THE HUMAN EXTREMITIES MECHANICAL DIAGNOSIS & THERAPY

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THE HUMAN EXTREMITIES MECHANICAL DIAGNOSIS AND THERAPY

BY ROBIN MCKENZIE AND STEPHEN MAY

The ability to treat musculoskeletal disorders affecting the soft tissues and joints of the body is a relatively modern concept. In the past, pain, stiffness and injury were simply tolerated until the body adapted or broke down completely. Repetitive Strain Injury, 'housemaid's knee', tennis elbow and general physical dysfunction were accepted as part of life's ageing process. However, improved social structure and treatment for these conditions mean that people expect to be able to regain good health, and want instant remedies for their pain.

Numerous health care providers – family physicians, physiotherapists, chiropractors, acupuncturists, naturopaths and osteopaths, among others – treat the painful symptoms of musculoskeletal conditions. These practitioners are sometimes loath to admit defeat when the patient does not respond to traditional treatment. Patients put their clinician in charge of curing their condition and expect them to "do something". Often the patient is the first to give up, resigned to the belief that they cannot regain their physical health.

The authors draw upon current published scientific evidence which overwhelmingly identifies controlled movement and exercise as the most effective means of alleviating pain and restoring function in patients with common musculoskeletal disorders. This latest text by Robin McKenzie and co-author Stephen May, allows the clinician to provide the same self-treatment philosophy and management strategies first described in McKenzie's text *The Lumbar Spine, Mechanical Diagnosis and Therapy*. These rarely discussed concepts are presented in straightforward language and are supported with over 100 photographs and illustrations.

Other books by Robin McKenzie include *Treat Your Own Back, Treat Your Own Neck, The Lumbar Spine: Mechanical Diagnosis and Therapy*, and *The Cervical and Thoracic Spine: Mechanical Diagnosis and Therapy.*

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The Human Extremities: Mechanical Diagnosis and Therapy

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Dedication

To my dear wife Joy.

Acknowledgments

I would like to acknowledge the very great contribution made by my friend and colleague Stephen May, MA, MCSP, Dip MDT, MSc, without whose assistance this book would never have been published. Mr May has created order out of chaos and provided this text with a credibility that I hope will help change the mode of physiotherapy management of musculoskeletal disorders worldwide.

I would also like to thank my daughter, Jan, who organised and coordinated the various specialists required to successfully complete a project such as this. I thank her for her patience and dedication.

I must also acknowledge the assistance of the many faculty members of the McKenzie Institute International for their helpful comments and criticism of the original text.

Robin McKenzie

For their helpful suggestions and comments concerning grammar and contents, I must thank Kenneth May and Julie Shepherd.

Stephen May

About the Authors

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

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

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

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

Robin McKenzie has authored four books: *Treat Your Own Back*; *Treat Your Own Neck*; *The Lumbar Spine: Mechanical Diagnosis and Therapy*; and *The Cervical and Thoracic Spine: Mechanical Diagnosis and Therapy*. With the publication of *The Human Extremities: Mechanical Diagnosis and Therapy*, Robin McKenzie, in collaboration

with Stephen May, describes the application of his methods for the management of musculoskeletal disorders in general. As with his publications dealing with spine-related problems, the emphasis in this text is directed at providing self-treatment strategies for pain and disability among the general population.

Stephen May was born in Kent, England, in 1958. He did his physiotherapy training in Leeds and graduated in 1990. Since qualifying as a physiotherapist, Stephen has worked in the National Health Service in England. For the last eight years this has been in Primary Care.

Early on he developed a passionate interest in musculoskeletal physiotherapy which ensured he was a keen consumer of the research evidence. In 1995 Stephen successfully completed the McKenzie Diploma programme and in 1998 an MSc in Health Services Research and Technology Assessment.

Stephen believes strongly that practice must be shaped by the evidence and that self-management concepts are the future of physiotherapy.

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Introduction

Mechanical disorders affecting the soft tissues and joints of the human extremities are encountered daily by general practice physicians, physiotherapists, chiropractors, osteopaths and a myriad of alternative health care providers. Whether the problems arise subsequent to injury or from inflammatory or degenerative processes, the consequences are pain, stiffness and limitation of function or activity, or all of these. The duration of impairment, pain or disability can vary from a few days in the case of minor injury to months or even years in some conditions. Fortunately all living bodies, human and animal, have remarkable capacity to heal and repair. Animals in the wild can recover and survive from apparently catastrophic injury. Skeletal remains of prehistoric man show similar ability to survive both injury and degeneration. The natural healing process has allowed living things to recover from all but fatal injury. The remodeling of soft tissue and bone alike is evidence of the body's inherent capacity to repair, adapt and survive.

Our ancient forebears had no knowledge of the mechanism of healing or medication to relieve pain. They must have discovered in very early times, however, that failure to restore activity levels meant certain rapid decline and eventual death. Fortunately mankind survived, and did so in those early times without the expertise of numerous therapists and physicians.

A century ago, only those suffering severe injury causing fracture or dislocation sought the services of a physician. Everyday problems such as sciatica, lumbago, tennis elbow, housemaid's knee and wry neck were most often managed with home remedies or by bonesetters. A 'hot iron on vinegar and brown paper' or 'stinging nettle' were commonly applied as counter-irritants to painful disorders until the natural healing process brought eventual relief. People in pain went about their daily tasks. Minor aches and pains were frequently disregarded, considered trivial and were viewed as another of life's hurdles either to overcome or to bear. They put up with the pain of repetitive strain and occupational overuse until their structures adapted. At the beginning of this century, to do otherwise would have meant starvation.

The chemical and mechanical tools and the economic capacity that allow us to apply many diverse treatments to musculoskeletal disorders for the alleviation of pain and disability have only been available in comparatively recent times. Improved social support structures now sanction sick leave for workers with musculoskeletal problems. These conditions have created an extremely costly situation for society.

In the Western world in modern times, the family medical practitioner, the physiotherapist, naturopath, acupuncturist, chiropractor, osteopath and others are consulted to provide relief and reassurance to those with injuries and aches and pains that long ago would have been silently borne until nature had her way. In modern times a common response to musculoskeletal complaints is to seek help from one or many of those offering a solution.

So successful have modern health care providers been in creating patient dependency on therapy and therapists that the advice usually proffered by one layman to another complaining of pain is routine: 'you had better go and see someone about that!' There exists in Western populations a widespread belief that for healing to occur it is essential to have some sort of treatment. Unfortunately, clinicians in training are commonly led to the same belief by their teachers. The desire of the public to receive treatment for many minor injuries or pains has led to an explosion in the demand for services and expenditure within health services worldwide.

This demand has occurred especially in the field of physiotherapy services. As an example, in 1953, in the city of Wellington, New Zealand there were seven physiotherapists serving a population of 138,000 people. In 1998, 61 clinics served a population of 159,000, a ratio of one therapist for every 2,600 people. There is no evidence to suggest that the prevalence of musculoskeletal problems in the community has escalated in a similar way.

The increase in consumption of therapy services seems hard to justify and more difficult to explain. Only a small percentage of musculoskeletal injuries actually require professional assistance during the period of healing and repair. Patients may need education and advice about the most effective way of returning to full function following soft tissue trauma, but offering treatment of dubious efficacy only detracts from the importance of the patient's role in this recovery.

Patients with degenerative joint disease and chronic musculoskeletal problems certainly require education in self-management, but it is highly unlikely that they require traditional modality-based passive physiotherapy.

Health care providers allied to medicine have two great persisting handicaps. The first is the lack of understanding of the meaning and significance of the phrase 'natural history'. The term is rarely part of the vocabulary of our teachers and is hard to find in textbooks used in physiotherapy, chiropractic and osteopathic schools of education. Yet it is upon the natural history of mechanical disorders that so many of us rely for our survival as health care professionals.

The second great handicap is the inability or reluctance of those in clinical practice to acknowledge defeat. There is a general tendency by clinicians to persist with treatment long after all available methods have been found wanting. There seems to be a reluctance among health care providers to acknowledge their limitations. The result is a continuing search for an elusive panacea – a new technique of manipulation or a new form of electrotherapy that will solve the problem when all else has failed.

It is not generally recognised that there is little scientific evidence supporting the use of any of the current physiotherapy modalities for the treatment of musculoskeletal disorders. There is, however, ample evidence that the application of movement and the repeated and prolonged functioning of all structures affected by injury provides the best stimulus for repair and the best environment in which to achieve full recovery of function and pain-free status.

Where significant injury causes the patient persisting pain, impairment of function and reduced mobility, physiotherapy by way of education and supervision may be indicated. Physiotherapy cannot accelerate healing, but education and supervision can ensure that healing is not disrupted and that the environment for recovery is optimal. The main function of the physiotherapist of the future will be the education and enlightenment of the injured and ignorant.

Patient education is one of the main themes of this text. Patients must be provided with sound explanations and should comprehend the need to maintain the correct environment for managing their problem. It is also necessary for the patient to understand that the

recovery of function can only commence following adequate healing and repair, and requires an appropriate rehabilitation process. These processes may take time, but recovery is impossible without the patient's active involvement.

This text does not attempt to provide the reader with a detailed description of diagnostic procedures usually expected in a book about musculoskeletal disorders of the extremities. I no longer believe that the precise identification of the structure involved is necessary or a prerequisite for the prescription and safe delivery of appropriate mechanical therapeutic interventions; nor do I believe it is always possible to make such an identification. Irrespective of the structure involved, ultimately the mechanical therapeutic strategy is determined solely from the responses obtained from tissue loading and the effect that loading has on symptomatology. This book is not intended to fill a gap for those in search of an additional technique of mobilisation or manipulation.

This book is written to encourage appropriate health care professionals to provide sound and effective self-management for their patients. The advice provided is aimed at allowing the patient to take responsibility for his or her own recovery. It is hoped that in departing from the usual 'hands on' pathway so dear to the hearts of many clinicians, the emphasis will shift from patient dependency on therapy to patient independence. The truth is that the vast number of treatments delivered by health care providers for the alleviation of musculoskeletal aches and pains are usually unnecessary; certainly their effectiveness is unproven. Eventually, all physiotherapists involved in the field of orthopaedic medicine and therapy must provide the education that will enable the patient to practice 'autotherapy'.

The main themes touched on here are developed throughout the book. The prevalence of musculoskeletal diseases, the large numbers of people who have persistent problems and the lack of efficacy of many commonly used treatments are examined. The healing process of soft tissue injuries, the mechanical, inflammatory and chronic pain mechanisms, and the mechanical syndromes of the McKenzie approach as related to the peripheral joints are described. Information on how to gather data about patient problems, assess changes and manage different patient conditions is also presented. Some common disorders are described and appropriate self-treatment strategies are

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recommended. It is hoped that this text will support those clinicians who are perplexed by the failure of many interventions to 'cure' their patients' problems, and who seek endorsement for an approach that demands patient involvement as the only logical way forward for the physiotherapy profession.

1: Musculoskeletal Problems

Introduction

This first chapter is intended to give a brief overview of the problem of musculoskeletal pain in the general population. Health problems are located in broad contexts and should not be considered only through the narrow focus of the clinic. If we know more about the prevalence, the natural history, and the effect of treatment upon a condition, then our understanding of a clinical problem is considerably improved. The size of the problem, its persistence and the health care services' ability to deal with it also have repercussions upon management.

Diagnosis in musculoskeletal medicine is in its infancy. Within spinal problems specific diagnoses have been replaced by broad, non-specific categories (Spitzer 1987; Rosen *et al.* 1994). In peripheral problems, specific diagnostic criteria are used more widely with an underlying assumption of reliability, which is, however, barely endorsed by the studies available. This chapter will also look briefly at some of the issues surrounding the problem of making a diagnosis. The implications that these issues have upon the management of musculoskeletal problems will be discussed.

Prevalence

Musculoskeletal conditions of all types are extremely common in the general population. They are frequently found in population surveys when individuals not receiving treatment are asked about problems. These studies give a reasonably accurate picture of the extent of a particular problem in the community. Population surveys in the UK show that musculoskeletal problems, spinal and non-spinal, are the most frequently reported long-standing illness in all age groups (Bennett *et al.* 1995).

There is a linear increase in these conditions with age. Less than 10% of those under 34 are affected, rising to 32 – 49% of those over 75 (Bennett *et al.* 1995; Badley and Tennant 1992). After 45 years of age over 10% of the population are affected by multiple joint problems. Difficulty with daily functions and lack of independence, as a consequence of these problems, rise linearly to about 50% in those over 85 (Badley and Tennant 1992).

Figure 1.1 Chronic conditions in the general population. Rate of reporting selected conditions per 1,000 adults. *UK* (*Bennett et al. 1995*).

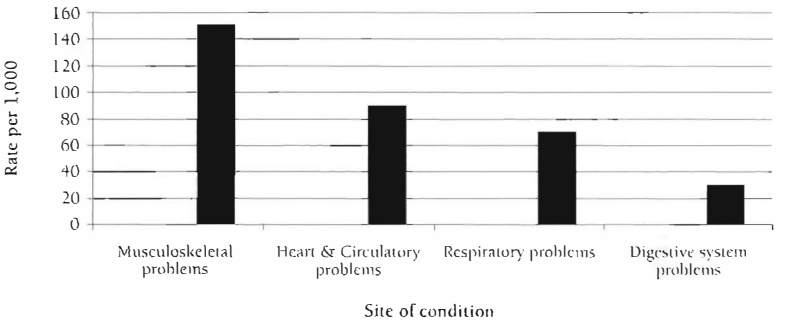
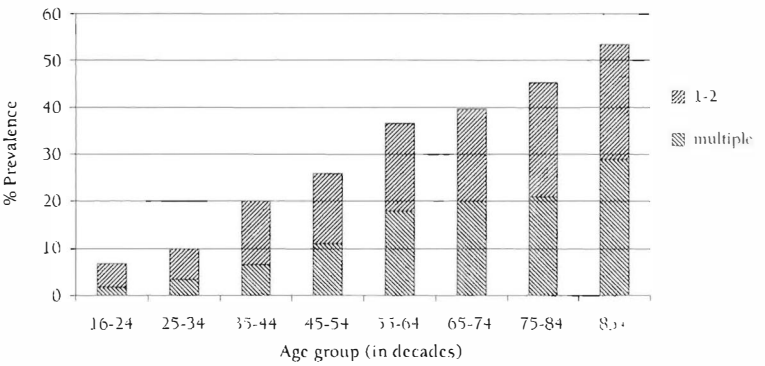


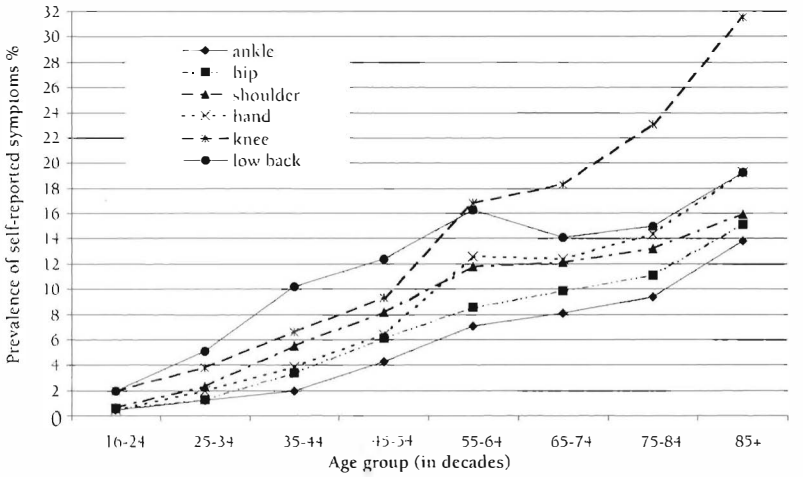
Figure 1.2 Prevalence of joint problems in the general population by age range. *UK* (*Badley and Tennant 1992*).



Problems were evenly divided between back problems, 'other bone and joint problems', and 'arthritis and rheumatism' (Bennett *et al.* 1995). While the prevalence of spinal problems reached a plateau in those older than 55, non-spinal problems continued to increase after this age. Musculoskeletal problems are more common in women at all ages.

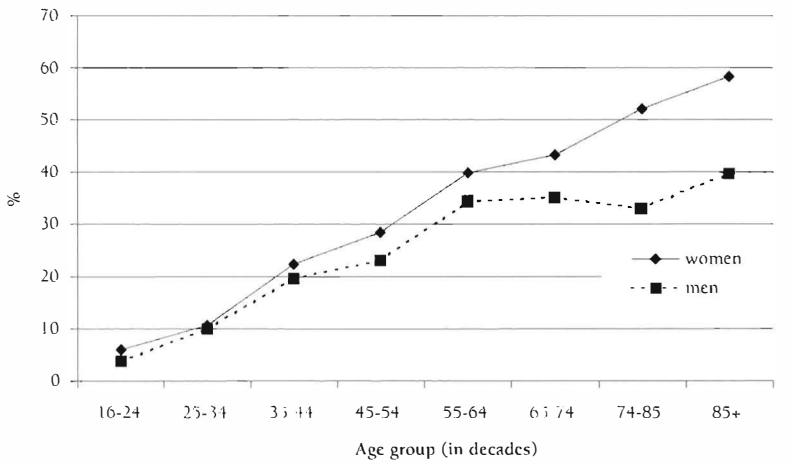
Surveys of general populations in other countries give similarly significant proportions that report all types of musculoskeletal symptoms. In Denmark, long-standing musculoskeletal problems are prevalent in 13% of the population, and make up 30% of all chronic illness (Bredkjaer 1991). They constitute the largest cause of chronic disease in Finland (Sievers and Klaukka 1991). In the US the prevalence rate of musculoskeletal illness is 30% (Cunningham and Kelsey 1984) and in Canada 16% (Lee *et al.* 1985). In Sweden

Figure 1.3 Reported prevalence in the general population of certain joint problems by age groups. *UK (Badley and Tennant 1992).*



10 – 20% of the sample population reported obvious musculoskeletal pain, most with pain persisting for more than six months (Brattberg 1989), and in those over 45 years of age, 35% reported long-lasting joint complaints (Larsson *et al.* 1991). There is some evidence of an increase in reporting of musculoskeletal problems and consequent health service use in Sweden in the last few decades (Sievers and Klaukka 1991; Slatis and Ruusinen 1991). There is some suggestion that social and economic factors, as well as medical ones, play a role in determining whether a person with these symptoms goes on to develop disability related to their musculoskeletal impairment (Cunningham and Kelsey 1984)

Figure 1.4 Reported prevalence in the general population of joint problems by men and women. *UK (Badley and Tennant 1992).*



A common musculoskeletal problem is shoulder pain. Populations in Sweden and England who were 50 or older reported painful shoulders in 14 – 34% of the samples investigated (Bergenudd *et al.* 1988; Allander 1974; Chard *et al.* 1991; Chakravarty and Webley 1990). In a Nigerian village, 14% of a community-based sample were found to have shoulder problems (Adebajo and Hazleman 1992) Across all ages, 7% of the population have been found to have shoulder conditions. This makes it the most frequent musculoskeletal complaint after back and knee problems (Badley and Tennant 1992)

Osteoarthritis is the most common source of joint problems throughout the world. This disease accounts for considerable disability to individuals, as well as a considerable cost in health services to society (Felson 1988; Felson and Zhang 1998). The prevalence of osteoarthritis from the middle years onwards is about 5%, although obviously this increases dramatically with the oldest groups (Petersson 1996; Felson and Zhang 1998). Radiographic degenerative changes become so common in older age groups that it can be said to be normal. These changes are present in the majority of people by 65 (Felson 1988); however, only 30 – 40% of those with radiographic changes have symptoms (Felson 1988). The hands, knees and hips are the most common sites of osteoarthritis, in that order (Petersson 1996). For instance, the prevalence of knee osteoarthritis reported by several studies ranges from 12 – 38% of subjects surveyed (Spector and Hart 1992). Osteoarthritis was the diagnosis given in about a third of patients attending an orthopaedic outpatient department in the UK (Byles and Ling 1989).

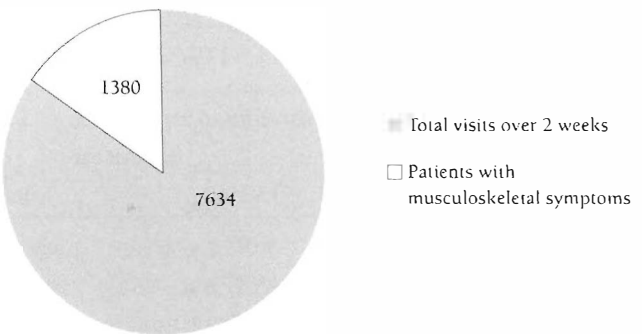
In summary, about a quarter of all populations surveyed have musculoskeletal conditions, including osteoarthritis. This is the figure of those affected in the general community, not those seeking treatment. There is increasing prevalence with age, and women are more at risk than men. Musculoskeletal conditions of all kinds constitute one of the biggest causes of chronic disease.

Health care

The high prevalence rate in the general population is reflected in the frequency with which individuals consult medical practitioners with musculoskeletal aches and pains. For instance, in the UK, 15% of the population consulted their GP because of a musculoskeletal complaint in 1991 (McCormick *et al.* 1995). In three general

practices, about 15% of GP consultations related to spinal and non-spinal joint and soft tissue problems (Hackett *et al.* 1993). The consulting rate was low among those under 44, but then increased steadily with age, with rates higher among women of all ages. The most common three groups of problems, with roughly equal proportions, related to back pain, arthropathies and 'rheumatic complaints', including other joint, muscular and soft tissue problems. Some 5% of general practice consultations are reported to be related to shoulder disorders (van der Heijden *et al.* 1997). Two per cent of the total population consulted for tendonitis or bursitis, mostly at the shoulder and elbow (McCormick *et al.* 1995). In a rural Finnish population, musculoskeletal problems accounted for 27% of adult visits to their primary care physician, and 21% of all visits (Rekola *et al.* 1993).

Figure 1.5 **Musculoskeletal symptoms in those attending primary care. *Finland (Rekola 1993).***



Not everyone consults with these types of problems. In Chard's (Chard and Hazleman 1987; Chard *et al.* 1991) surveys of 70-year-olds, about 25% had shoulder pain. Of the surveys, only 14% and 35% respectively of those with pain sought medical attention; of these, in one survey, only 40% were given any form of treatment. Frequently patients become disillusioned with health care, and after treatment failure they stop attending (van der Windt *et al.* 1996; Chakravarty and Webley 1990).

Musculoskeletal problems constitute an extremely common health problem in the community, with the burden being greatest in older populations. They represent the most common cause of persistent symptoms and functional disability in the general population. They are also a very common reason that people seek health care services.

The site of the problems

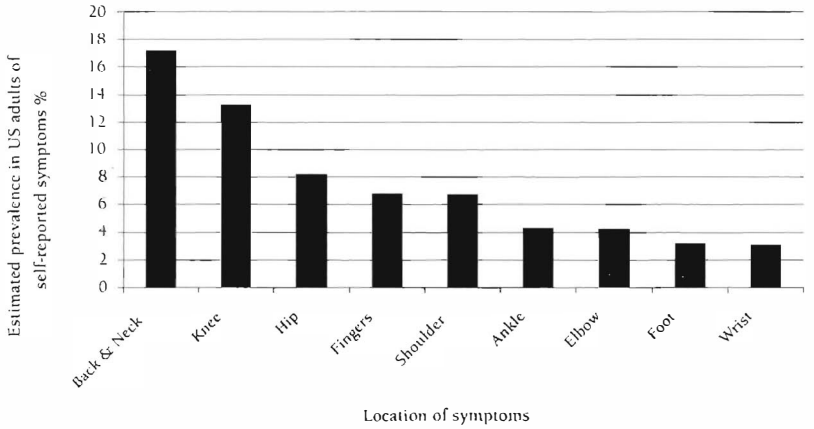
Clearly musculoskeletal complaints are a common cause of morbidity in the population at large and represent a large burden to health care organisations. In this instance we are concerned specifically with problems in the limbs. In the general population the prevalence of peripheral joint problems is much greater than spinal problems (Badley and Tennant 1992; Cunningham and Kelsey 1984). In surveys in which the site of all disorders has been collected less than 20% report spinal problems, whereas 44 – 49% report pain in the extremities (Badley and Tennant 1992; Cunningham and Kelsey 1984). Half, or more, have symptoms in more than one site (Larsson *et al.* 1991; Cunningham and Kelsey 1984), with the most common sites affected being the shoulders, hands, knees and hips.

Table 1.1 Prevalence of musculoskeletal problems by site in general population

Site of pain	*Badley*	*Cunningham*	*Larsson*
lumbar	10%	17% (all spinal)	
cervical	6%		
shoulder	7%	7%	15%
elbow	3%	4%	6%
wrist	3%	3%	9%
hand	7%	7%	15%
hip	5%	8%	13%
knee	10%	13%	17%
ankle	4%	4%	10%
foot	5%	3%	6%

However, when it comes to those who seek health care services for musculoskeletal problems, more people with spinal pain appear to come forward, as non-spinal joint and soft tissue problems represent about half of the load of musculoskeletal morbidity seen in clinics (Hackett *et al.* 1993; Peters *et al.* 1994; Hockin and Bannister 1994; Rekola *et al.* 1993). Comparing data from different countries suggests that spinal problems in the general population represent about 25% of musculoskeletal disorders (Cunningham and Kelsey 1984; Badley and Tennant 1992); however, in primary care they constitute nearly half of all musculoskeletal conditions (Rekola *et al.* 1993; Hackett *et al.* 1993; Peters *et al.* 1994). In surveys of one GP practice (Hackett *et al.* 1993), one orthopaedic out-patient department (Hockin and

Figure 1.6 Distribution of different locations of musculoskeletal symptoms in the general population. *US (Cunningham and Kelsey 1984).*



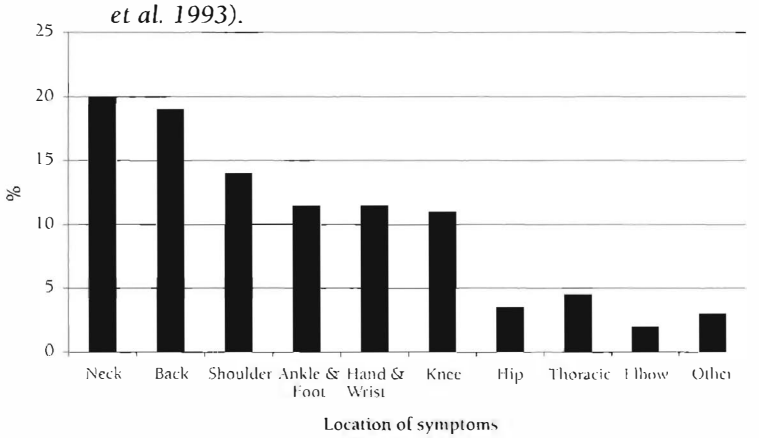
Bannister 1994), both in the UK, and one practice in a rural primary care setting in Finland, the average prevalence of problems by site was as follows:

Table 1.2 Prevalence of musculoskeletal problems in those who seek treatment

Site of pain	*Hackett*	*Hockin*	*Rekola*
lumbar spine	30%	35%	18%
cervical spine	15%	12%	20%
shoulder	22%	8%	14%
soft tissue injuries	11%		
knee	10%	21%	11%
ankle/foot	6%	20%	11%
tennis/golfer's elbow	4%	4%	2%
wrist/hand			11%
hip			4%

These figures suggest that peripheral musculoskeletal problems represent at least half of all musculoskeletal conditions seen in primary care and hospital settings, and that the most common sites of peripheral problems that patients seek help for are shoulders, knees and ankles. However, they also show that the proportion of different conditions may vary quite considerably in different clinical settings.

Figure 1.7 **Distribution of different locations of musculoskeletal symptoms in those visiting primary care.** ***Finland (Rekola et al. 1993).***



Natural history and clinical course

Although the natural history of musculoskeletal problems is frequently thought to be good and the outlook with treatment is often viewed optimistically, the evidence suggests otherwise. As several of the above studies make clear, large numbers of the population have these conditions for long periods of time (Bennett *et al.* 1995; Bredkjaer 1991; Sievers and Klaukka 1991; Brattberg *et al.* 1989). Studies show increasing prevalence of these conditions with age, suggesting that degenerative changes and cumulative trauma has a role in their aetiology and chronicity (Badley and Tennant 1992; Cunningham and Kelsey 1984).

For instance, many studies have found that shoulder pain has persisted or recurred in at least 40% of those patients studied when followed up for a year or more. Treatments including corticosteroid injections, anti-inflammatory medicine (NSAID), manipulation and physiotherapy have often conferred only short-term benefit (Croft *et al.* 1996; van der Windt *et al.* 1996; Bergenudd *et al.* 1988; Vecchio *et al.* 1995; Chard and Hazleman 1987; Winters *et al.* 1999). Variables that predicted poor outcome were concomitant neck pain (odds ratio 2:8), high baseline pain score (odds ratio 2:0), a long duration of symptoms at presentation (odds ratio 0:5), and initial referral to physiotherapy (odds ratio 0:4, compared to no treatment or medication only) (van der Windt *et al.* 1996).

Many patients do not persevere with seeking treatment despite persistent symptoms. They frequently try health care once or twice. When this does not bring relief they cease attending, but are left with chronic symptoms that they are forced to tolerate. This group with persistent symptoms may be as high as 50% of those originally coming forward for treatment (van der Windt *et al.* 1996; Chakravarty and Webley 1990). This can give the mistaken appearance of resolution of a problem that in fact persists and is simply tolerated, and an illusion of treatment efficacy that is illusory. There are suggestions in the literature that shoulder pain recurs, and that many individuals may have several episodes prior to the problem becoming chronic (Winters *et al.* 1997; van der Windt *et al.* 1996).

Frozen shoulder is a condition that is frequently said to recover spontaneously within six to eighteen months (Rizk and Pinals 1982). However, certain studies show its effect can last for much longer, and that treatment has little effect on outcome. Bulgen *et al.* (1984) compared intraarticular steroids, mobilisations, ice therapy and no treatment over a six-month period and found that all groups improved, but that there was little long-term advantage in any treatment regime. When these patients were reviewed around four years later, about half were still found to have persistent aching or restriction of movement, with greater restriction in those patients who had received mobilisation (Binder *et al.* 1984). Shaffer *et al.* (1992) also found about 50% of their study group had mild pain and/or stiffness when reviewed at about seven years. In a long-term follow-up of patients with rotator cuff tendonitis who were treated with NSAIDs, injection or exercise, nearly 50% were still symptomatic a year and a half later (Chard *et al.* 1988).

Wadsworth (1987) states that conservative management is successful in 90% of cases of lateral epicondylitis. Although patients frequently improve with ultrasound or injection, when reviewed up to five years later about 50% of study groups had pain and functional limitations, although few had sought further treatment for the problem (Binder and Hazleman 1983; Clarke and Woodland 1975; Hamilton 1986). 'Tennis elbow' clearly has a high rate of relapse and failure to resolve.

The evidence would thus suggest that the experience of persistent musculoskeletal problems, which are resistant to treatment, is very common in the general population. The lack of long-term follow-up and the withdrawal of patients from therapy generate the illusion of

therapeutic efficacy for many interventions. However, for a vast number of patients, the reality is persistent pain and functional limitations.

Systematic reviews of commonly used treatments

Systematic reviews of randomised controlled trials, because they pool the results of several studies, are said to be the optimal way to judge the efficacy of different treatments (Gray 1997). Systematic reviews frequently rate the trials poorly against the criteria by which they judge the methods used in that trial. They also generally find little firm evidence to support commonly used interventions for musculoskeletal problems.

"The evidence in favour of the efficacy of steroid injections for shoulder disorders is scarce" (van der Heijden *et al.* 1996).

"There is little evidence to support or refute the efficacy of common interventions for shoulder pain" (Green *et al.* 1998). This review looked at treatment with anti-inflammatory (NSAID) tablets, injections and physiotherapy.

"There is evidence that ultrasound therapy is ineffective in the treatment of soft tissue shoulder disorders... Evidence for the effectiveness of other methods of physiotherapy is inconclusive" (van der Heijden *et al.* 1997).

One group reviewed the use of ultrasound, ionisation, oral NSAID and steroid injections for lateral epicondylitis and *"concluded that there was insufficient scientific evidence to support any of the current methods of treatment"* (Labelle *et al.* 1992).

A systematic overview of steroid injections for lateral epicondylitis found a favourable treatment effect at six weeks, but a lack of long-term effect after this (Assendelft *et al.* 1996).

"There seems to be little evidence to support the use of ultrasound therapy in the treatment of musculoskeletal disorders" (van der Windt *et al.* 1999)

Systematic reviews and meta-analyses have an inherent problem in that they are only as good as the clinical trials they draw their conclusions from. In the attempt to make a general conclusion, trials with different procedures, measuring tools and outcomes are lumped together with the assumption that greater generalisable 'truths' can be gained from disparate studies. While the quality of methods is

strictly adhered to, the quality or exact type of intervention may be obscured. Although it is certainly pertinent to question the validity of their conclusions, in all instances the message from these reviews cannot be ignored. They testify to the inconclusive treatment effect of many commonly used interventions

These findings in fact accord with what is clear from the epidemiological literature outlined earlier – that many people have persistent or recurrent musculoskeletal problems that are either resistant to treatment or respond only temporarily. It suggests we should begin to reformulate the problem – not what can we do for our patients, but what can our patients do for themselves. This is bound to involve exercise therapy.

The only intervention that consistently appears beneficial across a wide range of spinal and non-spinal musculoskeletal problems is exercise.

"One can draw a number of conclusions, however. First, and importantly, exercise appears to be the best modality. Strengthening muscles protects the joint. Secondly, most of the machines that plug into the wall are acting as flashy placebos" (Clarke 1999).

After reviewing a wide range of interventions Feine found *"there is little evidence that any of the therapies under review cause improvements in symptoms of chronic musculoskeletal pain or in quality of life that outlast the therapy..."*. Only two trials reported positive long-term treatment effects. *"The treatment groups in both of these trials received exercise"* (Feine and Lund 1997).

"In common with other areas of musculoskeletal rehabilitation, the evidence strongly supports exercise (except possibly in the rare case of true radicular back pain) and a cognitive behavioural approach to pain management" (Haigh and Clarke 1999)

"Exercise reduces pain and improves function in patients with osteoarthritis of the knee" (Puett and Griffin 1994).

The available evidence does not support the treatment efficacy of many commonly used interventions for musculoskeletal problems. The one exception to this general rule is the widespread finding that exercise is beneficial across a variety of disorders. Rather than looking to short-term curative treatment solutions meted out by clinicians, we should be offering education and self-management strategies for patients to use which will be of long-term benefit.

The problem of diagnosis

Musculoskeletal problems about the shoulder are clearly common. This affects about 10% of the population at some point, and 5% of all consultations in general practice relate to pain about the shoulder (van der Heijden *et al.* 1996, 1997). What sort of problems are these? The commonly given diagnoses by GPs and physiotherapists in the UK and Holland are as follows:

Table 1.3 Diagnosis given by clinicians for shoulder conditions

Diagnosis	*Windt*	*Croft*	*Liesdek*	*Chard*
Rotator cuff injuries	30%		47%	70%
Capsulitis	22%	40%	17%	4%
Acute bursitis	17%		7%	
Chronic bursitis	13%		9%	
Acromio-clavicular joint	4%		6%	18%

These findings suggest considerable variety in what different clinicians think they are seeing. These kinds of diagnoses are commonly made in the clinic and are based on the ideas and criteria proposed by James Cyriax (1982), the founding father of orthopaedic medicine. The concepts, although widely accepted, have rarely been subjected to detailed scrutiny. Two key issues have not been fully investigated – the validity and reliability of the tests used to make these diagnoses. Validity relates to the ability to measure what is intended. Reliability is the extent to which a measure is reproducible, or gives the same results, in different situations (Streiner and Norman 1996). If different clinicians come to different conclusions using the same test, clearly that test is of little value in deciding treatment strategies. Reliability of a test between clinicians is a first step in evaluating the usefulness of diagnostic pathways to treatment.

Work done in this area is contradictory. In nearly 400 cases of shoulder pain, 141 were examined twice by their physician; in nearly half of these the initial diagnosis was changed (van der Windt *et al.* 1996). In other words, on subsequent analysis the GPs altered their diagnosis. Of these patients, 120 were referred to a physiotherapist who also diagnosed the patient according to the same diagnostic criteria. Cohen's Kappa is the statistical test used to evaluate agreement between clinicians as it takes into account the proportion of agreement that may occur by chance (Streiner and Norman 1996). The overall strength of agreement between the GPs and the physiotherapists was

Kappa 0.31 (Liesdek *et al.* 1997). This reflects only a 'fair' concordance between the clinicians and seriously questions the reliability of these diagnostic criteria if different examiners can reach such different conclusions. However, experienced therapists using the Cyriax evaluation procedure were highly reliable (Kappa = 0.87) in selecting diagnostic categories in 19 of 21 patients with shoulder pain (Pellechia *et al.* 1996)

Poor levels of agreement have been found in tests used in the clinical assessment of knee problems. Although 'within observer agreement' of various tests used to evaluate osteoarthritis is moderate to excellent, 'between observer agreement' is poor to moderate (Jones *et al.* 1992; Cushnaghan *et al.* 1990). For both within and between observer variation, history-taking is much more reliably assessed than physical signs, and the agreement between observers about physical signs only was poor (Jones *et al.* 1992). Certain elements of the Cyriax examination of the knee have also been shown to have only poor to fair reliability. These were the judgements therapists made about end-feel and the sequence of pain and resistance with movement (Hayes *et al.* 1994). Four commonly used tests to evaluate patellofemoral malalignment have also been shown to have only poor to fair reliability (Kappa = 0.1 to 0.36) (Fitzgerald and McClure 1995)

It is clearly wise to be cautious about the reliability and thus the value of commonly used tests. Although they often have a reasonably good record of producing the same results when repeated by the same clinician, they generally have a poor record of producing the same results when used by different clinicians. If the tests are suspect, then the diagnoses reached by these tests may also be suspect. In this instance should we again be reformulating the question? Should we sometimes favour general diagnostic patterns, which suggest treatment directions, rather than specific diagnostic criteria that are unreliable? Should these general diagnoses be based upon symptomatic history-taking, which often appears to be more reliable than physical signs?

The tests that are commonly used to make diagnoses in musculoskeletal disorders are neither proven to be reproducible nor to measure what is intended. The diagnoses themselves may therefore be suspect and represent an untrustworthy base upon which to establish treatment protocols.

Conclusions

This chapter has presented the contextual background of musculoskeletal problems. Certain facts stand out:

- musculoskeletal problems are one of the most common conditions that affect people in the community
- such conditions are prevalent in all body regions, but especially in the large joints of the shoulder, knee, ankle and hip
- these problems are very often persistent and chronic, even in those who have received treatment
- such patients frequently seek health care services and make up a significant proportion of those who attend primary care physicians and physiotherapists
- treatment, although sometimes conferring short-term relief, frequently does not result in a cure of the problem
- the only intervention that consistently does appear to be able to confer benefit is a self-management approach using exercise and behavioural modification
- disillusionment with professional help and opting out of further treatment is common
- commonly used treatment options in musculoskeletal conditions have a very inconclusive record of efficacy in controlled trials and systematic reviews
- the validity and reliability of accepted diagnostic categories are uncertain, and the extent of non-specific musculoskeletal problems in the periphery is unknown.

These facts suggest a failure on the part of the medical model, which purports to diagnose, treat and cure. The approach outlined in this book proposes a different model, based upon non-specific syndrome classification and the overwhelming logic of endorsing the key role of self-management strategies in musculoskeletal medicine. If a condition is very common, persistent and resistant to easy remedy, it is time that the patient is fully empowered to deal with these problems in an optimal and realistic fashion. As clinicians we should be offering this empowerment to our patients.

2: Soft Tissue Properties

Introduction

The previous chapter has shown that pain in the peripheral joints and soft tissues occurs frequently in the general population and is a common cause for seeking health care services. The concern of this text is the problems that affect the soft tissues of the limbs. It is thus useful to review some of the properties of these tissues including how injury occurs, the effect of injury, the healing process, and what can inhibit the healing process. As it is vital to recollect that tissues are part of a whole being, some of the impact of behaviour on these properties will also be considered. How these tissues maintain their function through use and atrophy through disuse are also essential elements of appropriate management. Recognition must also be made of the role of cumulative trauma and insidious degeneration in the onset of some musculoskeletal problems. The clinical implications of these factors will be discussed.

Tissue injury

Tissue damage may result from a discrete traumatic episode with a sudden onset of pain, or it may arise insidiously due to cumulative trauma when overuse or degeneration is present. Although there has been no overt incident in these cases, repetitive loads can be sufficient to cause micro-trauma or degenerative changes to the tissues, overwhelm reparative processes and evoke an inflammatory response. Shoulder capsulitis, tenosynovitis and lateral epicondylitis all commence with an acute inflammatory period, and usually an insidious onset. Thus injury is the result of intrinsic or extrinsic factors, either alone or in combination with chronic conditions representing an interaction between the two categories (Kannus *et al*. 1997). Whatever the cause, if tissue damage has occurred the inflammatory process will be triggered (Levine and Taiwo 1994).

Recovery following trauma

Following tissue injury the process that theoretically leads to recovery is divided into three overlapping phases – inflammation, repair and remodelling (Evans 1980; Hardy 1989; Enwemeka 1989; Barlow and

Willoughby 1992; Norris 1993). "*No inflammation / no repair is a valid dictum*" (Carrico *et al*. 1984). In fact, each part of this process is essential to the structure of the final result. Connective tissue and muscle do not regenerate if damaged, but are replaced by inferior fibrous scar tissue (Evans 1980; Hardy 1989). To produce optimal repair tissue, all phases of this process need to be completed at the appropriate time.

Inflammation

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

In optimum conditions the inflammatory stage lasts less than five days, with a gradual reduction of inflammatory cells thereafter and none present at the end of the third week (Enwemeka 1989). In this period a mesh of fibrin forms from the protein fibrinogen in the inflammatory exudate and seals the injury. During this time the application of ice, compression, elevation and gentle muscle movements are indicated to reduce the inflammatory exudate (Evans 1980). The greater the amount of exudate, the more fibrin will be formed and the more inextensible will be the repair. Ice, if applied in the first few days following injury, can reduce pain and oedema. Ice is of little value after the fifth day as the inflammatory cells are replaced by fibroblasts. These soon begin to lay down fibrils of collagen

Tissue repair

The fibroplastic or repair stage thus commences as the acute inflammatory stage subsides and lasts about three weeks (Enwemeka 1989). It is during this phase that the collagen and

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

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

It should be noted, however, that at this stage if an over-enthusiastic approach to treatment is adopted, the repair process can be delayed or disrupted and the presence of inflammatory chemical irritants and exudate will be prolonged or re-stimulated. During this early stage of healing, movements should be just into stiffness and pain and entirely under the patient's control. Any discomfort provoked by the movement should abate as soon as the movement is released. If lasting pain is provoked it is likely that re-injury has occurred, the inflammatory phase has been re-triggered and resolution of the problem will be further delayed.

Remodelling

Wound repair is only optimal if remodelling of the scar tissue occurs. This involves increasing strength and flexibility of the scar tissue through progressively increased normal usage and specific loading. Remodelling is the process of turning weak, immature and disorganised scar tissue into a functional structure able to perform

normal tasks. The repair is unlikely to achieve the strength of the original tissue, but progressive loading and mechanical stimulation enhances the tensile strength and improves the quality of the repair. This occurs over several months after the original injury. Tensile strength is increased by stabilisation of the fibres through cross-linking, alignment of the fibres along the lines of stress and synthesis of type I collagen (Barlow and Willoughby 1992; Witte and Barbul 1997).

Several factors can promote a less than optimal repair. The granulation tissue, which repaired the damage, can now act as glue to prevent movement between tissue interfaces. During the period when collagen turnover is accelerated, there is also increased molecular cross-linkage – these processes may produce adhesion formation and impair collagen gliding (Hunter 1994; Donatelli and Owens-Burkhart 1981). Newly synthesised collagen will tend to contract after three weeks; this naturally occurring shrinkage is said to continue for at least six months, if not forever (Evans 1980). Thus recently formed scar tissue will commence shortening unless it is repeatedly stretched. The stretching process should be commenced in the early stages following injury and continued until well after full recovery so no soft tissue shortening is likely to develop. Low-load regular application of stress will also help to increase the tensile strength of the repair tissue (Hardy 1989). Failure to perform the appropriate tissue loading will leave the repair process complete, but the remodelling stage incomplete; the individual may still be bothered by pain and limited function, and the tissue will remain weak and prone to re-injury. The nerves, which infiltrated the tissue during repair, can now be sources of pain each time the scar is stretched or loaded. This is a common cause of persistent symptoms in many patients.

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

In summary, no injury can be made to heal faster than its natural rate; whenever there has been tissue damage the processes of inflammation, repair and remodelling have to occur to allow full restoration of normal function. "*Failure of any of these processes may result in inadequate or ineffectual repair leading to either chronic pathological changes in the tissue or to repeated structural failure*" (Barlow and Willoughby 1992). These processes are essentially the same in tendons, muscles, ligaments and all soft tissues; however, intrinsic factors may be more likely to impair the recovery process in tendon injuries, especially if the onset is through overuse rather than trauma (Barlow and Willoughby 1992). Early progressive active rehabilitation is essential to optimise repair and function. No passive modality used within physiotherapy has yet been shown to reduce the time for the completion of natural healing. We can avoid delay to the healing process and ensure that the climate for repair is favourable (Evans 1980). Strenuous mechanical therapy applied when the pain from the injury is essentially chemical will delay recovery. The integrity of the repair must be established before more vigorous procedures are applied. However, of equal importance is the use of a progressive, controlled programme of loading the tissues at the appropriate time during the repair process in order to promote a fully functional structure *which the patient is confident to use*. Those factors that may restrict a return to normal function are listed in more detail later.

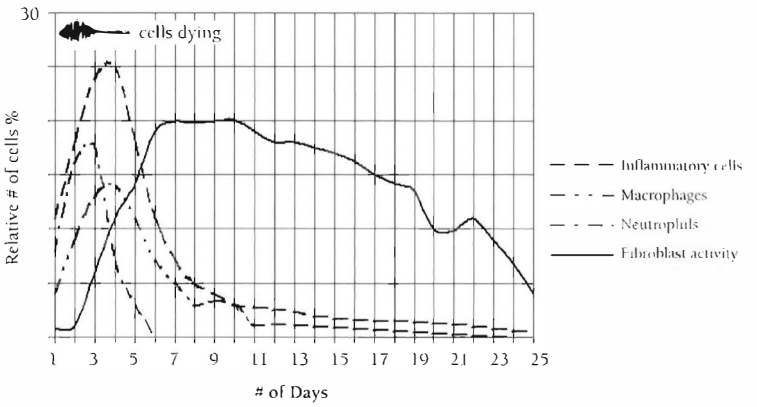
Table 2.1 Matching the stage of the condition to management

Injury & Inflammation	Protect from further damage. Prevent excessive inflammatory exudate. Reduce swelling
↓	↓
Repair & Healing	Gentle natural tension and loading. Progressive return to normal loads and tension.
↓	↓
Remodelling	Prevent contractures. Normal loading and tension to increase strength and flexibility.

Failure to remodel repair tissue

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

Figure 2.1 Time course of cellular activity following tissue injury



In some patients contracture resulting from previous injury may now prevent the performance of full range of motion. These patients will have been unwilling to stretch the recent injury, perceiving the 'stretch' pain as denoting further damage, and they will not have received appropriate rehabilitation advice at the time of the injury. They will present later with restricted range of movement and pain provoked by stressing the scar tissue. The tissue will become progressively more sensitised and deconditioned for normal function with lack of use.

In such cases the remodelling of collagen by applying a long-term structured exercise programme will be necessary. By applying regular stress sufficient to provide tension without damage, collagen undergoes chemical and structural changes that allow elongation and strengthening of the affected tissue. Because tissue turnover is slow, it must be recognised that it may be a slow process. If the contracture

has been present for some time, the remodelling programme will have to be followed for several months. Evans (1980) reports that some patients may have to exercise for the remaining years of their life. Stretching of old injuries should be routinely practised, especially prior to participation in sporting activities (Hunter 1994). The animal experiment of Arem and Madden (1976) showed that 'old' scar tissue might be unresponsive to a remodelling programme. Well established contractures, especially where the original healing process has been interrupted by repeated re-injury causing the production of more inflammatory exudate, may be resistant to improvement.

