number on each line)

	YES	NO
a. Cut down on the amount of time you spent on work or other activities	1	2
b. Accomplished less than you would like	1	2
c. Were limited in the kind of work or other activities	1	2
d. Had difficulty performing the work or other activities (for example, it took extra effort)	1	2

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

(circle one number on each line)

	YES	NO
a. Cut down on the amount of time you spent on work or other activities	1	2
b. Accomplished less than you would like	1	2
c. Didn't do work or other activities as carefully as usual	1	2

6. During the past four weeks, to what extent has your physical health or emotional problems interfered with your normal social activities with family, friends, neighbours, or groups?

(Circle one)

Not at all _____ 1
Slightly _____ 2
Moderately _____ 3
Quite a bit _____ 4
Extremely _____ 5

7. How much bodily pain have you had during the past four weeks?

(Circle one)

None _____ 1
Very mild _____ 2
Mild _____ 3
Moderate _____ 4
Severe _____ 5
Very severe _____ 6

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

(Circle one)

Not at all _____ 1
A little bit _____ 2
Moderately _____ 3
Quite a bit _____ 4
Extremely _____ 5

9. These questions are about how you feel and how things have been with you during the past four weeks. For each question, please give the one answer that comes closest to how you have been feeling.

How much of the time during the past four weeks -
(circle one number on each line)

	All of the time	**Most of the time**	**A good bit of the time**	**Some of the time**	**A little of the time**	**None of the time**
a. Did you feel full of life?	1	2	3	4	5	6
b. Have you been a very nervous person?	1	2	3	4	5	6
c. Have you felt so down in the dumps that nothing could cheer you up?	1	2	3	4	5	6
d. Have you felt calm and peaceful?	1	2	3	4	5	6
e. Did you have a lot of energy?	1	2	3	4	5	6
f. Have you felt downhearted and low?	1	2	3	4	5	6
g. Did you feel worn out?	1	2	3	4	5	6
h. Have you been a happy person?	1	2	3	4	5	6
i. Did you feel tired?	1	2	3	4	5	6

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

All of the time _____ 1
Most of the time _____ 2
Some of the time _____ 3
A little of the time _____ 4

None of the time _____ 5

11. How TRUE or FALSE is <u>each</u> of the following statements for you?
(circle one number on each line)

	Definitely True	**Mostly True**	**Don't Know**	**Mostly False**	**Definitely False**
a. I seem to get ill more easily than other people	1	2	3	4	5
b. I am as healthy as anybody I know	1	2	3	4	5
c. I expect my health to get worse	1	2	3	4	5
d. My health is excellent	1	2	3	4	5

By placing a tick in one box in each group below, please indicate which statements best describe YOUR OWN HEALTH STATE TODAY.
Do not tick more than one box in each group.

Mobility

I have no problems in walking about θ
I have some problems in walking about θ
I am confined to bed θ

Self-Care

I have no problems with self-care θ
I have some problems washing or dressing myself θ
I am unable to wash or dress myself θ

Usual Activities *(e.g. work, study, housework, family or leisure activities)*

I have no problems with performing my usual activities θ
I have some problems with performing my usual activities θ
I am unable to perform my usual activities θ

Pain/Discomfort

I have no pain or discomfort θ
I have moderate pain or discomfort θ
I have extreme pain or discomfort θ

Anxiety/Depression

I am not anxious or depressed θ
I am moderately anxious or depressed θ

I am extremely anxious or depressed θ

Compared with my general level of health over the past 12 months, my health state today is:

Better	θ	PLEASE
Much the same	θ	TICK
Worse	θ	ONE BOX ONLY

Section 2: About Your Back Problem

When your back hurts, you may find it difficult to do some of the things that you normally do. The list below contains some sentences that people have used to describe themselves when they have back pain. When you read them you may find that some of them stand out because they describe you *today*. As you read the list, think of yourself *today*. When you read a statement that describes you today, put a tick in the 'Yes' column. If the sentence does not describe you, tick the 'No' column.

REMEMBER, ONLY MARK THE BOX IF YOU ARE SURE THAT THE SENTENCE DESCRIBES YOU TODAY.