Degenerated tissue in which healing is suspended

Certain chronic conditions in which symptoms remain relatively stable over time appear to get 'stuck' in the repair process. This occurs in degenerated structures in which symptoms have arisen insidiously, as in such tissues the repair process is less effective (Buckwalter *et al.* 1993). Degenerative tendonopathy is thought to be due to hypoxia and micro-trauma and arises silently, so that complete ruptures of Achilles' tendons have occurred without prior symptoms (Leadbetter 1992; Kannus and Jozsa 1991). There is fibre disorientation, hypocellularity, scattered vascular ingrowth and occasional necrosis or calcification (Leadbetter 1992). Such degenerative changes in tendons are found in about a third of the general population over 35 (Kannus *et al.* 1997). In persistent tendon problems histological investigations do not reveal any inflammatory cells (Verhaar *et al.* 1993; Nirschl 1992; Chard *et al.* 1994; Kannus *et al.* 1997). In these instances the orderly phases of the healing process are aborted or absent (Leadbetter 1992). This failure to repair may result from a combination of slow or failed healing, avascularity, degenerated tissue and lack of appropriate stresses (Fukuda *et al.* 1994; Chard *et al.* 1994; Leadbetter 1992).

Failure to remodel adequately leads to imperfect repair. Symptoms will persist in this state. To stimulate the remodelling process in this degenerated tissue a different approach may be needed. In such instances vigorous stimulation of the tissue, so that the healing process is re-triggered, is often beneficial. There will be a temporary provocation of pain and aggravation of symptoms. Once this has been achieved the aggravating stimulus should be stopped, and progressive loading, as in the sub-acute model, should be introduced.

The effects of stress deprivation and exercise on connective tissue

Both following injury and in normal health the optimal function of musculoskeletal structures is maintained by activity. The detrimental effects of immobilisation on connective tissues are well known (Akeson *et al*. 1987, 1989, 1991; Frank *et al*. 1984). Repeated experiments, mostly with animals, have documented that in the absence of normal stresses these tissues weaken, atrophy, develop contractures and adhesions, and become generally unfit for normal function (Gelberman *et al*. 1982; Amiel *et al*. 1982; Houlbrooke *et al*. 1990; Videman 1987; Donatelli and Owens-Burkhart 1981; McDonough 1981; Woo *et al*. 1987). It is especially important to note that following injury connective tissues consistently respond better to early motion than to immobilisation (Gelberman *et al*. 1981, 1982; Houlbrooke *et al*. 1990; Arem and Madden 1976; Jarvinen and Lehto 1993).

Various authors (Akeson *et al*. 1987; Bland 1993; Videman 1987) have listed the negative effects of immobilisation on connective tissues. They include:

- increased random deposition of collagen fibrils
- increased collagen cross-links
- pannus formation over cartilage surfaces
- ulceration and loss of thickness of cartilage
- dramatic loss of tensile strength
- formation of adhesions and contractures in and between the synovial membrane, capsule and other tissues
- degradation of the ligament-bone interface
- generalised osteoporosis of cancellous and cortical bone

In summary, the effects of stress deprivation are thus to weaken and atrophy ligament, tendon and bone, as well as muscle; to degrade surfaces and tissue interfaces; and to cause disorganised tissue to bond randomly together. Bland (1993) makes clear the implications of these changes for the patient. "*The clinical issue is that the damage occurs very rapidly and the repair is extremely slow and less predictable, a heavy price to pay for immobilisation – total, partial or even relative immobilisation*" (p. 100)

The converse side of this picture is the beneficial effect which early motion after injury and exercise has on strengthening and normalising connective tissue function (Akeson *et al*. 1989, 1991; Salter 1989; Frank *et al*. 1984; Woo *et al*. 1987). Experimental and clinical work attests to the value of early motion in strengthening tendons and ligaments, restoring full range of movement, realigning collagen fibrils along lines of stress, preventing adhesion formation, and preserving articular cartilage (Frank *et al*. 1984; Gelberman *et al*. 1981, 1982; Evans 1989; Houlbrooke 1990; Woo *et al*. 1987). As with these other tissues, muscles atrophy in response to disuse, but hypertrophy when subjected to increased use (Pitman and Peterson 1989), and regain function more effectively when mobilised from a few days after injury (Jarvinen and Lehto 1993). Bone has the ability to respond to increased stress by remodelling and altering its structure to meet the physical demands placed on it (Nordin and Frankel 1989a).

This phenomenon, in which bone will alter its density and shape according to the mechanical stresses put upon it, is known as Wolff's Law (Bland 1993). The application of Wolff's Law has been criticised for being too limited, and many authorities see it rightly applied to all connective tissue, not only bone (Bland 1993; Akeson 1989; Akeson *et al*. 1991). It reflects the truism that anatomical tissues and structures will tend to reflect the functional stresses demanded of them. Progressively increased loads will strengthen tissues and enhance function; stress deprivation will cause atrophy and dysfunction.

Even in the instance of a compromised osteoarthritic joint, when destructive loading and stress deprivation may co-exist in different parts of the same joint, the possible value of exercise should not be forgotten (Akeson 1989). Animal experiments have shown that with continuous passive motion, healing and regeneration of cartilage may occur (Salter *et al*. 1980). Immobilised joints show signs of osteoarthritic lesions, while with normal loading the joint surfaces returned to normal; however, excessive exercise at this point aggravated the degenerative sequence (Bland 1993). Bland proposes that "*well governed and practised exercise and mechanical stimulation of the involved tissue may aid in arrest or reversal of the process*" (p. 107).

Finally, besides the physiological benefits which exercise brings to joints, connective tissues and muscles, as well as the cardiovascular

system, it is important to remember the psychosocial implications of inactivity versus activity. *"The result of a decrease in physical activity combined with a reluctance to maintain a level of fitness with increasing chronological age and a general failure to appreciate the dependence on physical stresses for continued functional integrity will lead to the degradation of all connective tissue; in a word, retirement – both intellectual and physical"* Bland (1993: 100). This applies even more in the presence of a painful condition. It has been proposed that fear of movement and re-injury leads to avoidance of activity and consequent disability, disuse and depression. A continuing vicious cycle is created in which the painful experience and reduced function persist. Those able to confront their pain and follow a progressive return to normal activity are more likely to make a full recovery (Vlaeyen 1998).

Numerous physiological improvements have been attributed to exercise; these relate not only to maintenance of normal function, but also to their favourable effects on the natural history of various diseases and degenerative conditions (Fentem 1992). Functions and structures that have been shown to be enhanced by regular exercise are the following:

- cardiovascular function
- skeletal muscle
- tendons and connective tissue
- the skeleton
- joints
- metabolic function
- psychological function.

Factors restricting a normal return to function

Injuries cannot be made to heal faster than their natural speed. Without completion of any of the three phases the scar tissue will not be adequate for normal function. Failure in any phase of the repair process may result in ineffectual healing leading to chronic degenerative changes, repeated structural failure, or less than optimal tissue (Evans 1980; Carrico *et al.* 1984; Barlow and Willoughby 1992). If the tissue is still stiff and painful long after the expected period of healing, the individual may be anxious and lose confidence

about using the structure properly. We must make sure that no contrary influences intrude and that the best possible conditions are encouraged. Various problems may prevent or inhibit healing and return to full function. Some of these factors are more amenable to influence than others. Age or pre-existing degeneration is an important prognostic variable to be aware of, but difficult to affect. Some of these factors relate to the tissue environment, some to the individual's response to injury; both elements may contribute to retarded healing. In the long-term return to full function reflects behavioural responses as much as the cellular healing process. Inappropriate action based on misconceptions, poor advice or avoidance can be as deleterious to full recovery as the wrong chemical environment.

Following are some factors that may inhibit the healing process and return to full function (Evans 1980; Carrico *et al*. 1984; Barlow and Willoughby 1992; Buckwalter *et al*. 1993; Linton 1996):

- poor blood supply or ischaemia – especially relevant in tendon injuries where intrinsic vascularity may be poor and where there is very high tissue pressure or absolute immobilisation
- lack of initial protection and reduction of swelling (first few days)
- lack of early mobilisation – gentle natural tension encourages good quality repair (starting within the first week post-injury)
- continued inflammation; anything that prolongs or re-triggers this phase will cause poor healing – for instance infection, a haematoma, or excessive premature vigour
- systemic or local steroid medication – decreases the tensile strength of wounds, slows the rate of wound closure and vascularisation
- nutrition – malnourishment inhibits the body's response to injury
- diabetes – mechanical problems and metabolic defects impair wound healing
- over-abundant deposition of collagen – keloid scar formation
- levels of physical activity; the level must be appropriate for the stage as outlined above. If too much vigour is employed too early, or if during remodelling inadequate stresses are applied

to the repair tissue, the end product can be the same – poor wound healing and impaired function

- age – this causes a decline in function, an increase in susceptibility to injury and disease, and a decrease in the ability to recover from injury and disease. This is not uniform, however, and occurs at extremely variable rates between individuals
- inappropriate behaviours in dealing with pain and its consequences. Responses which have been implicated in the development of chronic pain are belief that activity causes pain or injury leading to fear–avoidance behaviour, use of passive coping strategies, anxiety, low self-efficacy beliefs, external health locus of control beliefs, depression, catastrophising about the situation, lack of proper information
- inappropriate behaviours on the part of health care professionals – encouraging patients to adopt sick-roles, obtain sick leave, and offering passive, clinician-led treatment strategies which help maintain low self-efficacy and external health locus of control beliefs in the patient

Cumulative postural stresses on musculoskeletal tissue

So far the beneficial effect of appropriate tissue loading has been addressed. However, there are instances when repeated or sustained mechanical stress on tissues can be damaging. Many conditions start insidiously and so the role of daily recreational and occupational stresses on musculoskeletal structures must be recognised. Degenerative processes or repetitive micro-trauma, which overwhelms the tissue's reparative process, may produce symptoms (Kannus *et al.* 1997). Tendons in particular are prone to developing chronic overuse injuries.

Contractile tissue loading

Although contractile elements are commonly injured traumatically during sporting activities, muscular injuries also frequently arise in an insidious manner from excessive internal forces generated by occupational or domestic tasks. During such activities different muscles may be contracting to provide a stabilising or a movement-generating role. In other words, the contraction may be isometric,

during which a joint position is maintained, or concentric or eccentric, during which the joint is moved. Those muscles performing isometric work are under relatively constant loading and can suffer from ischaemia or micro-trauma of fibres even though they may not be required to cause any movement. Other muscles involved in the same activity may be required to relax and contract alternately in order to create movement. In performing the same task, some muscles will be required to be active both eccentrically and concentrically. It is therefore the case that in many activities, non-moving contractile structures can be statically loaded to the point of failure as readily as contractile structures involved in the actual movement.

In the extremities postural pain may arise from contractile structures as a consequence of prolonged static or repeated loading of muscles and tendons. Static loading can lead to ischaemic muscle pain and localised fatigue, and repetitive tasks can cause micro-trauma (Smith 1996). It is the pain often felt in the shoulders, arms, wrists and fingers of clerical workers who, in sustained postures, perform the same task for too long without interruption or variation. It may be the result of excessive mechanical strain on the structure resulting in deformation of the collagen fibres and pressure on the free nerve endings within (Bogduk 1993). Alternatively, in contractile structures undergoing constant contraction without brief periods of relaxation, symptoms may be the product of ischaemia (Newham 1991). Intramuscular pressure during activity can compress the blood vessels, impairing the removal of metabolites and the supply of oxygen, either of which may produce temporary pain (Mense 1997; Norris 1993; Smith 1996). No tissue damage has occurred as movement or a change of position removes the pain as the circulation returns and the metabolites are flushed away. These postural pains are thus produced by sustained mid-range activity in which abnormal stress is placed upon normal contractile structures.

However, continuing repetition of static tasks begins to take its toll, and tissue damage can occur (Smith 1996). For instance, pain may be felt in the shoulder from excessive performance of overhead tasks. This activity causes high intramuscular pressure in the rotator cuff muscles which can impair local circulation, already compromised by compression of the tendon under the acromion. Initially there is cellular degeneration and impaired vascularity, which may cause pain that abates when the activity ceases. However, if such loading is

repeated for many hours on a daily basis, the degenerated cells or ischaemic state can give rise to an inflammatory reaction, and a constant pain ensues (Grieco *et al.* 1998; Mense 1997). Local strain on muscles can thus produce ischaemic effects with time and load, as well as acute mechanical failures. If the strain is prolonged local hypoxia, ultrastructural ruptures, energy depletion, and cell death may induce symptoms (Herberts *et al.* 1984). Alternatively, hypovascularity may lead to degenerative changes in tendons so that, in a weakened state, the tissues are predisposed to early failure and pain, and prognosis is poorer (Herberts *et al.* 1984). Tendon degeneration can be severely symptomatic or entirely silent, with various aetiological factors being important in its pathogenesis, but reduced vascularity and impaired metabolic activity crucial in this respect (Kannus *et al.* 1997). It has been proposed that a sedentary lifestyle may cause poor circulation in tendons and so predispose to chronic disorders (Kannus *et al.* 1997).

Damage, if confined to fibres of the musculature, is unlikely to lead to long-term problems. The excellent vascularity provided to muscle ensures rapid repair following all but the most severe injuries. However, if the strain affects the less vascular fibrous tendons, aponeuroses or periosteal insertions, there is more likelihood of persistent problems resulting. Tendons are renowned sources of chronic symptoms in a wide range of upper- and lower-limb disorders. Hypoxic degeneration and innervated immature granulation tissue seems to produce prolonged pain and impaired function, which is highly resistant to full recovery (Leadbetter 1992; Gross 1992). This is not chronic inflammation, as inflammatory cells are never found in this degenerated tissue (Verhaar *et al.* 1993; Chard *et al.* 1994)

Thus during the early stages of the symptoms, discomfort or pain ceases entirely once the offending position is changed. With continued prolonged or repetitive loading over many months without adequate rest periods or relaxation of the muscle, micro-trauma may result and an inflammatory response is triggered which is no longer amenable to rest only. Cumulative trauma, rather than a discrete traumatic event, thus brings about pathological changes (Smith 1996; Thorson and Szabo 1992; Yassi 1997). Alternatively cumulative strains can produce occult degeneration in tendons, which leaves them susceptible to failure, with a corresponding poor prognosis.

Such disorders of cumulative trauma are the second most frequently reported category of occupational diseases after skin diseases (Thorson and Szabo 1992). Strains due to repetitive activities can occur in the hands, wrists, elbows, shoulders, neck, back, hips, knees and ankles, with upper-limb repetitive strain injuries being one of the fastest growing group of occupational disorders (Yassi 1997). The upper limb is very commonly affected by the consequences of prolonged or repetitive loading. Symptoms can be reported in the shoulder (bursitis or tendonitis), the elbow (medial and lateral epicondylitis), the wrist and thumb (tenosynovitis, writer's cramp, de Quervain's syndrome, carpal tunnel syndrome). A causative role has been attributed to repetitious work; unusual, constrained or static postures; and exposure to vibration and extremes of temperature. People who adopt extreme joint positions are at risk, and those who work in postures requiring excessive shoulder abduction, wrist flexion or radioulnar deviation are at high risk of developing tendonitis. Faulty workstations and equipment are also aggravating factors (Thorson and Szabo 1992; Smith 1996; Huskisson 1992). Keyboard operatives are frequent complainants of such disorders, but the prevalence of neck and arm pains apparently related to occupation are common across various industries (Buckle 1987; Bystrom *et al.* 1995).

Prolonged and abnormal postural stresses thus have a role in the pathogenesis of various musculoskeletal conditions. Furthermore, dynamic postural forces are significant in many of these conditions. For instance, pain can sometimes prevent abduction of the shoulder in the presence of a rotator cuff disorder, while abduction is possible with the arm laterally rotated. Individuals with lateral epicondylitis can find lifting with the arm supinated considerably more painful than lifting with the arm pronated. Patients with anterior knee pain, who get pain descending stairs or sitting for long periods because the patella is malaligned, are pain-free once this postural stress is removed. Thus static and dynamic postural forces are clearly relevant with many conditions and need addressing in their management

Clinical implications

Damage to soft tissues may be the result of a traumatic event, but equally can occur with the cumulative overload of repetitive strain, abnormal sustained postures or degeneration of tissues. Excessive external or internal forces can therefore produce mechanical strain

and tissue damage. Any damage that occurs to soft tissues unleashes the inflammatory response, the first stage on the road to recovery. This is followed by tissue repair and remodelling if all goes well. The entire process cannot be avoided or speeded up, but it can be impaired and inappropriate behaviour can prevent the remodelling, which should return the tissues to normal function. Mechanical forces are essential to this process and sometimes ignorance, bad advice or fear of doing further damage constrains individuals from doing what is best. In certain instances the process of repair seems to get 'stuck', leaving tissue that is immature, painful and not conditioned for normal use. This may be the result of failure to remodel adequately, or due to an aborted repair process or underlying degenerative changes in the tissues – tendons are particularly prone to persistent symptoms in this way.

The period when relative rest is appropriate, during the inflammatory stage, is very short. Progressive exposure to mechanical loading should start early. In fact, a large proportion of patients seen by physiotherapists are seen at a much later stage when rest is absolutely contraindicated. Repair has usually occurred, but pain and impaired function remain. Only through exposure to mechanical loading can remodelling occur, deconditioned structures be made functional and sensitised tissue be made normal. This is only achieved with regular exercise that challenges the unfit tissue. No pill, lotion or electronic gadgetry can even begin to promote this change, and to pretend otherwise is to condemn patients to continued suffering. Improvement in the state of the structure and the confidence of the patient to use it can only be brought about by gradually exposing the tissue to loading and movement.

Sir Astley Cooper's engravings

The following engravings were taken from the work of Sir Astley Cooper who practiced his profession as a Surgeon in London from the late 1700s through to about 1830. The engravings show the remarkable capacity of nature to heal even the most severe injuries and through the process of remodelling restore function; perhaps not entirely, but adequately in most cases.

The process of remodelling can only be achieved through persistent and regular movement. The examples that follow are from a wide range of articular specimens and show remodelling of bone and soft tissues such as tendons, ligaments, capsules and cartilage.

Cooper did not enjoy the benefits of modern technology and methods that are now available for the detection and reduction of dislocations and fractures. The natural processes of healing as demonstrated in the following specimens are rarely seen today. However, they demonstrate the power that normal functional activity has to remodel bone and soft tissue, in even the most extreme cases, and produce working pseudo-articulations.

The selected text and engravings shown here are unchanged from the original.

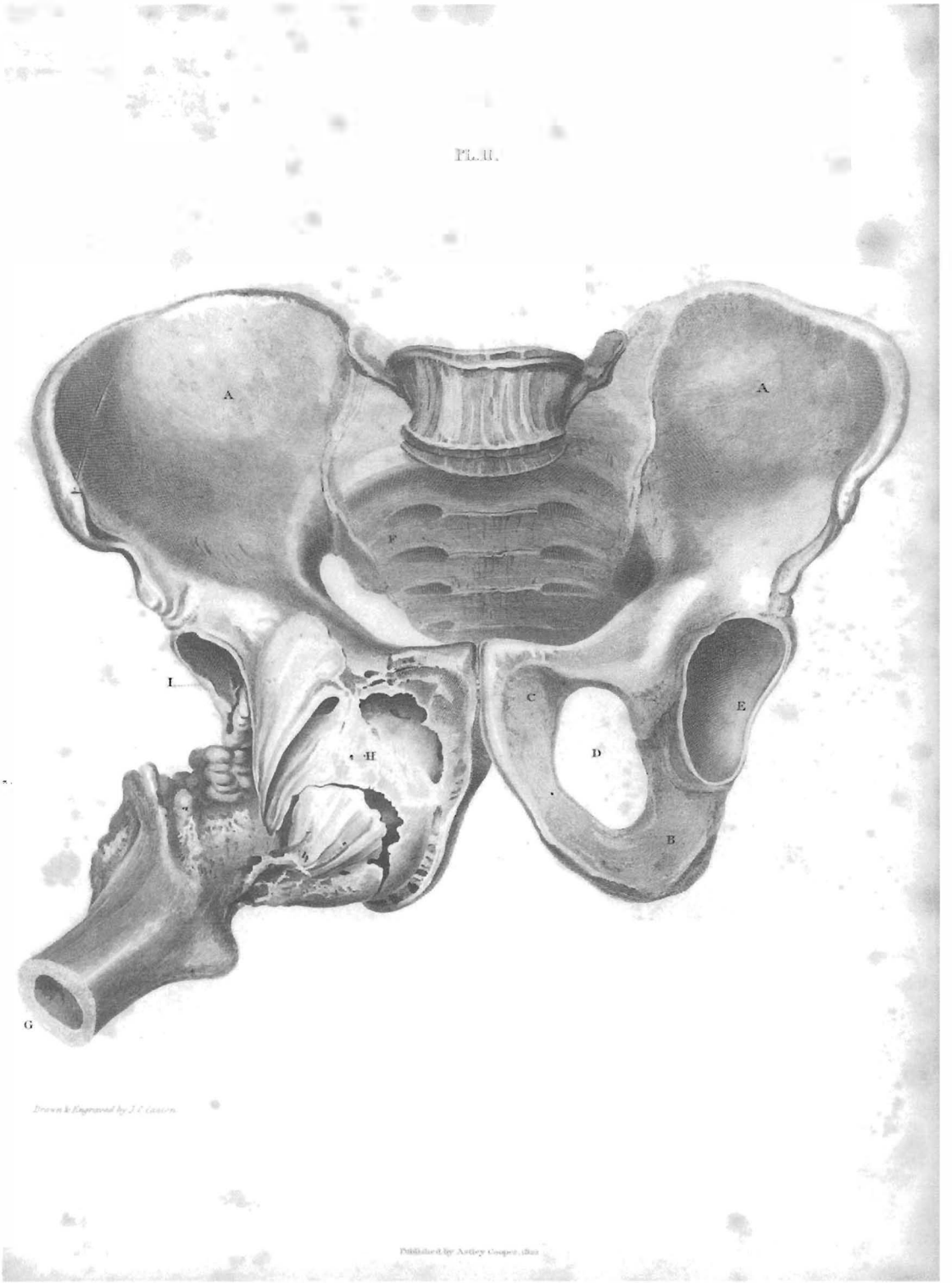
Treatise on Dislocations and Fractures of the Joints.
Sir Astley Cooper, BART. FRS.
Sergeant Surgeon to the King. Printed in 1831.
Seventh Edition printed by Messrs Longman, Reece, Vaughan-Brown and Green, Paternoster Row, South Highly, 174 Fleet St.
Drawn and engraved by CJ Canton, published by
Astley Cooper, 1822.

Plate II

Shews a dislocation into the foramen ovale which had never been reduced and beautifully exhibits the resources of nature in forming a new socket for the head of the bone and allowing of the restoration of a considerable degree of motion.

H – The new acetabulum formed in the foramen ovale in which the head of the thigh bone was contained and in which it was so completely enclosed that it became impossible to remove it unless a portion of the new socket was broken away. It was lined by a ligamentous substance on which the head of the bone moved to a considerable extent.

I – The original acetabulum situated above the level and to the outer side of the new cavity.


Pl. II.
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Drawn & Engraved by J. C. Cassers.
Published by Astley Cooper, Bart.

Plate III

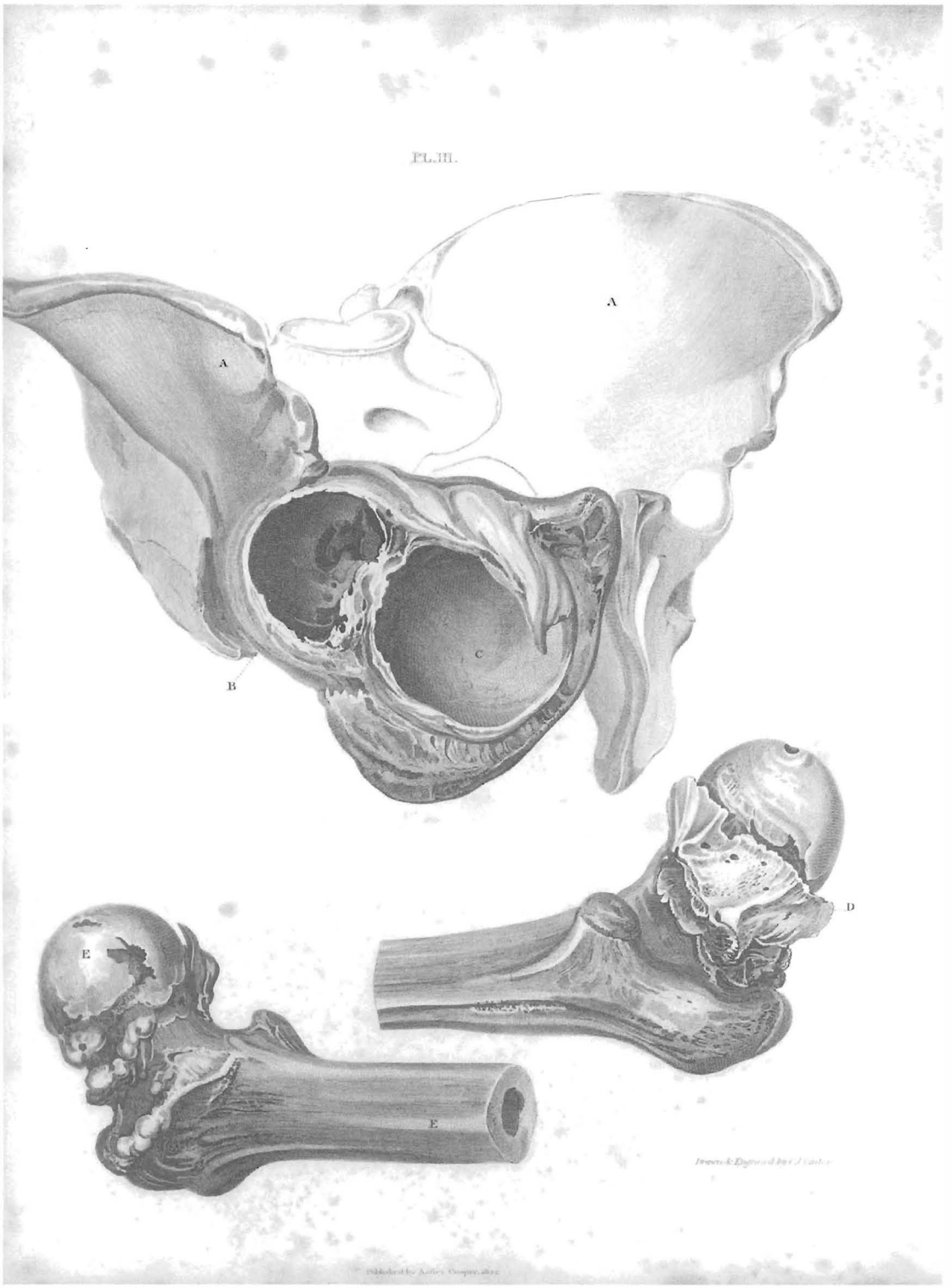
Exhibits another view of the same preparation shewing the relative situation and appearance of the new and the original acetabulum.

B – The original acetabulum little more than half its natural size, the edge of the new acetabulum occupying its lower and anterior part.

C – The new acetabulum formed in the foramen ovale, a deep ossific edge surrounding it. Its internal surfaces extremely smooth. The ligament of the foramen ovale has disappeared and ossific matter has been deposited in its stead.

D – The thigh bone removed and a portion of the new acetabulum is shewn which was obliged to be broken off to separate the thigh bone from its new socket.

E – Head and neck of the thigh bone; the former a little changed by absorption and the latter by ossific deposit.


PL.III.
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E

Plate IV

Shews a dislocation in the ischiatic notch. This is a side view of the exterior surface of the os inominatum.

G – Shews a new capsular ligament formed around the head of the bone and composed of cellular membrane condensed by inflammation.

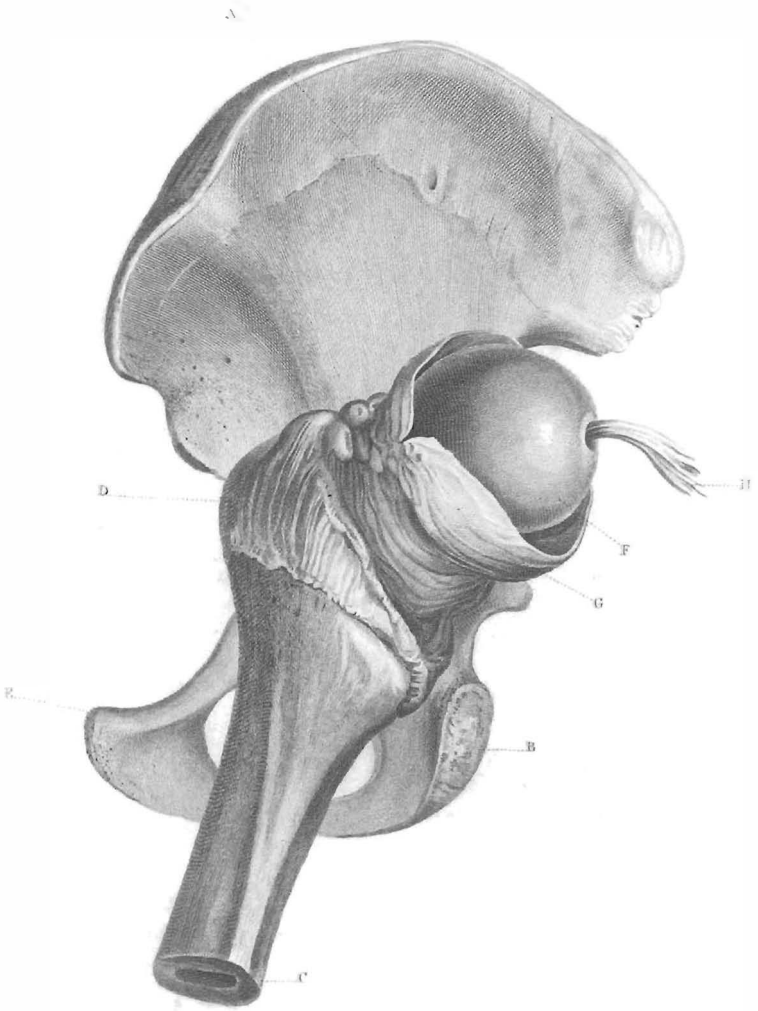
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Plate V

Exhibits a view of the dislocation of the os femoris upon the pubes or forwards and upwards. This preparation beautifully shews the power of nature in accommodating itself to new circumstances.

E – Trochantor major occupying the original acetabulum.

F – Head and neck os femoris upon the junction of the pubes and ilium.

G – The new cup formed for the neck of the os femoris.

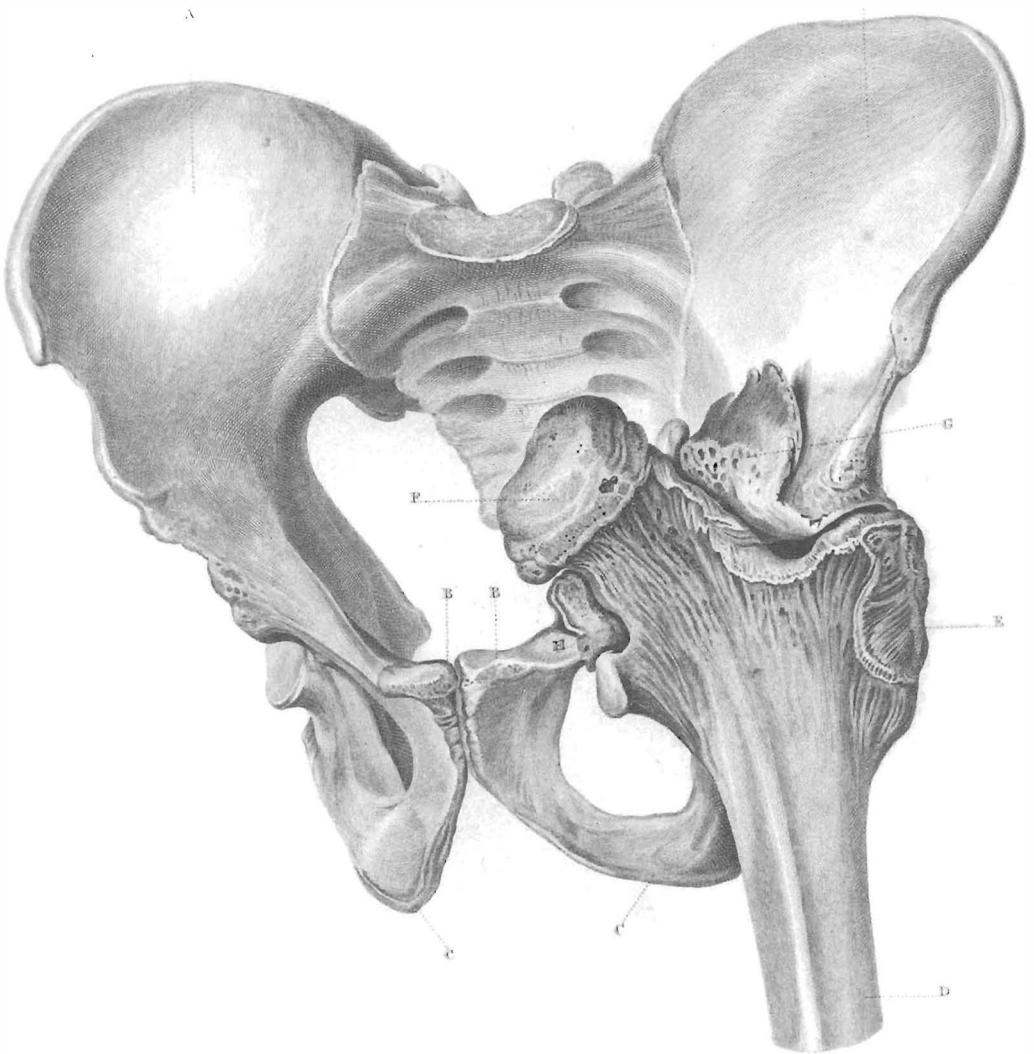
PL.V.
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Plate VI

Shews the same pelvis, with the thigh bone removed from it, to expose the new acetabulum formed by ossific inflammation in the junction of the pubis and ilium.

D – Acetabulum, which was occupied by the trochanter major.

E – F The new acetabulum.

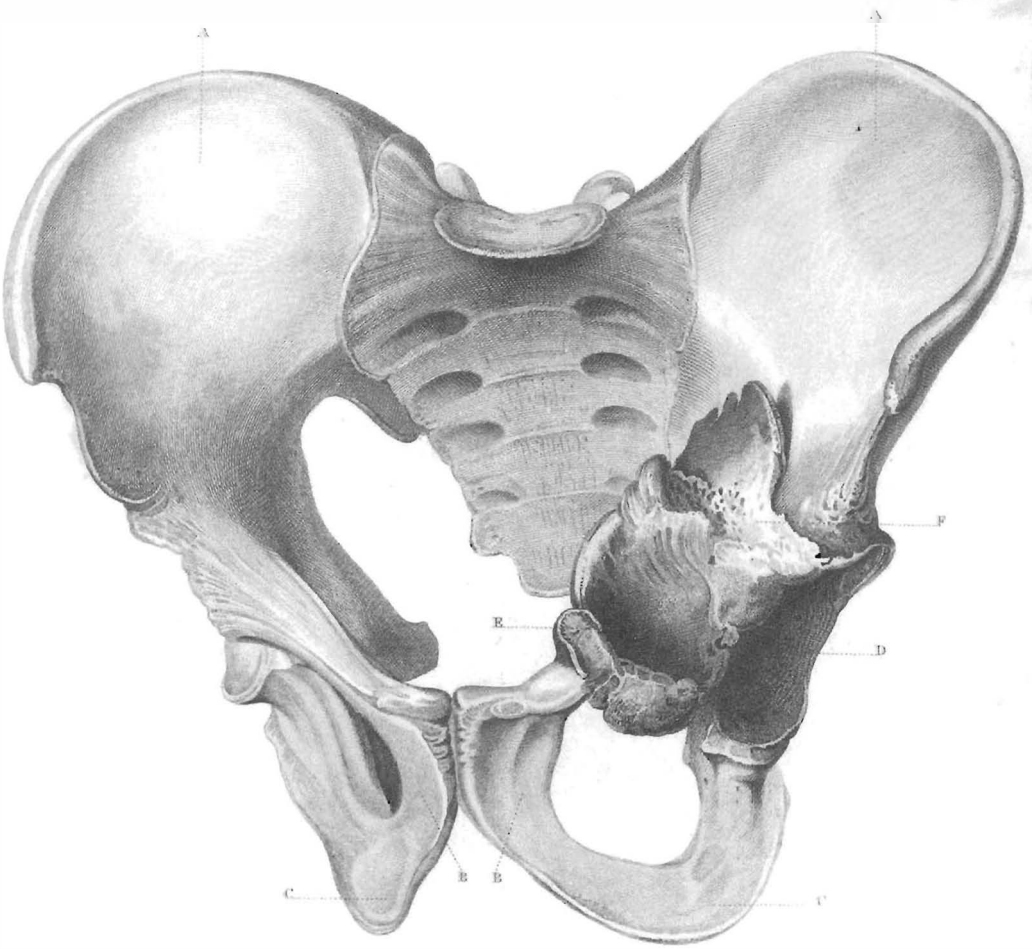
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Plate XXII

Fig 1

Shews the new socket which has been formed on the inner side of the inferior costa of the scapula in a dislocation of the os humeri into the axilla.

G – The new socket for the head of the os humeri.

Fig 2

D – New smooth cavity for the head of the os humeri – which extended from the edge of the glenoid cavity to the corocoid process of the scapula.

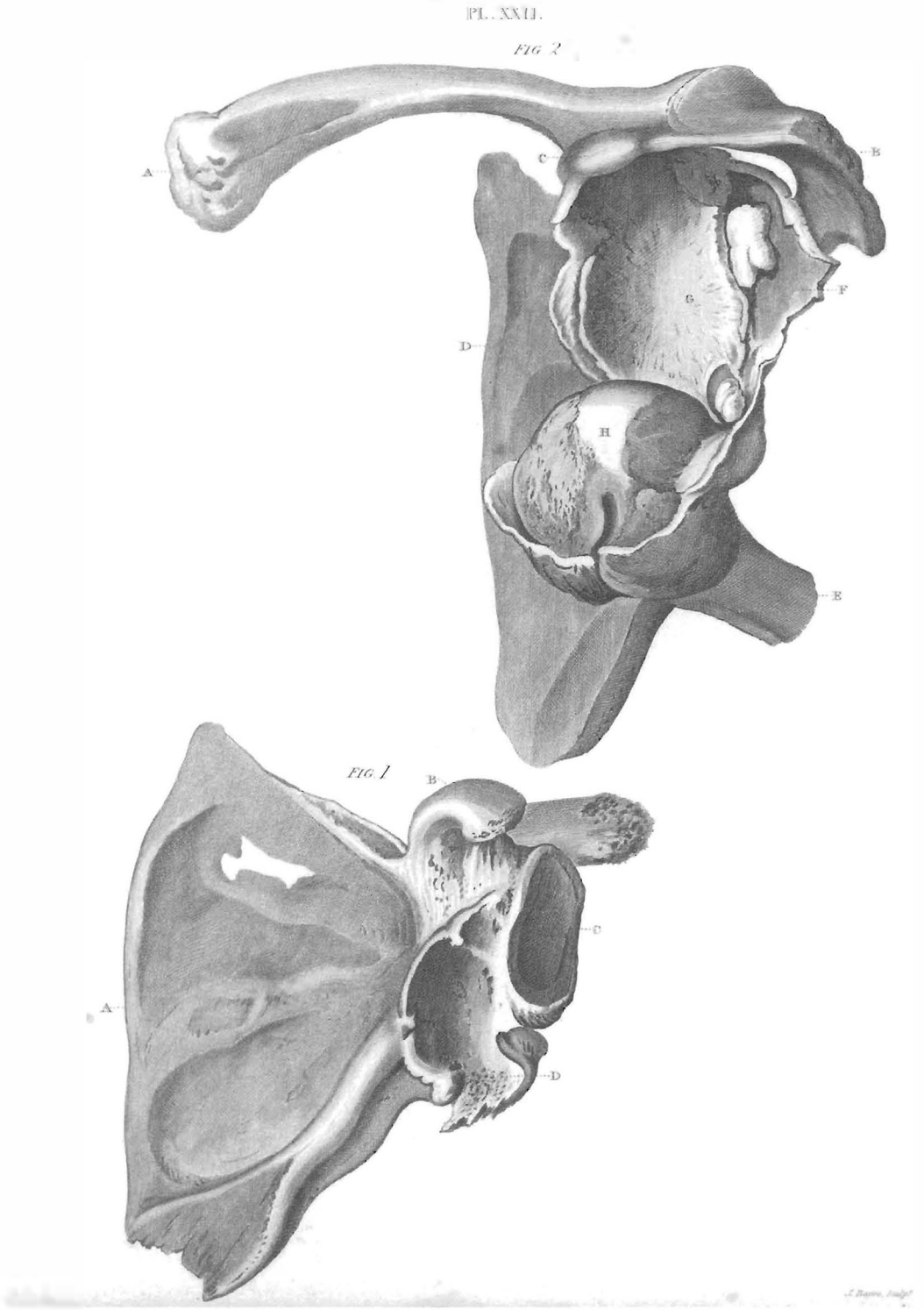
PL. XXII.
FIG. 2
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FIG. 1
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Plate XXIII

Fig 1

Dislocation of the os humeri forwards under the clavical and behind the pectoral muscle.

F – Portions of the new ligament which enclose the head of the bone.

G – The vacated glenoid cavity, from which the os humeri had been thrown.

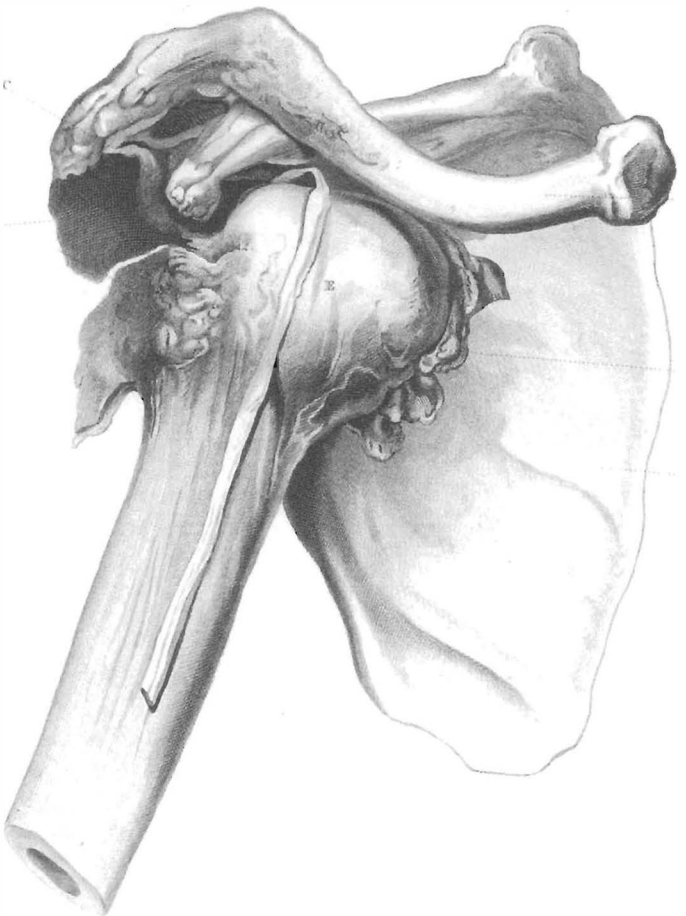

Plate XXV

Fig 1
Dislocation of the ulna backwards.

Fig 2
F – Head of the radius, which by its pressure against the external condyle of the os humeri has produced a socket there for itself.

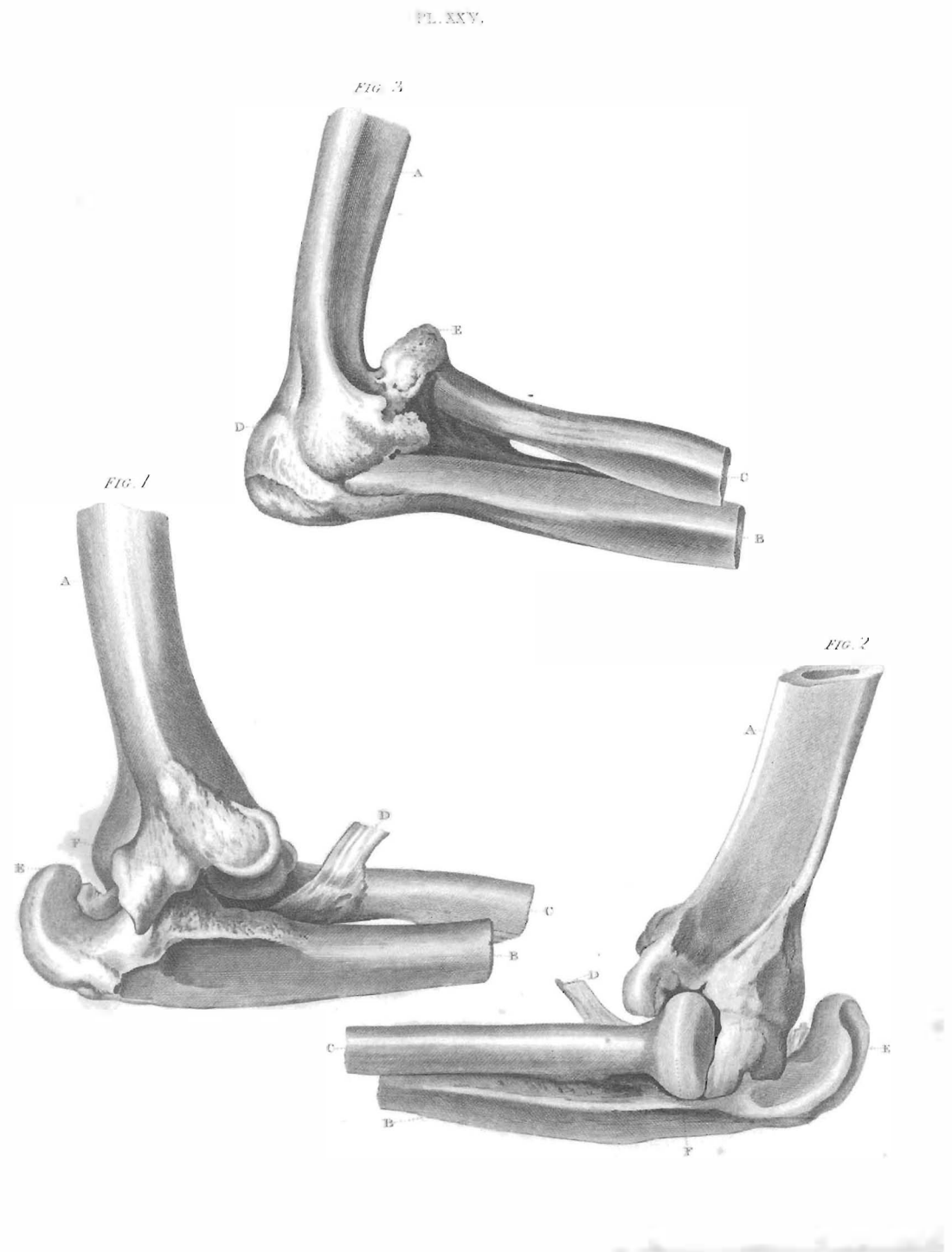
PL. XXV.
FIG. 3.
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FIG. 1
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FIG. 2
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Plate XXVII

Fig 2

E – External condyle fractured externally to the capsular ligaments; great attempts by nature to unite it, and the form of the bone changed.

Fig 3

G – The new ligament which has joined the olecranon to the ulnar.

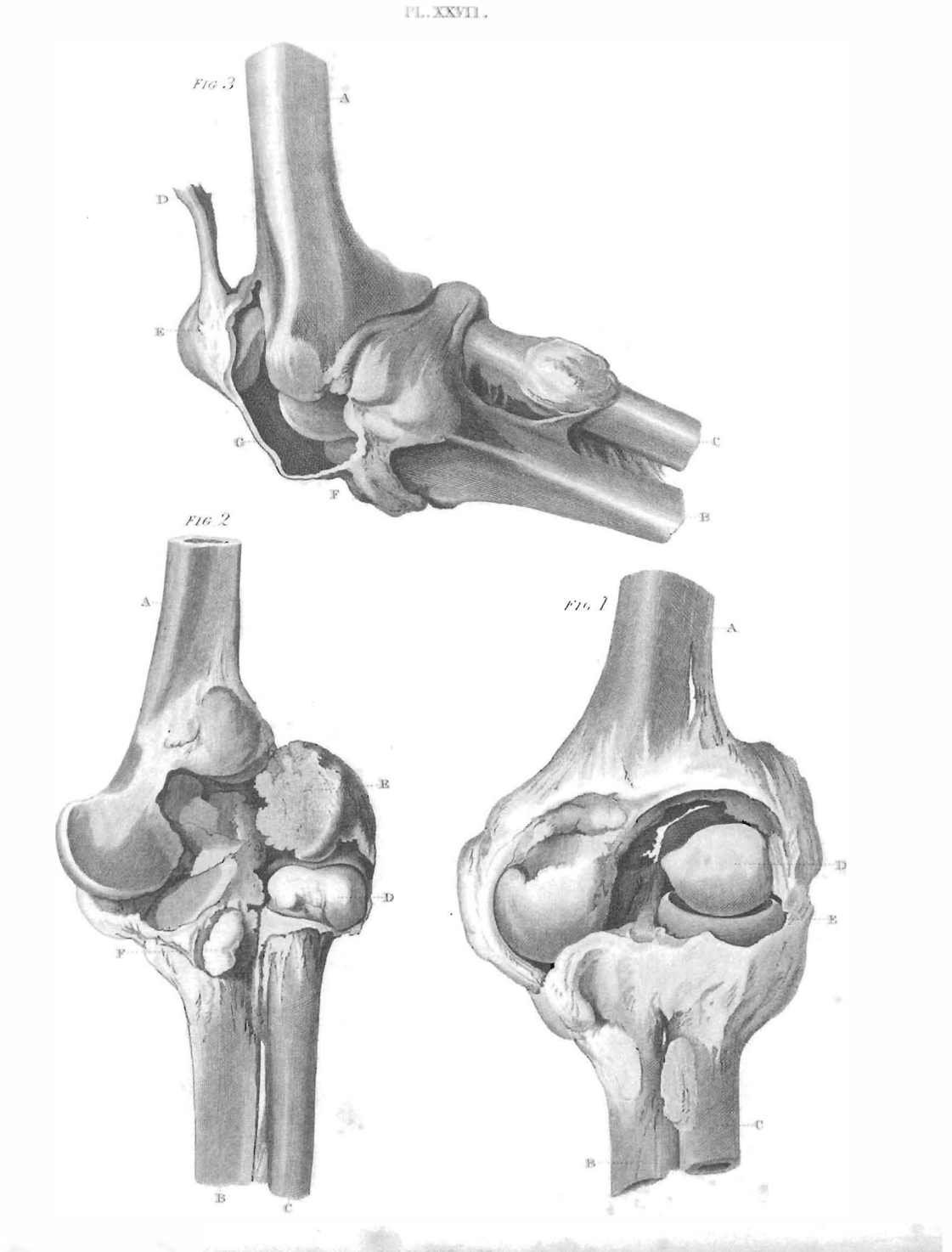
PL. XXVII.
FIG 3
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FIG 2
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FIG 1
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Plate XXVIII

Fig 3

Dislocation of the second phalanx of the finger forwards, and of the first, backwards.

F – The new capsular ligament covering the ends of the dislocated bones.

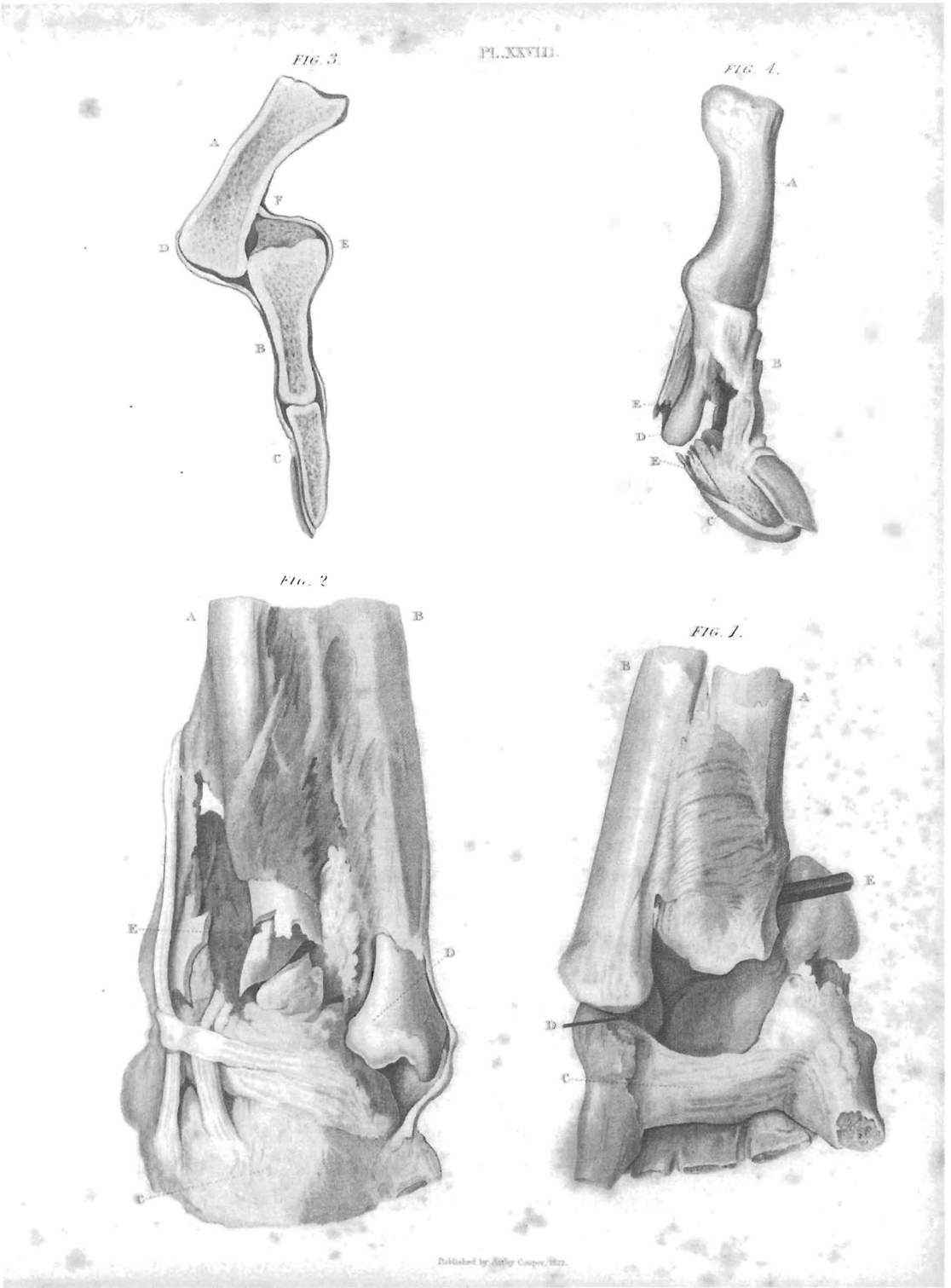
Pl. XXVIII.
FIG. 3.
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FIG. 4.
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FIG. 1.
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Published by Astley Cooper, 1822.

3: Pain

Introduction

Patients' prime concern is usually pain, although secondary to this may be concern with loss of function. It is important to have some ground rules by which to interpret and understand pain and the state of painful tissues so that the appropriate active management strategies can be implemented. This chapter looks at different causes of pain and relates these to the state of the tissues. By recognising typical patterns of pain behaviour early in the treatment process, we can predict those who will respond to certain management strategies. Atypical or partial responses will identify those patients with problems that are not going to fully resolve that require improved self-management strategies or may need a multi-disciplinary pain management approach. The clinical implications of these issues will be discussed.

Pain and nociception

The means by which information concerning tissue damage is experienced and transmitted to the cortex is termed nociception. Most tissues in the body possess a system of free nerve endings – nociceptors – which are activated if a noxious stimulus is experienced, thus triggering the nociceptive system. After tissue damage is detected this information is transmitted via the peripheral and central nervous system to the cortex and provides the means by which we are made aware of pain. En route the nociceptive message is modulated; that is, the central nervous system, including the higher centres, can exert inhibitory or excitatory influences on the nociceptive input (Bogduk 1993).

Pain has been defined as "*an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage*" (Merskey 1991). This widely accepted and much quoted definition recognises that the experience of pain is a cortical phenomenon influenced by affective and cognitive factors as well as sensory ones (Bogduk 1993). This definition also distinguishes between the experience of pain and the physiology of nociception. The implication to clinicians of this modern concept of

pain is that our management must encompass patients' responses to pain, as well as their sensation of pain.

It is important to recognise that the experience of pain involves patients' emotional and cognitive reactions to the process of nociception. Many issues are relevant here – patients' anxieties, fears and beliefs may strongly determine their response to injury, pain and treatment. Fear of pain and re-injury may lead to avoidance of activities that they believe will do more harm. Such lack of understanding of their condition causes inappropriate action in the face of pain and produces feelings of limited ability to control or affect the condition. Such avoidance in the long term will have a deleterious effect on the patient's recovery (Philips 1987). These factors can be addressed by providing patients with a thorough understanding of their problem and educating them in the appropriate use of activity and exercise to regain function and reduce pain. Facilitating patients' control over their problem, encouraging active coping strategies and helping them confront their fear of pain should all be part of management (Klaber Moffett and Richardson 1997). Strategies based upon education and patient activity are important as a means of addressing patient responses to a painful condition as well as the condition itself.

Failure to grasp this multifactorial, biopsychosocial model of pain and see it only as nociception leads to a failure to understand pain. The difficulty that researchers and clinicians have in grasping this concept is illustrated by the following comment: *"It is a marvel to me and a tribute to the persistence of selective attention that so many health care researchers seem unable to come to grips with what has been well known and repeatedly demonstrated: namely, that humans and the complex behaviours they emit can be understood only by considering their biomedical, genetic, psychological, and environmental/social contexts. To do less is to perpetuate a myth. Pain, particularly chronic pain, is transdermal. Put another way, perhaps pain ought to be seen as a verb, not a noun"* (Fordyce 1999).

Sources of pain

Any tissue which contains the free nerve endings involved in nociception is capable of being a source of pain. In relation to peripheral joint regions, nociceptors are located in the joint capsules

and ligaments, articular fat pads, perivascular sites, bone, periosteum, muscles and tendons, but probably not in the joint cartilage (Zimmermann 1989). The skin and subcutaneous tissues are also richly innervated (Williams and Warwick 1980). Bursae, fasciae and aponeuroses are also possible sources of pain, although the literature on their innervation is scant.

Two types of tissue are the main sources of musculoskeletal problems – muscles and connective tissues. Connective tissues include tendons, aponeuroses, fasciae, ligaments, bursae, synovial and intra-muscular sheaths, joint capsules, cartilage and bone. Cyriax (1982) referred to contractile and non-contractile structures, contractile tissues being:

- muscle belly
- tendon
- tendon–bone interface

Non-contractile tissues include:

- joint capsules
- ligaments
- bursae
- nerve roots
- dura maters
- fasciae

Activation of nociceptors

Only three mechanisms are known by which nociceptors can be activated – these are mechanical, chemical or thermal stimuli (Bogduk 1993; Zimmermann 1989). It is the first two that are our concern here. In normal circumstances the system of nociceptors are inactive, but as the stimulus crosses the threshold into the noxious range, the nociceptive process is triggered. This may be due to the application of mechanical forces that sufficiently stress, deform or damage tissues as would occur with pressure, distraction, distention, abrasion, contusion or laceration. The nerve endings may also be activated by their exposure to certain chemical substances present in the

surrounding tissue fluid following their release from traumatised, inflamed or necrosing tissue.

Pain arising from trauma is produced by a combination of mechanical deformation forces and noxious chemical irritation (Bogduk 1993). Initially, mechanical deformation causes damage to soft tissues and pain of mechanical origin will be felt. In most cases this is a sharp pain (Melzack and Wall 1988). When mechanical deformation is severe enough to traumatise soft tissues, it may be the result of an external force; it could also result from internal force, as in a sudden uncoordinated movement. Common examples might be spraining an ankle ligament when slipping off a pavement edge, falling onto an outstretched arm, a kick or blow during football or straining a calf muscle when playing tennis. Excessive external forces can cause certain conditions.

However, patients will frequently develop musculoskeletal pains without any appreciable trauma. When asked for their history, they will report that the pain came on for 'no apparent reason'. Diverse conditions such as shoulder capsulitis, supraspinatus tendonitis, lateral epicondylitis or tenosynovitis at the wrist are all known to start insidiously without a single traumatic incident. The pain related to joint degeneration is also more likely to start in a gradual way. Pain of insidious onset is more likely to occur following age-related changes. These cause a decline in musculoskeletal function, increasing the vulnerability of the tissues to injury and leading to slower and less effective healing (Buckwalter *et al.* 1993)

Mechanical stresses sufficient to cause trauma do not always arise from the application of abnormal external forces. Prolonged ***constant or repetitive loading*** repeated over many months or years might eventually cause cumulative tissue damage. Forces may become abnormal when the duration or repetitive nature of the applied force is excessive. Excessive internal forces can therefore cause certain conditions.

The nociceptive system can be activated by excessive mechanical stresses over long periods of time, by actual trauma, or by the chemicals associated with the inflammatory process. The degenerative process, which commonly occurs in peripheral joints and soft tissues,

can stimulate nociceptors by both mechanical and chemical irritants related to the pathology (Zimmermann 1989).

Because the cause of pain is an important determinant of the appropriateness of mechanical therapy, it is vital to distinguish between mechanical and chemical sources of nociception. We can begin to distinguish between these types of pain by certain factors gained during the history-taking and largely confirm this impression during the physical examination.

Differentiating between chemical and mechanical pain

A key characteristic which indicates the possibility of pain of chemical origin is constant pain. Not all constant pain is inflammatory in nature, but chemical pain is nearly always constant. The term 'constant pain' indicates that the patient is *never* without an ache or discomfort from the moment they wake until the moment they fall asleep. The ache may be exacerbated by movements and be less at times, but the dull, relentless ache never goes away entirely. Constant pain may result from chemical or mechanical causes, or be due to the changes associated with chronic pain. A description of the cause and behaviour of constant chemical and mechanical pain follows.

Constant pain – chemical cause (Constant Chemical Irritation)

Cause and onset

Pain is produced by chemical irritation as soon as the *concentration* of chemical substances is sufficient to irritate free nerve endings in the involved soft tissues. Such chemicals include histamine, bradykinin, serotonin, hydrogen and potassium ions. They are released by cells in damaged tissue or by inflammatory cells following tissue damage, and thus chemical nociception only occurs with actual tissue damage (Bogduk 1993). Systemic disease or infection can also cause this inflammatory process – for instance, with active rheumatoid arthritis, ankylosing spondylitis, tuberculosis or other bacterial infections. Treatment of inflammatory arthropathies lies outside the scope of this text. Our primary concern here is the inflammatory process that occurs in the first week or so following trauma.

An insidious onset of symptoms, without apparent trauma, does not discount an inflammatory component to the problem. This is more likely to occur when tissues are in a weakened state due to unseen degenerative changes or overuse. For instance, gradual arthritic degeneration can produce inflammatory mediated pain (Zimmermann 1989). Capsulitis of the shoulder is thought to have an early inflammatory stage, and frequently has a non-traumatic onset (Stam 1994). Sustained or repeated activities can lead to micro-trauma of soft tissues and insidious onset of conditions such as lateral epicondylitis (Binder and Hazleman 1983)

Behaviour of pain

Chemical irritants do not appear and disappear during the course of the day. Pain of chemical origin is constant, and therefore patients who experience periods of the day when no pain is present or who describe that certain positions are painless do not have pain of inflammation. Adoption of a certain position that stops all pain excludes the possibility of a chemical or inflammatory condition. The description of intermittence immediately excludes chemical or inflammatory origins for musculoskeletal pain. The provocation of symptoms only as a result of mechanical loading excludes inflammatory causes and allows for a completely mechanical approach to diagnosis and treatment.

Just as tissue damage always causes inflammation, so inflammation always causes the tissues to become hypersensitive (Levine and Taiwo 1994). The inflammatory irritants sensitise the local pain receptor system and lower the thresholds at which the system is triggered to create a state of 'peripheral sensitisation' (Cousins 1994; Woolf 1991) In this situation the application of relatively minor mechanical stresses causes pain that under normal circumstances would not occur – allodynia; noxious stimuli create exaggerated responses – primary hyperalgesia; and there may be a spread of hyper-responsiveness to non-injured tissue – secondary hyperalgesia (Cousins 1994; Levine and Taiwo 1994). Thus, at this stage, there will be aching at rest and exaggerated tenderness and pain with touch and movement (Levine and Taiwo 1994). Thus, movement may superimpose mechanical forces on an existing chemical pain and increase it, but they will never reduce or abolish chemical pain. This is significant in the differentiation between chemical and mechanical pain. Repeated movements are likely to cause a lasting worsening of symptoms.

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

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

Musculoskeletal symptoms due to inflammation need appropriate time-limited relative rest prior to mobilisation. Certain conditions can present at an inflammatory stage when the value of mechanical treatment is limited, or later when the problem has become mechanical and responds well to treatment. The assessment protocol described in later chapters is invaluable in determining whether pain is arising from the inflammatory process following trauma, infection

or disease. Some problems of recent onset, which are aggravated by early attempts to encourage movement, may, following a limited period of rest or relative rest, then begin to respond to progressive mobilisation. The diagnostic elements of an appropriate mechanical evaluation allow the identification of mechanical and symptomatic disorders amenable to mechanical therapy. It is important to recognise that clearly establishing the stage of the disorder is more important in determining management than a structural diagnosis.

Key factors in the identification of pain of an inflammatory nature

- constant pain
- shortly after onset (traumatic or possibly insidious)
- cardinal signs – swelling, redness, heat, tenderness
- lasting aggravation of pain in some or all movements
- no movement found which reduces or abolishes pain.

Stage and status of disorder

The same conditions can display chemical or mechanical sources of pain depending on the time of assessment – conditions often pass through different stages. Early after trauma or the insidious onset of an inflamed joint, tendon, bursa or capsule, constant pain will be experienced due to the inflammatory nature of the condition. Later on, when adhesions, scar tissue or stiff tissue become the main problem, intermittent pain is experienced only when the tissue is stressed. In the acute/sub-acute stage of the problem, repeated movements may cause a *lasting* increase in pain, and mechanical therapy must be applied with caution. This represents an interface between chemical and mechanical pain. In later stages repeated movements cause only a transient production of pain, which improves gradually on repetition, and mechanical therapy is appropriate. This situation represents an entirely mechanical source of pain. *The same disorder may present anywhere along this continuum, depending upon the time at which the patient attends for treatment.* In terms of appropriate management the establishment of this pain behaviour is more important than the structural diagnosis.

Following trauma the acute inflammatory stage should last no more than five days, although this may be prolonged if the delicate healing tissue is re-traumatised (Evans 1980). In some musculoskeletal

conditions, such as lateral epicondylitis and supraspinatus tendonitis, the persistence of symptoms long after the normal healing period is said to be due to chronic inflammation (Gross 1992; Perry 1992). In fact, histological studies of chronic tendon problems have never found inflammatory cells (Verhaar *et al.* 1993; Nirschl 1992; Chard *et al.* 1994). Instead, in patients with long-standing tennis elbow or rotator cuff disorders, the changes are degenerative and represent a failed attempt at repair and a response to the relative avascularity of the tendon (Fukuda *et al.* 1994; Chard *et al.* 1994). Chronic tendonopathies appear to be due to hypoxic degeneration and very sensitive granulation tissue rather than ongoing inflammation, with the orderly phased wound repair apparently absent or aborted (Leadbetter 1992). The tissues are left mechanically and structurally compromised, deconditioned for normal use, and hypersensitive to normal loading.

Management will also be guided by the status of the condition – that is, whether it is improving, worsening or unchanging. In these degenerated tissues in which healing is suspended, the problem will be relatively stable and unchanging over a persistent period despite normal use of the limb. In this instance temporary aggravation of the symptoms for a few days may be necessary to re-stimulate the repair process.

Constant pain – mechanical cause (Constant Tissue Deformation)

Cause and onset

Constant mechanical deformation of tissue containing pain receptors will cause constant pain, which may vary in intensity but will never disappear. Mechanical pain is a less well understood phenomenon because of the technical difficulties of studying it (Bogduk 1993). It is thought to be the result of the deformation of collagen networks so that free nerve endings are squeezed between the collagen fibres with the excessive pressure perceived as pain (Bogduk 1993). As long as applied forces remain to cause sufficient tissue deformation, pain will be experienced. This will occur most frequently because of internal derangement, dislocation and/or displacement of loose bodies such as sequestered cartilage or meniscal tissues. Internal derangement alters the tension in the structures within or about the segment, increasing mechanical deformation in some tissues and

decreasing it in others. The constant increase in tension resulting from the displacement produces constant pain that continues until the tension is decreased by reduction of the derangement or by adaptive changes occurring with time in the surrounding soft tissues.

Although a history of trauma will frequently have occurred, onset may also happen without apparent force. This is more likely when tissues are in a weakened state due to overuse or degeneration.

Behaviour of pain

Some movements or positions may be found that reduce symptoms, while other positions or movements may aggravate them. The term 'constant pain' must be used only when the symptoms are truly constant; that is, there is no time in the day when pain or aching is not present. Pain must be classified as 'intermittent' even if there is only half an hour in the day when the patient feels completely pain-free. In that half-hour there is no mechanical deformation present and we must examine the circumstances in which the patient is pain-free and utilise this information for treatment purposes. It should be noted that patients might adopt a strategy which brings some relief from pain but which does not cause resolution. The information that the patient provides about aggravating and relieving factors may help the clinician develop the appropriate strategy. Constant pain of mechanical origin will be associated with an obstruction to movement, and therefore an impaired range – this mechanical presentation should improve in line with the symptomatic presentation.

It is much more simple to treat a patient who has intermittent pain than one whose pain is constant and in whom mechanical deformation is continuous and present under all circumstances. Patients with constant pain are less likely to be able to provide clues as to the likely direction in which to provide therapeutic motion.

When a patient experiences truly constant pain, it can only be because of an inflammatory response to existing disease or recent injury, from some form of internal derangement, or from hypersensitised neural tissues in ongoing pain states. If an underlying inflammatory disease process is the cause of the symptoms, pain will be constant, all repeated movements will cause an increase in pain, and no movement or position will be found that reduces or stops the pain. If repeated movements or certain positions can be found that reduce or stop the

pain, the cause cannot be inflammatory and must be a result of internal derangement.

Key factors in identifying constant pain of mechanical origin

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

Intermittent pain – mechanical cause (Intermittent Tissue Deformation)

Cause and onset

Intermittent pain cannot be chemical in origin. Intermittent pain can arise from either prolonged positional (or postural) causes acting on normal tissue, or from mechanical stress applied to contracted, abnormal or displaced tissue during movement. It is thus the result of abnormal stresses on normal tissue, or normal stresses on abnormal tissues.

The development of abnormal tissue is usually a consequence of imperfect repair following injury. The abnormal tissue may be the result of a past traumatic event such as 'twisting an ankle' and producing a lateral ligament sprain, or pushing off too eagerly at the beginning of the football season and causing a calf muscle strain. Abnormal tissue can also develop without a history of trauma from repetitive overload, capsular reaction when joints have been irritated or degenerative changes.

Certain joints are also prone to developing loose intra-articular displacements; in the peripheral joints these are the knee and jaw, and the elbow, hip and tarsal joints less frequently (Cyriax 1982). These loose fragments will cause pain and obstruction to movement when trapped between the joint surfaces.

Mechanical pain in the musculoskeletal system may thus arise due to three situations:

- postural pains due to excessive stresses on normal tissues
- normal stresses on abnormally contracted or scarred tissue
- normal stresses on an articular derangement.

Behaviour of pain

Postural pains – normal tissue

It is not necessary actually to damage tissue containing pain receptors in order to provoke pain (Bogduk 1993; McKenzie 1981). Pain of mechanical origin will be experienced as soon as mechanical forces applied to innervated structures are sufficient to *stress or deform* free nerve endings contained within. Pain will disappear when the application of that force is terminated, and this often occurs by a mere change of position. This is termed 'physiological' or transient pain. It warns the body of potential damage and is characterised by a correlation with the mechanical stress producing the pain and lack of long-term sequelae, as there is no tissue damage (Woolf 1991; Woolf *et al.* 1998). A good example is the pain incurred during prolonged slouched sitting that disappears upon standing upright. Similarly, when a finger is bent backwards slowly, first a strain is felt. If a position just short of strain which would produce actual tissue damage is maintained for a few minutes a mild aching occurs. If the finger is bent suddenly by applying a brief, rapid end-range force, the pain receptor system will be activated immediately, but briefly. Pain can eventually appear after moderate prolonged loading or can appear immediately as a result of sudden short-lived overstretching. In both cases the pain will cease on release of stretching. In neither case, however, is damage to tissue necessary to cause the pain.

The greater the mechanical loading and deformation become, the greater the intensity of pain. If the painful position is prolonged, the pain will become more diffuse, widespread, and difficult to define and often expands distally (Harms-Ringdahl 1986; Kellgren 1977). If no structural damage is caused by such loading, on release and return to its normal resting position the pain will become less diffuse, less widespread, more localised to its point of origin and soon subsides.

Increasing pain intensity at the end-range of movement indicates the beginning of overstretching. Further movement in the same direction will result in damage. As in the case of the finger, for example, the joint is obviously being moved in the wrong direction as the pain intensity increases, and in the corrective direction as the pain intensity decreases. The example demonstrates that the use of increasing and decreasing levels of pain provides a reliable guide in the choice of direction in which to apply therapeutic motion.

As discussed in the previous chapter, temporary pains may also arise in contractile structures because of the accumulation of metabolites in tissues in which high intramuscular pressure prevents an adequate blood supply (Mense 1997; Smith 1996). Unlike the mechanical stress on peri-articular structures, as in the finger example, these stresses will be in physiological mid-range, or at whatever point in the range the muscle is working at, rather than at end-range. These examples demonstrate that the experience of pain is not dependent on the presence of pathology or trauma.

These postural pains will arise from persistent loading at mid-range for contractile structures and end-range for peri-articular structures. They come on gradually, but will go as soon as the position is altered or the muscle relaxed, and may be triggered more easily if the same posture is repeatedly adopted. Patients will rarely present with such conditions; they learn that they only have to move out of that position and the pain disappears. This discomfort is generally accepted as somehow 'normal'; it does not particularly concern the individual and medical help is rarely sought for it.

Abnormal tissue

Pain arising from abnormal tissue will eventually concern the patient because it occurs regularly, every time they undertake certain activities. It also does not get 'better', as they expect, but remains for months or even years, giving them the same pain every time they do the same thing. It may get gradually worse with time as the collagen continues to contract. It may limit their normal range of movement and thus produce functional problems. Capsular tightening at the shoulder may make it impossible to hang out the washing; at the hip it may make walking painful and difficult. Old groin or calf strains may prevent the resumption of previous sporting activity. These tissue abnormalities will present with end-range pain on stretching the affected tissue.

Abnormal tissue may also be revealed *during* movement rather than at end-range. Although this may be the result of derangement of intra-articular structures, upon repeated movements this should change, getting either worse or better. Pain that appears *consistently* during mid-range loaded movements (painful arc) is most likely to arise from contractile tissues. This may occur when the affected tissue is compressed or constricted, as in lesions of the rotator cuff. The pain may arise on static, resisted, mid-range testing, for instance with

resisted wrist extension in lateral epicondylitis. The abnormal tissue will be consistently painful upon repeated movements, neither getting better nor worse, unchanging from one day to the next. The pain will cease when the offending position is released, but will always reappear in that same position and at exactly the same point during the range.

No chemical treatment will prevent pain that arises solely from mechanical deformation of soft tissues. When intermittent mechanical pain is the main presenting symptom, drugs should never be the first treatment of choice.

Key factors in identifying intermittent pain of mechanical origin

- in normal tissue pain will only be produced after prolonged loading
- in abnormal tissue pain will be consistently produced every time that tissue is loaded or moved in a way that stresses it
- loading involves end-range movements in abnormal peri-articular structures
- a restricted range of movement will be present
- loading involves force generation at any point through the range in abnormal contractile structures
- there will be a characteristic symptomatic response to loading strategies – pain will be produced on loading, but will abate when this is released
- intra-articular derangements will show a preference for movement in one direction.

In summary, persistent problems may be due to poor repair tissue that will need a remodelling programme, or it may be due to intra-articular displacements. In some degenerated tissues the repair process appears to have been suspended or aborted. Some chronic pain can be due to dysfunctional elements in the central nervous system, in which tissues are hypersensitive and deconditioned, and when a graduated exercise programme is indicated. We will now consider these scenarios in more detail.

Failure to remodel repair tissue

It is a mistake to think that once repair is complete that pain will cease. It is also a mistake to think that as long as pain persists, repair is incomplete. Pain will be experienced until repair is complete and function is made full and free. Thus, long after healing is complete, structural impairment may continue to cause pain on static loading or with loading from movement or activity. Pain may persist long after healing is complete if the repair itself is contracted, adherent, impinged or suffers from compression by constriction. In the presence of imperfect repair, pain will appear whenever mid-range loading or end-range stress is applied to the shortened repair, when adherence restricts movement, or when affected tissue is itself compressed as occurs when tendons pass through constrictions or over bony prominences. The weakened state of the tissue will also leave it prone to further structural failure.

This imperfect repair tissue may follow from a failure to adequately remodel tissue damage after trauma. Equally abnormal tissue can present as the consequence of various degenerative conditions such as osteoarthritis or gleno-humeral capsulitis. These problems may present at a later stage when tissue contracture is the main cause of pain and loss of function – a similar remodelling programme is necessary. Abnormal tissue is also found in contractile structures as a consequence of insidious onset overuse conditions when the repair process has been incomplete.

Contractile structures, like all other musculoskeletal tissue, may go through the inflammatory and repair stages of healing, but not the remodelling stage. Adhesions, contractures or scar tissue may result from movement avoidance and the consequences of the repair process. This may leave the muscle or tendon with a nodule of abnormal tissue that is sensitised to normal loading. It may produce pain when the muscle is called upon to contract, if the muscle is stretched or when the sensitised nodule is compressed by other structures. Often this is the result of imperfect repair tissue that has been inadequately rehabilitated. In some instances in tendons, where degeneration pre-exists, this may result from an absent or aborted repair process. The failure to progress through the normal phases of healing is consequent upon intrinsic tissue properties, such as hypoxia or degeneration (Leadbetter 1992; Buckwalter *et al.* 1993).

Tension or compression loading of this abnormal tissue will cause pain immediately when the stretch or compression is applied at the point that challenges those particular tissues. In peri-articular structures we are used to seeing this phenomenon at end-range. In contractile tissues this scar tissue will be challenged during the range of movement. It represents a restriction of muscular function and the pain will appear whenever the contractile element is called upon to move either in an active or resisted way, or when the tissue is stretched. This will produce pain during movement, as well as pain at end-range. The pain should cease once the contraction, stretch or compression is avoided. Pain from this cause will not appear if the patient avoids the painful or limited movement. *Stretching or compression of imperfect fibrous repair is the most common cause of persisting musculotendonous, discomfort and pain in the extremities.*

Derangement

Intermittent mechanical pain may be due to deformation of articular structures by internal derangement. Cartilage fragments, other 'joint debris' or meniscal tissues may be interposed between joint surfaces causing blockage of movement and abnormal stresses on peri-articular structures. Inadvertent 'manipulation' by the patient may reduce the particle into the joint recesses, and so it no longer constitutes a blockage to movement or cause of pain. Cyriax (1982) stated that such internal derangement of joints is 'far commoner than was formerly supposed', especially at the knee, elbow, wrist, hip, acromioclavicular and tempero-mandibular joints. A degree of degeneration or trauma, which allows the production of the loose body, is a necessary prerequisite for the condition.

Examples include osteochondritis dissecans at the knee or elbow, in which repeated micro-trauma produces a patch of necrotic bone resulting in intra-articular loose bodies. These can cause locking of the joint, a blockage to flexion or extension, and may be visible using magnetic radiographic imaging (Ho 1995; Patten 1995). At the hip joint, tears of the acetabular labrum may provoke discrete episodes of sharp pain when twisting, sometimes associated with a click as the loose tissue is trapped in the joint. The condition may progress so that pain is felt more frequently or even becomes constant. The onset of symptoms in the majority of patients was associated with minor injuries or no trauma at all (Fitzgerald 1995)

Mulligan (1992) has suggested mobilisation with movement techniques for such conditions as 'tennis elbow' and lateral ligament of the ankle sprains. The problems are said to resolve rapidly, unlike true tennis elbow. He proposes minor articular positional faults for such pseudo-tennis elbows and ligament sprains, possibly akin to derangements.

Twinges and episodes of pain are likely to be associated with blockage of movement as in the examples above. The circumstances when the patient is pain-free may offer clues to treatment strategies. Sometimes the patient is even aware of certain movements that 'manipulate' the joint sufficiently to temporarily clear the painful obstruction. Although the patient with intermittent signs of derangement is certainly easier to treat than a patient with constant mechanical derangement, this condition is more likely to recur.

Chronic pain states

The acute and sub-acute model of tissue injury and healing described earlier is not an appropriate model for an understanding of chronic pain. If pain persists beyond the normal healing time, other factors may exist which complicate the picture (Johnson 1997). Persistent peripheral nociceptive input can induce changes in the central nervous system (Woolf 1991; Melzack 1988). This may lead to the sensitisation of neurones in the dorsal horn – a state characterised by reduced thresholds and increased responses to afferent input, such that normal mechanical stimuli is interpreted as pain. As well, there may be heightened responses to repeated stimuli, expansion of receptor fields and spontaneous generation of neuronal activity (Johnson 1997; Siddall and Cousins 1997; Dubner 1991; Cousins 1994). This is known as central sensitisation. Thus, nociceptive signals can also be initiated in altered parts of the peripheral or central nervous system which may produce the effect of localised 'phantom' pain in a part of the periphery where tissue damage no longer exists (Bogduk 1993; Zimmermann 1989). Pain may radiate into uninjured areas adjacent to the original problem (secondary hyperalgesia), normal movement can be painful (allodynia), repeated movements can exaggerate pain responses, and pain signals may fire off without any appropriate stimulus (ectopic pain signals).

Psychosocial factors certainly have a role in peoples' response to a painful experience and may also be important in maintaining chronic

pain (Bogduk 1993; Johnson 1997). Factors affecting pain responses are cultural, learned behaviour, meaning of pain, fear and anxiety, neuroticism, lack of control of events, passive coping style and focus on the pain (Cousins 1994). Clinicians' behaviour towards patients at all stages of a condition should guard against encouraging any of these passive responses to pain – especially in the chronic patient. It is hardly surprising that patients get depressed, anxious, fearful and focused on their persistent pain. Health care professionals seem unable to deal with it, some of whom imply it is primarily 'in their heads' as the pain is *'apparently discordant with discernible abnormalities'* (Awerbuch 1995). Maladaptive or inappropriate behaviour in the face of ongoing pain, however, does not represent malingering. It should be remembered that on the whole the emotional disturbance is more likely to be a consequence of chronic pain, rather than its cause (Gamsa 1990).

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

Gifford (1998) offers a useful approach to this small but difficult patient group. *"On-going pain states are best explained to patients in terms of an altered sensitivity state as a result of altered information processing throughout the system, and not solely a result of damaged and degenerating tissues. This helps patients accept the notion that hurt does not necessarily equate with harm – which leads on to the positive message that carefully graded increases in physical activity mean stronger and healthier tissues. By contrast, continued focus on a tissue as the pain source reinforces fear of movement and activity, the need to be constantly vigilant for pain and the desire for increasingly*

expensive passive therapeutic interventions that are yet to demonstrate convincing efficacy" (p 33)

Although these complicating factors may undermine treatment attempts, many patients with persistent symptoms will respond to mechanical therapy, and a mechanical assessment should never be denied patients according to the duration of their symptoms. However, in patients with persistent symptoms there is a need to recognise the possible importance of non-mechanical pain behaviour. This may involve peripheral sensitisation, central sensitisation or psychosocially mediated pain behaviour, or any combination of these factors, which will obscure or complicate any purely mechanical approach. The causes of chronic pain are different from the causes of acute pain. Although both problems may encourage reduced normal use of the hurting part, in the acute stage this is appropriate and relevant, whereas in the chronic stage this is inappropriate and irrelevant. Thus the management of these two stages of a condition should logically be driven by a different approach.

Conclusion – the state of the tissues

When patients present with painful musculoskeletal problems this may thus be due to different conditions in peripheral or central structures. Tissues may be in one of five abnormal states, with the pain maintained by different mechanisms:

Table 3.1 The state of the tissues

State of tissues	*Pain mechanism*
normal	(abnormal stress – mechanical)
inflamed (acute)	predominantly chemical
healing (sub-acute)	chemical/mechanical interface
abnormal (contracted/scar tissue)	mechanical
abnormal (derangement)	mechanical
persisting hypersensitivity (chronic)	peripheral/central sensitisation

From the history-taking and physical examination we need to be able to distinguish between these different states so that the different and appropriate management strategies can be introduced.

4: Mechanical Diagnosis

Introduction

Clinicians and patients like to have a diagnosis. The medical model is founded on the principle of a diagnosis preceding treatment. Indeed, it is necessary to have a precise diagnosis if one is about to commit a patient to any invasive therapeutic procedure. However, a tissue-specific diagnosis is not a prerequisite for the delivery of non-invasive therapies such as those provided by physiotherapists. While it is true that it is more satisfying professionally to have made a specific diagnosis, for physiotherapists the absence of a diagnosis need not impair the treatment process itself.

The medical model

Clinicians within different specialities require different levels of diagnostic precision. A surgeon about to perform a tendon transplant or repair must know precisely what is wrong with the affected structures. The exact location of the damage must be identified. There needs to be a precise correlation between the pathoanatomy and the source of the patient's symptoms. It must be clear what is to be done to modify and repair the anatomical disruption. The surgeon must therefore have a precise diagnosis, and the experience and expertise to perform the procedure and achieve a successful outcome. Without that diagnosis and expertise, surgery is hazardous and further injury inevitable.

The same precision in diagnosis is required, for example, when injections are contemplated for the treatment of tendonitis or capsulitis of the shoulder. The affected structure must be accurately identified. Precision in delivery is vital for the success of the procedure.

The role of diagnosis in musculoskeletal problems

It has been customary for clinicians to attempt to obtain a precise diagnosis when assessing musculoskeletal disorders. Traditionally, the affected anatomical structure is first identified. Then it is determined what the problem with that structure might be. This information allows the implementation of the treatment protocol

usually recommended for such a problem. Identification of the affected structure is achieved by following a recommended sequence of active, resisted and end-range movements. James Cyriax (1982) developed his system of selective tissue tension testing which attempts the specific identification of sources of musculoskeletal pain. This testing is an attempt to identify the precise location of lesions requiring either injection or frictional massage. Using this system it may be possible to locate the structure at fault.

As was noted in the introductory chapter, problems exist concerning the value of this structural diagnosis. First, there is no absolute evidence that tests, which purport to examine certain structures, actually do so – the problem of validity. Second, the evidence demonstrates that different clinicians will frequently come to different conclusions when using these tests – the problem of reliability (Liesdek *et al.* 1997; Cushnaghan *et al.* 1990). Within this area it has been shown that symptomatic responses can be assessed more reliably than physical signs (Jones *et al.* 1992).

Third, it might be questioned if it is really necessary for the clinician to obtain a specific diagnosis when invasive therapy is not under consideration. The answer might be 'no', as long as there is a general understanding of musculoskeletal tissue behaviour in health and pathology and the implications this has on management. What difference will a diagnostic label of structural pathology make to the treatment process? If the clinician is unable to reach a firm diagnosis because signs and symptoms are confusing or contradictory, does this mean they are incapable of offering a management strategy?

Furthermore, a tissue-specific diagnosis, even if readily obtainable, will not reveal the stage of a disorder which will determine the vigour with which to commence therapeutic activity and is a crucial determinant of management decisions. Whether a condition is acute or chronic is often more relevant to this decision than a precise diagnosis. A diagnosis that implicates a certain tissue also cannot determine what type of pain is present – whether the source is local nociceptive, central sensitisation or related to higher centres.

Whatever the diagnosis actually is, the clinician will assess the patient's response to certain movements and then manage the patient in light of the responses obtained. In many cases knowing which structure is the cause of the pain appears to be purely academic and is not

necessary for treatment to occur. Management of the patient may continue in the absence of a specific structural diagnosis, but will be based upon the patient's responses to treatment as reflected in changes in symptoms, movement and function. Within physiotherapy, management is often determined by a patient's impairment, disability and the stage of the disorder; that is, a general rather than a specific tissue diagnosis (Jette and Delitto 1997; van Baar *et al.* 1998; Dekker *et al.* 1993).

Making a non-specific diagnosis

The problems of diagnosis for non-specific mechanical disorders of the spine are well documented (Spitzer *et al.* 1987; Rosen *et al.* 1994). In order to overcome those difficulties, an alternative system of classifying spinal disorders was proposed by McKenzie (1981). This system is based on the symptomatic and mechanical responses of patients to various repeated movements or static loading forces (that is, a mechanical evaluation). It allows the classification of patients into broad rather than tissue-specific categories, and thus leads to the formulation of treatment. Rather than seeking to make a diagnosis, which is the identification of a disease by means of its signs and symptoms, the McKenzie system concentrates on syndrome identification. A syndrome is a group of symptoms and a pattern of responses characteristic of a particular problem. The system is now widely used to classify and treat patients with mechanical spinal disorders.

McKenzie (1981) suggested that this method of assessment was equally applicable and effective for the assessment and treatment of mechanical musculoskeletal disorders of the human extremities. By applying a structured sequence of loading forces, it is possible to identify three separate mechanical syndromes. The characteristics of each syndrome in response to repeated and/or sustained end-range loading are completely different from each other. Within these syndromes can be found the vast majority of mechanical disorders of the musculoskeletal system. The history-taking and examination described in later chapters therefore needs to determine the stage of the disorder and the response to mechanical loading. This assessment should allow classification into one of the following mechanical syndromes: an inflammatory problem following injury or a chronic pain syndrome.

The postural syndrome

Pain from the postural syndrome is caused by mechanical deformation of soft tissues or vascular deprivation arising from prolonged positional or postural stresses affecting the articular structures or the contractile muscles, their tendons or the periosteal insertions.

When postural pain arises from joint capsules or adjacent supportive ligaments, it is the result of prolonged end-range positioning. Moving from the end-range is sufficient to relieve pain immediately.

When postural pain arises from contractile tissues it is usually the result of prolonged static mid-range loading. Although the joint may be in mid-range, this will not reflect the state of the contractile tissues that are holding the joint in that position. This may cause mechanical deformation of tissues or a reduction in blood supply, so that there is a build-up of metabolites and relative ischaemia. Relief from pain can be obtained with a change of position that allows sufficient relaxation of the involved musculature. This removes tension from the contractile tissue and permits the return of normal vascularity.

Thus, the postural syndrome is characterised by intermittent pain brought on only by prolonged static loading of normal tissues. Patients with the postural syndrome experience no pain with movement or activity, nor do they suffer restriction of movement. No pathological changes occur in this syndrome.

Clinically, patients with pain of postural syndrome are not often seen. However, the role of postural stresses on the genesis and persistence of musculoskeletal conditions is very important. Postural syndrome is not a discrete entity, but a part of a continuum. These patients, if they do not alter their static and dynamic postural habits, can progress to cumulative trauma disorders or repetitive strain injuries. Dynamic postural stresses also have a role in prolonging a variety of disorders, for instance anterior knee pain and lateral epicondylitis.

The dysfunction syndrome

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

When structural changes and or impairment affect joint capsules or adjacent supporting ligaments, painful restriction of end-range movements in one or more directions will be experienced.

When structural changes and or impairment affect contractile muscles or tendons, pain may be experienced during resisted movement or loading at any point through the whole range. Pain on resisted movement will usually occur in one direction, and pain will also be provoked if the tissue can be stretched. In other words, the dysfunction will impair both types of movement that are required in the normal activity of a muscle – both as it contracts and as it is stretched.

In the dysfunction syndrome, pain is never constant and appears only as the affected structures are mechanically loaded. Pain will stop almost immediately on cessation of loading.

Thus, when affecting articular structures, the dysfunction syndrome is always characterised by intermittent pain and a restriction of end-range movement. When affecting the contractile elements, the dysfunction syndrome is characterised by intermittent pain occurring only during movement or loading of the structural impairment. This may be mid-range for the joint, but end-range for the contractile tissue.

Pain from the dysfunction syndrome will persist until remodelling of the affected structures has occurred. In dysfunction involving peri-articular structures, this will be attained once the range of motion has returned to normal. In contractile structures the remodelling process must affect the tissues' ability to contract as well as to be stretched. This can take months, and in some cases pain persists throughout life. Patients with dysfunction syndrome in peripheral tissues are very commonly seen in the clinic.

The derangement syndrome

Pain from the derangement syndrome, when affecting the spinal column, is caused by internal disruption and displacement of the fluid nucleus/annulus complex of the intervertebral disc. This in turn mechanically deforms the outer innervated annulus fibroses and/or adjacent soft tissues resulting in back pain alone or back and referred pain depending on the degree of internal displacement and whether or not this causes compression of the nerve root. The pain of

derangement is often constant and remains constant until the displaced tissue is reduced. As a result of displacement of tissue within the intact annulus, sudden obstruction to movement can occur at the time of the onset of pain. Repeated movements that increase the displacement also increase the obstruction that in turn increases the pain. Repeated movements that progressively reduce the pain also progressively reduce the obstruction and derangement and allow the restoration of normal pain-free movement.

Cyriax (1982) believed that internal derangement of joints is a common cause of many obscure pains in the extremities. When affecting the knee and other articulations containing meniscoid cartilage, tearing and or displacement of the deranged menisci can similarly cause painful locking and /or sudden obstruction to movement. Almost any articulation has the potential to develop loose bodies that can displace and cause similar symptoms. Joints most affected are the knee, wrist, elbow, acromioclavicular and ankle, hip and tempero-mandibular. Certain repeated movements that progressively reduce the pain also have the capacity to reduce the obstruction or locking

Internal derangement may cause a disturbance in the normal resting position of the affected joint surfaces. This will, in turn, deform the capsule and peri-articular supportive ligaments resulting in pain. ***Internal dislocation of articular tissue of whatever origin will cause pain to remain constant until such time as the displacement is reduced or adaptive changes have remodelled the displaced tissues.*** **Internal dislocation of articular tissue obstructs movement attempted towards the direction of displacement.**

The derangement syndrome is often characterised by constant pain. Intermittent pain may be described when the size of the displacement is smaller and when the patient's movements or positions during the day are able to reduce the derangement so that it is no longer causing symptoms. Movements that open the joint space may temporarily reduce the pain, but promote greater displacement and more pain when the limb is moved again. Thus certain positions can be found that alleviate the pain while the position is maintained, but which aggravate or perpetuate the pain afterwards.

Derangement requires movement that encourages reduction of displaced articular tissue. This process can often be achieved rapidly.

Days rather than weeks of exercise are required for the completion of the reduction process. In some cases, particularly when the wrist and elbow are involved, immediate reduction can be achieved in a few minutes.

Non-mechanical conditions

So far musculoskeletal conditions that behave in a straightforward mechanical manner have been described. Mechanical loading or deformation produces pain either immediately or after sustained loading in all three syndromes, and cessation of that tissue loading or deformation causes the pain to abate. In certain instances, although mechanical factors may be significant in pain behaviour, other factors must also be considered. These are the acute and chronic phases of musculoskeletal conditions.

Acute pain

Immediately following trauma or the insidious onset of a musculoskeletal condition, the prime pain generator is likely to be chemical. Although mechanical forces will aggravate the symptoms, they cannot be used to relieve them when the pain is primarily inflammatory in nature. However, the period when rest and relative rest are appropriate is short-lived – less than a week following an injury. If prolonged, this strategy will start to have a deleterious effect on recovery and function. During the sub-acute phase, tissue strength is low and re-injury is possible if functional rehabilitation is progressed too rapidly.

Chronic pain

Very often even symptoms that have persisted for months or years can behave in a mechanical fashion and can be easily classified into one of the mechanical syndromes outlined above. The persistence of a problem must never be used as the sole determinant of suitability for mechanical diagnosis and therapy. However, as has been discussed in Chapter Three, various non-mechanical factors may complicate interpretation of pain behaviour in chronic pain states.

The most relevant issue is the sensitisation of tissues that may occur peripherally and centrally in chronic pain. This will exaggerate responses to mechanical loading, producing pain when loading is absent, possibly causing constant symptoms and perpetuating maladaptive pain behaviour. Clinically this may look like chronic

inflammation, but rather than inflammatory chemicals mediating the pain this is largely the product of sensitised, deconditioned and abnormal tissue. This needs progressive functional loading to try to rectify the problem, and an initial mild aggravation of symptoms may occur. The exaggerated responses to mechanical loading that may be found in chronic conditions obscures and confuses normal mechanical responses. Once a progressive functional rehabilitation programme has been started and the patient is regularly and repeatedly loading those tissues, an underlying mechanical pain behaviour can sometimes be revealed. It is, at times, as if regular exercise therapy is able to desensitise the tissues so that normal mechanical responses are seen.