	YES	**NO**
1. I stay at home most of the time because of my back.	θ	θ
2. I change position frequently to try and get my back comfortable.	θ	θ
3. I walk more slowly than usual because of my back.	θ	θ
4. Because of my back, I am not doing any of the jobs that I usually do around the house.	θ	θ
5. Because of my back, I use a handrail to get upstairs.	θ	θ
6. Because of my back, I lie down to rest more often.	θ	θ
7. Because of my back, I have to hold on to something to get out of an easy chair.	θ	θ
8. Because of my back, I try to get other people to do things for me.	θ	θ
9. I get dressed more slowly than usual because of my back.	θ	θ
10. I only stand up for short periods of time because of my back.	θ	θ
11. Because of my back, I try not to bend or kneel down.	θ	θ
12. I find it difficult to get out of an easy chair because of my back.	θ	θ
13. My back is painful almost all of the time.	θ	θ
14. I find it difficult to turn over in bed because of my back.	θ	θ

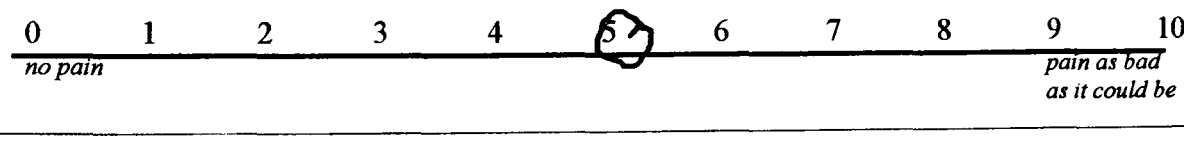
15. My appetite is not very good because of my back pain.	θ	θ
16. I have trouble putting on my socks (or stockings/tights) because of the pain in my back.	θ	θ
17. I only walk short distances because of my back pain.	θ	θ
18. I sleep less well because of my back.	θ	θ
19. Because of my back pain, I get dressed with help from someone else.	θ	θ
20. I sit down for most of the day because of my back.	θ	θ
21. I avoid heavy jobs around the house because of my back.	θ	θ
22. Because of my back pain, I am more irritable and bad tempered with people than usual.	θ	θ
23. Because of my back, I go upstairs more slowly than usual.	θ	θ
24. I stay in bed most of the time because of my back.	θ	θ

Please check that you have completed all of the questions before continuing.

The following questions assess the level (or severity) of any pain you might be experiencing with your back problem. Please answer each of the questions below by circling the appropriate number on the accompanying scale (see example).

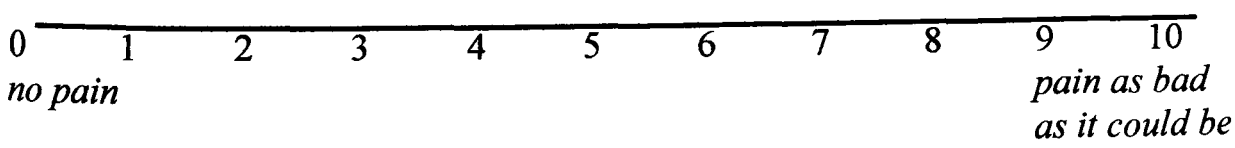
Example: this person rated the severity of their usual pain as 5 out of 10:

1. How would you rate your usual pain, on average over the past week, on a 0 to 10 scale?

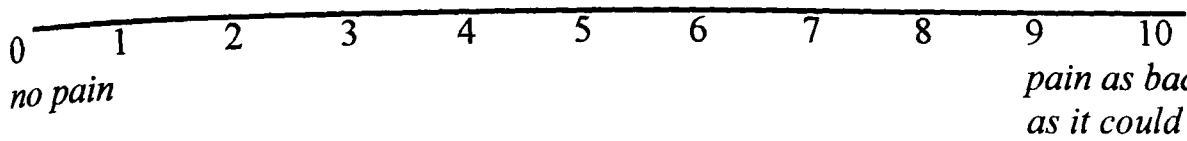


Please answer each of the following. For each scale 0 is equal to no pain at all, and 10 equal to pain as bad as it could be.

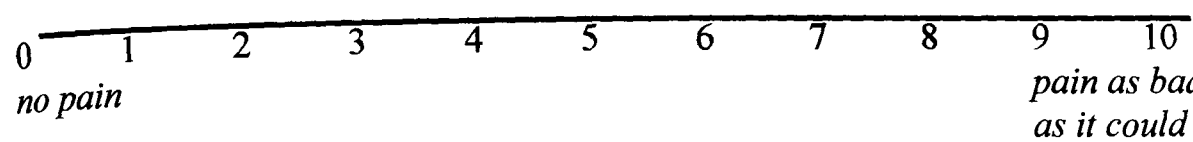
1. How would you rate your usual pain, on average over the past week, on a 0 to 10 scale?



2. How would you rate your pain at its least, on average over the past week, on a 0 to 10 scale?



3. How would you rate your pain at this moment in time, on a 0 to 10 scale?



Thank you for completing this section.

Section 3: About You

Please describe how you have felt in the PAST WEEK by making a tick (✓) in the appropriate box. Please answer all questions. Do not think too long before answering.

	Not at all	A little, slightly	A great deal, quite a bit	Extremely, could not have been worse
Heart rate increase				
Feeling hot all over				
Sweating all over				
Sweating in a particular part of the body				
Pulse in neck				
Pounding in head				
Dizziness				
Blurring of vision				
Feeling faint				
Everything appearing unreal				
Nausea				
Butterflies				
Pain or ache in stomach				
Stomach churning				
Desire to pass water				
Mouth becoming dry				
Difficulty swallowing				
Muscles in neck aching				
Legs feeling weak				
Muscles twitching or jumping				
Tense feeling across forehead				
Tense feeling in jaw muscles				

A number of statements which people use to describe themselves are given below. Read each statement and then circle the most appropriate number to the right of the statement to indicate how you feel right at this moment.

There are no right or wrong answers. Do not spend too much time on any one statement.

	NOT AT ALL	SOMEWHAT	MODERATELY	VERY MUCH
I feel calm	1	2	3	4
I feel tense	1	2	3	4
I feel upset	1	2	3	4
I feel relaxed	1	2	3	4
I feel content	1	2	3	4

I am worried 1 2 3 4

Please indicate for each of these questions which answer best describes how you have been feeling recently by marking X in the appropriate box.

	Rarely or none of the time (less than 1 day per week)	Some or little of the time (1-2 days per week)	A moderate amount of time (3-4 days per week)	Most of the time (5-7 days per week)
1) I feel downhearted and sad				
2) Morning is when I feel best				
3) I have crying spells or feel like it.				
4) I have trouble getting to sleep at night.				
5) I feel that nobody cares.				
6) I eat as much as I used to.				
7) I still enjoy sex.				
8) I notice I am losing weight.				
9) I have trouble with constipation.				
10) My heart beats faster than usual.				
11) I get tired for no reason.				
12) My mind is as clear as it used to be.				
13) I tend to wake up too early.				
14) I find it easy to do the things I used to.				
15) I am restless and can't keep still.				
16) I feel hopeful about the future.				
17) I am more irritable than usual.				
18) I find it easy to make a decision.				
19) I feel quite guilty.				

20) I feel that I am useful and needed.				
21) My life is pretty full.				
22) I feel that others would be better off if I were dead.				
23) I am still able to enjoy things I used to.				

We are interested in your own personal views of how you see your back problem.
In your opinion, what caused your present problem? _____
_____
_____

What are your main symptoms?_____
_____
_____

Do you feel that any of the following contributed to your back problem?

θ stress	θ an awkward movement (lifting/turning)	θ poor medical care in the past months
θ chance	θ hereditary (runs in the family)	θ other people
θ germ/virus	θ pollution/environment	θ my own state of mind
θ diet	θ workplace factors	θ it just happened

Thinking about your back problem, please indicate how much you agree or disagree with each of the following statements below. Tick (✓) one for each:

	STRONGLY AGREE	AGREE	NEITHER AGREE NOR DISAGREE	DISAGREE	STRONGLY DISAGREE
a) My back problem will last a short time					
b) What I do can determine whether my back problem gets better or worse					
c) My back problem will last for a long time					
d) My treatment will be effective in curing my back problem					
e) My back problem has had major consequences on my life					
f) There is a lot which I can do to control my symptoms					
g) My back problem has not had much effect on my life					
h) My back problem has strongly affected the way others see me					
i) My back problem has become easier to live with					
j) My back problem has strongly					

affected the way I see myself as a person					
k) My back problem will improve in time					
l) My back problem has serious economic and financial consequences					
m) There is very little that can be done to improve my back problem					
n) My back problem is a serious condition					
o) Recovery from my back problem is largely dependent on chance or fate					
p) My back problem is likely to be permanent rather than temporary					

Thank you for completing this last section.

Thank you for your time and assistance.

If you have any further questions please feel free to contact James McAuley at Brunel University on 0181 891 0121 x 2553.