Conclusion

The three mechanical syndromes presented describe completely different patterns of mechanical and symptomatic responses when subjected to a structured repeated end-range movement and static loading evaluation. Each syndrome response to repeated movements is different from the other syndromes, and each syndrome must be treated as an entity on its own. Different information needs to be given to patients with the different syndromes, and they need to be instructed in different exercise procedures with different symptomatic responses.

Patients in the acute phase of a problem have pain that is primarily mediated by inflammatory cells and so their management is different. Many patients with chronic conditions will respond in a normal mechanical way. However, in some cases exaggerated sensitivity will persist long after the acute phase and deter the patient from normal activity. Various behavioural factors may have a role in symptom prolongation such as fear–avoidance behaviour, poor self-efficacy or passive coping strategies. Patients need to be encouraged through the initial aggravation of symptoms that is likely to occur upon starting a programme of progressive functional restoration – an underlying mechanical response to tissue loading may be found. Instances where there have been major neurophysiological or behavioural changes may need a multi-disciplinary approach. Rates of failure to respond will be high in this group.

In order to identify which syndrome is present in a particular patient, a history must be established and an examination must be performed. Following the syndrome identification, a patient management plan must be devised. The method of history-taking, the mechanical evaluation and patient management plan are discussed in the following chapters.

5: History

Introduction

"Every patient contains a truth.... The [clinician] must adopt a conscious humility, not towards the patient, but towards the truth concealed within the patient" (Cyriax 1982: 45). The patient knows the details of the history, onset, symptom pattern and behaviour since onset, and aggravating and relieving factors. This information is vital to gain knowledge of the disorder. In order to access this truth, the clinician must approach the patient in a respectful and friendly way, they must have a logical format for collecting information and, most importantly, they must listen actively to the patient's responses. Only from the patient is it possible to gain insights into various aspects of the clinical presentation, which are essential to inform issues such as the stage and nature of the disorder, the prognosis and the management. Very often taking a history will provide information that is at least as important as that gained from the physical examination, if not more so.

One of the key aspects of an episode of health care that affects patient satisfaction is the relationship between the patient and the clinician. Of particular importance are communication, empathy and reassurance (Sitzia and Wood 1997; Wensing *et al.* 1994). It is important that we make the patient as comfortable and as relaxed as is possible. Patients will enter all clinical situations with their own expectations, feelings and fears (Brown *et al.* 1986). It is important that these issues are recognised by the clinician, fears are allayed and questions answered. It is important to avoid the use of medical words or phrases that may be foreign to the patient. Conversation should be conducted using terms and phrases with which the patient will feel comfortable. From the outset listening is a key skill to clinicians, without which the information that the patient can provide will be useless – the clinician's understanding of their condition will be limited and their management will not be optimal.

Another aspect of care which is important in terms of the patient's satisfaction is professional skills. These may be displayed by clinician's thoroughness, accuracy of assessment and understanding of the patient's problem (Sitzia and Wood 1997; Wensing *et al.* 1994). An

initial way to display these skills is the use of a structured but flexible interview format so that all pertinent factors from the history and behaviour of the condition are collected.

The standard assessment form includes the most important aspects of the history that need gathering; mostly it will be unnecessary to add to this information. Be wary of gathering excessive amounts of information – always consider 'how will the answer help in managing this patient?' If it is deemed essential to gather further details about a particular aspect of the clinical presentation that causes concern, the form should not prevent further specific questioning. It is best to gather the information using open-ended questions first so that patients may volunteer their own answers rather than using leading questions. Focused questions may be used to follow up if particular aspects need more detailed information. Thus, management decisions can be grounded in the particular patient's problem and their response to it.

Aims of history-taking

Using the form and the appropriate questioning technique after taking the history, ideally the following will have been obtained:

- an overall impression of the clinical presentation
- the stage of the disorder – acute / sub-acute / chronic
- the status of the condition – improving / unchanging / worsening
- a hypothetical diagnosis by syndrome and site of lesion
- baseline measurements of the symptomatic (and mechanical presentations) against which improvements can be judged
- factors that aggravate and relieve the problem, which may help guide future management
- the severity of the problem which may guide the intensity of the physical examination
- the functional limitations that the condition has imposed on the patient's quality of life
- an impression about the way the patient is responding to their condition, and how much encouragement, information, reassurance or convincing they may need to be active participants in their own management.

The interview

Age

We need to be aware of the patient's age. Certain disorders are associated with particular ages. Degenerative and overuse problems start to become more frequent in those over 40. Frozen shoulder is rare in those younger than this, except in those who have had trauma, but after this age is not unusual as a problem which starts without known cause. Anterior knee pain not due to patellar-femoral osteoarthritis is most common in the young – teenage girls and young men in their twenties are commonly bothered by this complaint.

The age of the patient may also be significant in their response to the problem. Increasing years not only raise the susceptibility to disease and injury, but also reduce the body's ability to recover from the effects of musculoskeletal disease and injury (Buckwalter *et al.* 1993). A patient's age is therefore important in their prognosis.

Work and leisure activities

Particular employment may be significant in the aetiology and persistence of the patient's problem. Find out what their job is and about the postures and stresses that it involves. Carpet-layers and those forced to kneel a lot of the time may develop knee problems – protective measures such as kneepads may be important in management. Those involved in clerical work, in activities with repetitive arm activities such as working on a conveyor belt, or in tasks that involve using the arms above the head may all be prone to developing overuse tendonitis problems of the arm. Relative rest, analysis of their activity and frequent interruption of the task may be an important part of management. A worker's inability to interrupt the activity may be significant in a poor prognostic outlook.

Equally, certain leisure activities may be important in the genesis of a problem, and the patient's normal level of sports and hobbies should always be discussed. This will also give an idea about the patient's general level of exercise or lack of it. If patients have stopped certain activities it is important to know if this is because it actually makes the pain worse or because they think it will make the pain worse.

Functional disability

Find out what limitations in normal activity the present episode has caused. It is important to know if the patient is on sick leave or has curtailed his or her usual leisure activities because of the problem.

Long-term work absence due to musculoskeletal disorders is the cause of major expense throughout the developed world, and return to work is seen as a key goal of any intervention. Loss of work can also cause the individual severe financial hardship, affect their societal and domestic roles and encourage inappropriate pain behaviours. As a result, "*a basic tenet of secondary prevention* [of chronic musculoskeletal pain] *is to intervene early to prevent the disruption of the patient's normal lifestyle, including work*" (Linton 1996) and to encourage an early return to normal activity, even if gradual.

The patient's level of function prior to the problem is the suitable goal for management, and so remodelling and rehabilitation exercises need to be targeted at the appropriate functional or sporting tasks for that individual. This has important implications for management.

Where is the present pain?

Discuss the location of the pain during this episode. This gives a general indication of the site or sites that need to be included in the physical examination. Find out where the pain was at onset and if there has been any change since then; for instance, the pain may have become more or less widespread since onset. It can generally be said that as the condition worsens the pain will become more widespread and frequently will move distally. Conversely, as the condition improves it is common to see diffuse symptoms contract to a localised point close to the site of the initial injury. It is not uncommon for shoulder, elbow, hip and knee problems, when at their worst, to refer symptoms down the arm or leg respectively. Only rarely are symptoms ever referred up the limb.

Ask if the pain is always located in the same place or if it changes location with movement or over time. If the pain is more widespread at the end of the day compared with the morning, it may be an indication that more rest is required or that aggravating factors are introduced during the day.

Often the anatomical source of distal extremity problems is reasonably clear. However, confusion occurs especially if the pain is located proximally in the limb. The site of pain of many musculoskeletal problems can be quite separate from the source. Pain from the hip can be felt entirely at the knee, articular shoulder problems are frequently felt at the top of the arm, isolated symptoms in the arm or leg can remain unchanged until the spine is tested. Only eliminate

the spine as a source of problems when peripheral tissue loading clearly brings lasting changes to the patient's symptoms.

Problems at the shoulder or hip are especially easy to confuse with spinal problems. The specific area of pain is one way of helping to clarify these ambiguities. Pain from the shoulder is usually felt around the shoulder joint or at the top of the arm, whereas pain that is felt around the scapular, posterior shoulder, or base of the neck is likely to be cervical in origin and necessitates an examination of the cervical spine. The occurrence of neck and shoulder pain together is very common, both problems being concurrent in nearly half of a sample of over 300 patients (van der Windt *et al.* 1995).

The area of referred pain in the leg may help distinguish a hip from a lumbar problem. The sites of referred pain in 89 patients with osteoarthritis of the hip were combinations of the greater trochanter, knee, anterior thigh, groin, shin and buttock (Wroblewski 1978). In patients who present with some combination of these pain sites, especially if the pain is worsened on weight-bearing and eased by sitting, the hip joint should be suspected. However, in both putative shoulder and hip problems it is always wise to exclude the spine first. The presence of numbness, tingling, wasting or weakness should be enquired about, and if present should clearly shift the index of suspicion to the spine.

How long has the pain been present?

Knowing the length of time that the problem has been present allows us to determine the state of the tissues. Days after onset tissues may be damaged and inflamed, whereas a few weeks later tissues may be healing. If the symptoms have been present for a couple of months, adaptive shortening may have occurred in the collagenous repair tissue indicating that dysfunction may be the cause of persisting symptoms. If the problem has been present for many months, although a straightforward mechanical condition may be present, the chance that the tissues are hypersensitive and deconditioned should be born in mind. Chronic pain syndromes often complicate the management of persistent pain and may, although not always, make treatment less effective. Pain that has been present for many months as well as having a poorer prognosis may respond more slowly when it does respond. Many patients have a long or recurrent history of their problem and therefore the educational component of management is particularly important to improve their future self-

care. The duration of the episode thus provides diagnostic and prognostic information.

This should be classified as acute, sub-acute or chronic. These terms tend to be interpreted slightly differently by different authors; thus there is a certain ambiguity and arbitrariness about these divisions. For sake of clarity they will be defined here as follows:

- acute – pain present for less than seven days
- sub-acute – pain present from one week to seven weeks
- chronic – pain present for more than seven weeks.

The length of time that the patient has had symptoms can also guide us in deciding how vigorous we can be with mechanical assessment procedures. If a patient has had symptoms for several months and has been able to work or remain active during this time, he or she will probably have placed more stress on the structures at fault than we are likely to apply during our assessment process. This allows us to be fairly intense with the overall mechanical assessment. However, someone who presents with an injury of very recent onset needs to be examined with more care and managed in a completely different way.

If the symptoms have been of recent onset and are improving, it is unlikely that any intervention other than education will be necessary. If the symptoms are of recent onset and are not improving, then it may be necessary for the clinician to encourage some form of active mechanical therapy.

If the symptoms have been present for several weeks or months, education will always be helpful, but the longer the symptoms have been present the more likely it will be that active mechanical therapy will be required at an early stage in the management process.

Did the pain commence following injury?

If the symptoms are of recent onset and have been caused by injury, an inflammatory source of pain is likely and caution may be required in the physical examination. The exact mechanism of the injury should be determined if at all possible. The direction and force of the trauma may give clues as to the extent and nature of damage caused. Where significant trauma is involved it may well be that radiological investigation is required. When it is clear that no fracture

or dislocation is present the extent of bruising or evidence of swelling should be determined. In any case, where patients have suffered recent trauma of any significance, the initial mechanical assessment procedures should always be applied with caution. In acute cases the patient should be educated about the value of Rest, Ice, Compression and Elevation.

Did the pain commence for no apparent reason?

Alternatively it should be asked if the symptoms came on for no apparent reason. Musculoskeletal complaints commonly start with no obvious trauma. For instance, a high proportion of cases of shoulder pain, lateral epicondylitis and anterior knee pain commence without any known precipitating cause (van der Windt *et al.* 1995; Binder and Hazleman 1983; Fulkerson and Shea 1990). Symptoms of pain or limitation of movement commencing for no apparent reason are usually a result of inflammation, early degeneration, repetitive activity or prolonged positioning required within the domestic, recreational or occupational environment. We all experience environmental stresses of daily living. When patients complain of musculoskeletal problems appearing for no apparent reason, the identification of the causative factors is important for successful management of the problem. Any of the foregoing causes could be responsible for any given problem and should be systematically sourced so that appropriate recommendations can be made regarding treatment and prevention of further unnecessary loading. It is sometimes impossible to discover a clear causative factor, but the high incidence of symptoms that appear for no apparent reason would suggest that environmental and degenerative factors play a key role in the aetiology of musculoskeletal conditions.

From the patient's history it may be possible to gain some insight into the likely presence of a degenerative disorder. If the older patient describes a slow insidious onset of stiffness and pain for no apparent reason, it is possible that degenerative problems affecting either the joint or adjacent soft tissue are causative. The most common peripheral sites of degenerative changes are the knees, hands and feet, with hips, ankles and shoulders affected less frequently, and the elbows and wrists rarely (Petersson 1996; Huskisson and Hart 1987). Usually several sites become affected, and the majority describe stiffness in the morning and after inactivity. It should be remembered that radiographic signs of degeneration are almost universal after 55,

but that symptoms are present in only about 50% of those with such changes (Huskisson and Hart 1987)

Onset of symptoms for no apparent reason may also result from environmental factors. Everyday lifestyle activities can be, without the patient being aware, the cause of many common musculoskeletal problems. Certain sleeping postures may cause shoulder arm or hip and leg symptoms. Certain sitting postures may also cause symptoms in the upper or lower limbs. The relationship between sitting postures and the onset of peripheral symptoms should be examined in detail as the origin of the pain may well arise from the spine. Symptoms may also arise from repetitive loading incurred at work or during domestic or sporting activity.

Sometimes patients have commenced a new occupation or a sedentary worker has become involved in more physical employment, and symptoms develop because of unaccustomed activities. These activities on the surface may appear to the patient to be insufficient to cause their problem. However, if the individual is not conditioned to cope with the unaccustomed exertion, the relationship between the new occupation or activity and the onset of symptoms should always be considered.

Often patients are reluctant to accept that there is no obvious reason for the onset of symptoms and ascribe a causative role to some recent event. When questioned more closely it is apparent that the event, such as taking some unusual exercise or a fall, is a week removed from when the problem actually started. It is especially important to disabuse patients of misconceived causality when it is apportioned to an attempt at exercise.

Where symptoms have commenced for no apparent reason it is always possible that some more sinister cause may be present. The likelihood of the presence or otherwise of serious pathologies should be determined from further development of the history and from the mechanical evaluation.

Is the problem improving, worsening or unchanging?

This important question determines the stage and status of the disorder, which will guide management decisions. Where the patient records that their problem is steadily resolving it will be necessary to provide only education at this point. Should improvement cease at

some future point in time, the need for mechanical intervention can be considered then.

In the event that the patient reports that their condition is worsening, measures must immediately be taken to evaluate and identify the appropriate course of management. Certainly education must be provided on the first day.

If the patient reports that their problem is unchanging, education and possibly mechanical intervention may be required. Frequently patients report that after an initial period of improvement the condition has been unchanged for several months, getting neither better nor worse despite limitation of activities. Such a history suggests a need for a new more active approach to management, and a stable condition that will benefit from exercise. This type of history may begin to suggest the presence of persistent dysfunction, a chronic pain state, or a structure that has become 'stuck' in the healing process. A fairly vigorous management approach can be proposed in these relatively stable conditions.

Is the pain constant?

This is one of the most important questions with musculoskeletal disorders. Be sure that by 'constant pain' the patient does actually mean that their pain is there '100 per cent of their waking day', 'from the moment you get up to the moment you fall asleep'. Some patients can always produce their pain with certain movements, and they interpret this as constancy.

Constant pain is caused by inflammatory diseases and is also present where patients have suffered recent trauma causing an inflammatory response. Constant pain may also result from internal articular derangement. There will be various clues in the history which will suggest which of these options are the most likely. Mode of onset, age, associated stiffness and pain behaviour, for instance, will reveal this.

From the mechanical evaluation it will be possible to confirm whether the cause is inflammatory or not. If the cause of the constant ache or pain is chemical the symptoms will not be reduced or abolished by mechanical assessment procedures. If the cause is mechanical in origin, movements and positions will usually be found that reduce or stop the aching or pain. Constant mechanical pain is the result of

internal derangement. The derangement syndrome can be associated with a constant ache, whereas patients with the postural and dysfunction syndromes do not experience constant pain. Constant pain is more difficult to treat than intermittent pain because the patient is usually unable to identify a directional or postural preference.

Chronic pain syndromes may also present with constant pain, which also may worsen initially on mechanical assessment. The history will reveal the length of time symptoms have been present, and also may relate previous failed treatments. Constant pain may also be caused rarely by serious non-musculoskeletal pathology.

Is the pain intermittent?

A description of intermittent pain generally excludes the possibility that the pain is inflammatory. Should the pain cease at rest or when the patient is still, the pain is not a result of an inflammatory response and must therefore be mechanical in origin.

It could be postural or result from dysfunction or derangement. Intermittent pain is relatively easy to treat because if there is one hour in the day when no mechanical deformation is present, it is possible gradually to extend that pain-free period by duplicating the favourable circumstances. In addition, the patient is usually already aware of certain movements or positions that bring relief, thus indicating the likelihood of the presence of a directional or postural preference.

A change in the frequency of the pain from constant to intermittent, or reduction of intermittent pain from being present most of the time to present only sometimes represents improvement in symptomatic presentation. Such improvement should help in the overall assessment of management strategies. It is therefore useful to ask about the proportion of the day that pain is present. This could be expressed as a percentage; for instance, "on average are your symptoms there for 80% of the day, 50% of the day, 20% or how much of the time?"

What activities produce or worsen the pain? What activities stop or reduce the pain? Does it remain worse or better afterwards?

These questions allow the patient to provide the information that is likely to lead to the appropriate management. They are designed to determine what movements or positions produce or abolish, or

increase or decrease, mechanical loading and or deformation of the affected structures. It is important to record those movements, positions or activities that specifically reduce or relieve the pain as this information will be utilised in our management protocols. It is also important to record which movements or activities aggravate the symptoms. As part of the educational strategy it may be necessary to temporarily avoid such causative factors, or alter the way an activity is performed so that stresses are lessened.

If symptoms are intermittent it is important to know the positions, movements or activities that produce the pain. We also wish to know if these activities consistently produce the pain, and what happens when the activity is stopped. If pain is always brought on by the same activity and ceases shortly after cessation of that activity, we may begin to suspect dysfunction.

It is helpful to know if the movement that relieves or aggravates the pain brings about a lasting improvement or worsening. A lasting improvement following a particular loading strategy will give a useful idea about self-treatment procedures. If pain is produced by certain activities but quickly abates once the movement ceases, the disorder is at reasonably stable stage. If pain remains worse for hours after a relatively trivial movement, the disorder may still be at an inflammatory stage. Thus knowing if the pain remains worse or better afterwards has important implications for self-treatment procedures.

If uncertainty exists as to whether a condition is spinal or peripheral, the activities that produce or aggravate symptoms may also be helpful in determining the source of the problem.

Does continued use of the affected area increase, decrease or have no effect on your pain?

In certain cases, pain felt initially with activity diminishes and then stops after a period of time, even though the activity is continued. Although the pain may reappear later, this sequence of events indicates the value of repeating the movements or activities involved. Conversely, if pain progressively increases, the indication in this event is for immediate cessation of the movement or activity, especially if it remains worse after. Should the activity have little or no effect on the symptoms at the time of application, there is no indication that the activity should be avoided.

It is not uncommon for patients with musculoskeletal problems to describe stiffness and aching first thing in the morning or after resting which abates with gentle activity. Such pain behaviour may be due to degenerative joint conditions, but equally happens in healing collagenous tissue that has been insufficiently remodelled. Whatever its cause it indicates regular movement as being an appropriate part of management.

Do you have pain if the limb or part is relaxed at rest?
If the patient describes that lying in bed or resting or relaxing the affected part stops his or her symptoms, the presence of an inflammatory source of pain is excluded. However, when all mechanical stresses are removed but the aching continues unabated, it must be due to inflammation, derangement or serious pathology.

It is not uncommon for certain mechanical conditions to wake people at night. This may not be the result of inflammatory pain but due to mechanical stresses. For instance, the adoption of certain positions while in bed, such as lying on the side, produces compression of the shoulder which can be painful in capsulitis or rotator cuff problems, whereas lying in a different position is painless.

Can you find a position that stops your pain? If so, please describe.
Although the pain may have been reported to be constant, a question clarifying the situation is helpful as often the patient will reply that if, for instance, they rest the arm behind the neck, pain in the shoulder ceases.

Do you get better or worse as the day progresses? Is it a problem at night?
Patients who experience improvement as the day progresses are usually already improving generally and the report may be regarded as a sign of good prognosis. Musculoskeletal conditions frequently respond adversely to the enforced rest of nighttime. Such patients report that upon waking they are stiff and painful for a while, but that as they become active during the day the ache improves. They certainly require education in appropriate activities and increased exercise levels.

If patients report that their condition worsens throughout the day then their normal activities are aggravating the problem, and these may need to be temporarily restricted or adapted.

It is important to enquire if patients are woken at night by the problem. Various conditions are particularly troublesome at night – shoulder conditions and carpal tunnel syndrome, for instance, often wake people. This is another functional aspect against which to judge management efficacy at a later stage.

Have you had any previous treatment for this episode?
The details of and response to any previous treatment, if any has been received, should be briefly enquired about.

Have you ever had this problem before?
If the patient reports having had similar symptoms in the past this may indicate that poor repair of the initial injury has predisposed the affected structures to repeated failure. It is also possible that degenerative changes have left the tissues weakened and prone to failure. Where the problem is one of recurrence, particular attention must be given to educating the patient so that they avoid aggravating or causative factors. Their management must also include appropriate graded exercises to try to remedy the underlying tissue abnormality and weakness.

What treatment was given for that problem?
Determine what previous treatment may have been provided for similar problems in the past. If the treatment was passive, did the condition respond to that treatment or was the recovery prolonged over the period of the natural history? If recovery followed the use of medication or NSAIDs, it may be appropriate for that treatment to be repeated in conjunction with active management. However, such management may not have restored the full functional integrity of the tissue, which will need to be evaluated. If a regular exercise programme has not been previously implemented, then this may be needed for the future management of the problem.

Do you get back or neck pain?
If the history provides little or no evidence to indicate a peripheral problem, mechanical assessment of the neck or back is needed to exclude the spine as the site of pain generation. Previous spinal problems, the site of the pain, the presence of paraesthesia and the aggravating and relieving factors may raise the index of suspicion concerning the source of symptoms.

Differentiation between neck and shoulder problems can be difficult. It should be remembered that these problems frequently occur together (van der Windt *et al.* 1995), and that cervical conditions

can present clinically as apparent 'shoulder' problems (Grieve 1993; Schneider 1989; Wells 1982). The presence of a concomitant neck condition at presentation has been related to a high risk of persistent shoulder problems (van der Windt *et al.* 1996). The situation may arise that both problems need addressing, or that one site is the source of their major complaint. One method of determining the relevance of the spinal component of a problem is to observe the mechanical presentation of the shoulder both before and after performance of repeated cervical movements. If the shoulder movements improve or worsen, clearly treatment needs to start with the spine. If a spinal problem is present an appropriate spinal mechanical evaluation should be carried out (McKenzie 1981, 1990)

Are you taking any medication for this problem?

Find out what medication and what amounts patients are currently taking for this problem and for any other problems. A later decrease in their quantity of medication will be another sign of improvement. Ask about the effect of the medication on their problem. Patients are generally well aware if medication is helping or having no effect, and if helping whether it is a temporary or lasting improvement. Appropriate pain medication such as paracetamol (or in the United States acetaminophen) may be safer and more cost-effective than NSAIDs (Hochberg *et al.* 1995a). Medication is also less time-consuming and in many cases more cost-effective than the provision of physiotherapy. Some doctors prescribe a course of NSAID at the same time as referring to physiotherapy, but the source of any improvement in symptoms in such a situation is confusing.

Also, the dispensation of medication does not necessarily mean that the recovery of full function will follow. Some patients may have taken various courses of tablets which have brought temporary relief, but the problem always seems to recur; at this time they may be keen to try a longer-lasting solution for their problem involving education and active management.

Is your general health good or poor?

Reports of ill health or feeling unwell could indicate the presence of other factors which may be of higher priority than a musculoskeletal problem. Referral for further investigation is desirable under such circumstances.

Have you been x-rayed for this problem?
Radiological or imaging studies are not required in the early stages of treatment for musculoskeletal problems with the exception that any significant trauma may cause fractures in which case radiological screening will be required. Otherwise, in the event that responses to management are poor, only then might such investigations be considered.

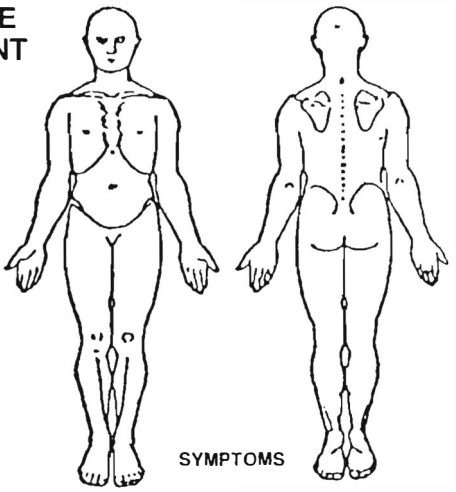
Conclusion

It can be helpful at this point briefly to sum up your understanding of the problem to the patient – have you got a correct interpretation of their presentation? What was the mode of onset? How long they have had the problem? Is it constant or intermittent? What makes it better and worse? It will help to focus on the key elements of the condition. After taking the history certain elements of the clinical presentation should be clear. It will be known what site or sites will need to be looked at in the physical examination. In the case of some peripheral conditions, differentiation from spinal problems may need further elucidation during the mechanical assessment. The time-scale and onset will be known, which should give an idea about the likely state of the affected tissues – whether they are damaged and inflamed, healing, abnormal or deconditioned. A recent traumatic onset should alert the clinician to the possible need to proceed cautiously with the mechanical evaluation. This can be summarised as an acute, sub-acute or chronic condition.

The history will also reveal if the pain generation is constant or intermittent. This gives a vital clue to the nature of the problem, whether it is chemically or mechanically mediated, and often also to the prognosis. There will be an impression of the way the patient has responded to his or her problem – whether, for instance, they are anxious or fearful of causing further damage and therefore restrict their activities in a detrimental way.

Figure 5.1 The McKenzie Institute Peripheral Assessment sheet: history-gathering

THE McKENZIE INSTITUTE PERIPHERAL ASSESSMENT



Date ...../...../.....
Name ..... M / F
Address .....
Date of birth ...../...../..... Age .....
Work / Leisure .....
Postures / Stresses .....
.....
Functional disability from present episode .....
.....
.....

HISTORY

Present symptoms .....
Present since ...../...../..... Improving / Unchanging / Worsening
Commenced as a result of ..... or no apparent reason
Symptoms at onset .....
Constant symptoms ..... Intermittent symptoms .....
What produces or worsens .....
.....
.....
What stops or reduces .....
.....
.....
Continued use makes the pain Better Worse No Effect
Pain at rest Yes / No
Disturbed night Yes / No .....
Other questions .....
.....
.....
Treatment this episode .....
Previous episodes .....
Previous treatment .....
Spinal history .....
..... Paraesthesia Yes / No
Medications: tried ..... Effect .....
Present medication .....
General health .....
Imaging .....
Summary: Acute / Sub-acute / Chronic Trauma / Insidious onset
Sites for physical examination .....
.....
.....

6: Physical Examination

Introduction

The mechanical assessment should relate closely to the findings from the initial interview. From the history provided by the patient, the clinician will already have formed conclusions regarding the likely origin of the symptoms. The clinical examination is designed to confirm the initial findings and fully expose the mechanical nature and extent of the problem. The two parts of the first day's assessment should thus produce a good general picture of the patient's symptomatic and mechanical presentations. From these findings will come the optimal management of the condition. It also provides baseline measures of pain, movement and function against which to establish the value of any subsequent intervention. The information also gives prognostic indicators, derived from such items as the duration of the problem, the previous history, the age of the patient or the constancy of the pain. The examination format and sequence described here can be applied to the articular and motor elements in both the upper and lower limbs.

The physical examination involves various observations and movements about which the clinician must make judgements. It should be remembered that such perceptual tests, in which a human being is the measuring device, are bedevilled by variability of results. Intraobserver and interobserver variability is seen as the inevitable consequence of such perceptual tests (Gray 1997). Although this phenomenon cannot be totally prevented, its impact can be limited by conducting the examination in the same way each time it is done. Clinicians need to perform tests consistently on each occasion, and patients must always start from the same position. In this way we can be more certain that different test results reflect changes in the mechanical presentation rather than being the fault of inconsistent examination technique.

Frequently if the symptoms are in the shoulder or hip areas it is essential first to have ruled out spinal sources of pain. The effect of single and repeated end-range spinal movements on symptoms and on the mechanical presentation should be noted. If cervical movements affect pain at the shoulder or improve a painful arc, clearly

the spine needs to be addressed first. However, concomitant neck and shoulder problems are common and at times both problems may need management strategies.

With the information gathered from the patient's history, the physical examination should allow differentiation into one of the three mechanical syndromes, a chemically mediated pain or a chronic pain syndrome. Using passive resisted and repeated movements, we can further differentiate between symptoms that arise from peri-articular or contractile structures. If the cause is found to be articular, then mechanical therapy will most often be targeted at end-range movement restriction in the case of dysfunction, or end-range movement obstruction in the case of derangement. If the lesion affects contractile structures, treatment of the dysfunction will require repetitive loading in mid-range.

The mechanical evaluation will also indicate the preferred direction in which to apply loading, the precise point in the range where loading must be targeted, the amount of loading to be applied to the affected structure, and the frequency of its application. It will also be determined from the assessment whether or not the patient will require assistance to achieve the desired effect or whether it is within the patient's capacity to manage his or her own recovery.

Aims of physical examination

Using the form and the appropriate testing procedures at the end of the physical examination, ideally the following will have been obtained:

- classification into inflammatory, postural, dysfunction, derangement or chronic syndrome
- inconclusive and needing further mechanical testing over a few days
- baseline measurements of symptomatic and mechanical presentations, functional disability, and present analgesic and NSAID consumption against which to judge effects of management strategy
- explanation of problem to patient and reason for required exercise programme

- time scale for improvement explained to the patient
- appropriate loading strategy, or strategies, needed to manage the condition demonstrated to and practised by the patient
- the repetitions and regularity of exercise programme explained to the patient
- expected pain response explained to the patient.

Observation

The affected part should be uncovered and examined for signs of redness, heat, swelling, bruising or abrasion. Localised redness, heat or swelling will indicate the presence of an inflammatory response to trauma or infection. Where generalised joint swelling is present, especially if bilateral and in the hands, feet, wrists or ankles, rheumatoid arthritis may be suspected (Berkow *et al.* 1987). Where indicated, such patients should be referred for appropriate management.

Mechanical evaluation

The affected structures require a mechanical evaluation to determine the effects that various movements have on the patient's symptoms. In order to stress the joints and relevant surrounding soft tissues in a controlled manner, test movements should be applied in the sequence described below. The information gained from the history and this mechanical evaluation should allow differentiation between problems that are mechanically or chemically mediated. Subsequent to this, differentiation is made between one of the three mechanical syndromes.

To explore the relationship between exercise and the patient's symptoms, test movements must be performed in such a way that they produce a change to those symptoms. This change may be expressed in various ways. If prior to movement pain is present, the test movement may increase or reduce its intensity; it may alter the location of the pain by abolishing one pain and introducing another. If prior to movement no pain is present, the test movements may produce the pain. Changes that occur during test movements may remain better or worse afterwards or may return to the former level of pain. Prior to the application of each of the various mechanical tests required for the assessment, it must be established and recorded whether or not pain is already present. The consequences of various mechanical loading forces are determined by comparison of 'before

and after' levels of pain. See Chapter Seven for a detailed description of the evaluation of symptomatic responses to loading strategies.

If there is no change in the patient's symptoms during or immediately following the test movements, it may be that the structures have not been stressed adequately. If so, the process should be repeated more vigorously. It could also be that the pain is not of mechanical origin, because mechanical pain must be and always is affected by movements or positions. Alternatively, the structures under assessment may not be causing the symptoms and other areas should be investigated, most importantly the spine.

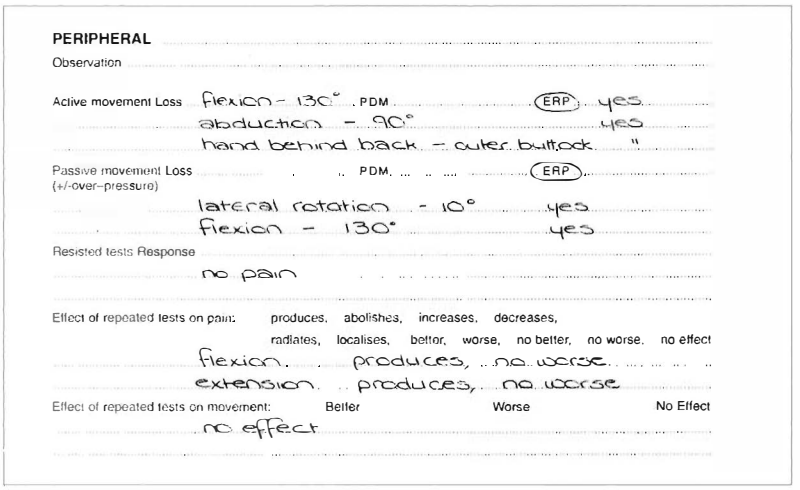
Role of palpation

The most effective way to assess the state of a joint or muscle is through testing function. Exploring the symptomatic responses to different loading strategies does this. Palpation offers nothing extra in the assessment of function. In repeated studies of manual therapists' palpatory skills in the spine, poor intertester reliability has consistently been shown (Mootz *et al.* 1989; McKenzie and Taylor 1997; Binkley *et al.* 1995). Palpation for tenderness can be very misleading due to the phenomena of primary and secondary hyperalgesia, and is generally unnecessary. It may be used to determine warmth, swelling, or feel for crepitus during joint movement. However, this should always come after the functional assessment so that palpation merely endorses the examination findings rather than serving as a means of establishing the site of lesion.

Use of the assessment form

The physical examination part of the assessment sheet allows selection of movements in different planes – flexion, extension, adduction, abduction, lateral rotation, medial rotation, etc. The amount of loss of movement should be recorded as minimal, moderate or major, or else range of movement can be recorded in degrees. The effect of repeated movements should be recorded in the normal way (see Chapter Seven for a description of the standardised terminology to be used to describe symptomatic responses). If there is pain during the movement or a painful arc, the PDM column should be ticked; if it is end-range pain only, ERP should be ticked. The example below demonstrates how a part of the form might be used in describing a shoulder joint dysfunction or 'frozen shoulder'. The part of the form relating to the physical examination is at the end of this chapter.

Table 6.1 Description of a shoulder joint dysfunction



PERIPHERAL

Observation

Active movement Loss flexion - 130° . PDM ERP yes
abduction - 90° yes
hand behind back - outer buttock "

Passive movement Loss (+/-over-pressure) PDM ERP
lateral rotation - 10° yes
flexion - 130° yes

Resisted tests Response
no pain

Effect of repeated tests on pain: produces, abolishes, increases, decreases, radiates, localises, better, worse, no better, no worse, no effect
flexion produces, no worse
extension produces, no worse

Effect of repeated tests on movement: Better Worse No Effect
no effect

Test movements used will be as follows. Not all test movements need be used every time: active movements; passive movements (+/-over-pressure); resisted movements; repeated movements; sustained postures.

Active movement

From the history, the patient will have described the *general* direction and nature of the movement or movements producing the most pain. The examination will identify the *precise* direction of movement that provokes or aggravates the symptoms. From examination of the movement involved, it will be possible to identify the anatomical location of pain as it occurs. This may assist in the search for the origin of the problem in an adjacent articulation, muscle or tendon.

The range of active movement and the presence of pain must be noted in different planes of movement. Movement may be full or reduced; compare with the other limb if in doubt. Movement may be pain-free or painful during the movement (PDM) or at the end-range of movement (ERP). Record findings for each movement on the assessment sheet.

End-range pain is commonly seen in peripheral musculoskeletal conditions. Contracted tissues that produce pain when stretched may limit movements; in this case the range achieved with passive movements of the limb will be very similar to the active range. Limited active and passive movements, which are painful at end-range, are characteristic of capsular contraction and scar tissue that has not

been adequately remodelled. Degenerative changes can lead to restrictions in range of movement, for instance loss of extension and flexion at the knee (Altman *et al.* 1986; Hayes *et al.* 1994; Bijl *et al.* 1998), or flexion, medial rotation and abduction at the hip (Dieppe 1995). Peri-articular problems such as 'frozen shoulder' produce similar movement restriction; in this condition especially flexion, abduction and lateral and medial rotation (Jacobs *et al.* 1991). The more severe the degeneration, the more likely it will be that significant active movement restriction will be found. These restrictions are what Cyriax (1982) termed capsular patterns of movement loss, which he considered to be outward signs of arthritis in a joint from any cause. His descriptions of proportional movement losses in set patterns have not been validated (Hayes *et al.* 1994; Bijl *et al.* 1998). Movement losses occur, but in individuals with hip osteoarthritis this has been shown to be in all six directions of movement, not the set pattern described by Cyriax (Bijl *et al.* 1998). End-range pain is also a characteristic of traumatised tissue which has healed with contractures or adhesions that limit normal flexibility.

Rather than pain being produced at end-range, it may be produced or aggravated during the movement. Pain during a movement may be produced because of a problem with a contractile structure or because of an articular problem. An arc of pain in mid-range is due either to the compression of irritated soft tissues by a bony constriction or to an articular internal derangement. Painful arcs occur quite frequently at the shoulder, where it is thought to indicate constriction of abnormal tendons or bursa. They are also seen at the knee and lumbar joints, indicating internal derangements (Cyriax 1982; van der Windt *et al.* 1995). Degenerative conditions can produce pain during movement. Passive and resisted movement testing will clarify the situation.

In a few very severe cases active movements may be limited, while passive movements are full or cannot be fully tested because of extreme pain. This sort of presentation is seen sometimes at the shoulder in elderly patients.

Active movement is almost always a painful experience whether the problem is articular or musculotendonous in origin. Exceptions may be found, however, where injury has occurred to ligaments restraining the non-voluntary movements. These structures are best tested using passive movement tests with overpressure (see appropriate section)

Active movement summary

Pain produced by active movement is mechanical and can be articular, ligamentous or musculotendonous in origin. Active movements can provide us with the following:

- mechanical presentation (range of movement or willingness to move) which can be used to evaluate management
- symptomatic presentation that can be used to evaluate management
- whether pain and/or stiffness limits movement (passive movements may be needed also to confirm this)
- arc of pain, which has diagnostic implications, for instance implicating tendons or bursa at the shoulder
- end-range pain, which has diagnostic implications, generally implicating articular or peri-articular structures.

Passive movement

Movement performed passively does not rely on contraction or loading of musculotendonous structures for its completion. Thus these tests stress non-contractile structures in and around the joint and tend to implicate articular or peri-articular structures, and tissue contractures in any connective tissues. Pain may appear during the passive movement or at the end of range; it may be due to articular derangement or dysfunction

Passive movement, which produces end-range pain in a restricted range of movement, is characteristic of dysfunction. As discussed in the active movement section, this may be in the soft tissues around a joint, as in the restrictions seen in osteoarthritis of the hip or capsulitis at the shoulder, or it may be found when stretching healed, scarred tissue following trauma. For instance, in someone who has injured his or her calf muscle, but failed to remodel it adequately during healing, dorsiflexion would be limited, stiff and painful.

A painful arc occurring during passive movement, with or without a painful limitation of movement, occurs in derangement. In this case, when end-range overpressure is also painful, the attainment of end of range is obstructed by dislocated tissue or some anatomical disruption in an articular structure.

To distinguish between end-range pain due to derangement and that due to dysfunction, two options are available. With repeated

movements there will be no change in the symptomatic response in dysfunction even if the movement is repeated a hundred times a day for several weeks – pain of dysfunction only changes slowly as the tissue remodels. Given the slow turnover of collagen this takes time, usually to be measured in weeks and months rather than days. With derangement repeated movements will change the pain. It may worsen on repetition, in which case the opposite direction should be explored, or it may improve – the key differentiation is that it will not be consistent every time as it will be with dysfunction. Overpressure may also be used to distinguish between the two syndromes (see next section).

In patients who have recently injured a muscle resulting in the formation of a haematoma there would also be pain on passive movement testing. Such patients also present with pain appearing towards the end-range of passive movement. Distension of muscle consequent to the swelling prevents normal elongation of the affected muscle. Passive end-range movement in such cases is always painful, but the site of pain will occur near the location of the haematoma and away from the joint.

Passive movement summary

Passive movements stress the non-contractile tissues, for instance joint capsules and intra-articular contents. They also test the flexibility of any connective or contractile tissue after trauma. Tissue that has healed without adequate remodelling may be symptomatic and tight when that shortened, scarred tissue is stretched. Passive movement tests can be useful:

- to confirm a restricted range of movement
- if accompanied by reproduction of the patient's symptomatic complaint, it may result from internal articular derangement or from tissue dysfunction (for example, capsular contracture or unremodelled scar tissue)
- repeated movements and overpressures aid differentiation
- if passive movement is full, but accompanied by reproduction of the patient's complaint during the movement, articular derangement is likely.

Passive movement with overpressure

There are various instances in which extra force applied at the end-range of normal active or passive movements may provide additional

information. As noted above, overpressure can be used to help distinguish between end-range pain due to dysfunction or derangement. In dysfunction once the pain is produced more pressure will produce more pain, but this will rapidly abate once the stress is released. In derangement the application of more pressure will either increase or decrease the pain and bring about a lasting change in pain, either worse or better.

There are also instances in which other movement tests fail to reproduce the patient's pain. In some less acute articular conditions, pain may not be provoked by active movement or during the passive testing sequence. Pain may only be elicited when overpressure is applied at the end of the range. Due to soft tissue apposition, active movements at certain joints fail to reach end-range; this occurs with flexion at the hip or knee. In such instances passive movements with overpressure are more informative. Overpressure, can, if excessive, cause pain in normal articulations. It is important therefore that the pain reported is the 'familiar' pain and not a 'strain' pain caused by excessive force. Overpressure should be applied at first with a gentle firmness that may increase gradually with two or three repetitions. If the condition is articular, familiar pain in one or more directions should be reported.

Where limitation of movement is moderate to severe, the passive end-range 'feel' can be described either as a 'bony' or 'hard' obstruction, or a 'rubbery' or 'soft' obstruction. It is unlikely that attempts at recovery of movement will be successful when the end feel is 'hard'. A 'soft' end feel, on the other hand, is indicative of a malleable tissue that may respond to remodelling.

When a patient describes that normal voluntary movements are painless, it is unlikely that the problem will be identified using active, passive or resisted movements. Where voluntary movement through full range is painless, it is likely that the patient experiences pain only when a position is adopted or external force is applied against or across the planes of voluntary motion peculiar to that joint. Testing articular ligamentous structures that prevent non-voluntary movements at the joint may identify the source of pain. Instances of this are the varus and valgus forces used by clinicians to test medial and lateral collateral ligaments at the knee and elbow. See, for instance, Evans (1986) or Magee (1987) for thorough descriptions of these ligament stress tests. In both these cases it becomes necessary to

identify the direction of pain provocation in order to structure the appropriate management programme. This usually requires manual overpressure in the required plane and will vary from one joint to another.

Passive movement with overpressure summary

In assessing the pain response from any articulation, it may be necessary to apply overpressure at the end-range of joint motion if other movements evoke no painful response. Overpressure is also useful in the following instances:

- the end feel may be a prognostic indicator, with a 'hard end feel' being unlikely to respond to a remodelling programme
- to help differentiate dysfunction from derangement
- to provide ligamentous stress tests at right angles to the normal plane of joint movement.

Resisted tests

Resisted tests load the tissues involved in contraction and thus test for any painful impairment of contractile structures – whether muscle, tendon or periosteal insertion. By ensuring that no motion occurs in the adjacent articulation at the time of applying resistance to the muscle group, the experience of any pain occurring during this test is likely to arise from the contractile structures alone. A positive resisted test is the production of the patient's pain complaint. All other resisted tests should be pain-free, or virtually so, and passive movements likewise, excepting one (see below). Weakness will often accompany the pain.

It is essential that the limb to be tested be completely stabilised and all movement prevented. Any disturbance of the limb that occurs invalidates the resisted test result as pain may arise from other than contractile structures. Examples of these tests are illustrated, for instance, in Laslett (1996) and Cyriax and Cyriax (1997). Resisted movements should have no effect in the presence of articular derangement or articular dysfunction, or where capsular or ligamentous structures are the source of pain as long as all movement is prevented.

Resisted movements will provoke pain in the presence of dysfunction or trauma affecting the contractile structures of the extremities. The pain will be elicited immediately when the loading is sufficient to deform the structurally impaired or inflamed tissue. In some instances

small amounts of resistance will provoke severe and rapid pain, while on other occasions strongly resisted movements will only just generate enough loading to be felt. In more severe cases pain may be accompanied by weakness and a giving way of resistance as the loading stresses the damaged tissue.

Because of the large excursion of motion available to most joints of the extremities, they require lengthy contractile elements in order to function efficiently. This anatomical construct provides abundant opportunity for contraction or adhesion formation during the repair process following injury. Dysfunction developing from imperfect repair of injured muscle or tendon will provoke pain whenever the structure is subjected to loading, irrespective of where in the range of movement that loading may occur. Thus the patient may report that pain will appear whether loading is applied at the innermost, at mid-range, or at the outermost point in the range. Pain will also appear during either concentric or eccentric loading.

Dysfunction of contractile structures will produce pain when the tissue is loaded in mid-range with the appropriate resisted test. Muscles and tendons also have to be able to stretch as well as contract. Thus it will be found that stretching the dysfunctional tissue, either actively or passively, in the opposite direction from its contractile role is painful also. For instance, in dysfunction of the hip adductors in a 'groin strain', both resisted adduction and end-range abduction will be painful.

Resisted tests summary

Tissue loading of contractile structures, achieved through resisting and preventing a certain movement, can isolate impairment of these structures by reproducing the patient's symptoms. All other movement must be prevented.

- painful resisted tests indicate the involvement of muscle, tendon or periosteal insertion
- painless resisted tests exclude the possibility of the involvement of contractile structures
- stretching the muscle unit is also likely to be painful.

Repeated movement or loading

Repeated movements when applied to an inflammatory disorder, or when applied too early or too vigorously to an injury that is healing,

will produce a lasting increase in the pain, and movement may become more difficult. Repeated movements when applied to a chronic disorder may produce a temporary increase in symptoms. Repeated movements when applied to mechanical disorders of the musculoskeletal system evoke different responses when applied to the three syndromes, and thus can be diagnostic.

The two main mechanical syndromes found in clinical practice are derangement and dysfunction. Differentiation between them is important because the management for derangement requires the application of movement that *reduces* the pain, which occurs as a consequence of the reduction of the derangement. On the other hand, dysfunction requires the application of movement that *produces* the symptoms, a requirement for stimulating the remodelling of contracted or adherent tissue. As mentioned previously, repeated movements are key to this differentiation.

Derangements can demonstrate lasting and rapid changes in symptomatic and mechanical presentations in short periods of time with repeated movements. Pain arising from internally deranged and displaced articular tissue increases or decreases according to whether the direction of repeated movement increases or decreases the displacement. Thus repeated movements will cause the pain to increase in one direction and decrease in the other, often with a concomitant change in range. After the test movements the resulting pain remains worse or remains better accordingly.

In dysfunction the tissue abnormality is not amenable to rapid change. It requires remodelling over weeks or months, and it responds consistently to repeated movements. The essential nature of a dysfunction is its consistency. Hundreds of movements a day repeated over a week or more will produce the same pain that the patient complains of time after time. When the stress is removed the pain will abate within minutes. There will be no lasting change. Repetitions will neither make the pain increase nor decrease in intensity; it will be the same each time. The movement that produces the patient's pain may be active, passive, passive with overpressure or resisted depending on the nature of the dysfunction.

Apart from differentiating between derangement and dysfunction syndromes, repeated movements are essential in determining whether

the time is appropriate to commence stretching procedures following trauma. When repeated movements applied in a given direction produce less and less pain with each repetition, exercise in that direction is indicated. If repetitive loading from the same movement produces pain at mid- or end-range that ceases on unloading, exercise in that direction is also indicated. If pain progressively increases during repeated movement, exercising in that particular direction is not indicated. If pain only worsens in part of the range, repeated movements are indicated for that part of the range that is painless or to that particular point in the range that does not worsen the pain.