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Appendix VII: Clinical Reasoning Form A

GROUP A GOALS & CLINICAL REASONING

PATIENT AGREED GOALS (2 maximum)

Functional Short Term Goals	**Anticipated Treatments**	**Time (weeks)**	**Actual Treatments**	**Time (weeks)**
Functional Discharge Goals	**Anticipated Treatments**	**Time (weeks)**	**Actual Treatments**	**Time (weeks)**

SOURCE OF SYMPTOMS (Structures from which the symptoms are emanating)			
Component	**Structure**	**Supporting evidence**	**Negating evidence**

PAIN MECHANISMS (Evidence for mechanisms by which the symptoms are being initiated and/or maintained neurologically)	
Nociceptive mechanism	
Peripheral neurogenic	
Central mechanisms	
Autonomic	
Psychological/mental processing	

NATURE OF THE DYSFUNCTION			
Component	**Structure**	**Supporting evidence**	**Negating evidence**

CONTRIBUTING FACTORS
(Predisposing or associated factors involved in the development or maintenance of the patients problem)

Physical

Psychosocial

SEVERITY, IRRITABILITY & CAUTION
(Incl stability, progression GH & SQ)

Pain		Mild ☐	Moderate ☐	Severe ☐
Irritability	Non ☐	Mild ☐	Moderate ☐	Severe ☐

other

Do the signs fit with the symptoms?

Clinical impression/principal hypothesis regarding the primary syndrome/disorder

PROGNOSIS	
Favourable	**Unfavourable**

MANAGEMENT PLAN

Is initial treatment to be directed primarily to:	
Source of symptoms	☐
Contributing factors	☐

Objectives of physiotherapy treatment (prioritise)	**Treatment indicators** (based on which assessment findings)

Technique/s of treatment for each objective	Criteria for achievement of objectives	Date

Additional factors influencing first choice
Most appropriate to achieve functional goals (explain)   Patients level of understanding Patients expected the treatment Treatment worked before Expected compliance of patient Personal preference of therapist Time available Newly acquired skill Increase patient satisfaction/confidence Enhance motivation of patient Other:

Therapist:

Date:

REFLECTION ON PATIENT AGREED FUNCTIONAL GOALS
DATE:

Have the short terms goals been achieved?
YES ☐ NO ☐

If "no", why do you think they have not been achieved?
(what clues, if any, can you now recognise that you initially missed, misinterpreted, under or over-weighted)

How has your understanding of the patients problem changed from your interpretations made after the initial assessment?

Modified Functional Short Term Goals	**Anticipated Treatments**	**Time (weeks)**	**Actual Treatments**	**Time (weeks)**
? Modified Functional Discharge Goals	**Anticipated Treatments**	**Time (weeks)**	**Actual Treatments**	**Time (weeks)**

REVIEW OF FUNCTIONAL MILESTONES

	N/A	**Y**	**N**
Is the patient still off work	☐	☐	☐
Are usual leisure and social activities still affected	☐	☐	☐
Is the patients usual sleeping pattern still disturbed	☐	☐	☐

Time to resumption
(weeks from onset)_____

ON DISCHARGE

DATE:

Have functional discharge goals been achieved?
YES ☐ NO ☐

Persisting symptoms:

Pain		Mild ☐	Moderate ☐	Severe ☐
Irritability	Non ☐	Mild ☐	Moderate ☐	Severe ☐

Medication:

Persisting signs:
ROM:

Joint:

Neural:
Slump:

SLR R L

Other:

Prognosis (do you feel the problem is likely to recur and why?)

REVIEW OF FUNCTIONAL MILESTONES

	N/A	Y	N
Is the patient still off work	☐	☐	☐
Are usual leisure and social activities still affected	☐	☐	☐
Is the patients usual sleeping pattern still disturbed	☐	☐	☐

Time to resumption
(weeks from onset)_____

Appendix VIII: Clinical Reasoning Form B

GROUP B GOALS & CLINICAL REASONING

EXPECTED GOALS (2 maximum)

Functional Short Term Goals	**Anticipated Treatments**	**Time (weeks)**	**Actual Treatments**	**Time (weeks)**
Functional Discharge Goals	**Anticipated Treatments**	**Time (weeks)**	**Actual Treatments**	**Time (weeks)**

SOURCE OF SYMPTOMS (Structures from which the symptoms are emanating)			
Component	**Structure**	**Supporting evidence**	**Negating evidence**

PAIN MECHANISMS (Evidence for mechanisms by which the symptoms are being initiated and/or maintained neurologically)			
Nociceptive mechanism			
Peripheral neurogenic			
Central mechanisms			
Autonomic			
Psychological/mental processing			

NATURE OF THE DYSFUNCTION			
Component	**Structure**	**Supporting evidence**	**Negating evidence**

CONTRIBUTING FACTORS
(Predisposing or associated factors involved in the development or maintenance of the patients problem)

SEVERITY, IRRITABILITY & CAUTION
(Incl stability, progression GH & SQ)

Pain		Mild ☐	Moderate ☐	Severe ☐
Irritability	Non ☐	Mild ☐	Moderate ☐	Severe ☐

other

Do the signs fit with the symptoms?