Repeated movements that cause the pain to be worse afterwards may indicate the re-triggering of the inflammatory response in sub-acute conditions. However, in the event that pain is aggravated following repeated movement in a particular direction, repeated movement in the opposite direction should be explored. If the change in direction brings about a reduction in pain, the derangement syndrome must be causative and the reductive movement has been identified. In chronic pain syndromes in which tissues are deconditioned and hypersensitive, there may also be an initial worsening of pain with repeated movements. However, under these circumstances, this should not be interpreted as a contraindication to active mechanical therapy.

Repeated movements should also always be used when syndrome classification is uncertain and diagnostic acumen is low. In these instances the response to repeated movements allows determination of the appropriateness of an exercise programme. If following certain movements the patient is no worse or better, regular performance of those movements is indicated to improve functional impairment. If the patient is worse following all movements, then restoration of function must start more gently.

It is not necessary or even desirable to conduct repeated movement testing in all directions. This may aggravate the problem and obscure the value of symptomatic responses. For each condition only certain directions should be involved in repeated movement testing. The planes of movement to be tested in this way are most likely to be those that involve the greatest impairment; this may be loss of movement or pain on resisted tests. Thus repeated movement testing

will vary between conditions and between individuals and will be guided by responses to the earlier part of the mechanical evaluation.

Repeated movement in the postural syndrome

When applied to patients with the postural syndrome, repeated movement has no effect and all findings will be negative. In the postural syndrome, pain only arises in the affected area after prolonged static loading.

Repeated movement in the dysfunction syndrome

In the dysfunction syndrome, no pain will be felt at rest. Repeated movements or tissue loading will cause pain with each repetition; the appropriate tissue loading may be active and passive or resisted. The pain will cease on unloading. End-range pain with limitation of movement will indicate an articular or capsular origin for pain. Pain felt during repeated static loading or during part or the whole arc of full movement indicates a contractile origin for the symptom. Pain may increase slightly with ten or twelve repetitions, but usually subsides rapidly and remains neither better nor worse as a result. No rapid changes in pain or range of motion will occur.

Repeated movement in the derangement syndrome

In the case of derangement pain may rapidly reduce or cease and remain better following the application of repeated movements applied in the direction that reduces the degree of internal derangement. At the same time, repeated movements will be seen to reduce the degree of the obstruction to movement. Symptoms can also be made to increase and remain worse following repeated movement applied in the direction that increases internal derangement. Simultaneous increase in the obstruction to movement will also occur. This clear-cut indication of the direction for the application of therapeutic movement is known as 'directional preference', a term coined by Donelson *et al.* (1991), an orthopaedic surgeon involved in research and practice of mechanical diagnosis and therapy.

The directional preference identified in patients with this syndrome provides a reliable guide when determining the most appropriate therapeutic exercise programme. Provided the reductive movements are practised and the causative movements avoided for a few days, patients with the derangement syndrome are rapidly restored to full pain-free function.

Summary

Table 6.2 Directional preference

The response to repeated movements	*Implication*
no pain during repeated movements	Postural Syndrome
pain produced only at limited end-range, no worse afterwards	Dysfunction Syndrome (peri-articular)
pain produced only by resisted tests, no worse afterwards	Dysfunction Syndrome (contractile tissues)
increasing symptoms in one direction decreasing symptoms in the other	Derangement Syndrome
all directions cause lasting increase in pain in sub-acute condition	Chemical pain
persistent pain in which initial active therapy causes some temporary aggravation of symptoms	Chronic pain state

Neurological examination

During the course of history-taking and mechanical evaluation, it is sometimes the case that a sensory or motor deficit is noted. That is, the presence of tingling or numbness may be voiced by the patient, or weakness not attributable to pain, or loss of reflexes may be found on examination. The most likely origin of such neurological impairment is the spine. Radicular pain in the arm or leg will also be present, although spinal pain may not. Tension tests such as straight leg raise, neck flexion, or upper limb tension tests are also likely to be positive. Sometimes these tests are positive without the sensory or motor deficits. Usually the source of these problems is the spine and examination and management should be directed at this area by conducting a normal mechanical evaluation (McKenzie 1981, 1990).

Occasionally a thorough examination and testing of the spine is unable to offer a clear source of the symptoms. In these instances further examination of neural tension tests may still unearth mechanically sensitised neural tissue, which upon testing reproduces the patient's pain. Before using these tests in treatment it is essential to make sure that both spinal and local articular structures are not involved in the

problem – both will make these tests false positive. The tests used in the upper arm are described in Butler (1991), as is their use. Great care must be taken in their use, as it is very easy to aggravate sensitised neural tissue. It is possible, again with care, to use movements based on these tests for self-treatment.

Conclusions

The collection of information from the history, examination and mechanical evaluation will indicate whether the patient is suffering from the postural, dysfunction or derangement syndrome, or a non-mechanical cause. The differentiation is necessary, as the treatment of each will be completely different.

In patients with the postural syndrome, following identification of the causative factors, adjustment to the position or posture responsible for the symptoms is all that is required

In those patients with the dysfunction syndrome, in their treatment those movements are applied that in the examination provoked their pain. When applied for treatment or remodelling purposes, it is imperative that some pain be experienced during exercise. This is particularly important in the initial stages of treatment when contracted structures must be stretched enough to assist them to remodel to their original length and regain their former elasticity. Patients in treatment for dysfunction must experience some pain or discomfort. An articular dysfunction will require end-range stretches. A dysfunction of contractile structures will require resisted mid-range loading and end-range stretches in the opposite direction from its contractile role.

In those patients with the derangement syndrome, in their treatment are applied those movements which in the examination reduced, stopped or localised the pain. Predisposing postures or movements will be identified so that the patient may avoid recurrence and in that event apply self-treatment.

If lasting aggravation of a recent condition results from repeated movements further relative rest may be needed, or on the next occasion a more gentle movement or different starting position should be used.

In the case of chronic conditions an initial temporary aggravation of symptoms after commencing exercise is quite common. This should be explained to patients and should not be so great as to deter them from following the necessary progressive reconditioning programme.

Inconclusive results

Following the complete mechanical assessment of the upper or lower extremities, if it is not possible to reproduce or affect the patient's symptoms, it may well be that the source of pain lies within the spinal column and is either referred or radiating from the spinal segments. To exclude the spine as a cause of pain experienced in the extremities, a mechanical evaluation as described in *The Cervical and Thoracic Spine: Mechanical Diagnosis and Therapy* (McKenzie 1990) or *The Lumbar Spine: Mechanical Diagnosis and Therapy* (McKenzie 1981) should be applied. Following the spinal assessment process, if no movement or position has been found to affect change in the symptomatic or mechanical presentation in the periphery, the condition is unlikely to be caused by mechanical musculoskeletal disorders. It may be necessary to refer the patient for further investigation.

After the history and examination have been completed it may appear at first that the prognosis is not encouraging. It is a mistake to provide the patient with this clinical impression before we have had confirmation of our prediction from the result of a trial period of mechanical assessment over a few days. It is not always possible for the prognosis to be so certain. Therefore it is best to avoid giving an incorrect impression of the nature and extent of the problem before this is fully apparent. However, once it is clear that the nature of the problem is such that mechanical therapy will have little or no impact on the condition, it is important that a realistic picture is conveyed to the patient. This allows the patient to come to terms with a persistent problem and also makes it more likely that they will adopt the necessary strategies for managing it in the best possible way. Prolonged physiotherapy care for intractable problems is certainly a waste of resources; it is also bad practice as it detracts from the need to educate patients to manage persistent pain states in the optimal way.

Figure 6.1 The McKenzie Institute Peripheral Assessment sheet: physical examination

PERIPHERAL

Observation .....

Active movement Loss ..... PDM ..... ERP .....

Passive movement Loss ..... PDM ..... ERP .....
(+/-over–pressure)

Resisted tests Response .....

Effect of repeated tests on pain: produces, abolishes, increases, decreases,
radiates, localises, better, worse, no better, no worse, no effect

Effect of repeated tests on movement: Better Worse No Effect

Static Positioning Effects .....

Loaded / Unloaded .....

SPINE

Movement Loss .....

Effect of repeated movements: .....

Effect of static positioning: .....

Spine testing: Not relevant / Relevant / Secondary problem

CONCLUSION Peripheral ..... Spine .....

Dysfunction : Articular ..... Contractile .....

Derangement ..... Postural .....

Other ..... Uncertain .....

PLAN

Exercise Frequency

7: Assessment of Symptomatic and Mechanical Presentations and Responses

Introduction

At the first assessment and on all subsequent occasions clinicians must be evaluating the patient's problem and the effect of the management strategies being used. This evaluation needs to address both the pain, which is frequently the patient's main complaint, and the impaired function. The two elements should be evaluated on each occasion and generally improve or worsen in tandem – in other words, if the pain eases the function should also improve. Depending on the effect of the management strategies these should be continued, abandoned or supplemented with overpressures or possibly clinician techniques.

Symptomatic presentation

Pain is usually the main complaint of patients with musculoskeletal problems, although paraesthesia, numbness or weakness may also be relevant. Pain as an outcome measure is criticised as 'soft' data, which lacks objectivity. However, while pain is by its very nature a subjective experience, it can be recorded and assessed in a reliable way, especially when using serial measurements of pain taken from a single individual (Sim and Watefield 1997). Numerous articles and books present ways of assessing pain and some of the associated problems (for instance, Jadad and McQuay 1993; Sim and Watefield 1997; Adams 1997). The most common tools are rating scales of pain intensity or relief, visual analogue scales, analgesic consumption, pain frequency and pain questionnaires that investigate multi-dimensional aspects of pain (Jadad and McQuay 1993). For any therapeutic intervention whose goal is the reduction of pain, the assessment of pain must rank as one of the most important and relevant measures of improvement (Holmes and Rudland 1991). Pain has various dimensions by which changes can be assessed:

- Temporal component. A patient may complain of constant pain. If this subsequently becomes intermittent an improvement has been made. If an intermittent pain which is present for most of the day is reduced to being present only twenty per cent of the day, this also is an improvement.

- Site. Pain of spinal origin may centralise or peripheralise (Donelson *et al.* 1990, 1991). The further the pain spreads into the limb, the worse the presentation; if the area of symptoms can be reduced this is an improvement. Radiation of pain also commonly occurs from some peripheral joints, especially the hip and shoulder. Cyriax (1982) proposed that the spread of symptoms was partly a reflection of the strength of the painful stimulus. As the noxious stimulus increases, the pain becomes more diffuse and spreads distally – referred pain from the hip can spread to the antero-medial shin, and from the shoulder into the hand. A reversal of this process, and thus an improvement, is pain localised to the particular joint – thus *localisation* is the peripheral equivalent of the spinal phenomenon of centralisation.
- Severity. Intensity of the pain can be assessed in various ways. This can be done formally using a Visual Analogue Scale, with the patient marking the pain intensity on a line between 'no pain' and 'pain as bad as it could be' (Huskisson 1974). They can be asked the same question at a later date for comparison to determine whether the pain intensity is severe, moderate or mild. Alternatively at re-assessment patients can be asked how their present symptoms compare to when they first attended. 'If you had 100 units of pain when we commenced treatment – how many do you have now?' Some patients even volunteer that they are '80% better'.
- Consumption of analgesics and NSAIDs. The number of tablets being taken daily at the beginning of the episode should be recorded and compared with consumption later.
- Pain on movement. Even if movement appears to be full-range, pain may be felt during the movement or at end-range. An improvement is made if the patient reports that over time the difficulty of performing the movement and the degree of discomfort it engenders has lessened.

Although very often pain responses are a useful determinant of appropriate mechanical therapy, it should also be born in mind that excessive attention to the pain can heighten the pain response (Arntz *et al.* 1991). By contrast, use of distraction techniques can minimise the pain (Klaber Moffett and Richardson 1995). With some patients

the focus should be on function rather than pain; this is especially relevant in patients with persistent pain problems.

Mechanical presentation and its assessment

The mechanical presentation refers to the outward manifestations of the problem that may limit or alter normal movement and posture. The mechanical presentation has various dimensions by which it can be assessed. Broadly these address the issues of impairment and disability. Impairment refers to abnormality of normal physiological function because of the problem, while disability is the ensuing restriction of normal function. The dimensions are:

- loss of normal range of movement
- deformity (more common in spinal problems)
- deviation of movements
- velocity of movement; pain often makes people move more cautiously, and return to normal speed represents an improvement (McGregor *et al.* 1995; Paquet *et al.* 1994; Magnusson *et al.* 1998)
- loss of normal function; for instance, abstinence from usual sporting activities, inability to perform normal domestic tasks or absence from work

The assessment of these changes is relatively straightforward. Range of movement tests should always be conducted from the same starting position and in consistent ways so that the results can be compared on different occasions. More formal ways of assessing the patient's function should be done using established disability questionnaires relevant to different joints. These questionnaires, completed by the patients, are a very good measure of treatment efficacy and address issues relevant and important to both patient and clinician. Examples of such questionnaires are given below:

Table 7.1 Disability questionnaires

Shoulder	van der Windt *et al.* (1998)
	Croft *et al.* (1994)
Knee	Irrgang *et al.* (1998)
Lower extremities	Binkley *et al.* (1999)

Assessment of the symptomatic presentation

Standardised terms (McKenzie 1981; van Wijmen 1994) are used to evaluate the patient's pain responses during mechanical testing. The pain status is established before, during and after test procedures. If we wish to compare the effect of movements on the symptoms *it is vital we know the pain status prior to testing* – it is too late to establish this once the patient has started to do the movements. We are most interested in the effect of the movements a minute or so after testing.

At baseline the patient is either with or without pain. During the test movements this can be *increased, decreased, abolished* or *produced*. The significant response which will help determine the treatment strategy is not, however, to a single movement. The key symptomatic response follows repeated movements when different terms are used to describe any changes. At this point pain which was increased or produced by the movements can either remain *worse* or be *no worse*; while pain which was decreased or abolished during the movements can either remain *better* or be *no better*. If during and after the movements the symptoms remain completely unchanged they are said to have *no effect*. These terms are listed and defined in the glossary.

Use of symptom responses to determine loading strategies

Using these standardised terms to define the patient's responses to repeated movements allows us to determine the appropriateness of those particular movements. A system called the Traffic Light Guide, devised by W. and J. Rath (Robinson 1994), addresses the therapeutic implications of the pain response after the test movements.

The Traffic Light Guide allows a logical formulation of appropriate mechanical loading strategies based on the patient's symptom responses. If the patient is better after the test movements, or in the case of dysfunction if pain produced by movement ceases when the stretch or contraction is released, the correct movement has been selected and treatment should be continued unaltered. This is a 'green light' to more of the same procedure. *If the patient is showing improvements with the prescribed management, there is no justification for changing or supplementing it in any way*.

Table 7.2 Traffic Light Guide

Pain status prior to test	*Pain response during test*	*Pain response after test*	*Implications (Traffic Light Guide)*
Pain	Increase	Worse	Red
		Not worse	Amber
	Decrease	Better	Green
	Abolish	Not Better	Amber
No Pain	Produce	Worse	Red
		Not Worse	Amber/Green

If the pain remains worse after test movements, then either the direction or speed of movements is wrong and that particular exercise should be stopped. Movements in the opposite direction should be tested as well as lateral movements. If movements still aggravate symptoms, non-mechanical problems might be suspected. Movement may be starting too early during the healing process and the inflammatory process is being reactivated, the wrong starting position is being used, or the procedure may be too vigorous for the stage of the disorder. This is a 'red light' to that particular procedure. If everything appears to aggravate the patient's symptoms, they should be spared further testing and reviewed in a week's time. If a similar response is generated with minimal testing, the disorder is inappropriate for mechanical therapy at this stage.

In the case of the 'amber light', essentially nothing is changed by the test movements. Although they may produce, increase, abolish or decrease symptoms at the time, afterwards the patient reports that they are just the same as they were before. This allows the application of more force to see if the traffic light changes to give a clearer indication of the appropriate direction of therapeutic exercise. Only with an 'amber light' response is it justifiable to use force progressions, altho

patient exercising more regularly, or with patient overpressure, or it may involve clinician force if this is thought to be necessary. In dysfunction this response is a 'green light' for that particular exercise. If pain is produced on contraction of contractile structures or on

end-range stretch of peri-articular structures which ceases on release, that loading strategy should be continued unaltered.

Most conditions will fit into one of the above categories, especially if the problem is of recent onset. If symptoms have been present for some time the response may be the equivocal 'amber light'. It is often helpful at this point to get the patient to test out a specific procedure for one or two days and gauge the response following this. Other ways of facilitating the diagnostic process when it is unclear are (van Wijmen 1994):

- ensure that movements are to end-range
- apply mechanical forces more vigorously
- sustain postures
- increase the number of repetitions
- increase the frequency of repetitions
- use alternate starting positions
- stress the joints in one direction and check the effects on pain and movement range in the opposite direction
- test provocative procedures over two to three days.

Chronic pain

In the situation of chronic pain when peripheral tissue and central nervous system elements may be sensitised and deconditioned to normal movement, the criteria of symptom response needs to be different. Under these circumstances normal mechanical stimuli can produce pain, repeated movements may have a 'wind up' effect on pain production, there may be a spread of painful areas, and there may be ectopic nociceptive signals (Dubner 1991; Johnson 1997). These changes make the interpretation of mechanically produced symptom responses difficult and invalidate diagnostic labels applied to particular responses (Zusman 1992, 1994). These examples suggest that we should interpret the behaviour of chronic pain to repeated movements somewhat less rigidly. Non-mechanical factors may be more significant than simple tissue abnormality.

The above effects are unlikely to be present to the same degree, or even in all patients with chronic pain; some such patients respond

straightforwardly to mechanical therapy. Some patients with chronic pain can have ongoing tissue damage or a degenerating disease (Johnson 1997). Most patients with chronic musculoskeletal pain have only mild or moderate symptoms and do not suffer major functional impairment (Magni *et al.* 1990; von Korff *et al.* 1990); many also will leave the pool of persistent pain sufferers if followed over a few years (Crook *et al.* 1989). Only a small proportion of patients with persistent pain are at the severe, disabled end of the spectrum; many will respond normally to a mechanical evaluation. If the response is equivocal, test out the patient's response over twenty-four hours or use one of the other suggestions above for facilitating the diagnostic process when it is unclear.

However, in the instance of chronic pain patients, it is sometimes permissible to allow a slight worsening of symptoms initially. Sometimes the response to mechanical therapy takes a while to elucidate, thus it is valuable to follow the approach for a few sessions rather than abandoning it as soon as there is a slight worsening of symptoms. Sometimes the sensitisation induced by chronic pain states needs to be desensitised by encouraging gentle regular movement prior to establishing a more mechanical pattern of response. With chronic pain patients it is necessary to concentrate more on trying to improve coping strategies and function rather than focusing on pain. Often improvement in general function and the psychological effect of doing something active about their problem can produce a reduction in pain. Patients with severe levels of dysfunctional behaviour due to persistent pain problems are best treated in a multi-disciplinary pain programme rather than on a one-to-one basis.

As has been discussed in Chapter Two, some chronic tendon problems represent a failed repair process in degenerated tissue in which the normal phases of healing are suspended or absent. In these conditions a vigorous exercise stimulus can often re-trigger the process of healing. Initially with these degenerated structures a temporary aggravation for a day or two is thus desirable. Once this reaction has been achieved the vigorous stimulus should cease, and a graduated exercise programme should be instigated as for a sub-acute problem.

Review process

On the second day and at each subsequent visit, a structured, logical and informative review process needs to be conducted. This is to determine what the patient has been doing regarding previous instructions, the immediate effect of any procedures being done, and if there have been any overall changes. Find out from the patient if as a result of following the instructions there has been any change:

- With doing the exercises over the last few days, is the patient better, worse or the same overall?

If the patient is better there is no need to change management in any way, and they should continue with more of the same. If they are worse or unchanged they must be questioned more closely about what they have been doing:

- Have they been exercising?
- How frequently?
- What exercise have they been doing? Get them to show you. However clear you think you may have been, patients unfortunately frequently 'modify' the exercise.
- Are there any problems with this?
- What is the symptom response when they do the exercise?
- Check symptomatic presentation.
- If there is a change, is this definite or doubtful?
- Check if there is any change in functional problems.
- Check mechanical presentation

It will then be known how regularly they have been doing their exercises and if they are doing them correctly. Their technique may need correcting, but wait until you have heard how it affects their symptoms – they may be doing it 'wrong', but it may be helping! They may need encouragement to be exercising more regularly, or they might have been doing too much. If patients are having problems with the demands of a regular exercise routine, the importance of doing it regularly needs to be emphasised.

Encouraging patients to solve difficulties with the regime or the exercise itself promotes self-management. Some patients are very reluctant to do things that hurt and are still very anxious about pain responses. They need extra reassurance 'that hurt does not equal harm', that reduced activity is only briefly beneficial at the onset of pain, and that the *only* way to try to re-establish normal function is graded exposure to normal activity.

From the review you will also know their symptomatic and mechanical response to performing exercises over a day or two – they will either be better, worse or unchanged. As outlined earlier this gives a 'green light' for more of the same, a 'red light' or an 'amber light' respectively. It is important to ensure that they are actually in these states. Detailed questioning and close analysis of symptomatic and mechanical responses, as outlined earlier, are sometimes necessary to elucidate the true picture. If the patient is better, nothing needs to be changed, only encouragement given. If the patient is worse, exercises and symptom responses need to be checked, but ensure the patient is actually worse, rather than the exercises simply 'hurt'. When starting any unfamiliar exercise programme new pains may be generated. This is not unusual, but it may confuse the patient. If unchanged, are they exercising regularly enough and doing the right exercise? Is force progression necessary? In the case of dysfunction no change would be expected, and they should be encouraged to continue.

Always ensure that the patient understands why they are doing what they are doing and are happy about doing it. Self-management demands that the patient is convinced; it is the clinician's responsibility to convince them.

8: Patient Management

Introduction

Patient management means the organisation, supervision and implementation of the strategies to be applied for the successful education and treatment of the patient. Clinicians will already have their own systems in place for patient management. These systems vary, of course, but the broad principles outlined below remain the same for all.

The patient has been questioned regarding the history of their complaint. An examination of appearance and function of the affected region has been performed. The findings from the history and the examination allow the patient to be classified according to a mechanical, chemical or chronic syndrome. From this classification the therapeutic management can be structured to suit the patient's requirements. The therapeutic management will consist first of an educational component and second, an active mechanical therapy component. The educational component will provide the patient with an understanding of his or her problem and the role of movement in their rehabilitation, while the active therapy component will provide them with instructions in the appropriate exercises.

Education component of management

All patients have certain needs for information about their disorder from health care professionals (Charles *et al.* 1997). In a group of patients attending their general practitioner in London, an 'explanation of the problem' was the most wanted item during the consultation (Williams *et al.* 1995). However, doctors frequently underestimate patients' desire for information about their condition (Ong *et al.* 1995). The failure to provide adequate information is a common cause of dissatisfaction among patients in general, and among patients with musculoskeletal problems in particular (Deyo and Diehl 1986; Cherkin and MacCormack 1989; Fitzpatrick *et al.* 1987; Hall and Dornan 1988; Locker and Dunt 1978). The information that patients want concerns four key areas (Williams *et al.* 1995; Hall and Dornan 1988; Cherkin *et al.* 1991; Fitzpatrick *et al.* 1987):

- information about the problem itself
- what patients can do to help themselves
- information about tests, diagnosis and interventions
- an idea about the prognosis of the problem.

Patients' specific requirements for information will individuals and between presentations. The depth and detail of information given should be appropriate for that patient, but must be sure to cover the essentials that are necessary for the self-management of their problem. Essential components of the information provided should reassure patients' anxieties and fears about the pain, and encourage the earliest possible restoration of function. Failure to provide the appropriate information is likely to result in patient dissatisfaction and will seriously undermine the patients' ability to manage their problem in the optimum way. Patient education should not be seen as a 'nice extra', but as an effective treatment in itself.

For instance, in the field of arthritis, individual studies document the ability of patient education to decrease pain, depression, disability and tender joints (Lorig 1995; Mazzuca *et al.* 1997). Evidence suggests these health care gains can be sustained long-term (Lorig *et al.* 1985, 1993). Current meta-analyses suggest that pain, tender joints, depression, exercise, coping behaviours and health services' use can all be improved by educational programmes for rheumatology patients (Lorish and Boutagh 1997). For patients with back pain, although the evidence concerning group educational programmes is weak (Cohen *et al.* 1994), more recent studies have shown the value of information provision and a self-management approach in reducing sick leave and anxiety and improving self-care and function (Indahl *et al.* 1995, 1998; von Korff *et al.* 1998). Two factors have been identified that seem important in achieving these beneficial effects. They are improved self-efficacy, which is the belief that people have about being able to control aspects of their life; and an internal health locus of control, which is the belief that they can affect their health without the help of 'external' others (Lorig *et al.* 1993; Indahl *et al.* 1998).

If the condition is of recent onset, a description of the healing and remodelling process should be given. The patient must understand the nature of the environment necessary for the creation of good

quality repair and the importance of a graduated exercise programme for the restoration of full function. Some explanation should be given about the characteristic of scar tissue to contract with time and lack of movement. To create an extensible and strong repair, patients should be encouraged to commence gentle movements in the first week, and progress as the healing tissue strengthens. Reassurance should be given about the pain, the prognosis, and any other particular concerns that the patient has, so that fear–avoidance behaviour does not retard the reparative process. Once the repair is well established the patient should be encouraged to restore any restriction of motion and recover any lost function. *Treat Your Own Strains, Sprains and Bruises* (Lindsay *et al.* 1994) is an excellent patient-directed text on the self-treatment of many common injuries. This book presents a structured format by which patients can manage their own rehabilitation in a variety of acute and sub-acute soft tissue injuries. It offers a brief overview of the healing process and gives daily progressions of specific exercises.

If the patient consults with a problem that has persisted for many months, they will often have been avoiding moving the limb normally, fully or into the pain. This behaviour may have been appropriate in the initial stage, but later is more likely to perpetuate problems. Reassurance is vital to convince the patient that even though exercises may provoke their pain, they are not only appropriate but also an essential part of their rehabilitation. The recovery of full pain-free movement must be the ultimate aim in the treatment; therefore all patients must learn the progressions of exercise necessary to achieve total functional recovery. If the problem has persisted for some time, rapid changes in symptoms or mobility will be less likely in some conditions. The patient should be informed
gradual change in their condition, and this should not be used as an excuse to implement passive treatment modalities of dubious worth.

Finally, the patient must be instructed sufficiently so that he or she can, in the future, avoid recurrence of the problem. In the event of recurrence they must know how they can minimise the consequences. If complete freedom from symptoms is not attainable patients need to be acquainted with the optimal self-management strategies that allow them to cope with their problem in the best way possible. Thus patients have a significant role to play in their own rehabilitation – only they can influence the state of the tissues repeatedly and regularly

day after day, but they need to be educated in the appropriate behaviours.

If clinicians are to become educators it is necessary to be aware of some of the tools involved in education. Giving the patient too much information, especially on the first visit, will be confusing. What are the most important messages you want to get across? Keep them to a minimum and be clear about them. Repeat them several times. If the patient is having problems grasping something, get them to repeat it back to you. Explain again using a different example. Use patient-friendly language and avoid being judgemental – this is likely to antagonise them rather than make them receptive to your message. When teaching exercises and appropriate symptomatic responses, show them what to do first, then get them to do it and repeat it. Performance mastery of a task is the most effective way of influencing self-efficacy and adherence (O'Leary 1985). Action – getting patients to perform an exercise – is a better teaching model than demonstration, information or persuasion. Talk through the pain response, giving the explicit assurance that it's all right if it hurts a bit. Check that the patient knows what they are doing before they leave. As well as providing a model of pain behaviour that is acceptable, warn them about excessive aggravation of symptoms that is not acceptable, and when they should stop.

However, if we give the patients all the answers they totally rely on us and are less likely to maintain control of their problem after the intervention has finished. It is important to get the patients to solve their own difficulties and dilemmas as much as possible and get them to work through how individual problems can be tackled. One way to do this is to give them options; for instance, alternative ways of avoiding postural stresses or overcoming problems concerning maintenance of an exercise regime. Patients who are reluctant to follow a programme can be offered the choice of doing it and changing their present state or not doing it and remaining as they are. The more they can cope without health care providers the more their independence is enhanced. In effect the episode of therapy should be a process during which information and strategies are discussed and control is gradually ceded to the patient, so that at discharge they feel confident that they can manage their problem effectively and independently.

Active mechanical therapy component

Patients wish to have both an understanding of the problem and to know what they might do to help themselves. Once an explanation has been provided of the role that exercise has in restoration of normal function, it is then necessary to show the patient the active mechanical therapy component of their self-management. The specific exercises should be demonstrated to the patients with instructions to practice these. The expected pain response should be explained, as well as any warnings against lasting aggravation of their condition. As necessary, progressions and alterations should be given. In certain chronic conditions a temporary exacerbation of symptoms is possible and necessary prior to any improvement. The situations in which this is relevant will be noted in the text. In most circumstances exercises should be repeated about ten times each session. Initially, to test that the response is satisfactory, these should occur three or four times a day. Once the safety and appropriateness of this routine is established, exercise sessions should be held every two hours, or as regularly as possible.

The clinician's initial role is to provide reassurance and encouragement to the patient that they are performing the appropriate strategies to regain their function. They must isolate the relevant movements which the patient should be performing and make sure these are being performed correctly. The clinician's role is to gently guide the patient through and beyond the healing process to the point where recovery of function calls for and allows more vigorous measures. The management will first consist of self-treatment procedures and *only in the event that these fail to provide improvement* should procedures that require the assistance of the clinician be added. At times, if improvements are too slow in coming, patient techniques may be supplemented with clinician techniques, but the emphasis must always be on what the patient is doing for themselves ('What I'm doing is only 20%, what you're doing is 80%'). There is no guarantee, however, that the addition of clinician procedures will hasten the speed of improvements.

The clinician will identify the direction of functional impairment and may supplement patient exercises with techniques that restore full range of pain-free movement. For instance, in capsular restrictions at the hip, stretching into medial rotation can be difficult for the

patient and clinician assistance may be required. The clinician may be required to aid in the reduction of internal derangement. Reduction by self-applied procedures is sometimes difficult, and it may be necessary for the clinician to mobilise or manipulate to achieve reduction. The clinician must also identify postural forces which, if not removed, may delay recovery following trauma.

Compliance or therapeutic alliance?

When self-treatment programmes are discussed, the issue of patient compliance or non-compliance with instructions is often raised. Patients are frequently seen as reluctant followers of medical or physiotherapy advice. Various studies have found that average compliance with medication regimes is only found in 50 – 60% of patients, while compliance with physiotherapy programmes was consistently worse at about 40% of patients (Deyo 1982). Factors that have been found to be important in non-compliance with exercise regimes are the barriers that patients perceive or encounter to doing the regime, the lack of positive feedback and the degree of perceived helplessness (Sluijs *et al.* 1993). Other factors that have consistently appeared to influence non-compliance are patients who have an external health locus of control, complex regimes, regimes that are not tailored to the patient's daily routine, unclear explanations, and lack of explanation concerning the reason for performing the programme (Sluijs *et al.* 1993). However, it is suggested that as compliance is defined as the extent to which patients adhere to health advice, it implies coercion or passive submission. An alternative has been proposed, termed *therapeutic alliance*, which implies negotiation and shared decision-making (Brady 1998).

In ongoing disease states it is proposed that active patient involvement rather than passive submission to health instructions is the most appropriate model. "*The passive sick role is incongruent with rheumatology findings that control beliefs such as self-efficacy and helplessness are important influences on health outcomes*" (Brady 1998). Patients and health care providers are each partially knowledgeable and appropriate decisions can only be made with the active participation of the patient in the decision-making process. Clinicians must inform the patient about the condition and self-management strategies, but can only evaluate the effectiveness of

those strategies with the patient. In turn, the patient with a chronic problem has certain responsibilities, these being to:

- learn about the condition and its management
- take responsibility for self-management and joint decision-making
- evaluate the health care experience.

Active self-management rather than compliance is the preferred outcome (Brady 1998). This can only be achieved if the patient is sufficiently informed and empowered. It is the clinician's responsibility to try to create this state of mind.

To treat or not to treat?

Irrespective of the nature of the problem, whether post-traumatic, non-traumatic, inflammatory or degenerative, it should never be assumed that treatment by physiotherapy is always necessary. There can be no justification for applying physical treatment unless that treatment is known to accelerate the natural history or will assist in the recovery of function. There is no evidence that many commonly used physiotherapy modalities actually do this (Clarke 1999; Feine and Lund 1997; van der Windt *et al.* 1999). However, the patient must always be given comprehensive guidance and education in the appropriate strategies to assist with the healing process and to regain normal function. All patients are entitled to that guidance and any educational tools that may be relevant to their problem. Not to do so is to deny to patients the right to self-manage their problem which, given the physiological and psychological aspects of the pain experience outlined earlier in the book, may lead to less than optimum treatment and can engender iatrogenic disability. All clinicians should be obliged to provide the relevant information to secure the maximum response in the manner best suited to the patient's physical, social and financial wellbeing

Treatment is usually perceived as doing something to patients – patients may feel cheated if something is not 'done to them', and clinicians find it difficult to resist the urge to use 'hands on' techniques or make use of some modality. These influences are especially strong in a climate of financial exchange. The evidence previously presented

makes clear that the value of these interventions is largely unproven. However, it is clear that many patients want to be informed about their condition and what they can do about it.

In physiological terms, regularly repeated movements strengthen and normalise healing tissue and restore to normal function tissue that is abnormal, over-sensitised or de-conditioned. Only patients can do these exercises. In psychological terms, patients' involvement in treatment has the ability to decrease fear–avoidance behaviour and allow patients to have greater control over their problem. Optimum treatment of many musculoskeletal conditions is founded on patient involvement. Thus the first and most powerful management option that should be used is educational. It is not always necessary to do something to the patient; this should only occur if other routes have been exhausted. Treatment can and should always comprise the provision of advice, information, encouragement and monitoring of patient-managed progress.

At the first interview, and mainly from the patient's history, we can usually identify whether or not the patient will require education or treatment, or a combination of both. Key factors in this decision-making relate to the stage and status of the patient's condition. It is important to know the likely state of the tissue – whether inflamed, healing or abnormal – and whether things are improving, unchanging or worsening.

Condition improving

When the patient reports that their condition is improving, a review of the problem and its prognosis is all that is required. Avoid the inclination to embark on a programme of passive therapies. Whatever the origin of the symptoms, whether inflammatory, degenerative or post-traumatic, improvement in the patient's condition is the ultimate aim of treatment. If that process is already under way, continuing at a steady rate, and accompanied at the same time by improvement in function, there is no justification for any intervention other than education and assurance. Provide guidelines for the progression of activity and exercise where necessary, but such patients do not require attending a clinic for regular 'treatment'.

An example could be shoulder pain, which commenced following lifting or reaching, and is improving from the date of onset. Where

that is the case, treatment is unnecessary. Instruction to temporarily avoid the painful movements and education to prevent loss of function through a graduated exercise programme will suffice in the management of the problem. A telephone call in a few days should be made to determine if a further consultation is necessary. There is as yet no evidence that physical treatment accelerates the time required for recovery from such minor problems.

Condition unchanging

Where the patient reports that a slow insidious onset of pain with movement has developed over time and is now unchanging, a routine approach to the assessment can proceed and education and instruction for a suspected dysfunction can be provided immediately. A similar routine approach can be adopted where the patient reports only partial but stable recovery following a sudden or traumatic onset of pain in the past. A dysfunction resulting from imperfect repair might be suspected. Whatever the type of onset, stable and persistent symptoms generally suggest the need for a reasonably vigorous approach to assessment and management.

If the patient describes that symptoms resulting from trauma two to three weeks ago *are unchanged since onset*, it could mean that healing has not occurred or is suffering frequent disruption. It is uncommon but not impossible for healing to take longer than three or four weeks, especially if the patient is overactive and disrupts the process of repair. Athletes and sportspeople are frequently guilty of returning prematurely to full activity. If applied too soon after injury, movement may further delay repair. On the other hand, if the commencement of active movement is unduly delayed, the stimulus to repair that is provided by early movement does not occur. Management must take account of the need to reach the right balance between relative temporary rest and active, graduated movement in sub-acute problems.

Derangement may cause pain and functional impairment that may continue unabated for weeks until reduction occurs spontaneously as a result of a chance event or is reduced by manipulation. Unlike pain from the inflammatory process, certain repeated movements or sustained positions will reduce the pain when derangement is causative. This will be clarified after the mechanical assessment. Derangement may be the cause of persistent pain of recent onset

– sub-acute conditions are not always the product of chemical pain states.

If the patient reports pain that has persisted for many months, which may be constant or intermittent, and classification according to one of the mechanical syndromes is unclear, then a chronic pain syndrome may be suspected. Getting such patients started on regular, graduated exercise programmes frequently leads to an improvement in symptoms, function and patient's perceived self-efficacy. Initially they may experience an exaggeration in symptoms due to the nature of chronicity, which is likely to involve sensitisation of certain tissues. They should be encouraged to pace their activities, not do too much too soon, and alternate activity with rest. Unless findings emerge from the assessment process that suggest further tests or more caution is required, *education and instruction* in a vigorous self-treatment programme are indicated. Clinician intervention at this point is unnecessary but may follow at a later date should self-treatment and guidance fail to provide improvement.

Chronic tendon conditions with relatively stable symptoms can represent a failed attempt at repair which has got 'stuck' at some stage. An initially vigorous management approach can provoke symptoms by stressing the tissue to re-stimulate the healing process. The temporary aggravation of symptoms should only last a day or two, and once provoked a more gentle and graduated approach should replace the vigorous exercise programme.

Condition worsening

In the event that the patient describes that his or her symptoms are worsening since onset, it will be necessary to investigate the cause of deterioration. Should the symptoms be localised and of recent onset, it may be that inadequate avoidance of the causative activity is all that has worsened the condition. Certain inflammatory diseases, including 'frozen shoulder' in its early stage, can progress over time, gradually causing increasingly severe pain. Therefore the possibility of the presence of an underlying inflammatory or more serious disease process should always be considered. If the patient has been or looks unwell, or if the reactions to mechanical evaluation are atypical or if they fail to affect the symptoms, the patient should be referred for further investigation. Appropriate blood tests or radiological

assessment may shed light on the origin of the symptoms in such cases.

A rather gentle approach to the mechanical evaluation is always required if the patient describes that their pain is progressively increasing. Increasing pain intensity could indicate that early movement is not immediately appropriate. Under these circumstances a purely educational approach may be indicated, certainly for the first 24 to 48 hours

Patients whose symptoms are worsening should be seen on a regular basis until stability or improvement occurs, or until it becomes obvious that referral for further investigation is necessary. Certain conditions such as early stage 'frozen shoulder' may be aggravated by even gentle attempts to begin to regain function. Such patients can be advised on relative rest, use of analgesics or NSAIDs, and then reviewed at a later date.

Conclusions

Following an analysis of the history and the mechanical assessment, it will be possible to classify the patient into at least one of the categories outlined below. Most patients will fit a single category, although some may describe symptoms of more than one syndrome. It is possible for the patient intermittently to experience postural symptoms resulting from prolonged loading and at the same time to experience constant pain from internal derangement. A patient who has the raised sensitivity to normal movement associated with chronic pain may also have an underlying mechanical response to movements.

From that classification it should be possible to determine the required management programme for the patient or to refer him or her to a more appropriate source of care in the event that the condition is unsuited to the mechanical approach.

It should also be possible at this stage to decide whether or not the patient is likely to require clinician intervention or a purely educational and self-treatment programme. The programme details should be provided in writing to improve compliance. This is especially important if there is any doubt regarding the patient's ability to comprehend and comply with it.

The patient must fit one of the following classifications:

Table 8.1 Classification chart

Tissue state	*Management strategy*
trauma/inflammatory	rest
healing	restorative exercise programme
postural stress	re-educate
articular dysfunction	remodel end-range
musculotendonous dysfunction	remodel through range
articular derangement	reduce
chronic pain state	recondition and desensitise.

9: Management and Self-Treatment

Introduction

Previous chapters have described the various pathological states that the tissues may be in during different stages of a musculoskeletal disorder. Conditions are continuums that present with different tissue states at different stages of the disorder. A traumatic or insidious onset unleashes an inflammatory response. This is followed by the healing and remodelling processes, which if incomplete may leave tissues that are abnormal, dysfunctional, deconditioned or sensitised. Obviously management that is appropriate at one stage will be completely inappropriate at another. Patients may present with pain behaviours that are appropriate for an acute problem, while the same behaviours in someone with chronic pain is inappropriate and maladaptive. Treatment required to reduce an articular derangement will differ from that needed to remodel a dysfunction. A straightforward postural syndrome patient who fails to correct persistent poor posture and/or faulty technique in manual or clerical labour may cause his or her problem to progress so that it becomes no longer reversible with a simple correction of posture. Thus management strategies are determined more by the stage or nature of the disorder rather than the structural diagnosis. The status of the condition, whether it is improving, unchanging or worsening, is also an important determinant of management decisions.

This chapter provides the general principles by which different stages of a condition and different disorders need to be managed. It should always be remembered that it is not simply disordered tissue states that are being treated, but individuals with various anxieties, fears, misconceptions or inappropriate behavioural responses to their painful condition. At every stage of a disorder patients need to be provided with the relevant information to allow them to manage the problem effectively. Their questions need answering and any worries or fears that they have about the pain need to be addressed. This is an integral part of ensuring optimal management of their condition.

This chapter contains general principles on the management and treatment of:

- postural syndrome
- tissue injury (acute)
- tissue healing (sub-acute)
- articular dysfunction (chronic)
- musculotendonous dysfunction (chronic)
- derangement syndrome (acute through to chronic)
- chronic pain states.

This chapter also briefly considers the role of clinician techniques and passive modalities in the treatment of musculoskeletal problems as these are so widely used. The philosophy behind this approach is firmly committed to patient education and involvement in treatment; these interventions detract from this. However, at times there may be a role for additional clinician forces, although this should never be done as a first option. In terms of passive modalities there is both very little evidence to support their use, and such treatment, based on a passive recipient role for the patient, undermines the message that confers prime importance on self-management.

Normal tissues, abnormal stresses – Postural Syndrome

Pain occurring in the extremities as a result of prolonged postural or positional loading is common. Individuals will report that after a period of time they experience tiredness or aching in the limb. The longer the position is maintained the worse the pain becomes. Relief is usually obtained immediately by a mere change of position or by moving the affected muscles.

During the early stages of the symptoms, discomfort or pain ceases entirely once the offending position is changed. Education at this stage while the symptoms are purely postural would probably avert the more disabling stages from developing. It is in this phase that education is most effective, although few receive it. With continued prolonged or repetitive loading over many months without adequate rest periods and/or relaxation, micro-trauma may result and an inflammatory response is triggered which is not amenable to

avoidance techniques. Cumulative trauma rather than a discrete traumatic event thus bring about pathological changes (Smith 1996; Thorson and Szabo 1992; Yassi 1997).

The need for the provision of appropriate work stations for keyboard operators and others performing stationary tasks is now well recognised. Employers have an obligation to provide employees with ergonomically designed workstations that minimise postural loading. There is an obligation on the part of the employee to use the workstation in the manner required for efficient operation. The best ergonomically designed chairs and desks are of no use if the employee sits slouched at the keyboard. The correction of the working posture is the sole responsibility of the employee. Education about the importance of posture and interruption of sustained working positions should be provided.

If the patient reports that pain only arises under conditions of static loading, and if movement is painless and symptoms cannot be provoked with active, resisted or end-range passive overpressure, the problem is postural. This can be confirmed by applying a prolonged loading assessment.

Education towards self-management

The patient should be given an explanation of the cause of the pain and how self-care involves frequent interruption and breaks from the repetitive or sustained activity, if they are not able to avoid the task completely. Frequent interruption of the causative position or posture is essential to prevent the condition from worsening. Repetitive full-range movement of the affected structures achieves this, if done very regularly – every half-hour for one minute. Regular 15-minute breaks from keyboard work have been recommended, for intensive work every hour and for ordinary work every two hours – rotation of job tasks is a more efficient solution (Huskisson 1992). Infrequent exercises would appear to have no effect upon workers' symptoms. For instance, two seven-minute on-the-job exercise routines performed each shift for a year produced no significant difference in discomfort compared to those not participating (Silverstein *et al.* 1988).

Overall body posture should be altered regularly and be well supported. If work is being performed in constrained or inefficient postures, patients need to be informed how to attain and maintain a

better working position. They should be warned especially about sustaining abnormal or extreme joint positions. Ergonomic interventions, which are considered to be important in the prevention and treatment of repetitive strain injuries, seek to reduce the repetition of tasks, their abnormal mechanical stresses and vibration, and improve working postures. However, as the physical environment improves, work organisation and psychosocial factors become more important and also need to be addressed (Yassi 1997). The possible implications of continuing with the same occupational stresses should be made clear to the patient.

Self-treatment procedures required

The patient must be shown how to modify or avoid the causative posture. Teaching the patient to reduce or stop their pain simply by modifying their working posture is an essential step in the prevention of further problems. Duplicating the offending position or posture until pain is provoked best does this. Once symptoms have appeared, have the patient change posture or, if necessary, perform appropriate movements until the pain goes. For example, a typist may develop pain in the upper arm after several hours of typing. Such symptoms are common and frequently result from sitting at the keyboard for prolonged periods in too low a chair. This requires the upper arm or the forearm to be raised in order to operate the keyboard. Raising the height of the seat is usually sufficient to allow the upper arm to hang limply by the side with the elbow at a right angle. This will relieve the static loading of the muscles involved. Most such problems are responsive to postural or positional modification of the workstation. Encourage people to think that posture is a habit that can be changed over a few weeks if the necessary consciousness is maintained – like all habits it can be changed, but sincere effort must be made.

In certain situations, however, it is impossible to alter the physical environment sufficiently to avoid pain. Under these circumstances, patients must be taught repeatedly to move the affected structures through a full range of flexion or extension for about a minute every half an hour.

Dynamic postural stresses have a role in the onset and aggravation of various musculoskeletal problems such as anterior knee pain, tennis elbow, supraspinatus, carpal tunnel syndrome, etc. These factors will be mentioned in the next chapter in the appropriate sections.

Acute presentations

Traumatic onset – tissue injury

First aid measures following trauma will vary depending on the severity of the injury. Serious injuries that require hospitalisation are beyond the scope of this book. It is nonetheless necessary for all those involved in the later management of the patient to familiarise themselves with the extent of the injuries and the nature of the immediate post-traumatic care. Where injuries warrant emergency interventions, it will be impractical to commence immediate physiotherapy or rehabilitation. Nevertheless, orthopaedic physiotherapy may sooner or later be required to recover function and rehabilitate the injured.

In the event that the extent of the injury did not require hospitalisation but the trauma was still considerable, the chance of a fracture or dislocation should be born in mind. This is especially pertinent following impact injuries such as during contact sports or motor vehicle accidents, and in certain groups such as young children or older women when trivial events can produce disproportionate injury. Thorough radiographic examination is the best way to rule out such eventualities. A protocol for selecting patients with injured extremities who may need x-ray examination has been developed. The set of criteria that best discriminated those with fractures or dislocations were bone deformity, instability or crepitus, point tenderness, ecchymosis or severe swelling in the upper limb, and moderate or severe pain in the thigh with weight-bearing (Brand *et al.* 1982). This text considers soft tissue injuries only, in which any more serious damage has been properly discounted.

Management

Shortly after trauma the inflammatory process will commence. The tissue damage causes a release of chemicals, one effect of which is to increase capillary permeability. This causes localised swelling from inflammatory exudate, which develops within a few hours – immediate swelling is a sign of substantial bleeding. The exudate is rich in protein, including fibrinogen; from this will come a network of fibrin that will hold the wound edges together. However, excessive amounts of exudate, and thus fibrin, promotes large quantities of scar tissue which will be a problem later. At this very early stage the essential aspects of management are protection from further damage and limitation of further bleeding or swelling. This is achieved using

the basic formula of **R**est **I**ce **C**ompression **E**levation (**RICE**). Gentle muscle contractions may also be useful to promote venous drainage (Evans 1980; Hardy 1989; Enwemeka 1989). Recent evidence-based guidelines have been produced by the Association of Chartered Physiotherapists in Sports Medicine in the UK which endorse the general rest, ice, compression and elevation model, but also include protection of the injured area (ACPSM 1998). It is recommended that these guidelines be used by all 'in the immediate (up to 72 hours) post-injury management'. There is no indication from the evidence for the use of ultrasound; this will be addressed in detail later in the chapter.

For the first few days following injury it is best not to commence the process of recovering function. The inflammatory phase lasts about five days during which time the RICE regime with gentle movements should be pursued. Protection of the wound from further damage, which might maintain this phase or cause more exudate, and limitation of the swelling are still the main points of management. Animal experiments have shown that following injury two days' rest is insufficient and causes further damage and increased quantities of abnormal scar tissue. Five days' rest allows the newly formed granulation tissue to cover the damaged area and to gain sufficient strength to withstand the subsequent mobilisation. Longer rest than this promoted weak and abnormal scar tissue, which was contracted and of poor structural organisation (Lehto *et al.* 1985; Jarvinen and Lehto 1993). Within the first week gentle movements must be commenced to start to influence the quality of repair and stimulate circulation. Exact timing to start mobilisation will depend upon the extent of the injury and be determined by pain response to gentle movements.

Treat Your Own Strains, Sprains and Bruises (Lindsay *et al.* 1994) provides an excellent self-management programme for the immediate and later guidance of the patient in his or her own home. This book also shows patients how to progress injury rehabilitation over the first three weeks. Where attendance at an outpatient clinic is sought, management during the first week following injury remains the same and the RICE strategy plus early mobilisation should follow routinely. Some injuries may require temporary immobilisation, but this should be used as sparingly as possible and be interjected with some regular movement, however limited.

Sub-acute presentations

Post-trauma – tissue healing

From about the fifth day following trauma, fibroblasts proliferate and collagen is laid down, with maximal deposition occurring by about three weeks. The strength of the wound increases but is still far from normal. An animal experiment found that skin wounds at sixty days were only ten to fifteen per cent of control values, and even at 100 days were only about one-third the strength of intact skin (Douglas *et al.* 1969). Mobilising thus proceeds gradually, *but never so that aching is aggravated or worsened*. By the third or fourth week after injury cross-links between collagen fibres begin to increase the tensile strength of the repair tissue. It is essential during this reparative phase of the healing process that careful tension is applied to the wound, but well within the patient's tolerance of discomfort. The tension encourages the correct orientation of the new collagen so that it aligns along the line of stress of that structure, and thus produces the best functional outcome. If tension is not applied to healing tissue, the new collagen is laid down randomly in a way that lacks tensile strength, is prone to developing adhesions, contractures, and excessive cross-links between collagen fibres, therefore hindering function. As the weeks pass the degree of tension applied should be increased in a progressive manner (Witte and Barbul 1997; Evans 1980; Hunter 1994; Hardy 1989; Enwemeka 1989)

From about the third week collagen synthesis is matched by collagen lysis so the quantity of collagen is no longer on the increase. Cross-linkage between collagen fibres continues the process of strengthening the repair tissue. The same need to apply tensile and contractile loading to the healing tissues applies as before. This prevents the formation of restraints to normal function and encourages the strength of structures appropriate to need. During the remodelling phase, full range of movement and tensile and contractile loading as well as functional capabilities should incrementally return to normal levels. The tendency for wound contracture to occur, and therefore the need to continue to stretch the area long after healing, must be conveyed to the patient (Evans 1980; Hunter 1994).

Education towards self-management

The patient should be given a clear explanation regarding the overall steps that they need to take to manage and rehabilitate their problem to full function and pain-free status. This may include some

information about the healing process that their injury is going through, and the risk of lasting impairment if the appropriate rehabilitative process is not performed. They should understand that complete rest of the injured part is necessary for only the first few days, if that. Gentle attempts at movement should be made at some time every day as early as possible. Instruction in movement and exercise should commence as soon as possible depending upon the patient's condition and pain. Both the number and frequency of repeated movements and the anticipated progression required for recovery should be explained.

Patients must be given a clear model of acceptable pain behaviour. If pain is produced by movement but abates when the movement stops or decreases upon repetition, this is acceptable. If pain, which is produced during movement, remains worse afterwards or if pain progressively increases with repetition, this is not acceptable at this acute stage. With the latter response mobilisation is applied either too soon or too vigorously and needs to be modified. See Chapter Seven for a more detailed description of symptomatic and mechanical responses to loading.

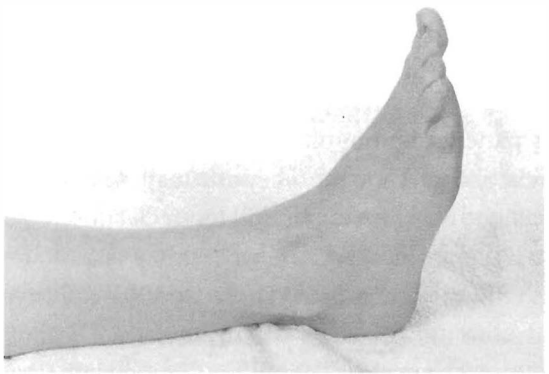
It should be stressed here that this cautious approach to encouraging patient activity is only appropriate at this early stage when prevention of further damage is a relevant management goal. While exaggerated pain behaviour at this stage allows the individual to aid recovery and protect from excess vigour, just a few weeks later the same pain behaviour is inappropriate, maladaptive and liable to cause increased pain and disability.

Self-treatment procedures

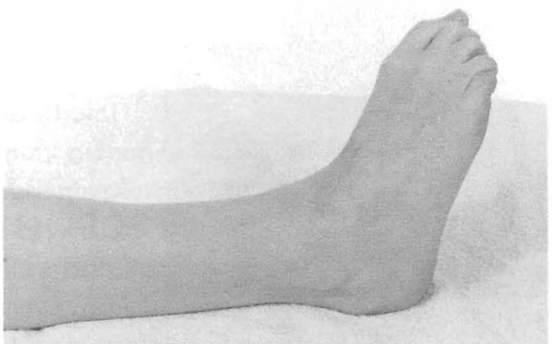
The patient should understand that initially the number of movements, their frequency and the range should be guided by pain response. The patient should attain as full a range of movement as pain allows. The number and frequency of movements will vary, but a minimum goal of five movements every two hours should be set and more often if pain permits. Both the number of movements and the range achieved should progress daily. As a means of progression to improve both strength and function, all patients should be taught self-resisted movements. These can be commenced once pain from resistance can be tolerated.

For different injuries and parts of the body different programmes will be required. Most physiotherapists are well aware of the range of exercises needed to make the appropriate progressions. Progressions may involve any of the following factors: non-weight-bearing/weight-bearing; inner range/outer range movements; isometric/isotonic loading; concentric/eccentric loading; and velocity of movements. Some examples of different progressions are shown below.

Photos 1, 2
Early non-weight-bearing active movement into the discomfort zone, in this case following a lateral ligament sprain of the ankle.

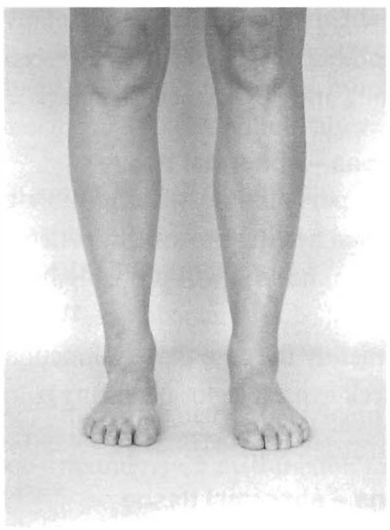


1

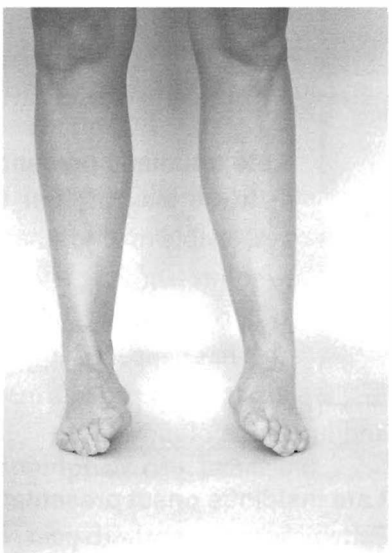


2

Photos 3, 4
Later weight-bearing active movement following a lateral ligament sprain of the ankle.



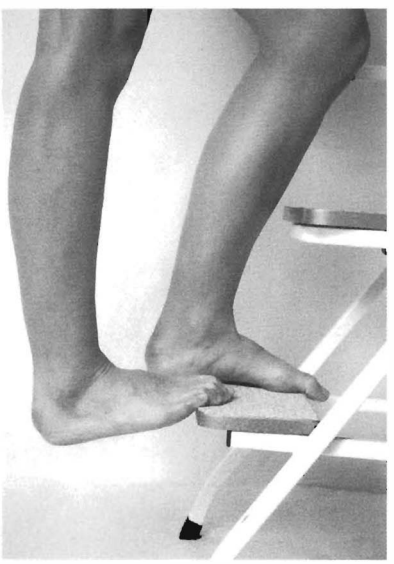
3



4

Photos 5, 6
Eccentric loading of Achilles' tendon during late stage rehabilitation of a chronic problem. The affected leg (here the right) starts in plantar-flexion and unloaded. The weight is transferred to that leg and the heel lowered to provide eccentric loading.

5



6

More vigorous exercise is important in the prevention of dysfunction or structural impairment, especially in patients undertaking demanding sporting, recreational or occupational activities. Exercises should relate to the individual patient's functional demands. Where significant disability or weakness persists, resisted strengthening exercises with gymnasium equipment may be needed once pain and range of motion permit.

Chronic presentations

The majority of patients seen by physiotherapists are not in the early stages following trauma. What has been explored so far is the straightforward rehabilitation of a soft tissue injury with the optimal, appropriately timed management. Unfortunately patients are not often like this; of course this is why they are seeking treatment. They are not getting better – pain has remained and function is still limited. Those who recovered in a linear fashion and are where they want to be functionally do not seek treatment. Those we see are always likely to be the more 'difficult cases' where the normal healing process has not gone through all its stages due to inappropriate, over-protective behaviours, failure to remodel, pre-existing degeneration in the tissues, sensitisation caused by malfunction in the central nervous system, or for some other reason the condition has become 'stuck' during the process of repair. As shown in the introduction, many common musculoskeletal conditions that are frequently seen by physiotherapists have long and persistent histories.

Late traumatic presentations – abnormal tissue

Patients may present with persistent pain long after a reasonable time for the normal healing process. In patients who present long after trauma, healing will probably have occurred without the benefits of education or advice. Where this has happened it is frequently the case that dysfunction exists. Such patients may seek assistance for persisting pain and limitation of movement.

Late insidious onset presentations – abnormal tissue

Patients who present with pain of insidious onset can go through the same tissue responses as those following trauma – inflammation,

repair and remodelling – or they can also become 'stuck' at one of these stages. 'Frozen shoulder', 'tennis elbow' or osteoarthritis of the hip or knee, for example, can be seen at different times as an inflammatory or mechanical disorder. Tendon disorders in particular are prone to lengthy, protracted and aborted recovery. The insidious onset is likely to indicate pre-existing overuse or degeneration which has undermined the capability of the tissue to heal normally (Leadbetter 1992; Buckwalter *et al.* 1993). Because of this the timeframe for such problems is generally much longer. Despite their weakened state, it is important to remember that all connective tissue need loading stresses in order to improve function.

Intra-articular derangements

Alternatively, following a traumatic or non-traumatic onset patients may have persistent symptoms and limitation of normal function because of intra-articular derangements.

Chronic pain states – abnormal tissue / abnormal response

Another scenario is that tissue healing may be complete, but sensitisation to normal movement or fear of pain may be preventing the individual from regaining full function. With pain that has persisted for long periods it is important to recognise the role that neuronal plasticity and/or psychosocial factors can have in modulating pain sensation irrespective of the local tissue state. Chronic musculoskeletal pain has been shown to be maintained by pathological sensitisation in peripheral and central nervous systems – a state where the dysfunctional tissue is neural rather than somatic (Cohen 1996). Patients' fear of pain, beliefs about pain, or feelings of lack of control may all be factors that maintain an ongoing pain state. In patients with persisting musculoskeletal pain, the role of non-mechanical factors should always be considered.

In summary, these different forms of tissue abnormality may be present in conditions that have persisted after the normal recovery period. These equate to:

- somatic tissue dysfunction – articular
  – contractile
- intra-articular derangement
- neural tissue malfunction / inappropriate pain behaviour.

The management of these problems will be reviewed in the following sections.

Abnormal tissue, normal stresses – Dysfunction Syndrome

Dysfunction in the extremities commonly affects either articular or musculotendonous structures. Contraction or adherence of soft tissues is a very frequent reason for loss of function. This abnormal, shortened tissue may have arisen from a past traumatic event or be the product of an overuse or degenerative condition. Normal stresses reproduce the patient's pain. Remodelling of these structurally impaired tissues occurs over many weeks and months. Structural impairment cannot be rapidly normalised. A slow but gradual improvement must be expected. Therefore, irrespective of the treatment provided, little change in pain intensity or range of motion is seen from day to day in the management of patients with dysfunction.

There is a highly characteristic symptomatic response to loading strategies in dysfunctional tissue. The performance of repeated movements in the direction that produces loading of structurally impaired, contracted, adherent, fibrosed or adaptively shortened structures will produce pain. Repetition of the movement does not make the pain progressively worse. Range of motion will neither be increased nor reduced. When the limb or structure is returned to the neutral position the pain will cease. The pain that the patient complains of will be reproduced by end-range stretch in articular dysfunction and through range contraction in musculotendonous dysfunction. The test movements most commonly required for assessing joint and adjacent soft tissue function are flexion and extension. Rotation may be required for the shoulder and hip. However, all joint movements should be assessed to determine the full range of motion and the most salient responses. Generally speaking, the movements that produce the greatest amount of mechanical deformation and therefore pain can, when modified appropriately, be used to have the greatest therapeutic effect.

Articular dysfunction

Articular dysfunction may present with a history of trauma or insidious onset. Patients with articular dysfunction resulting from an old injury will have reported in their history that they have had the symptoms for several months. The symptoms date back to some event or injury that has never completely resolved. Initially the symptoms were more severe or more noticeable and have improved, but are persisting and no improvement has occurred for some time. There will be restriction of movement of the affected joint and pain

will commence if movement towards the restriction is actively or passively performed.

Degenerative joint disease can cause adaptive changes which eventually impact on the joint capsule and surrounding soft tissues. This in turn causes contraction of the capsule and results in progressive loss of movement; thus osteoarthritis is also commonly the cause of articular dysfunction. Another cause of insidious onset articular dysfunction is late-stage capsulitis of the gleno-humeral joint. After the initial inflammatory stage the capsule can be left with severe loss of movement, but pain at end-range only.

If the subject's pain is produced at active end-range movement that is limited, and not through the arc of movement, the problem is probably capsular. If the pain is produced only at passive but mechanically limited end-range and not through the arc of movement, the problem is probably capsular. If only overpressure at end-range is painful, the problem is probably capsular. If all three tests are painful, the problem is capsular dysfunction. This will be confirmed after applying resisted movements, none of which cause pain. Repeated end-range movements will confirm this. They will, each time, reproduce the patient's pain, which get neither better nor worse, but abate within minutes of ceasing the movements.

The loss of function occurring in the joints of the extremities is frequently in the sagittal plane. As is most common in the human spinal column, extension is frequently reduced at the elbow, wrist, fingers, hip, knee and ankle. Flexion may be reduced at the shoulder, elbow, wrist, hip, knee and ankle joints. Loss of rotation and abduction is a particular problem at the shoulder and hip. Thus it is the case that the procedures required depend on the particular movement losses observed at that individual joint.

In the case of articular dysfunction, remodelling of the involved structures will require repeated movement loading sufficient to reproduce the patient's pain at end-range.

Education towards self-management

It is very important to give the patient a thorough explanation of the cause of the tissue abnormality, the reason for the necessary management and the expected prognosis. Patients will be presenting with pain and abnormal function; their chief complaint is usually

pain. They are only likely to tolerate a management strategy that involves producing that pain at regular intervals if they are given a convincing reason for such a course of action. A description of the healing process and possible structural imperfections that may occur during this process using the example of contracted scar tissue should be given. The capability of tissue to be remodelled by repeated stresses and the self-treatment procedures needed to remodel should be explained. A timeframe for recovery should be provided. The condition will not change quickly – especially if it has been present for some time – and they must be warned not to be over-optimistic about the time needed for improvement. Remodelling of tissue takes weeks or months of regular repeated movements – the patient's committed input is vital for recovery, for only the patient can perform the appropriate exercises with enough frequency.

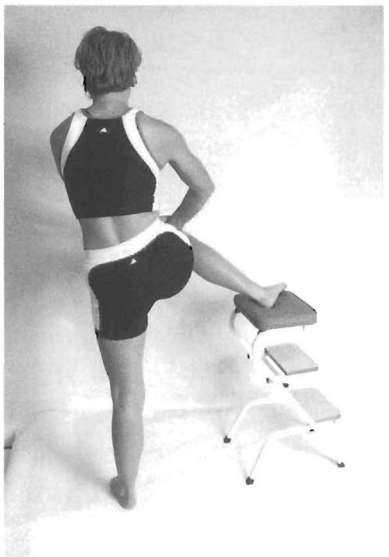
The patient must be warned of the need to stress structures to the point of pain, but not to the point of damage and lasting pain. By showing them that pain, although easily and consistently produced, abates within minutes of ceasing the stressful manoeuvre, the patient is provided with a simple example of pain behaviour to follow. Very often in dysfunction the clinician's main role is to ensure that the appropriate exercises and progressions are being done correctly and with enough regularity, to make sure that the patient understands the expected pain response, and to provide encouragement and monitoring of the progress in symptomatic and mechanical presentations. The patient should be reviewed in two days and at the end of the first week to correct and check exercise techniques, regularity, pain responses and the patient's understanding of the concept of dysfunction. Appropriate progressions of resisted exercises or modified techniques should be added as indicated. Subsequent reviews can frequently be conducted over the telephone on a weekly basis or with irregular visits to the clinic.

Self-treatment procedures required to remodel articular dysfunction

When the diagnosis of dysfunction has been made in articular structures, it follows that treatment designed to remodel affected soft tissues is indicated. To ensure that a remodelling process can be initiated successfully, it is frequently necessary to apply sufficient stress to the affected structures to stimulate the formation of better quality collagen repair or scar tissue.

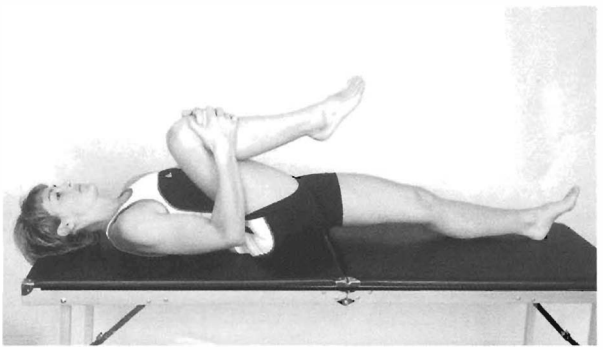
Examples of end-range self-mobilisations for use in articular dysfunction

Photo 7
Medial rotation of the hip with or without overpressure.



7

Photos 8, 9, 10
Different ways of achieving flexion of the hip with overpressure. The method with the stool may be easier to do in the work environment.



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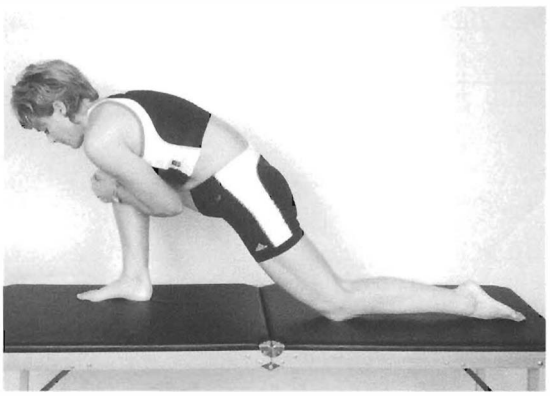

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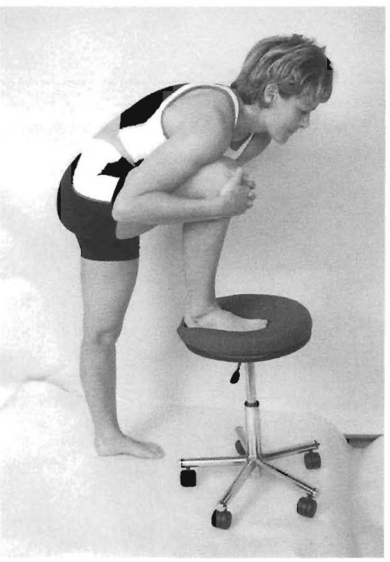

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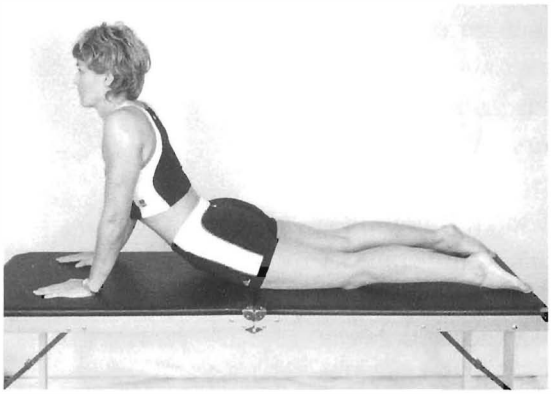
Photos 11, 12, 13
Different ways of achieving hip extension, bilaterally or unilaterally (left hip only). The method in photo 13 may succeed in producing pain at end-range that is not provoked by the other procedure.



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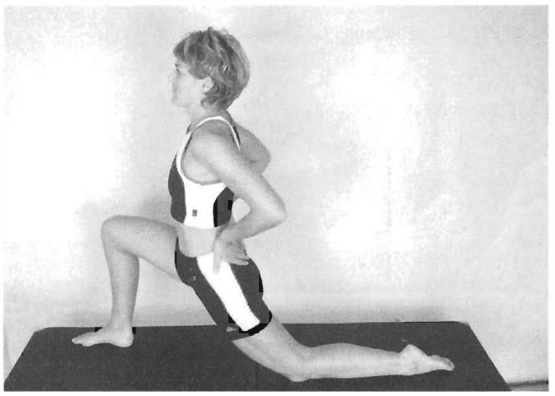
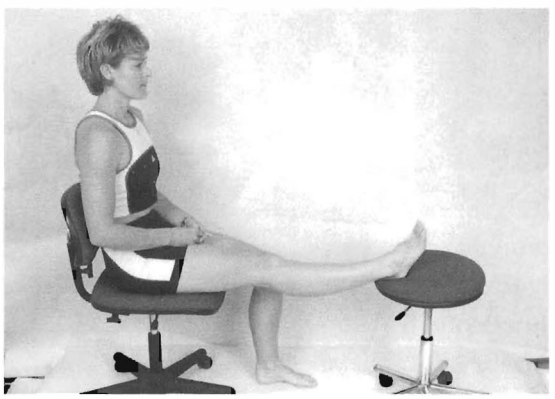


13

Photos 14, 15
Knee extension, sustained or with intermittent overpressure.



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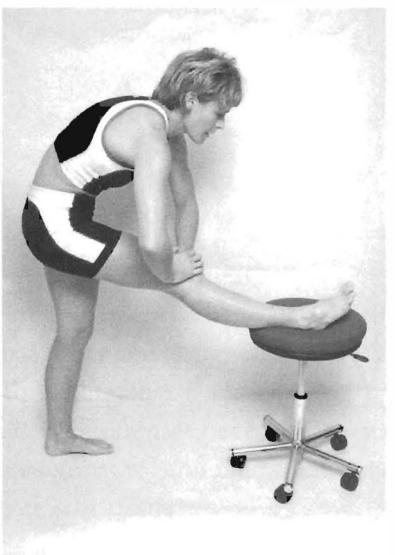

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In the case of articular dysfunction, the patient must be instructed actively to move the affected joint towards the restriction until pain is felt. The movement should be repeated ten or twelve times to the point of pain, three or four times daily. If this is done without exacerbation of symptoms, the exercise should be increased to every two hours. After the first week or ten days, the patient should be shown how to progress to self-apply overpressure using the healthy arm or leg. In many joints it is only the addition of overpressure that will get the joint to end-range. For instance, full hip and knee flexion is impossible to obtain by active movement only. Overpressure may sometimes be necessary from day one.

The appropriate exercises should be continued until function and pain are improved. Frequently the movement will improve as symptoms do. This is not always the case, however; sometimes the restriction is little changed, but symptoms improve. When movements are especially stiff with a hard end-feel, the ability to remodel the tissue and improve symptoms is much less, and failure to help this group is high. After an improvement in pain and function, exercises performed as one session each day should help to maintain the improvements that have been achieved – this is especially important in degenerative joint conditions.

Musculotendonous or contractile dysfunction

The patient may describe either a traumatic or insidious onset. There may be, for instance, a history of a groin or calf strain, or they may describe long-term symptoms of repetitive or cumulative strain such as from a tennis elbow or rotator cuff disorder. Any inflammatory disorder of tendon or sheath also has the capacity to cause lasting impairment of the affected structures. Symptoms will have been present for several months, but would initially have been more severe. The condition has improved, but persisted with no further improvement.

Resisted mid-range movement provokes the symptoms. If the pain is provoked by resisted movement applied at any point in the arc short of end-range, the problem must arise from one or more of the contractile structures. Mid-range repeated and or resisted movements are indicated. The contractile structure has to fulfill two functions – it must be able to contract and work against load, but it must also be able to stretch its full length (Fyfe and Stanish 1992). Musculotendonous dysfunctional tissue is painful on resisted movements and

if the flexibility of the unit is tested. If the contractile structure can be stretched, this also provokes discomfort and is restricted compared to the other limb. For instance, dysfunction of the Achilles' tendon will be symptomatic when pushing up onto the toes and when passively stretching into dorsiflexion. Remodelling is required where a tendon has become adherent to its sheath, where excessive cross-linkage of collagen repair within the contractile structure has occurred, or where fibrosis has developed following resolution of haematoma.

If pain occurs midway through any movement, it could arise from either derangement within the joint or from musculotendonous dysfunction – the cause needs to be differentiated. Passive movement will provoke the symptoms if they arise from internal derangement and will be painless in contractile dysfunction. Resisted movements applied in the 'target zone' will provoke the symptoms if they arise from the tendon or muscle, but will generally be painless in derangement. Repeated resisted mid-range movements are indicated. As is the case with articular dysfunction, musculotendonous dysfunction will require repeated movement loading sufficient to reproduce the patient's pain; unlike articular dysfunction, this is usually required short of end-range.

Education towards self-management

See *Education towards self-management* section above under articular dysfunction.

Self-treatment procedures required to remodel musculotendonous dysfunction

In the case of musculotendonous dysfunction, the movement that causes the greatest amount of pain also provides the greatest stimulus or irritation to the affected tissues. This will readily be found by testing out those movements described by the patient as being painful, if necessary with resistance. One of these will consistently provoke the patient's pain. Having identified the most painful movement, it is then necessary to find the point in the range where resisted exercises should first be applied.

Static or dynamic loading may be used to remodel musculotendonous dysfunction. Exercises should generally be performed without applying so much loading that there is a risk of causing damage or disruption to the remodelling process and re-triggering a constant inflammatory pain. The model of pain behaviour is that pain is

produced by the resisted exercise, but abates once the loading ceases. The exercise programme commences where the loading is sufficient to generate the patient's pain, but ceases when the loading is removed. There is an exception to this general rule in the case of certain chronic tendon problems that are associated with degenerative changes; this will be discussed later. For these conditions a temporary aggravation of symptoms is sometimes necessary to re-stimulate the healing process.

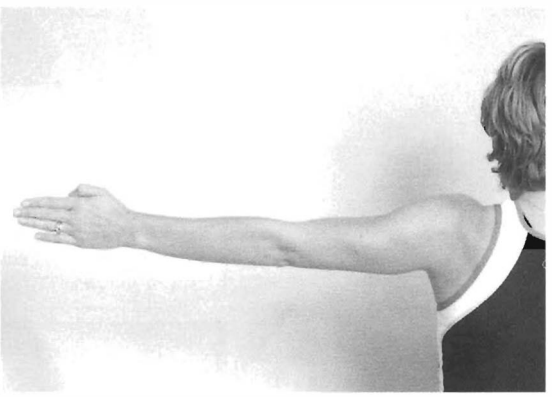
The point in the arc of movement that provokes pain or where pain is at its maximum is a reliable guide when deciding where in the range it is best to apply loading for the purpose of remodelling. This is the 'target zone' for the patient's focus. If the pain is felt at the same intensity throughout the range it is not necessary to seek a 'target zone'.

Having identified the point during the painful movement the patient should then be shown the procedures that will apply either static or dynamic loading sufficient to cause reproduction of the pain. The appropriateness of these procedures should be established over a test period and progressions given as necessary. Options available include active movements, static resisted movements and concentric and eccentric loading. Exercises can be performed in outer range, inner range, short of the 'target zone' and in the 'target zone' itself.

Eccentric loading in particular has been shown to be extremely useful in the rehabilitation of chronic tendon problems (Alfredson *et al*. 1989; Fyfe and Stanish 1992; Stanish *et al*. 1986). Stanish *et al*. (1986) for instance reports an uncontrolled study of 200 patients with chronic tendon problems, with marked restrictions of their normal function and failure to respond to a variety of physiotherapy

Photos 16, 17, 18
Finding the target zone. In this case it is at the shoulder. The patient exercises in mid-range, through the arc of greatest pain. If pain is too severe, exercises short of the target zone should be carried out.

16



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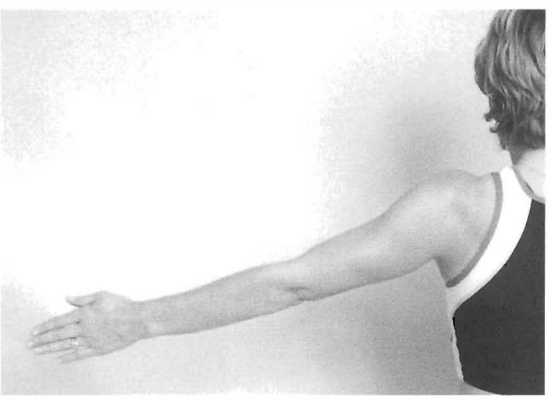
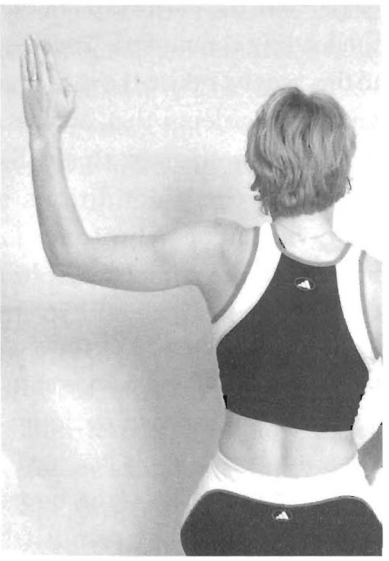


18

Photos 19, 20
Finding the target zone at the shoulder, in abduction and rotation in this case. Variation of the position of the articulation at any joint may be necessary to find the best target zone.

19



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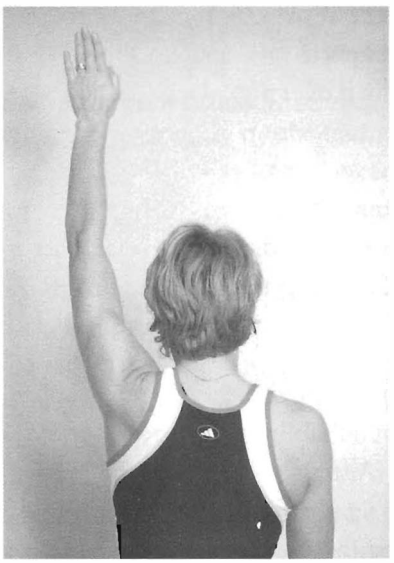
Photos 21, 22, 23, 24, 25
No target zone at the shoulder when pain is similar throughout the movement. Exercises are performed through full range and similarly for all movements at other joints. In this case, low-grade eccentric loading is achieved by lowering the arm to the side.



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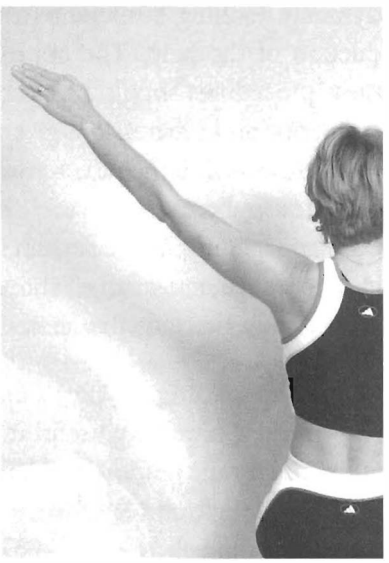

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programmes and corticosteroid injections. Following an eccentric strength-training programme performed daily over a six-week period, patients were followed up for an average of sixteen months. For 44% of patients there was complete relief of symptoms and return to normal function, 43% had a marked decrease in the problem (mild aching after sporting activities), 9% were unchanged, and only 2% were worse.

Low-grade eccentric loading can be achieved with active movements alone, for instance at the shoulder in lowering the arm to the side. The

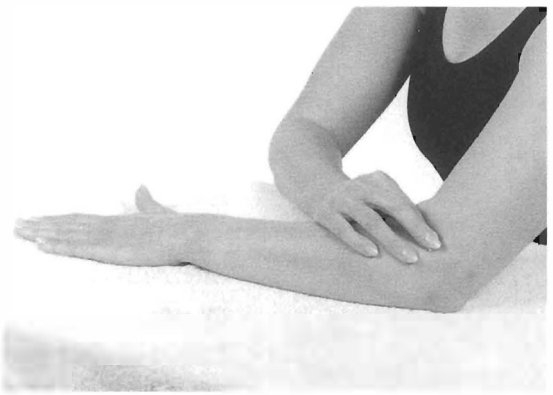
weight of the limb can be supplemented by a weight in the hand. If at the shoulder active exercises are inhibited by pain, the starting position can be changed to supine lying, which usually permits improved mobility. Not every individual will have to go through all the progressions mentioned. Necessary starting points and progressions are determined in individual cases by symptomatic and mechanical responses.

Isometric contractions exert less tension than dynamic contractions, and thus might be appropriate for initial use if there is concern about the stability of the stage of healing (Fyfe and Stanish 1992). However, isometric contractions can induce hypoxia in the contracting muscle within minutes, so it is recommended they be held for a minimal length of time (Solomonow and D'Ambrosia 1987). Static loading can be used where the same pain intensity is experienced in all parts of the range of movement. In this case, applying tension without movement usually provokes the pain. Static loading can be applied at different angles to evaluate where the best response is gained. The patient must be taught to apply the right amount of resistance to prevent movement but to cause some pain. The pain should be provoked briefly, ten to fifteen times in each session, relaxing completely after each contraction, and repeated three or four times a day. If the response to this is acceptable the frequency should be increased to every two or three hours. Isometric exercises only increase strength at the joint angle at which resistance is applied (Norris 1993), therefore dynamic exercises may be necessary to progress treatment. If pain is equal throughout the range of movement, loading can also be done through the entire range.

Dynamic loading is preferable when a 'target zone' is identified – in which case loading is carried out through the painful arc. Appropriate stimulation is best obtained by initially applying low amplitude alternating concentric and eccentric minimal loading ten to fifteen times. The amplitude of concentric and eccentric loading as well as the degree and speed of loading can be increased or decreased according to the patient's pain responses (Fyfe and Stanish 1992). After initially testing out the response when the exercise is applied three or four times a day, the series should then be repeated every two hours.

In the event that a slow but steady improvement is made, no change needs to be made to the management at this time. Should pain from loading levels gradually reduce, it may be necessary to increase loading progressively to provide the necessary stimulus for remodelling. If strengthening is needed for a specific task, sporting or occupational, then the exercise must mimic that task as closely as possible; this is in line with the specificity of strength training (Norris 1993). Eccentric loading in particular has been found to accelerate recovery and hasten strength gains, and has been shown to be very valuable in the rehabilitation of chronic tendon problems that have failed to improve with other treatments (Alfredson *et al.* 1989). However, it is more fatiguing than concentric work and more prone to induce muscle soreness (Fyfe and Stanish 1992; Norris 1993).

Photos 26, 27
Compression during active movement, in this case for lateral epicondylitis. The patient firmly applies pressure to the painful extensor origin with the thumb or index finger reinforced by the middle finger. The wrist is then extended and relaxed ten times every two hours.



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In the event that the patient has increased symptoms at follow-up, it will be necessary to review the exercise and the effects on pain. It may be that the patient has increased pain only for a short time after exercising, in which case the recommended exercises should be continued for a further two or three days. However, should the patient have increased pain which remains worse long after exercise, the recommended programme should be modified. Modification of the programme may take several forms. The first option should be to exercise in the ***least*** painful range, if one can be found. This may involve static holds well away from the 'target zone'. Alternatively, in some cases it may be necessary to exercise in a non-painful range as closely adjacent as possible to the point where pain commences. If exercise in this form causes no increase in pain, the modified programme should continue for a further ten to fourteen days. Alternatively, changing the starting position can help – for instance, various shoulder exercises, which may be impossible or aggravating in standing, can be done with ease when lying

Should no improvement follow after two to three weeks it may be necessary to increase the degree of loading temporarily to irritate the tissue. This is more likely to be necessary in chronic tendon problems which have been stable over time and that arise in degenerated tissue. This occurs silently in many individuals and is likely when there has been an insidious onset of symptoms. The brief period of stimulation of the tissues serves the purpose of re-triggering the healing process which appears to be absent or suspended, with the repair process 'stuck'. After an initial provocation, which worsens the condition for a day or two and generates change, loading needs to be reduced. To restimulate the tissue increased loading is required, for instance by working in the target zone or with the addition of weights. Alternatively, increased stimulus can be obtained during the exercise by applying pressure locally against the painful region using the fingers of the healthy limb. While the patient actively performs the movement, he or she applies static overpressure or compression at the site of maximum tenderness.

Once these procedures have stirred up the condition so that constant aching has been achieved, the increased loading should be stopped. The aching should only last a day or two, following which active range of movement, and later resisted, exercises should be initiated to rehabilitate the structure. If these measures fail to provide improvement, the patient should be instructed to stop all provocative loading for four to five days. Sometimes a period of relative rest after the stimulus of exercise exposes an improving status.

Treatment

Should a purely self-management protocol fail to produce some improvement within a four-week period, a review of the management programme should be considered. If compliance with exercising has been less than required, attempts must be made to seek better cooperation from the patient. If compliance with exercise has been adequate, the addition of more vigorous general activity and or exercise is indicated and clinician procedures in the form of resisted eccentric exercise may be indicated. A fully equipped modern gymnasium is a necessary adjunct to the rehabilitation and physical reactivation of certain patients with musculoskeletal disability and impairment, especially when the patient is required to exert more than average forces through the muscle because of occupational or sporting activities. Attendance at a gymnasium where specialised equipment is available must always be considered.

In the event that the patient has complied with all instructions regarding exercise and remains unimproved, it is sometimes the case that the recent increased activity is sufficient to stimulate the remodelling process without the patient necessarily being aware of the changes occurring. When conditions have got 'stuck' during the remodelling stage the stimulus of direct tissue loading, such as working in the 'target zone', may often be sufficient to initiate further change. Following this the stimulation of the exercise needs to be stopped for a period of re-assessment. A cessation of all prescribed exercise is sometimes sufficient for the patient to recognise that improvement is indeed occurring. Consideration should be given to instructing the patient to use the limb as normal for up to two weeks

In some contractile conditions strapping may be considered as a supplement to the management outlined above. Some individuals with persistent tennis elbow problems, for instance, are able to perform their normal sporting or recreational pursuits provided they use a suitable strap. A trial period may be necessary, as these items will not help everyone.

Articular Derangement Syndrome

It is common for internal derangement to occur quite suddenly in the joints of the extremities. This is unlike internal derangement affecting the cervical and lumbar intervertebral discs where a slow gradual onset over several days or weeks is common. The reason for this may be the absence of nuclear gel in the joints of the extremities. The menisci of the knee and tempero-mandibular joint are known to tear and cause locking or obstruction to movement. Painful obstruction to movement or pain in the mid-range of movement without obstruction is a feature of derangement occurring in the joints of the extremities. Loose bodies from degeneration of articular cartilage and bony fragments have been found at surgery within the knee, elbow, shoulder and hip joints. Osteochondritis dissecans at the knee or elbow can produce intra-articular bodies that restrict full movement (Patten 1995). Impingement of synovial membrane may also be a cause of obstruction to joint motion. Tears and catching of the acetabular labrum at the hip can produce painful episodes when twisting or moving (Fitzgerald 1995). Whatever the true causes may be, internal derangement of the extremity joints occurs and in some cases recurs frequently. There will generally be a rapid onset producing a painful limitation to certain movements at a joint.

As rapid onset is a common characteristic of derangement, it is also the case that rapid reduction of derangement may be achieved. Unlike the slow improvement that occurs with the remodelling of dysfunction, reduction of derangement may occur in two to three weeks or less. It is therefore common to observe daily improvement in levels of pain and increased range of motion as a result of applying reductive loading forces to the obstruction. Rapid and lasting changes occur when derangement is the cause of pain.

Sudden onset constant pain may therefore be the result of constant mechanical deformation due to articular derangement. Clinicians should always be aware that constant acute pain is not necessarily due to inflammation, but may result from a mechanical disorder; the symptom response should be clear. Although in derangement some movements may cause a worsening, there may be dramatic improvements with repeated movements in a certain direction. The rapid onset of painful obstruction to the attainment of end-range movement excludes the possibility of dysfunction as a diagnosis. An alternative but less common presentation of derangement is pain produced during the arc of active movement, especially in one direction, with end-range movement painless and not obstructed.

In the history, the patient will describe that certain movements cause his or her pain to increase, and other movements will cause the pain to decrease or stop completely. As the pain increases, movement becomes more difficult. The converse also applies – as pain reduces, movement should become easier. If during the mechanical assessment process the patient experiences an increase in pain or the pain becomes more widespread, a worsening of the condition is likely. Conversely, if there is a decrease in pain intensity or localisation of the pain the condition will be improved by procedures or movement repeated in that direction. In derangement, the performance of repeated movements in the direction that increases pain will almost certainly increase the derangement. There is an increase in pain intensity and symptoms will become more widespread, usually distally. The performance of repeated movements in the opposite direction results in a reduction of the derangement and reduction in pain, generally or distally initially; that is, localisation of the pain to its source. This characteristic of a derangement to favour movement in one direction is termed 'preferential direction'.

Although there is no satisfactory explanation, some apparent musculotendonous problems cause pains that respond to loading in an identical manner to articular derangement. The clinical presentation of these problems is virtually identical to certain soft tissue injuries. However, they can make rapid and lasting improvements that are more suggestive of articular derangement than a soft tissue injury. Certain shoulder conditions, apparently affecting the tendons, can become progressively worse when repeated extreme end-range loading is applied in internal rotation. The symptoms will remain worse until repeated extreme end-range loading is applied in external rotation. Although conjectural, it is possible that a fold in the synovial lining or the capsule itself could be alternately impinged and released. Similar instant changes of symptoms have been observed with apparent tennis elbows and lateral ligament strains of the ankle (Mulligan 1992). With these 'pseudo' tennis elbows, for instance, a lateral glide at the elbow while the patient contracts their wrist extensors makes the movement pain-free; after repetition, the contraction remains pain-free.

Education in self-management

The nature and cause of the problem must be explained to the patient – that a displacement within the joint is preventing movement and causing pain. It should be explained that this can be made worse by certain movements and reduced by opposite ones. They should temporarily avoid the aggravating positions and movements, but at the same time regularly do the reductive movements. These may cause some increase in pain at the time, but afterwards the joint should feel better and movements should be easier. There should be no lasting aggravation of pain. Symptomatic and mechanical presentations should worsen and improve in tandem. The self-reductive procedures should be demonstrated and then practised by the patient until correct. The timeframe of a few weeks for recovery should be offered to the patient if it appears that reductive pressures are bringing about a lasting change in symptoms and movement. The importance of regularly performing the exercise must be made clear to the patient. This can be performed ten times three or four times daily in the first few days, and then more regularly once the procedure and responses have been checked.

The patient should be reviewed in a few days. It should be checked whether and how frequently they have been doing the exercise. The

procedure should be checked to ensure it is done correctly, and response to the procedure should be determined. If they are doing as instructed and improvements are occurring, the present management should continue. After a couple of visits review can be by telephone.

Self-treatment procedures required to reduce internal derangement

In derangement, reduction of displaced tissue will require repeated end-range movement loading in the direction that progressively reduces pain. It is usual that accompanying the reduction of pain there will be a simultaneous improvement in the range of motion as the obstruction is reduced.

To achieve reduction the patient first applies repeated active exercise to end-range. Providing the pain becomes progressively less with repetition or the range of movement increases, or both, the patient may continue the basic exercise without modification until recovery is complete. Should improvement cease, the first progression of self-applied overpressure should be made. The patient must be instructed to apply this using the healthy hand in the case of upper extremity problems or both hands in the case of lower extremity problems. More forceful overpressure can frequently be gained using an article of furniture to assist. If the overpressure is in the correct direction, the extra force will produce more reductive pressure and improvements in mechanical and symptomatic presentations will follow.

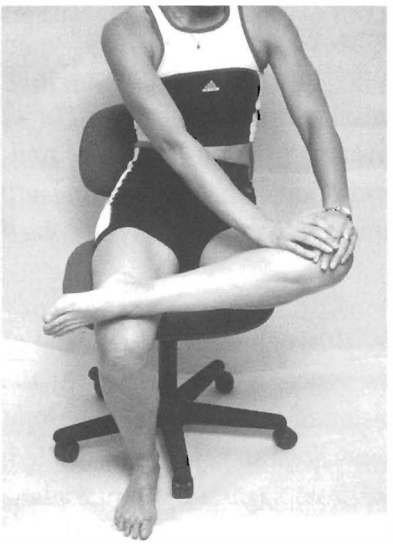
In the event that improvement ceases following the application of overpressure, the patient should be instructed in the application of resistance towards the direction of obstruction. In derangement it is sometimes the case that resisted movement towards the obstruction, that is towards end-range, results in increased range and decreased pain. Where end-range is neither limited nor painful but the derangement presents with an arc of pain, resisted movements may be the treatment of choice. In these cases, repeated movements, resisted through the arc of pain, should be applied first in the most painful direction. If repetition reduces the intensity of pain, the patient should be taught how to perform the exercise with resistance.

The preferred direction of end-range reductive movements needed for reducing peripheral derangements varies at different joints. Generally, the more planes of movement that are available at the joint, for instance at the hip and shoulder, then the greater the variability of reductive directions that may need to be tried. Symptomatic and mechanical responses need to be evaluated rather than relying on recipe-book solutions. Each condition is different and often the reductive movements can only be found by fully evaluating symptomatic and mechanical responses to loading. Forces may be required in any plane of movement, weight-bearing or non-weight-bearing, with or without the addition of traction, loading into the blockage or static resisted loading, and may or may not need clinician forces.

At the shoulder response is variable, although loading in medial or lateral rotation, with or without resistance, is often useful. At the elbow response is common to extension or flexion, with or without resistance, although lateral movements are also sometimes needed. At the wrist response can be gained from sagittal or frontal plane movements, often with better results when traction is added. The tempero-mandibular joint frequently needs opening, rarely ever closing. The hip may respond to flexion, extension or rotation movements. Alternative procedures are weight-bearing rotation performed by twisting on the affected leg, and a non-weight-bearing movement from flexion to extension, performed by standing with the unaffected leg on a step and quickly straightening the leg. At the knee, flexion and extension are the most common reductive movements.

The following photos represent some examples of end-range loading that may be required in the reduction of articular derangements

Photos 28, 29, 30
External rotation of the hip with and without overpressure to be applied ten times - different starting positions.



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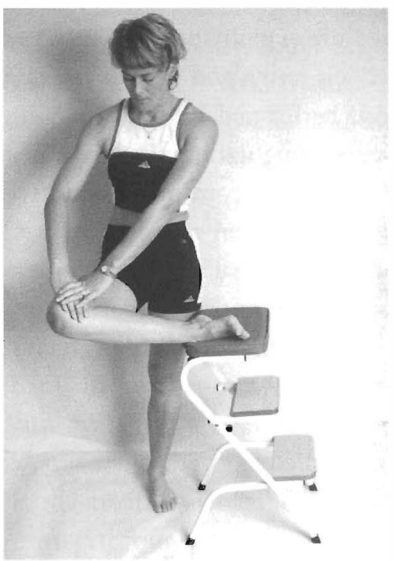

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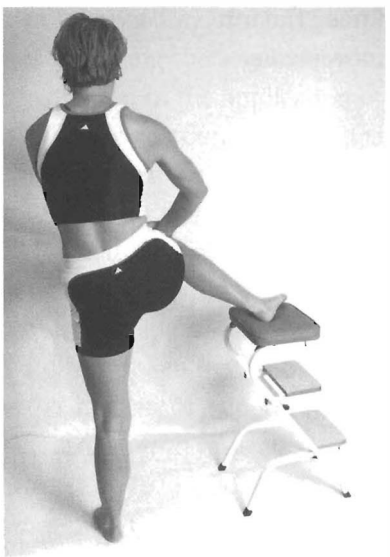
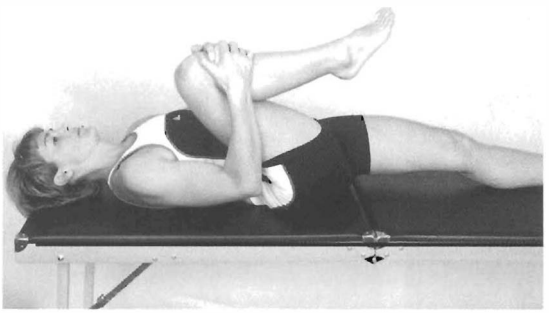
Photo 31
Internal rotation of the hip with and without overpressure to be applied ten times.