Clinical impression/principal hypothesis regarding the primary syndrome/disorder

PROGNOSIS	
Favourable	**Unfavourable**

Therapist: **Date:**

MANAGEMENT PLAN

Is initial treatment to be directed primarily to:

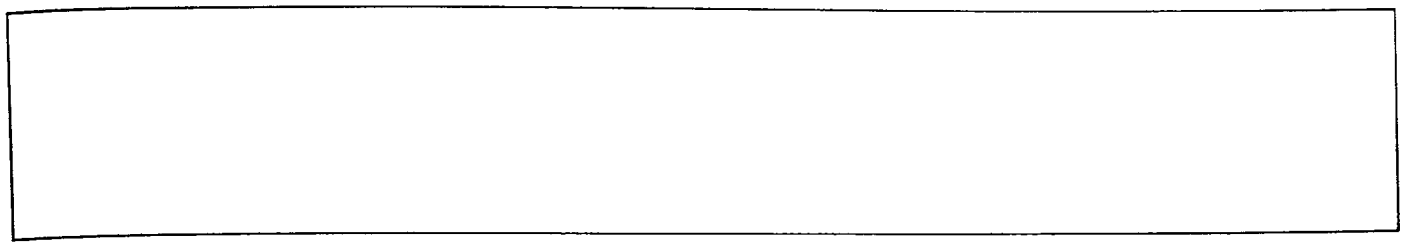
Source of symptoms ☐

Contributing factors ☐

Objectives of physiotherapy treatment (prioritise)	**Treatment indicators** (based on which assessment findings)

Technique/s of treatment for each objective	**Criteria for achievement of objectives**	**Date**

Additional factors influencing first choice
Most appropriate to achieve functional goals (explain)   Patients level of understanding Patients expected the treatment Treatment worked before Expected compliance of patient Personal preference of therapist Time available Newly acquired skill Increase patient satisfaction/confidence Enhance motivation of patient Other:



Therapist:

Date:

REFLECTION ON PATIENT AGREED FUNCTIONAL GOALS
DATE:

Have the short terms goals been achieved?
YES ☐ NO ☐

If "no", why do you think they have not been achieved?
(what clues, if any, can you now recognise that you initially missed, misinterpreted, under or over-weighted)

How has your understanding of the patients problem changed from your interpretations made after the initial assessment?

Modified Functional Short Term Goals	**Anticipated Treatments**	**Time (weeks)**	**Actual Treatments**	**Time (weeks)**
? Modified Functional Discharge Goals	**Anticipated Treatments**	**Time (weeks)**	**Actual Treatments**	**Time (weeks)**

REVIEW OF FUNCTIONAL MILESTONES

	N/A	**Y**	**N**
Is the patient still off work	☐	☐	☐
Are usual leisure and social activities still affected	☐	☐	☐
Is the patients usual sleeping pattern still disturbed	☐	☐	☐

Time to resumption(weeks)_____

ON DISCHARGE

DATE:

Have functional discharge goals been achieved?
YES ☐ NO ☐

Persisting symptoms:

Pain		Mild ☐	Moderate ☐	Severe ☐
Irritability	Non ☐	Mild ☐	Moderate ☐	Severe ☐

Medication:

Persisting signs:
ROM:

Joint:

Neural:
Slump:

SLR R L

Other:

Prognosis (do you feel the problem is likely to recur and why?)

REVIEW OF FUNCTIONAL MILESTONES

	N/A	Y	N
Is the patient still off work	☐	☐	☐
Are usual leisure and social activities still affected	☐	☐	☐
Is the patients usual sleeping pattern still disturbed	☐	☐	☐

Time to resumption (weeks)_____

APPENDIX IX: PEDro SCORING SYSTEM

One point is assigned for each of the following categories.

1. Random allocation
2. Concealed allocation
3. Baseline comparability
4. Blind assessor
5. Blind Subjects
6. Blind therapist
7. Adequate follow up
8. Intention to treat analysis
9. Between group comparisons
10. Point estimates and variability