31

Photos 32, 33, 34
Different methods of applying flexion at the hip with overpressure (ten times).



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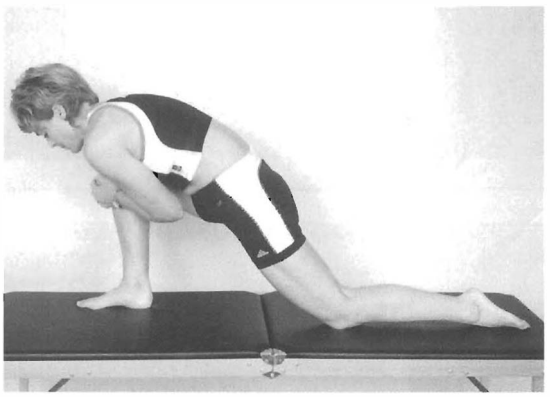

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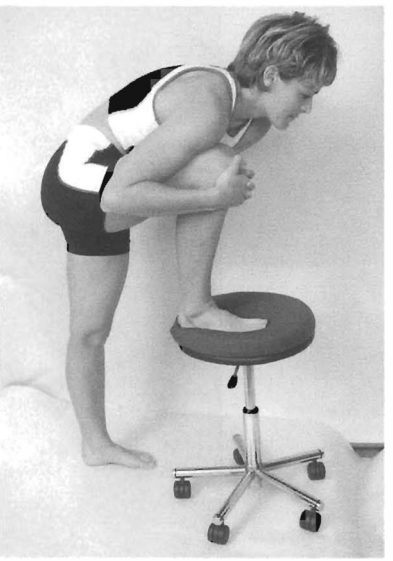

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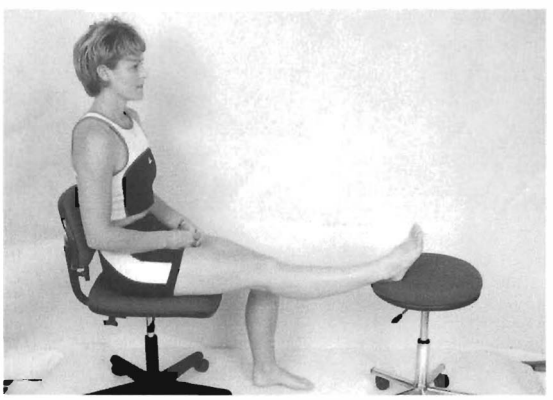
Photo 35
Sustained knee extension, most useful in treating derangement obstructing the last few degrees of extension. Hold for 20-30 seconds, relax. Repeat up to ten times.



35

Photo 36
Knee extension with or without overpressure. Pressure on, pressure off, ten times.

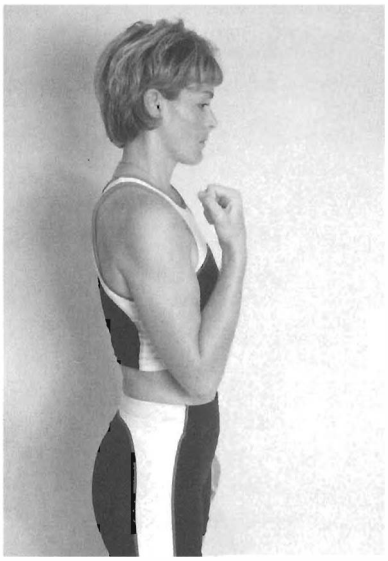
36

Photos 37, 38
Knee extension with overpressure with internal or external rotation. Pressure on, pressure off, ten times.

37

38

Photos 39, 40, 41, 42
Elbow extension with overpressure. Starting with the arm in full flexion, the patient rapidly and abruptly 'throws' the arm into end-range extension. Overpressure is achieved from the high velocity of the procedure



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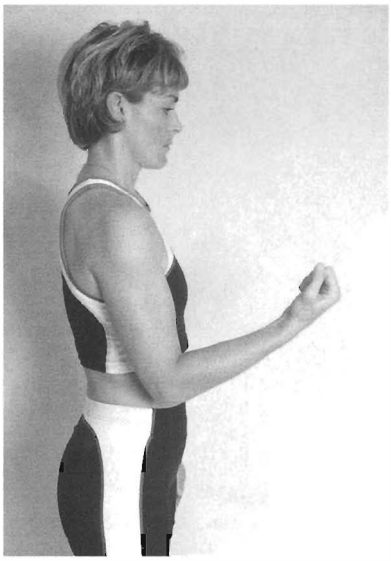

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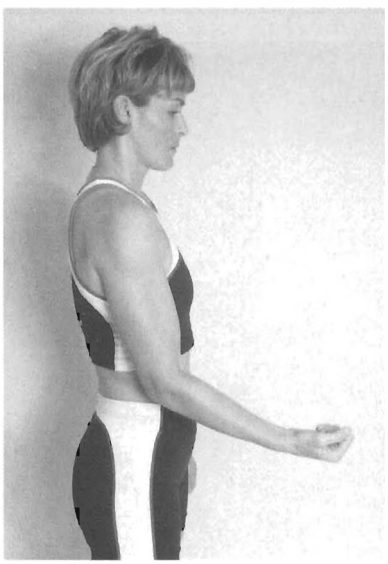

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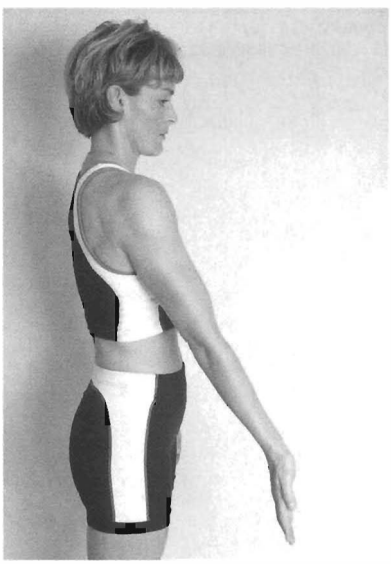
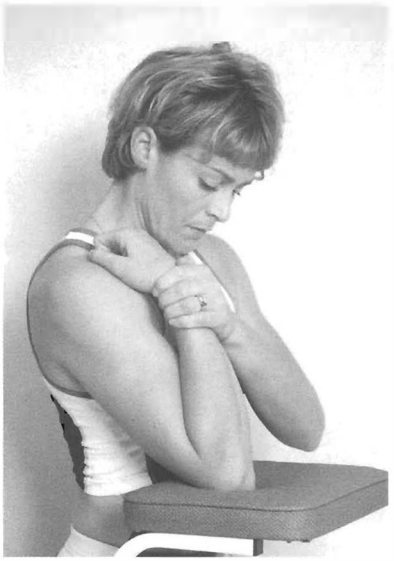


42

Photos 43, 44
Elbow flexion with overpressure with forearm supine or prone. Pressure on, pressure off, ten times.



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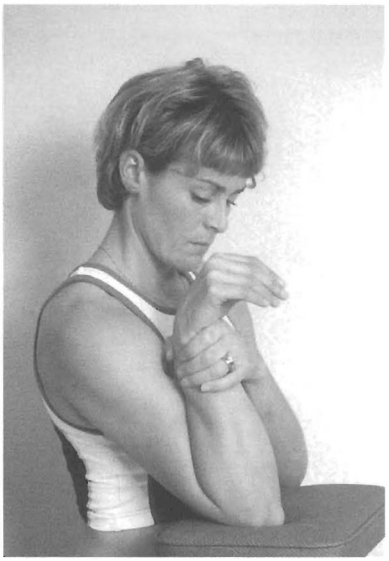
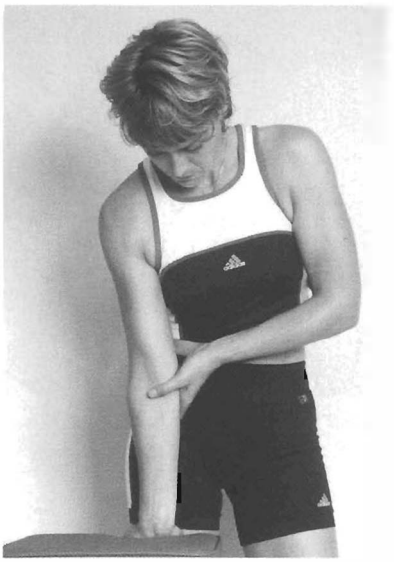


44

Photos 45, 46
Lateral overpressure in extension at the elbow. The affected arm is held in extension by the other hand and counterpressure from the table, on which the patient is leaning. The patient then pushes the elbow laterally as far as they can ten times.



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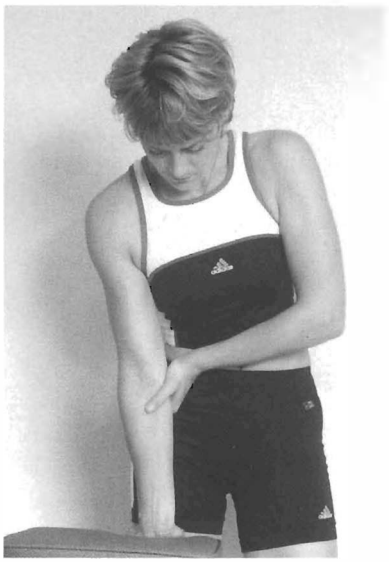
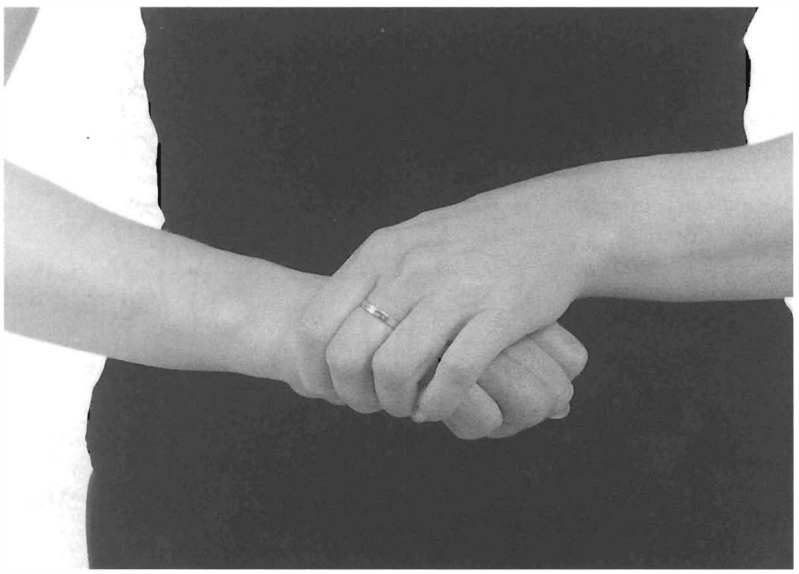


46

Photos 47, 48
Traction with dorsal/ventral gliding for derangement of the wrist. Fix the forearm against the body. Raise and lower the wrist while maintaining traction with the other hand. Lateral gliding, or translation, can be applied in the same manner.

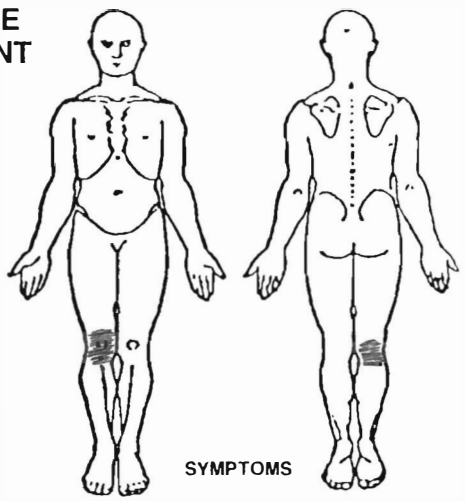


47

48

Figure 9.1 Assessment sheet – knee derangement

THE McKENZIE INSTITUTE PERIPHERAL ASSESSMENT



Date ...../...../.....

Name ..... (M) F

Address .....

Date of birth ...../...../..... Age 38

Work / Leisure Electrician - kneeling

Postures / Stresses squatting

used to play football & jog.

Functional disability from present episode

working

stopped football / jogging

HISTORY

Present symptoms Right knee - anterior & posterior

Present since ...../...../ 4 months better than it was Improving / (Unchanging) / Worsening

Commenced as a result of after squatting on a job all day or no apparent reason

Symptoms at onset same

Constant symptoms ..... Intermittent symptoms about 50% of day

What produces or worsens Running (immediately); walking (> ½ mile); squatting, kneeling, knee extension

What stops or reduces Rest; holding knee bent at about 90°

Continued use makes the pain Better (Worse) No Effect

Pain at rest Yes / No sometimes

Disturbed night Yes (No)

Other questions knee feels a bit swollen; sometimes locks, twisting knee about unlocks it; get sharp pains then ach

Treatment this episode Tablets

Previous episodes none

Previous treatment .....

Spinal history back pain - several episodes, last one a year ago Paraesthesia Yes (No)

Medications: tried NSAID Effect not helped

Present medication none

General health good

Imaging none ?

Summary: Acute / Sub-acute / (Chronic) (Trauma) / Insidious onset

Sites for physical examination knee

PERIPHERAL knee

Observation minimal swelling ; quads wasting

Active movement Loss PDM ERP

flexion - minimal (ERP)

extension - minimal (ERP)

Passive movement Loss (+/-over-pressure) PDM ERP

flexion & extension - minimal loss (ERP)

Resisted tests Response no pain

right quads weaker

Effect of repeated tests on pain: produces, abolishes, increases, decreases, radiates, localises, better, worse, no better, no worse, no effect

flexion produced worse

extension abolished better

Effect of repeated tests on movement: Better Worse No Effect

flexion (Worse)

extension (better)

Static Positioning Effects

Loaded / Unloaded

SPINE

Movement Loss

Effect of repeated movements:

Effect of static positioning:

Spine testing: (Not relevant) / Relevant / Secondary problem

CONCLUSION (Peripheral) knee Spine

Dysfunction : Articular Contractile

(Derangement) extension Postural

Other Uncertain

PLAN

Exercise Frequency

extension with over-pressure x 10 every 2 hours

Avoidance of squatting / kneeling.

Chronic pain states

As is clear from the epidemiological studies reviewed in Chapter One, persistent musculoskeletal symptoms are commonly found throughout the population and are frequently encountered in health services. The evidence also suggests that commonly used treatments often fail to provide these patients with lasting relief. Chronic pain by its very nature is resistant to treatment, but these patients deserve the optimum management strategies, even if complete relief from symptoms is not possible. Duration of symptoms should never be seen as a reason to deny patients a thorough mechanical examination. Many patients with persistent problems will be classifiable in one of the mechanical syndromes, and many can achieve resolution, improvement or better self-management of their problem relatively easily.

However, in some patients symptom responses may be contradictory, confusing or non-specific, and no clear-cut mechanical diagnosis can be made. In such instances a test period of repeated movements over a few days may help to clarify the response. Chronic pain responses and behaviour can sometimes mask and confuse an underlying mechanical disorder. Once the abnormal sensitivity has settled, a mechanical response may be found. If this fails to unearth a specific exercise approach, the patient should be classified as chronic and a general graded exercise programme be implemented. Various non-mechanical factors may be modulating chronic pain – for instance, neurophysiological changes in the peripheral and central nervous system, or beliefs about pain.

The complexity of chronic pain has been most widely explored relative to spinal problems; less work has been done which pertains directly to extremity joints. Nonetheless, it is probably fair to extrapolate certain components of this complex picture to all musculoskeletal pains. The evidence suggests that peripheral joint and muscle lesions that have an inflammatory element are quite capable of inducing both peripheral and central sensitisation. This involves the reduction of pain thresholds, an amplification of responses, prolonged post-stimulus sensations, pain response to normally innocuous mechanical stimuli, spread of heightened sensitivity to uninjured tissue and spontaneous pain (Cohen 1996; Mense 1997; Levine 1996). In other words, neurophysiological mechanisms mean that the link between the intensity of the noxious stimuli to the tissue and the pain experienced by the patient need not be correlated at all. Pain may be

the product of plastic changes in the central nervous system and not related to local tissue damage. A malfunctioning nervous system can exaggerate pain responses.

Psychological factors have also been associated with chronic pain. Specific personality traits which may predispose to persistent symptoms relate to fear–avoidance behaviour, heightened anxiety and somatic concern, low self-efficacy, belief in an external health locus of control, and passive coping strategies (Philips 1987; Ackerman and Stevens 1989; Jensen *et al.* 1991). Thus, aspects of the patient's personality can cause exaggerated and disproportionate pain behaviours. The factors that would appear to be most significant are fear–avoidance behaviour and self-efficacy.

Social and economic factors also appear to have a role in the development of pain and disability. Musculoskeletal symptoms and consequent disability have been associated with lower educational achievement and family income (Cunningham and Kelsey 1984), and symptoms of stress have predicted the occurrence of musculoskeletal disorders (Leino 1989). Most health problems inflict disproportionately on those who enjoy the least prosperous lifestyle – generally there is a linear correlation between lower social classes and increasing mortality and morbidity (Radical Statistics 1987). Musculoskeletal problems are no exception to this reality (Cunningham and Kelsey 1984). It is suggested that chronic pain be seen as a biopsychosocial phenomenon, with interdependent somatic, cognitive and environmental dimensions (Cohen 1996). A clinician's interaction with patients thus needs to embrace this complexity.

In summary, various non-mechanical factors can affect the modulation of pain. The pain science perspective stresses the neurophysiological changes that can happen in the central nervous system. The psychology perspective stresses the fear of pain, the lack of self-efficacy, and the passive coping strategies. The societal perspective stresses the environmental context such as work and family which influences that individual. It is likely that different factors, or a combination of factors, may be more important in different individuals. It is likely that in some these factors are reversible while in others they are not. Presumably in individual patients these different factors can be more or less relevant – as in so many clinical phenomena a continuum is likely to exist, with severe irreversible changes at one end, and mild reversible changes at the other. The

prevalence of the more extreme cases as a proportion of all those with persistent pain is unknown but is likely to be small.

The chronic pain population is a heterogeneous group with the majority at the less severely disabled and not irreversible end of the continuum. In this context it is worth noting that, in those with chronic spinal pain at least, 60% of this group define their pain as mild and 30% as moderate (Magni *et al.* 1990). In those with severe and persistent pain, nearly half have no activity limitation as a result (von Korff *et al.* 1990). After two years, 13 – 36% of those who had had chronic pain no longer reported pain as a problem, while for the rest pain had become less frequent and caused less distress (Crook *et al.* 1989). It is clear from this epidemiological data that patients with chronic pain are a heterogeneous group. It is likely that they will present with a variety of mechanical and non-mechanical factors affecting their pain, and varying degrees of severity and disability. It is clear that individuals may leave the chronic pain pool as well as enter it. The clinical implications of this are that patients with persistent pain may respond easily to mechanical therapy, or they may respond only partially, or some may not respond at all. Improved self-management strategies for their problem may benefit all patients with chronic problems.

Management

Often persistent symptoms will respond positively to an exercise programme. Sometimes the response will be slower, with an initial aggravation, and often the outcome is improved management rather than resolution of symptoms. A trial of progressive, graded exposure to an exercise and activity programme should be encouraged.

Management of chronic pain is a complex field beyond the scope of this text. However, as it so commonly encountered, some guidelines for a general approach will be given. These will be adapted from experience gained in managing those with chronic spinal pain. For chronic musculoskeletal problems it is recommended that a cognitive–behavioural framework is used for interaction with the patient (Turner 1996). This requires:

- awareness of and enquiries into psychological 'yellow flags' that suggest inappropriate pain behaviours and beliefs about pain, and that can be risk factors for the development of persistent pain

- appropriate information provision – the importance of the self-management principle for ongoing health problems, activity for musculoskeletal conditions, and reassurance that pain on movement does not mean an exacerbation of the problem
- encouragement of a graduated, systematic resumption of activities

Common features of successful programmes for back problems have been identified (Linton 1998). These might easily be applied to all musculoskeletal conditions:

- multidimensional view of the problem, including psychosocial aspects
- a thorough 'low tech' examination
- communication of the findings to the patient, and an explanation of why it hurts and how to best manage it
- emphasis on self-care and explanation that the way the patient behaves is integral to the recovery process
- reduce any unfounded fears or anxiety about the pain ('hurt does not mean harm')
- clear recommendations about starting normal activities and a graded approach to exercises
- do not medicalise the problem; avoid 'high tech' investigations, long-term sick leave, and advising the patient to 'take it easy'.

Therapist techniques and passive modalities

This text is primarily concerned with endorsing a self-management approach to peripheral musculoskeletal problems. This is logical in the light of the epidemiology, the poor efficacy of commonly used treatment, and the importance, physiologically and psychologically, of the patient taking control of their treatment. This book does not prescribe a list of clinician techniques; many such books already exist. Most techniques described and pictured will be ones that the patient themselves can use; few books have previously concentrated on this aspect of management

Clinician procedures

If no improvement follows from the application of the procedures recommended for the patient, it may be that techniques of frictional massage, mobilisation or manipulation applied to the affected structures could assist recovery. However, in line with the philosophy behind this text, it is always recommended that hands-on techniques should not be employed as a first option, always as a last option. The use of clinician techniques before the patient has been able to explore their own ability to promote recovery only serves to underline the power of the clinician and the dependence of the patient. Furthermore, there is no guarantee that hands-on techniques will necessarily result in a better outcome. If that option is chosen, the procedures described by those with long experience in the diagnosis and treatment of mechanical musculoskeletal disorders should be considered. It is not the purpose of this book to provide a description of those manual techniques as they are already well documented. See, for example, Cyriax and Cyriax (1997); Maitland (1991); Laslett (1996); and Kesson and Atkins (1998).

Passive modalities

Physiotherapy students spend many hours learning about the theory and practice of various electrotherapy modalities. Although their value is negligible, some electrotherapy interventions are still widely used. Ultrasound, for instance, is said to be used in about 10% of ankle and knee disorders (van Baar *et al.* 1998a; Dekker *et al.* 1993; Jette and Delitto 1997; Roebroeck *et al.* 1998a). However, these studies may seriously under-report daily physiotherapy practice. Surveys from the UK, Australia and Canada show that ultrasound is available to virtually all responding departments and practices and is used on a daily basis in over 90% of them (Pope *et al.* 1995; Robertson and Spurritt 1998; Lindsay *et al.* 1990, 1995). It would appear that ultrasound is the most widely available and most commonly used electrophysical agent internationally (Robertson and Spurritt 1998).

Because it appears to be the most widely used electrotherapy modality among physiotherapists, it is useful to examine ultrasound as an example of passive therapies. The proponents of ultrasound advocate its use to relieve inflammation, reduce oedema, relieve pain, accelerate tissue repair, increase circulation and modify scar formation (Maxwell 1992). It is used in a wide range of musculoskeletal conditions (ter

Haar 1988). Logically, such a tool should be used primarily during the acute phase of any musculoskeletal disorder, whereas in fact many clinicians carry on using it beyond the first few weeks (van Baar *et al.* 1998; Jette and Delitto 1997; Roebroeck *et al.* 1998a, 1998b).

While it is thought that ultrasound has an anti-inflammatory effect and enhances tissue repair following soft tissue damage, its actual effect is largely unknown (Maxwell 1992). Those investigations that have been done are mostly experimental, frequently using animal models rather than clinical studies. Clinical studies that have been done so far are generally of very poor quality (Kitchen and Partridge 1990; Gam and Johannsen 1995; van der Windt *et al.* 1999; Holmes and Rudland 1991). The experimental work is generally contradictory (Kitchen and Partridge 1990; Maxwell 1992), and the evidence that ultrasound has some anti-inflammatory effect is lacking. In an animal experiment ultrasound has been shown to have no anti-inflammatory effect (Goddard *et al.* 1983). In patients the anti-inflammatory activity has been attributed to the placebo effect as the results were similar in the real and sham ultrasound groups (Nyanzi *et al.* 1999; Hashish *et al.* 1986) and found to be so small as to lack clinical relevance (Snow and Johnson 1988).

In the treatment of specific conditions the results of ultrasound therapy have been equally disappointing. A very poor success rate has been found when using the modality for lateral epicondylitis (Stratford *et al.* 1989), and in the treatment of acute lateral ligament sprains ultrasound was no better than placebo (Nyanzi *et al.* 1999). In a review of treatments for lateral epicondylitis, it was concluded that although ultrasound may have a therapeutic effect it was unclear if this was any better than placebo (Labelle *et al.* 1992). In a systematic review of treatments for shoulder disorders it was concluded that there is evidence that ultrasound is *ineffective* for these conditions (van der Heijden *et al.* 1997). A meta-analysis performed to evaluate the pain-relieving value of ultrasound concluded that it had an unimportant analgesic effect across a variety of disorders (Gam and Johannsen 1995). Another meta-analysis concluded, *"there seems to be little evidence to support the use of ultrasound therapy in the treatment of musculoskeletal disorders. The large majority of 13 randomised placebo-controlled trials with adequate methods did not support the existence of clinically important or statistically significant differences in favour of ultrasound therapy"* (van der Windt *et al.*

1999). The fact that ultrasound is widely available and regularly used despite an absence of supporting clinical research is testimony to the optimistic claims of the manufacturers, who obviously benefit from its availability. The naïve acceptance of these claims by the physiotherapy profession is augmented by the placebo effect (Nyanzi *et al.* 1999; Hashish *et al.* 1986), which gives the comforting appearance of efficacy; this explains clinicians' empirical 'feeling' that it works.

The evidence that is available not only seriously questions the worth of this intervention, but also raises the possibility that it may be doing more harm than good (Maxwell 1992; Kitchen and Partridge 1990). Certain studies, for instance, suggest that ultrasound may cause vasoconstriction rather than increase blood supply; increase histamine release and consequently produce more inflammatory exudate; lower the effectiveness of macrophages in fighting infection; and also produce excessive amounts of collagen and scar tissue (Maxwell 1992). *"The dosage should be considered carefully in the light of existing evidence; ultrasound can be damaging at high intensities within the 'therapeutic' range"* (Kitchen and Partridge 1990).

The empirical proposal that ultrasound has an anti-inflammatory and pain-relieving effect is clearly not supported by the available evidence. Furthermore, in the acute phase incorrect usage may actually be making the patient worse. Reliance upon such passive modalities ignores the physiological importance of movement in musculoskeletal disorders. When used as part of a 'shotgun' approach to these problems it implicitly downplays the importance of patient involvement in treatment and is still of little value. There seems to be evidence that ultrasound is not effective as an adjunct to exercise (van der Windt *et al.* 1999; Holmes and Rudland 1991). There is no reason to believe that the combined effects of two or more treatments will produce a summation of the individual therapies (Holmes and Rudland 1991). The more therapy that is given the greater the placebo effect, but there is no evidence that ultrasound produces any change that outlasts the giving of therapy (Feine and Lund 1997).

The psychological ramifications of such an approach may be to promote iatrogenic disability. As with all electrical modalities, the treatment is predicated on the role of the passive patient receiving therapy from the dispensing clinician. Such a passive, uninvolved role for the patient contradicts the actively involved role of the patient that is the optimum course of management for musculoskeletal conditions. It has been suggested that in the field of back pain such passive management strategies have helped promote iatrogenic disability (Waddell 1998).

10: Common Disorders

Introduction

As has been discussed earlier in the book, the issue of diagnosis presents problems about reliability and validity. It is often less relevant to management than the stage of the disorder and the patient's response to their painful condition. However, some specific musculoskeletal conditions are relatively simple to identify and are commonly seen. The application of mechanical diagnosis and therapy can be most simply exemplified by discussion of some recognised disorders. Other patients present with non-specific disorders to which it is difficult to apply a clear-cut diagnostic label. Lack of a diagnosis, however, does not mean an inability to treat. Categorising patients' conditions by the mechanical syndromes outlined earlier and with knowledge of connective tissue responses to injury and stress deprivation, the appropriate management strategies should be apparent from the history-taking, physical examination and symptom responses

Generally in the first week following injury relative rest is briefly needed to prevent disruption of the healing tissue, but from that time on most non-inflammatory musculoskeletal conditions need progressive movement and loading. The quality of connective tissues decline with lack of use and reach optimum function with incremental usage. Painful tissues become sensitised and deconditioned when not used so that normal movement becomes more painful and the area around the injury becomes tender to the touch. The individual is discouraged from using the limb normally, and develops avoidance behaviour. By introducing patients to gradual progressive loading strategies and an active, patient-centred management approach, clinicians offer patients a way to improve tissue health and function, and allow them some understanding and control of their problem so that it is no longer of such concern. With some conditions the loading needed is very specific to stress the tissues in a specific way, but with other conditions the important thing is simply to get the patients moving and using the limb.

Clinicians may find that patients often do not present with an easily recognisable specific condition. Signs and symptoms may be vague and non-specific, and a diagnosis cannot be reached. This does not

mean that the clinician is unable to institute treatment. As long as attention is paid to the stage of the disorder and the symptomatic response to repeated movements, it should be perfectly feasible to commence an exercise programme. This will be based upon the patient's responses to repeated active, active assisted, active plus overpressure or resisted exercises. Frequently the patient's response to repeated movements will in fact provide more useful information than a diagnosis. A diagnosis does not, for instance, distinguish between the inflammatory and dysfunctional stage of soft tissue conditions.

This chapter contains descriptions of some specific musculoskeletal conditions that are commonly seen in physiotherapy departments. The natural history and appropriate patient management strategies are also described. The amount of detail relating to each condition is in part a reflection of literature that is available, which is frequently limited in its scope. These conditions are intended to exemplify how mechanical diagnosis and therapy can be applied to problems in peripheral joints. **Mechanical diagnosis and therapy have a role in all musculoskeletal conditions.** This chapter is not a complete account of all instances when this approach may be used. It seeks to give concrete examples of how to apply the mechanical evaluation and treatment strategies outlined earlier in the book to certain specific conditions. It is hoped that interested clinicians, applying sound clinical reasoning skills, will then be able to apply the same approach to other peripheral musculoskeletal problems.

The spine and peripheral joint problems

It is very common for lumbar or cervical spine problems to refer pain into the limbs. This may occur with or without nerve root involvement, as somatic spinal structures are implicated in diffuse radiating pain, sometimes as far as the foot or hand. Such symptoms can occur with very little local spinal pain. Usually such problems are clearly related to the spine and there is no cause of confusion between spinal and non-spinal disorders. In extremity joint problems the pain is usually localised around the involved structure, sometimes with distal referral as well. For instance, shoulder problems can refer as far as the hand, lateral epicondylitis can refer down the forearm, and osteoarthritis of the hip can be felt in the groin, anterior thigh and even medial shin. Hip osteoarthritis can also present as localised knee pain with no groin or buttock pain. Again, in these instances

the differential diagnosis between spinal and extremity joints is relatively straightforward.

Any peripheral joint pain that is associated with proximal pain, especially in the scapular, top of the shoulder or buttock areas, strongly suggests that there is a spinal component to the problem. The presence of numbness or paraesthesia is a further clue to the problem originating in the spine. When symptoms are felt in the extremity but are not affected by mechanical testing of the local joints and muscles, again it is important to test the spine. **Given the propensity of spinal structures to cause referral of symptoms into the limbs, it is paramount always to discount this as a source of pain before focusing on the extremity.** Relative to the lower limb, a McKenzie lumbar examination procedure should be carried out (McKenzie 1981).

With all shoulder problems it is wise to first exclude involvement of the cervical spine before commencing treatment at the shoulder. Cervical problems can mimic shoulder lesions to the extent of painful and even restricted shoulder movements (Wells 1982; Schneider 1989). Involvement of the spine should always first be discounted with the use of a McKenzie cervical examination procedure (McKenzie 1990). Sometimes it may be helpful to test range of movement, or pain during movement at the shoulder both before and after repeated cervical movements; if the shoulder movements improve, treatment should start at the spine. If neck movements have no effect and the primary movement dysfunction is at the shoulder, assessment and treatment should concentrate there. Concomitant neck pain is also very common in those with separate shoulder problems, and is a high risk factor for persistent or recurrent shoulder problems (van der Windt *et al.* 1996). In such cases it is generally better to start treatment at the neck, although both problems should be addressed. It is important to start with the problem that is of prime concern to the patient.

Rotator cuff tendonitis

Pathology of rotator cuff muscles would appear to be the most common cause of shoulder problems – different studies cite this as the cause of symptoms in 30 – 75% of their sample (Chakravarty and Webley 1990; Chard and Hazleman 1987; Chard *et al.* 1991; van der Windt *et al.* 1995; Vecchio *et al.* 1995). The supraspinatus tendon is most frequently affected (Cofield 1985; Fukuda *et al.* 1994;

Chard *et al.* 1988; Herberts *et al.* 1981). The pathology may involve micro-failure, acute or chronic inflammation, fibrosis, degenerative changes, partial or complete rupture of the tendon, and impingement of the tendon under the acromion (Cofield 1985; Blevins *et al.* 1997; Herberts *et al.* 1981). Because of the last factor pain may be produced during abduction of the arm in a specific arc of the movement. Because of the variety of pathological changes that may be found, the condition is best viewed as a continuum in which the state of the tissues differs in individual patients. The condition has several synonyms: rotator cuff disease, supraspinatus tendonitis, painful arc or impingement syndrome, etc.

Patients may present with a constant or intermittent ache depending on the stage of the condition. Generally range of movement at the shoulder is preserved and there is frequently a painful arc on abduction. In cases of complete rupture active movement is lost, but passive movement testing reveals a full range of movement. Resisted movements are the key tests. With supraspinatus tendonitis, resisted abduction will be weak and will reproduce the patient's pain; there may also be some pain on resisted lateral rotation. Resisted lateral or medial rotation is painful if infraspinatus or subscapularis are involved. The average age of patients is over 50, they come from manual and non-manual occupations, and they may be retired or classified as housewives. Onset is rarely due to trauma, but related to overuse at work or domestically, and is frequently of unknown cause (Chard *et al.* 1988).

Four elements are commonly said to be involved in the pathogenesis of rotator cuff tendonitis; these are either intrinsic to the tissues or extrinsic (Cofield 1985; Blevins *et al.* 1997; Neviaser and Neviaser 1990; Herberts *et al.* 1984; Neer 1983; Uhthoff and Sarkar 1990; Fu *et al.* 1991; Bjelle 1981; Fukuda 1994; Chard *et al.* 1994; Uhthoff and Sano 1997; Soslowsky *et al.* 1997). The consensus seems to be emerging that the pathogenesis is multifactorial, and thus that the relative contributions of each will vary between individuals. These causes are:

- vascular
- degenerative
- mechanical stress
- anatomy.

The area of the tendon that is frequently affected is a site of reduced vascularity. This appears to accelerate degenerative changes and leaves the tissue vulnerable to further pathology. The relative avascularity also means that healing and repair processes are impeded. The tendons degenerate with age, with the incidence of cuff tears increasing markedly in those over 40. The changes seen include cellular distortion and necrosis, micro-failure and tears of the tendons, calcium deposition, fibrinoid thickening and disruption of the site of insertion into the bone.

The anatomical factor that is of most significance is the impingement of the rotator cuff caused by certain structures. 'Hooked' acromions are associated strongly with rotator cuff tears. Spurs and osteophytes from the acromion or the acromioclavicular joint can exaggerate impingement of the tendon. The circulation to the tendon may be impaired by compression between the humeral head and the coracoacromial arch. Due to the close juxtaposition of all the tendons of the rotator cuff, the capsule, the sub-acromial bursa, and the coracohumeral and glenohumeral ligaments lesions of the supraspinatus tendon may easily involve and be complicated by one of these other structures (Clark and Harryman 1992; Fukuda *et al.* 1994).

Mechanical factors generally relate to overuse and static arm positions, with the compressive effect of the humeral head aggravated by raised arm working conditions, especially overhead. Rotator cuff tendonitis in occupational groups who work with their arms at shoulder level showed an odds ratio of 11 (Hagberg and Wegman 1987). The prevalence in welders has been found to be 18% compared to 2% in clerical workers (Herberts *et al.* 1981). Repetitive micro-trauma is thought to be a prime cause of degeneration in the tendons, with localised failures leading to reparative, inflammatory or degenerative changes. Although fibroblastic activity and vascular granulation-like tissue have been observed, this reparative process has never been seen to be successful in actually restoring the normal structure of tendon tears (Chard 1994). It is likely that a combination of factors trigger symptoms in different patients, but a common denominator would appear to be the role played by degeneration, leaving the tendons susceptible to pathology.

The condition can pass through various stages, starting with an inflammatory reaction to overuse-induced tissue trauma. Fibrosis and

thickening of the tendon and adjacent bursa may follow. At later stages inflammatory cells are rarely seen, and the normal healing process appears to be aborted. Chronic tendonitis is thought to result from a failure to complete the reparative process so the tissue is never remodelled for normal use. Histological changes related to hypoxia are characteristic of chronic degenerative tendonopathy (Leadbetter 1992). Further degeneration, impingement wear and cumulative ischaemic trauma can finally produce complete tears of the rotator cuff (Neer 1983; Blevins *et al.* 1997; Fu *et al.* 1991; Gross 1992). At different stages of the continuum of rotator cuff pathology the tissues may be inflamed, healing, abnormally fibrotic, chronically degenerated or ruptured. The pain-generating mechanism may be chemical or mechanical, or both, due to abnormal tissue – the impingement effect may also add an ischaemic element.

Natural history and clinical course of rotator cuff tendonitis

Various studies have identified patients with rotator cuff tendonitis and then monitored their symptoms over the subsequent year or over several years. These patients have often received standard treatments such as steroid injections, physiotherapy or NSAIDs. The long-term outcome of many of these patients is not good. At one year about 50% of such patients are still reporting problems (van der Windt *et al.* 1996; Chard *et al.* 1988); in a population of over 70s some 70% were still symptomatic at three years (Vecchio *et al.* 1995). Having treatment seems to confer no long-term advantage. Frequently individuals will have such problems but not seek help, accepting their symptoms and disability as a part of the ageing process (Chakravarty 1990; Chard and Hazleman 1987; Chard *et al.* 1991). One systematic review of interventions for rotator cuff disease found that there is no clear advantage for corticosteroid injections over placebos (van der Heijden *et al.* 1996), while in another the only positive finding was that a subacromial steroid injection was better than placebo in improving the range of abduction (Green 1998). Steroid injections have been found to be better (Adebajo *et al.* 1990) and no better than NSAIDs (White *et al.* 1986). It is not clearly proven that steroid injections aid recovery.

It would appear from these studies that the natural history of rotator cuff tendonitis is frequently one of persistent and lasting symptoms, and that standard treatments frequently do not make the outlook much better. This rather pessimistic picture accords with the role that degeneration has in producing and maintaining the condition.

Management and self-treatment exercises

If seen during the inflammatory period, the patient is encouraged to restrict usage of the arm, especially any overhead or aggravating activities. Elevation by flexion is less likely to cause impingement than abduction, which is best avoided. Then from early on progressive loading of the involved tissues is needed to promote a strong tendon that can withstand the stresses of daily work, recreational and sporting activities (Gross 1992).

Active movements through part of the abduction range, in the case of a supraspinatus problem, can be used to load the tendon initially. As improvement occurs the range can be increased. Isometric resisted abduction exercises can also be used early. These should reproduce the patient's symptoms, which fade within a few minutes of stopping. Exercises should be repeated about ten times three or four times during the day, increased to every two hours if the response is all right. These can be done pushing against a wall. To progress the exercises resistance can be offered at different points in the range, moving towards the target zone. As long as these are not aggravating the symptoms, movement remains full and the painful arc becomes easier, these can be progressed onto concentric and eccentric exercises. Dynamic exercises can be done in the target zone, where one is found, or throughout the range of movement when no target zone is found.

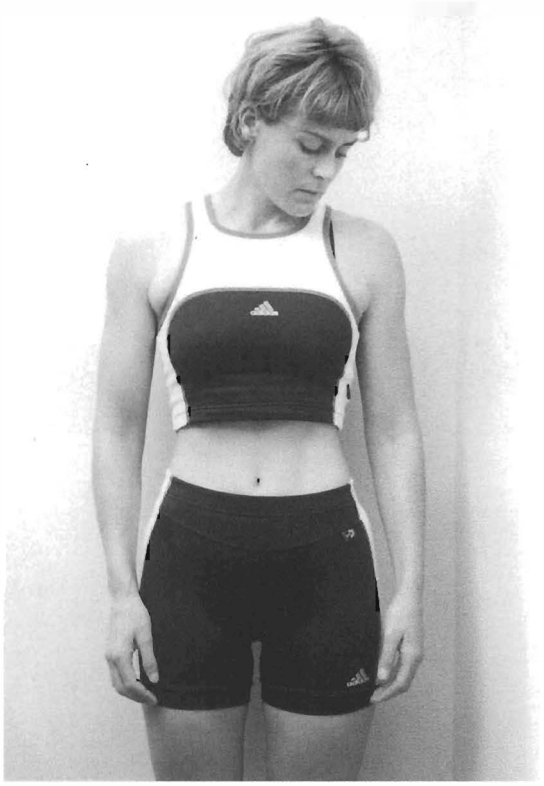
If the patient is seen with chronic symptoms, progressive exercises are again very important to provide the stimulus for collagen fibril realignment and maturation, and reconditioning of sensitised tissue. Dynamic and static abduction exercises could be used. An adequate range of lateral rotation is also important to reduce impingement; if this is limited, the humerus impinges much earlier upon the coraco-acromial ligament (Caillet 1981b). The role of the rotator cuff as a humeral head stabiliser should be remembered, as well as postural components such as thoracic and shoulder girdle mobility (Dalton 1994).

The value of eccentric exercises for chronic tendon problems is their ability to facilitate maturation of a strong tendon and reduce the risk of injury recurrence (Gross 1992; Fyfe and Stanish 1992). Given the greater loading that these exercises generate, they should only be considered during the terminal stages of rehabilitation. Again, these

may be temporarily painful, but this should quickly abate on stopping the exercises.

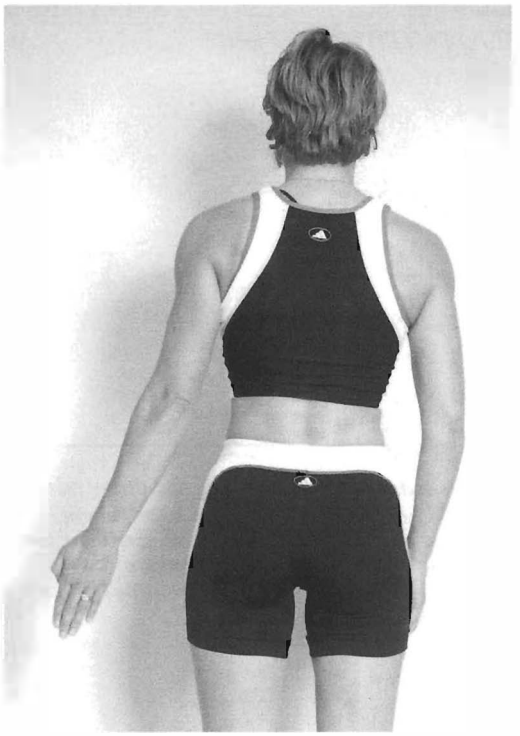
If needed, dynamic or static exercises can be used to stimulate tissue in which the healing process is suspended. The patient will have had the problem some time, remaining relatively stable. Once the exercises achieve a temporary aggravation of symptoms, a less vigorous loading strategy is adopted.

Photos 49
Isometric abduction of the shoulder. The patient stands with the painful side near a wall, with feet apart and the affected arm relaxed by their side.



Photos 50
Isometric abduction of the shoulder. Stabilising their body so they do not lean into the wall, the patient abducts the arm into the wall, generating tension, but no movement, in the supraspinatus tendon. This position is held briefly and then relaxed. The isometric exercise is repeated up to ten times.

Photos 51, 52, 53, 54
Finding the 'target zone' in a rotator cuff problem. The patient exercises in mid-range through the arc of greatest pain up to ten times. If the pain is too severe, exercise short of the target zone. A similar plan for all movements at other joints should be used.



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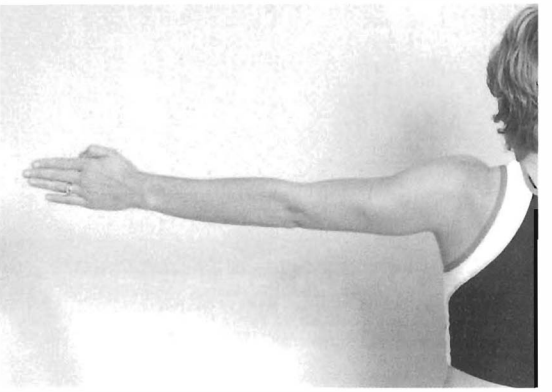

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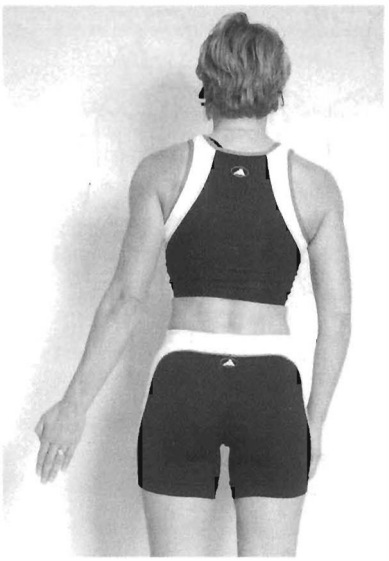
Photos 55, 56, 57, 58
No 'target zone' - pain on abduction is similar at all points. Exercise through full range up to ten times and use a similar plan for all movements at other joints.



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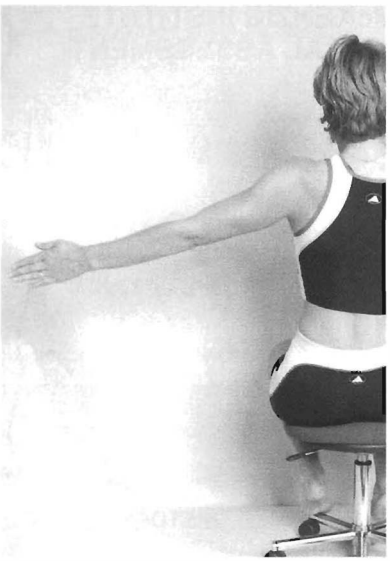

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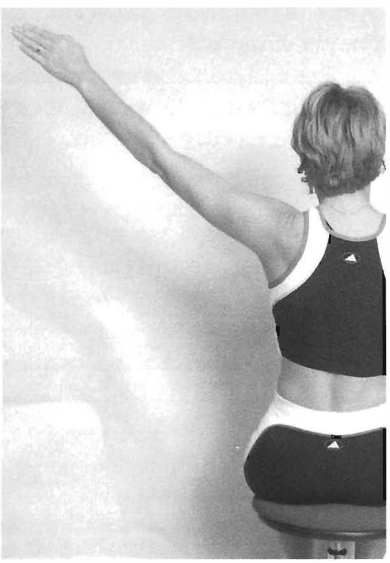

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Figure 10.1 Assessment sheet – rotator cuff suprinatus dysfunction

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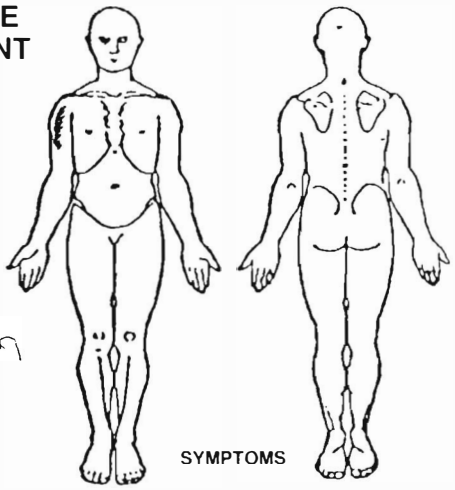


Date ... / ... / ...
Name ... (M) F
Address ...
Date of birth ... / ... / ... Age 36
Work / Leisure ... welder
Postures / Stresses ... arms raised, lifting
Functional disability from present episode ... nil, at work

HISTORY

Present symptoms ... right shoulder
Present since ... / ... / ... one year Improving (Unchanging) Worsening
Commenced as a result of ... fell onto shoulder or no apparent reason
Symptoms at onset ... right shoulder
Constant symptoms ... Intermittent symptoms ... yes
What produces or worsens ... lying on it, hand behind back, abduction, elevation not worse
What stops or reduces ... gentle use
Continued use makes the pain (Better) Worse No Effect
Pain at rest Yes (No)
Disturbed night Yes (No) ... if he avoids lying on right
Other questions ...
Treatment this episode ... Tablets
Previous episodes ... none
Previous treatment ...
Spinal history ... none
... Paraesthesia Yes / No
Medications: tried ... NSAID Effect ... no effect
Present medication ... nil
General health ... good
Imaging ... X-ray
Summary: Acute / Sub-acute (Chronic) (Trauma) Insidious onset
Sites for physical examination ... shoulder

PERIPHERAL Right shoulder

Observation N.A.D.

Active movement Loss (PDM) ERP

flexion - nil at 90°

abduction - nil at 90°

hands behind back - nil

Passive movement Loss PDM ERP
(+/-over-pressure)

lateral rotation - nil

gleno-humeral abduction - nil

Resisted tests Response abduction - pain

lateral rotation - pain

rest - NAD

Effect of repeated tests on pain: produces, abolishes, increases, decreases, radiates, localises, better, worse, no better, no worse, no effect

resisted abduction produces no worse

flexion produces no worse

Effect of repeated tests on movement: Better Worse (No Effect)

Static Positioning Effects

Loaded / Unloaded

SPINE

Movement Loss

Effect of repeated movements:

Effect of static positioning:

Spine testing: (Not relevant) / Relevant / Secondary problem

CONCLUSION (Peripheral) Spine

(Dysfunction) : Articular (Contractile) supraspinatus

Derangement Postural

Other Uncertain

PLAN

Exercise	Frequency
flexion	x 10 x 3/4 daily
resisted abduction	x 10 x 3/4 daily

Capsulitis of the gleno-humeral joint ('frozen shoulder')

'Frozen shoulder', adhesive capsulitis and periarthritis are some of the many synonyms used to describe a condition that is frequently seen by physiotherapists. It may account for about a fifth of patients with shoulder problems who visit doctors and physiotherapists (van der Windt *et al.* 1995; Liesdek *et al.* 1997; Chard and Hazleman 1987; Chard *et al.* 1991; Chakravarty and Webley 1990; Herberts *et al.* 1981; Vecchio 1995). It has been the subject of several reviews (Rizk and Pinals 1982; Owens-Burkhart 1987; Wadsworth 1986; Stam 1994). Although there are certain aspects of the condition that still generate debate, clinical features make it relatively easy to recognise after a certain period of time. Involvement of the other shoulder is not at all unusual (Shaffer *et al.* 1992; Rizk and Pinals 1982).

Generally it has a spontaneous onset affecting people over 40, sometimes following an injury such as a fall onto the shoulder – in these cases pain is felt immediately and the stiffening progresses rapidly. It is said to go through three stages: painful / 'freezing', stiff / 'frozen', and recovery / 'thawing'; these stages often extend over several years (Reeves 1975; Rizk and Pinals 1982). The actual presentation and pathology will depend at which stage the patient is reviewed.

At its most severe, the patient complains of continuous aching in the shoulder with radiating pain spreading down the arm as far as the wrist. The aching is felt even at rest, especially at night; patients are unable to lie on the painful side when it wakes them. All movements cause a significant increase in pain, and activities of daily living are seriously impaired. Patients frequently describe difficulties with dressing, doing their hair and reaching behind their back. There is usually a gross loss of all movements with flexion, abduction, internal and external rotation being the most noticed, but the exact pattern of loss varies between individuals. The key pathognomonic finding in capsulitis is that the movement loss is both passive and active (van der Windt *et al.* 1995; Croft *et al.* 1996; Jacobs *et al.* 1991; Dacre *et al.* 1989; Bulgen *et al.* 1984; Rizk *et al.* 1991; Ekelund and Rydell 1992).

Natural history

The first and most painful stage may last from a couple of months to eight or nine months, at the end of which the shoulder will be maximally contracted (Reeves 1975). Arthroscopic and surgical studies suggest that there is an early inflammatory process commencing in the capsule or rotator cuff, which as it abates is replaced by adhesions and contractures (Neviaser and Neviaser 1987; Fareed and Gallivan 1989; Stam 1994). This accounts for the varied clinical presentations and pathological findings. The condition should be viewed as a continuum in which initial chemical pain is replaced by mechanical pain as the problem changes from intense pain to pain and stiffness.

The stiff period, with pain now present when any movement is taken to its restricted end-range, can last from four months to a year; the recovery of movement (the 'thawing' stage) has lasted more than two years (Reeves 1975). The mean duration of symptoms from onset to recovery has been shown to be thirty months, considerably more than what is often optimistically quoted (Reeves 1975). However, the total duration varied from one year to three and a half years, and even at this point over 50% of the sample still displayed persisting loss of some movements (Reeves 1975). The natural history is thus basically self-limiting, but in some cases can run an extremely protracted length of time. Nearly 50% of patients who have been followed over many years report some symptoms and demonstrate residual restricted mobility, although few rate their problem as serious at this stage (Shaffer *et al.* 1992; Binder *et al.* 1984; Reeves 1975).

Management

Active treatment of patients with frozen shoulder prior to the cessation of aching at rest is unwise. The condition at this time is primarily inflammatory and exercises or mobilisation of almost any type will aggravate symptoms and may delay recovery. Attempting active therapy too early can easily cause the patient unnecessary increased pain on movement and increased aching at rest which may last for hours or even days. More gentle mobilisations are equally futile and achieve nothing. All but painless movement of the shoulder should be avoided; sometimes pendulum exercises reduce the intensity of the ache.

If the patient is seen during the inflammatory stage, appropriate NSAIDs or injections are generally deemed to be the treatment of choice. However, although frequently recommended anecdotally and

common sense would suggest that this is appropriate treatment, their effects are less clear in the literature. Some studies have found a useful role for intra-articular steroid injections in increasing movement and reducing pain (Jacobs *et al.* 1991), while others have found that they give benefit in the early stages of the condition only, with no long-term advantage over no treatment at all (Bulgen *et al.* 1984; Binder *et al.* 1984). Systematic reviews have found the overall evidence across multiple studies for injections for capsulitis to be unclear (Green *et al.* 1998; van der Heijden *et al.* 1996). The studies do, however, have various methodological problems in their construction, including lack of uniformity in definition of shoulder disorders, heterogeneity of interventions, and time of application – all relevant in order to make an informed judgement about the efficacy of an intervention.

The role of NSAIDs in the treatment of shoulder complaints in general has been addressed by one systematic review (van der Windt *et al.* 1994). The three trials with the best methods score reported superior short-term efficacy compared to placebo. In those trials where the indication for use was periarthritis, NSAIDs were generally found to be less effective than a reference treatment or conferred no significant benefits. The authors comment that in view of the adverse reactions to NSAIDs reported by 8 – 76% of patients, future studies should investigate the benefit–risk ratios of NSAIDs and analgesics. In other words, as anti-inflammatories appear to confer, at best, only short-term gain, but carry with them risk of gastrointestinal adverse reactions, perhaps ordinary painkillers should be the first treatment of choice.

Self-treatment exercises

Well into the course of the disorder, and provided all aching at rest has subsided, a mechanical assessment can be considered. The patient should first receive a full explanation regarding the nature of the problem, the natural history and reassurance *that the condition will improve even without treatment*. The choices open to the patient should be clearly made. The patient may do nothing, in which case they can expect the pain and limited movement to eventually but slowly recover over time. Alternatively, he or she may choose to take a more active approach and commence exercises, which at this stage is usually possible without risking aggravation. This option is usually taken if restricted and painful movement is persisting with little improvement. The patient should be given a structured exercise programme to follow.

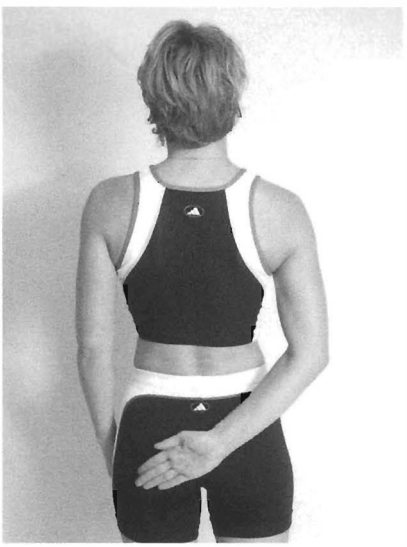
It is worth noting that there is no advantage in passive mobilisations over and above exercises alone. Several trials have looked at the effect of passive mobilisations applied two or three times weekly for four weeks or more compared to regular exercises and other interventions, and no significant differences were found. In one trial the mobilised group actually had greater restriction in range of movement at long-term review (Bulgen *et al.* 1984; Binder *et al.* 1984). Similarly, Nicholson (1985) found that whether the patients exercised only or received passive mobilisation as well, the movement increased and the pain decreased, but with no clinically important differences between the two treatment groups.

The pain behaviour, which is characteristic of dysfunction, should be consistently evoked – pain is produced on each movement, but is not worse afterwards and goes shortly after the movement is stopped. An increase of pain following the stretching sometimes lasts for up to half an hour, but if increased pain lasts for much longer, the frequency of the exercise, the number of movements or the degree of stretching should be reduced. Patients must always be given a clear explanation of the pain response expected and warned to await re-assessment if there is a serious aggravation of their condition. All exercises should be repeated ten to twelve times in three or four sessions daily for a few days. Once the response is clear and acceptable, the frequency can be increased so that they are done every two hours.

The exercises given to the patient will depend upon their particular movement loss. Self-assisted flexion is usually the easiest movement to start with when the joint is severely restricted; this can be augmented by self-assisted extension. As movements get easier or if the restriction is less severe initially, then the loss of rotation should be worked on. Movements behind the back and head, which involve medial and lateral rotation, are frequently the most severely affected and the last movements to recover. It is generally better to try to recover medial before lateral rotation. Attempts to simultaneously recover both movements or progress the exercise programme too quickly may result in aggravation of the symptoms.

Photos 59, 60, 61, 62, 63
Recovery of medial rotation in frozen shoulder or dysfunction of shoulder. Patient must progress exercise over months from the restricted position to full range, using assistance from the other hand as necessary.

59



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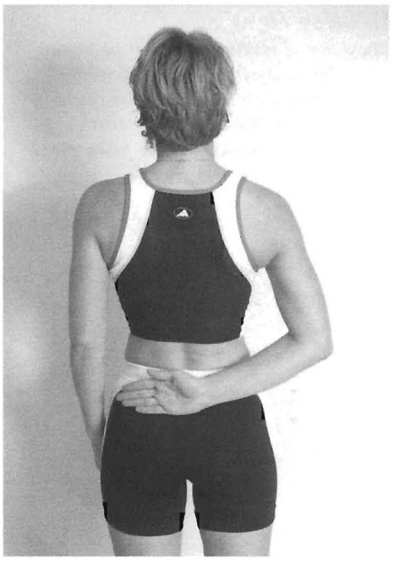
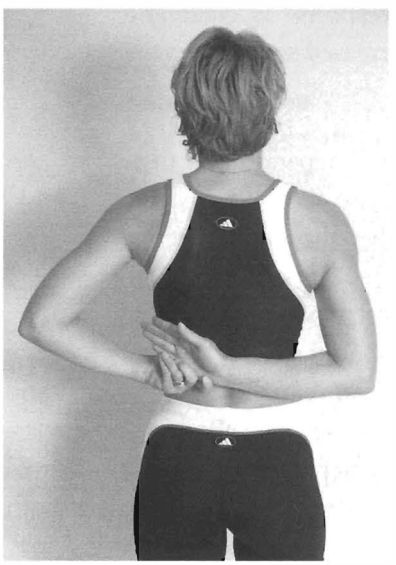


61

For assisted-flexion the patient should be shown how to clasp hands together and, keeping the elbows straight, lift the arms as high as possible over the head. As with all the exercises, the patient is encouraged to take the movement as far as possible and to try to increase the range over time. The limit of movement will be due to a mixture of pain and stiffness.

Assisted-extension may need work prior to medial rotation. For extension, clasp hands together behind the back and lift backwards; caution against leaning forward. If they are unable to link hands behind their back, they may use a stick to do so.

For internal rotation the patient should be instructed to place the hand of the affected arm behind the buttock. If the patient can reach even further, perhaps across to the buttock on the other side, they should be encouraged to do so. After reaching as far as possible the hand should be allowed to return to the side and rest briefly. The movement should be repeated, and with each repetition the hand should move across behind the buttocks and attempt to slide higher up the back. By using the healthy limb to assist with the movement a better range of movement can be obtained. If clasping hands together is initially difficult, a towel can be used to achieve this.



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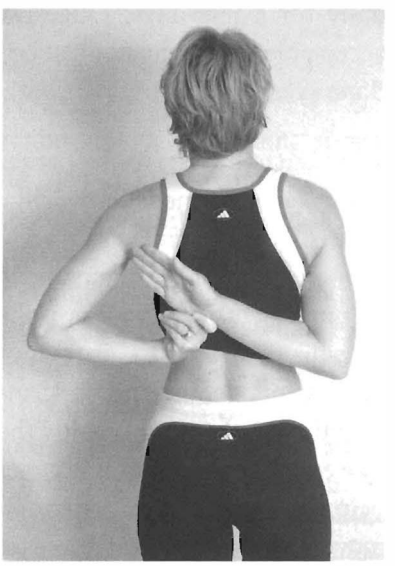

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Provided there is no lasting increase in aching following the exercises, the patient should be instructed progressively to increase the range of internal rotation performed by this means. Once the hand on the affected side can reach the level of the waist, the recovery of external rotation and elevation can be commenced. Attempts to recover external rotation and elevation of the shoulder before recovering a degree of internal rotation may cause a painful reaction, which in some cases can be severe.

The recovery of external rotation is most simply achieved by having the patient stand facing against a wall. By sliding the hand as far as possible up the wall, elevation and external rotation are both encouraged. The patient lowers the arm after having reached as far as possible or as far as pain will permit. So that the patient may have a visible indication of their progress it is helpful to mark the daily achievements on a strip of paper pinned to the wall.

During any part of the programme should the intensity of the patient's pain increase and remain through to the following day, a reduction of the frequency and intensity of the exercises is indicated. If the reaction persists over a week to ten days despite reducing the amount of exercise, attempts to recover function should be delayed or even abandoned.

Clinician mobilisations

If patient-generated forces alone are failing to bring about a satisfactory improvement in the symptomatic or mechanical presentations, the clinician may consider supplementing the self-treatment programme with an increase in force by adding in clinician-generated mobilisations. As noted above, these are certainly not guaranteed to be any more efficacious than exercise alone (Bulgen *et al.* 1984; Binder *et al.* 1984; Nicholson 1985), but a few sessions will show if they are of value in bringing about some changes. If after a few sessions little has changed, the patient should be advised to continue their home exercise programme, told about the final good prognosis, but warned about the protracted nature of the condition.

Clinician-generated mobilisations should reproduce the patient's pain during the manoeuvre, which should abate quickly afterwards. Any long-lasting aggravation of symptoms means that this increase in force level is not appropriate. The mobilisations can address different aspects of the movement loss. Physiological stretches can be

performed into flexion, extension, adduction, lateral rotation, etc. Accessory movements can be performed at end-range physiological movements. If there is no rapid, clear improvement in the symptomatic or mechanical presentations, these procedures should be abandoned and only the patient strategies continued.

Photos 64, 65, 66
Recovery of lateral rotation and elevation in frozen shoulder or dysfunction of the shoulder. The patient should strive for a higher mark up the wall over time.

64

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Figure 10.2 Assessment sheet – capsulitis: early stage

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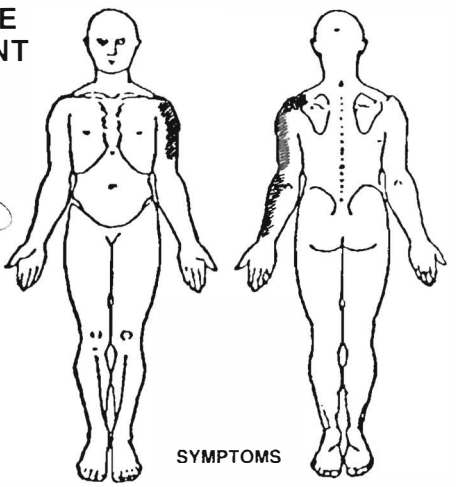


Date ... / ... / ... ...
Name ... M / F
Address ...
Date of birth ... / ... / ... Age ... 60
Work / Leisure ... office worker
Postures / Stresses ... sedentary
... swims
Functional disability from present episode ...
... working; activities - daily
living difficult; not swimming

HISTORY

Present symptoms ... left arm & forearm
Present since ... / ... / ... one month Improving / Unchanging / Worsening
Commenced as a result of ... or no apparent reason
Symptoms at onset ... same
Constant symptoms ... arm Intermittent symptoms ... forearm
What produces or worsens ... any arm movements, even if minimal
lying on it, difficulty with dressing, hair, reaching
hand behind back
What stops or reduces ... rest (sometimes)
...
...

Continued use makes the pain Better Worse No Effect
Pain at rest Yes / No
Disturbed night Yes / No ... 3/4 x ... every night
Other questions ...
...
...
Treatment this episode ... tablets
Previous episodes ... right shoulder, similar problem 3 years ago
Previous treatment ... injections, physiotherapy
Spinal history ... none
... Paraesthesia Yes / No
Medications: tried ... NSAID ... Effect ... not helping
Present medication ... NSAID, analgesics
General health ... good
Imaging ... none
Summary: Acute / Sub-acute / Chronic Trauma / Insidious onset
Sites for physical examination ...
... shoulder
...

PERIPHERAL shoulder

Observation N.A.D

Active movement Loss flexion 100° PDM (ERP)
abduction 80° (ERP)
hand behind back outer buttock (ERP)

Passive movement Loss PDM ERP
(+/-over-pressure)
flexion 110° abduction 90°
lateral rotation 15° gleno-humeral abduction 30°

Resisted tests Response

Effect of repeated tests on pain: produces, abolishes, increases, decreases,
radiates, localises, better, worse, no better, no worse, no effect
flexion increase worse
extension increase worse

Effect of repeated tests on movement: Better (Worse) No Effect

Static Positioning Effects

Loaded (Unloaded) flexion increase worse

SPINE

Movement Loss

Effect of repeated movements:

Effect of static positioning:

Spine testing: (Not relevant) / Relevant / Secondary problem

CONCLUSION (Peripheral) Spine

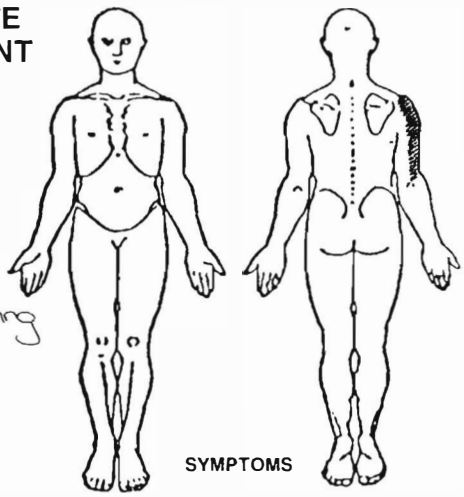
Dysfunction : Articular Contractile
Derangement Postural
Other capsulitis - inflammatory stage Uncertain

PLAN

Exercise Frequency
none given - advice - review in 2 weeks

Figure 10.3 Assessment sheet – frozen shoulder: late stage

THE McKENZIE INSTITUTE PERIPHERAL ASSESSMENT



Date ... / ... / ...

Name ... (M) / F

Address ...

Date of birth ... / ... / ... Age 63

Work / Leisure manager - desk, driving

Postures / Stresses ... gliding

Functional disability from present episode problems activities of daily living

HISTORY

Present symptoms Right shoulder

Present since ... / ... / 6 months Improving / Unchanging / Worsening

Commenced as a result of ... or no apparent reason

Symptoms at onset same

Constant symptoms ... Intermittent symptoms with use

What produces or worsens Twisting arm Reaching up / out Hand behind back no worse

What stops or reduces arm by side

Continued use makes the pain Better (Worse) No Effect

Pain at rest Yes (No)

Disturbed night (Yes) / No sometimes 2/3 x a week

Other questions ...

Treatment this episode tablets

Previous episodes none

Previous treatment none

Spinal history none

Paraesthesia Yes / No

Medications: tried NSAID Effect no help

Present medication none

General health good

Imaging none

Summary: Acute / Sub-acute (Chronic) Trauma / (Insidious onset)

Sites for physical examination shoulder

PERIPHERAL shoulder

Observation N.A.D.

Active movement Loss flexion 140° PDM (ERP)
abduction 100° (ERP)
hand behind back - mid buttock (ERP)

Passive movement Loss PDM ERP
(+/-over-pressure)
flexion 140° Abduction 100°
lateral rotation 30° Gleno-humeral abduction 45°

Resisted tests Response
NAD

Effect of repeated tests on pain: produces, abolishes, increases, decreases, radiates, localises, better, worse, no better, no worse, no effect
flexion produce no worse
hand behind back produce no worse

Effect of repeated tests on movement: Better Worse (No Effect)

Static Positioning Effects

Loaded / Unloaded

SPINE

Movement Loss

Effect of repeated movements:

Effect of static positioning:

Spine testing: (Not relevant) / Relevant / Secondary problem

CONCLUSION Peripheral Spine

Dysfunction: (Articular) frozen shoulder Contractile
Derangement Postural
Other Uncertain

PLAN

Exercise	Frequency
flexion	x 10 x 3/4 daily
hand behind back	x 10 x 3/4

Lateral epicondylitis ('tennis elbow')

'Tennis elbow' is a common complaint with an incidence of four to seven per 1000 patients, with a peak in the 35 to 54 age group, affecting individuals from all occupational groups (Hamilton 1986; Assendelft *et al.* 1996). It results from participation in many activities that involve the use of the wrist and elbow, causing a strain at the common origin of the extensors of the wrist. It is only sometimes caused by tennis, but tennis players themselves are frequently affected. It is considerably more common than medial epicondylitis (or 'golfer's elbow'), and is the most common musculoskeletal complaint of the elbow (Hamilton 1986; Coonrad and Hooper 1973).

Pain is felt at the tip of the external epicondyle of the humerus, but can radiate down the dorsal aspect of the forearm. Depending on the severity of the condition, patients can present with a constant ache that is aggravated by relatively trivial activities or an intermittent ache produced by gripping or clenching of the hand and wrist. Recognition of a pure lateral epicondylitis is straightforward. Most active and passive movements at the wrist and elbow do not cause a problem, except sometimes wrist flexion with elbow extension, which stretches the contractile structure. All resisted movements are pain-free except loaded or resisted extension of the wrist, worsened when the elbow is extended. The movement reproduces the patient's pain, sometimes to such an extent that they are unable to hold the position.

Degenerative changes of overuse or ageing frequently have left the fibres of the extensor origins in an asymptomatic but weakened state. If rest from the aggravating activity is permitted the tissues may recover, but continued and cumulative activity produces pathological changes and an insidious onset of symptoms. The pathology, observed at surgery, is macroscopic and microscopic tears in the tendon. Granulation and scar tissue formation is an attempt at repair – this contains free-nerve endings and thus is very painful when stressed. The repair process is frequently aborted or incomplete. Acute inflammatory cells are not found when patients get to surgery. The tissue proceeds from trauma to reparative, immature granulation tissue and adhesions which remain sensitive to normal use. At the chronic stage the tissues are degenerated, deconditioned for normal use, and not remodelled for full function (Boyd and Mcleod 1973; Coonrad and Hooper 1973; Wadsworth 1987; Nirschl and Pettrone 1979; Nirschl 1992; Leach and Miller 1987; Noteboom *et al.* 1994).

Initially, then, tennis elbow appears to be an inflammatory problem, while later it becomes a mechanical one with abnormal tissue of a dysfunction. This needs remodelling for normal use, starting with gentle loads and then progressing. Alternatively the healing process is suspended, in which case vigorous stimulation may be required to encourage a resumption of the repair process. Again, it is important to view this condition as a continuum, with different mechanical and non-mechanical factors present at different stages and in different individuals.

The natural history and clinical course of lateral epicondylitis

Common treatments for this condition do not result in automatic resolution of the problem. A systematic review of corticosteroid injections for lateral epicondylitis found a general trend that they were effective in the short term at bestowing benefit, but not if there was long-term follow-up greater than six weeks (Assendelft *et al.* 1996). Another review of treatments was unable to find a clear therapeutic value in ultrasound, NSAIDs or injections (Labelle *et al.* 1992). The key issue in the clinical course of lateral epicondylitis is that although ultrasound or steroid injections may confer short-term benefit with the latter being more effective but associated with more recurrences, the relapse rate is considerable (Binder and Hazleman 1983, 1985; Clarke and Woodland 1975). At six months to a year in over 50% of patients the symptoms have persisted or relapsed. Few patients seek medical help again, but may tolerate severe pain on use and accept disability (Clarke and Woodland 1975; Hamilton 1986; Binder and Hazleman 1983). It is clear that persistent, recurrent and chronic symptoms are extremely common in lateral epicondylitis, and the condition can frequently defy all manner of treatments. The role of therapeutic exercise to manage the problem has not been much explored. However, patients with chronic lateral epicondylitis who had failed to respond to injection, medication or physiotherapy were treated with a progressive stretching and conditioning programme and compared to treatment with ultrasound (Pienmaki *et al.* 1996). The exercise group had significantly less pain at rest and, under effort, was more able to work and function more normally.

Self-treatment exercises

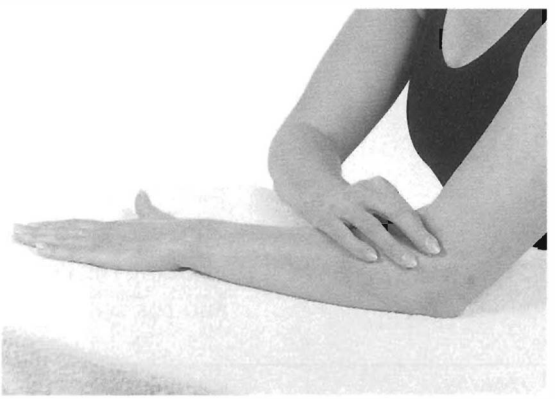
Patients with constant symptoms of recent onset that are easily aggravated may not tolerate mechanical therapy. The condition may still be at a chemical stage and rest, relative rest or avoidance of aggravating factors may be necessary.

If the patient presents with more persistent symptoms, even if constant, a programme of exercises to try to regain normal function should be tried. If the patient presents with intermittent symptoms, their response to a remodelling programme is often good.

The remodelling programme needs to address both the flexibility and contractile strength of the tendon. Stretching involves elbow extension, wrist flexion and medial rotation of the whole arm – usually the other hand will be needed to generate enough tension to the tissues for it to be felt. For strengthening exercises the patient needs to start at a level that generates enough contraction for it to be felt without aggravating the condition in a lasting way. Thus it is often better to start with static muscle contractions to test the response. The patient making a fist or offering self-resistance to wrist extension can do this. All exercises should reproduce the patient's pain, but it should not last on ceasing the movement. Exercises should be repeated 10–12 times three or four times a day initially, progressing to every two hours if this is well tolerated.

Photos 67, 68
Local compression in lateral epicondylitis. The patient firmly applies pressure to the painful extensor origin with the thumb, or the index finger reinforced with the middle finger. With compression maintained, the patient extends and relaxes the wrist ten times.

67



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If static resisted exercises are being easily tolerated or are no longer causing discomfort, the programme should progress onto concentric and then eccentric work. Such dynamic muscle work is better at producing a strength training effect throughout the range of movement. Eccentric training develops greater tension in the muscle than concentric work, which may cause more delayed onset muscle soreness, but is thought to have a superior training effect (Norris 1993). Such dynamic training can be achieved using self-resistance through range, weights, flexi-band or gym equipment. See Pienmaki ***et al.*** (1996) for a progression of exercises.

If improvements cease to occur at any point a progression can be made involving the patient performing wrist extension or clenching his or her fist, but at the same time applying static compression at

the tender site with the fingers of the other hand. The middle finger can be used to reinforce the second finger as it applies pressure to the extensor origin while they repeatedly extend and relax the wrist.

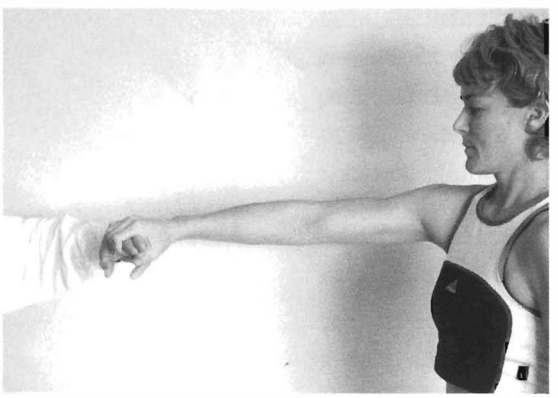
Resisted exercises or active movements with compression can be used if the condition is not responding to remodelling and needs to be stimulated more vigorously. This would be useful for patients with stable symptoms of a persistent nature in whom the healing process has been suspended. Once a temporary aggravation of symptoms has been achieved the more vigorous procedures should be stopped.

Some patients also find a compression clasp worn just below the elbow to be useful in allowing them to perform otherwise painful activities. This should be recommended from day one in all cases. Relief from pain in these cases is immediate but returns once the strapping is removed. It is difficult to identify a plausible explanation for such relief.

'Pseudo' tennis elbow or elbow derangement

Many patients with the typical symptom of 'tennis elbow', which is pain on resisted wrist extension, also demonstrate a painful obstruction to full extension of the elbow. If the problem is one affecting the common extensor origin, why is full extension, active and passive, restricted and painful in a substantial minority of such problems? Why is wrist extension with the elbow fully extended more painful than when it is slightly flexed? Why do the symptoms resolve with the recovery of full extension? Some as yet unexplained internal derangement mechanism must be the cause in a significant number of cases.

Photo 69
Grip test to differentiate derangement from tennis elbow. The patient attempts to lift against the clinician's resistance while keeping their elbow straight. If the elbow gives way into flexion, suspect derangement and test for loss of extension. Obstruction to extension confirms derangement.



69

A grip test may help to differentiate between 'tennis elbow' arising from derangement and 'tennis elbow' arising from strain of the origin of the wrist extensors. The patient strongly grips the clinician's fingers at arm's length and, keeping their elbow maximally extended, pulls up as the clinician resists. Pain of tennis elbow may cause giving way at the wrist, whereas in derangement the elbow itself buckles into

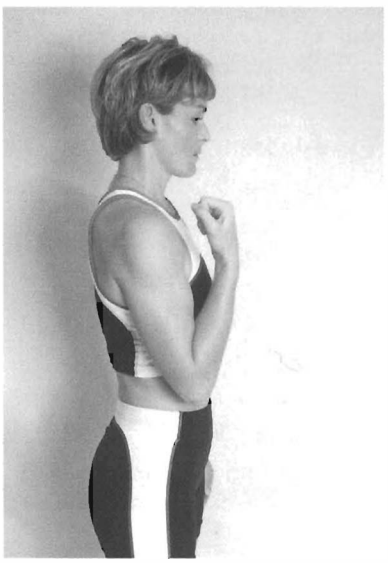
flexion. Lack of full extension should confirm the differential diagnosis.

Self-management exercises

In such cases exercise must address the articular derangement rather than the apparent contractile problem. This is presumably the reason for Mulligan's (1992) proposed method of treating this condition using mobilisation with movement, which he claims can give dramatic improvements.

The reductive force recommended here is a combination of extension and supination. The patient must be instructed from maximal flexion to straighten the elbow as far as possible with the forearm in a position of maximum supination. While maintaining maximum supination the patient must extend the elbow rapidly and abruptly, release the strain for a brief moment and then repeat the process ten to fifteen times as often as the opportunity presents. The movement must be forced further with each excursion and will cause some pain at the maximum point of extension. This will immediately subside with each cycle of relaxation. Ten to fifteen movements into extension should be completed in one minute.

Photos 70, 71, 72, 73
Extension self-mobilisation for extension derangement at the elbow, with obstruction of extension. Commencing with the arm in full flexion, the patient rapidly and abruptly extends the arm to end-range, keeping the forearm in supination. Overpressure is obtained from the high velocity generated by the movement. This is repeated up to ten times.



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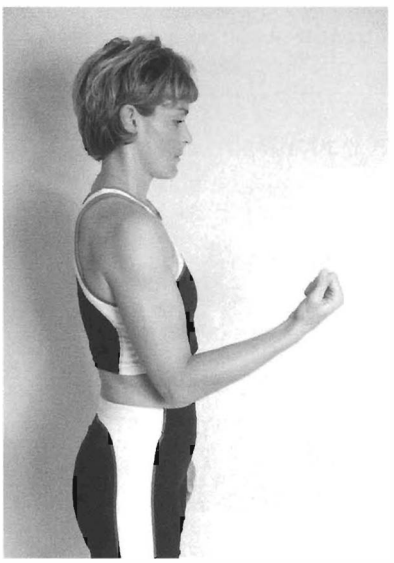

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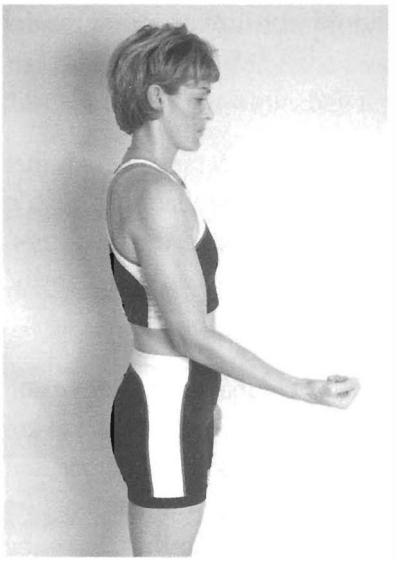

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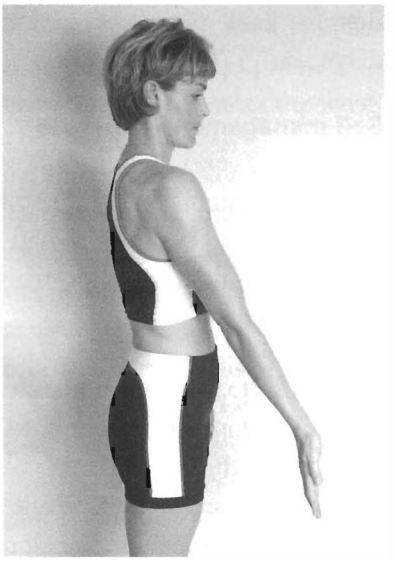


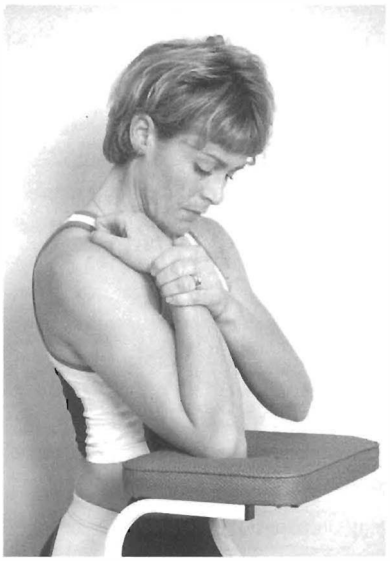
73

In the event that repeated extension of the elbow fails to reduce the obstruction, the patient must be shown how to apply overpressure using the healthy hand, with the arm stabilised by leaning onto a table. If no improvement follows, several options are available. If it seems that further force progressions are necessary to produce lasting reduction of the derangement, the extension manipulation devised by Cyriax may be used (see Kesson and Atkins 1998 or Cyriax and Cyriax 1997). Alternatively, if symptoms are being worsened by extension and the blockage is not being reduced, other directions should be explored. One possibility is the use of flexion, with the forearm supinated and then pronated, with overpressure provided by the other arm.

A lateral force may be used. As in the extension overpressure, the arm should be stabilised by leaning onto a table, the other hand keeping the arm in extension and producing the lateral force by pushing onto the lower part of the humerus.

After all manoeuvres, their value in reducing the derangement should be checked by re-assessing the response to extension.

Photos 74, 75
Flexion self-mobilisation with overpressure for extension derangement at the elbow, with obstruction to extension. The patient applies overpressure with the other hand. The procedure is done with the forearm supinated, then pronated. If symptoms are being worsened by extension and the blockage is not being reduced, the procedure is done with the forearm supinated, then pronated.



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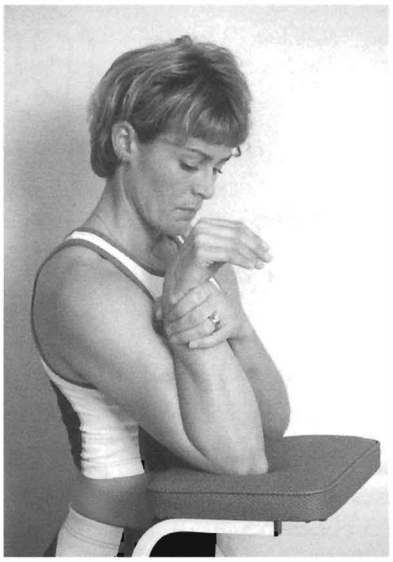

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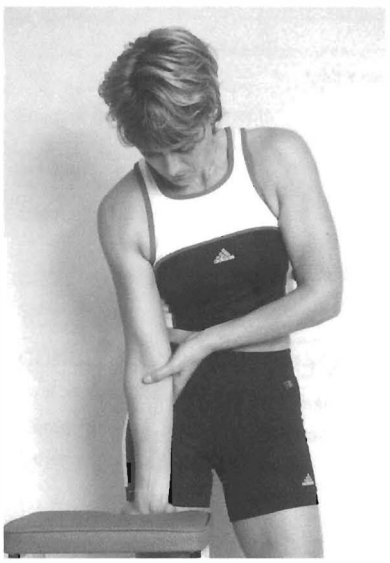
Photos 76, 77
Adduction self-mobilisation for derangement at the elbow. The arm is held in extension with the other hand and counterpressure applied by using the table. The patient then pushes the maximally extended elbow laterally, as far as possible, ten times.



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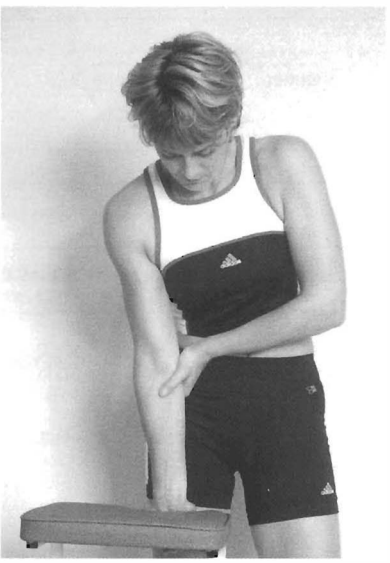

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Figure 10.4 Assessment sheet – tennis elbow

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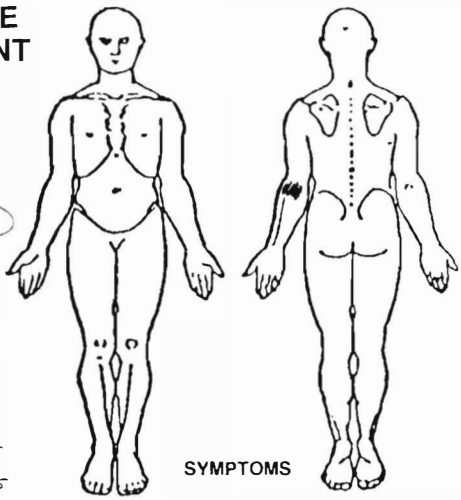


Date ...../...../.....

Name ..... M / F

Address .....

Date of birth ...../...../..... Age ...40...

Work / Leisure ...cook...

Postures / Stresses ...lifting & cleaning...

Functional disability from present episode ...working... has been off work - improved but symptoms returned when back at work

HISTORY

Present symptoms ...Left lateral elbow & forearm... better but now

Present since ...../...../...5 months... Improving / Unchanging / Worsening

Commenced as a result of ... or no apparent reason

Symptoms at onset ...same...

Constant symptoms ...elbow... Intermittent symptoms ...fore arm...

What produces or worsens ...lifting weights, gripping objects... now not worsened

What stops or reduces ...rest, gentle elbow/wrist movements...

Continued use makes the pain Better Worse No Effect

Pain at rest Yes / No

Disturbed night Yes / No

Other questions ...

Treatment this episode ...steroid injections (1) reduced temporarily (2) no effect

Previous episodes ...one...

Previous treatment ...

Spinal history ...Episodes of neck pain - nothing in last 2 years

Paraesthesia Yes / No

Medications: tried ...NSAIDS... Effect ...initially helped, now no effect

Present medication ...analgesics (irregularly)

General health ...good...

Imaging ...none...

Summary: Acute / Sub-acute / Chronic Trauma / Insidious onset

Sites for physical examination ...elbow...

PERIPHERAL elbow

Observation N.A.D.

Active movement Loss PDM ERP

elbow : flexion / extension - pronation / supination

wrist : flexion / extension - all full ROM.
- no increased pain

Passive movement Loss PDM ERP
(+/-over-pressure)

elbow flexion + wrist flexion with O.P (ERP)

Repeated test : increased, no worse. All else - NE

Resisted tests Response elbow : extension / flexion / pronation / supination - NE

elbow extension + wrist extension - pain

Effect of repeated tests on pain: produces, abolishes, increases, decreases,
radiates, localises, better, worse, no better, no worse, no effect

elbow extension + wrist extension : increase no worse

gripping (end of towel) increase no worse

Effect of repeated tests on movement: Better Worse (No Effect)

Static Positioning Effects

Loaded / Unloaded

SPINE

Movement Loss

Effect of repeated movements:

Effect of static positioning:

Spine testing: (Not relevant) / Relevant / Secondary problem

CONCLUSION (Peripheral, Tennis elbow) Spine

Dysfunction : Articular (Contractile) Deconditioned

Derangement Postural

Other Uncertain

PLAN

Exercise	Frequency
Gripping (piece of towel) x 10	x 3/4 daily
Elbow extension + wrist flexion with O.P. x 10	x 3/4 daily

Carpal Tunnel Syndrome

The carpal tunnel is a ligamentous-osseus foramen just below the wrist comprising the transverse carpal ligament and the carpal bones. Through it pass the tendons of flexor digitorum superficialis and profundus and pollicis longus and the median nerve. Decrease in the cross-sectional area of the tunnel or enlargement of the tunnel contents can cause compression of the median nerve (Bleecker 1987). This causes numbness and tingling in the digits innervated by the median nerve – usually the middle and index fingers, the medial aspect of the thumb and the lateral aspect of the ring finger; the little finger is spared. However, atypical innervation patterns are said to be common, and thus the little finger and whole hand can be involved (Keenan 1991). Sensation may be reduced and the patient might complain of dropping things. As the disease progresses motor function is also affected, and in extreme cases there may be thenar atrophy. Pain may be localised to the wrist, but may also show retrograde spread to the elbow or shoulder – one of the few instances in which pain is referred from distal to proximal. Typically the patient wakes in the middle of the night, disturbed by the symptoms, and shakes their hand to gain some relief (Caillet 1981a; Szabo and Madison 1992; Sola 1994; Yassi 1997). In the early stages symptoms are frequently only felt at night and no symptoms felt during the day. As the disorder progresses symptoms become more frequent or constant.

Assessment and examination of suspected carpal tunnel syndrome should always be done with a high index of suspicion concerning the cervical spine. Neck problems commonly can radiate symptoms into the hand in a similar distribution. It is likely that if attention is not paid to possible cervical involvement carpal tunnel can be mistakenly diagnosed.

Tapping firmly over the carpal tunnel (Tinel's sign) and sustained full passive wrist flexion (Phalen's test) are said to provoke the patient's symptoms and to be diagnostic of the disorder. However, these tests have only moderate predictive value and the diagnosis is usually established on history-taking, although numerous other tests exist (Szabo and Madison 1992). Use of a hand diagram to reach the diagnosis has been shown to have good sensitivity and specificity (Katz and Stirrat 1990a; Katz *et al.* 1990b).

Carpal tunnel syndrome is the most frequently encountered peripheral compression neuropathy, and is the second most common industrial injury in the US. Two types of patients have been described: the typical patient, in whom the condition was first recognised, is a woman older than 40. More recently a second distinct population with this problem has been recognised – they are workers of either sex who perform repetitive manual labour that involves gripping. Studies have shown that occupational risk factors increase the likelihood of developing the condition. Work that requires heavy wrist activity, repetitive flexion and extension of the wrist, finger motion with wrist extension or exposure to vibration is considered to be high risk. Biomechanical studies have shown that intracarpal pressure is particularly exaggerated by wrist flexion and ulnar deviation (Szabo and Madison 1992; Sola 1994).

Although other systemic risk factors exist such as diabetes or thyroid disorders, it has been estimated that up to 47% of all cases may be caused by workplace factors (Baker and Ehrenberg 1990). Typically those involved in sustained or repetitive grasping, hand/wrist activity or use of vibrating tools are more likely to suffer the disorder. Increased association with carpal tunnel syndrome has been found in butchers, meat packers, checkout workers, electronic assembly workers, musicians, typists, carpenters and garment workers (Baker and Ehrenberg 1990; Masear *et al.* 1986; Feldman *et al.* 1987). Such situations where initially there are intermittent symptoms, abating with rest and returning with sustained activity of the hand and wrist, have been defined as ***dynamic*** carpal tunnel syndrome (Braun *et al.* 1989). Continuing with the provocative activity is likely to cause a worsening of symptoms and a deterioration of function.

The syndrome is also associated with pregnancy – it is the second most frequent musculoskeletal symptom of pregnancy after back pain. It usually occurs in the second and third trimesters, and arises from fluid retention and swelling. Wrist splints are very effective, and when worn at night frequently bring relief. The symptoms virtually always resolve rapidly after delivery, although they may recur during subsequent pregnancies (Heckman and Sassard 1994). Other diseases and conditions may be involved in the pathogenesis of carpal tunnel syndrome (Szabo and Madison 1992).

Natural history

The pathophysiology of carpal tunnel syndrome relates to alteration of pressures due to changed fluid and tendon dynamics leading to ischaemic impairment of the nerve (Szabo and Gelberman 1987). Pressure within the carpal tunnel is markedly elevated in those with the syndrome pre-operatively compared with control patients and post-operatively (Gelberman *et al.* 1981; Okutsu *et al.* 1989). Pressures are increased by movements of the wrist and local muscle contractions (Szabo and Chidgey 1989; Werner *et al.* 1983).

Like many overuse conditions onset is gradual, symptoms being initially intermittent and mild; the pathology is reversible. It is thought that continued aggravation eventually produces irreversible changes. Continued pressure and reduced circulation produces oedema and further swelling. Nerve conductivity is further impeded and without relief these changes may produce extensive intraneural fibrosis (Sunderland 1976; Gelberman *et al.* 1987). As the condition worsens, symptoms become constant, motor impairment develops and gross morphological changes may be found in the median nerve. At this point symptoms are no longer reversible and surgical release of the transverse carpal ligament may be necessary to bring relief, although this is not always successful (Szabo and Madison 1992).

Management

Patients may present with carpal tunnel syndrome either at the inflammatory or chronic stage. The former will usually have constant symptoms, which are easily aggravated or produced. At the chronic stage patients may present with a dysfunction of the median nerve, which produces intermittent symptoms with activity. The distinction between the two should be clear from the history, but can be confirmed by their response to repeated extension of the wrist with elbow extension. This manoeuvre stretches the nerve and is likely to worsen an inflammatory condition. If a dysfunction is present the stretch should reproduce the symptoms, repetition should not worsen the symptoms, and these should abate once the stretch is released.

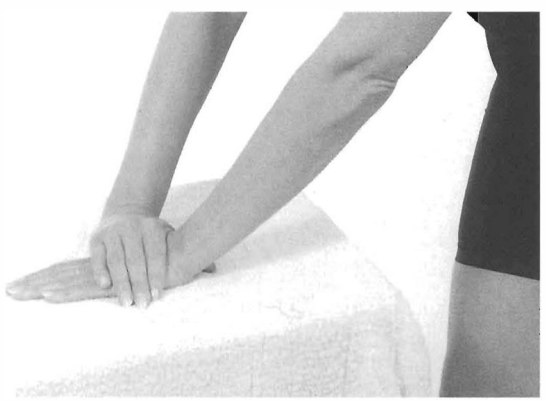
During the inflammatory stage therapy has little to offer. Use of a wrist splint at night and during aggravating activities, and avoidance of aggravating activities may help. As noted above the pathophysiology is a mixture of mechanical factors causing physiological changes – cessation of factors that are clearly aggravating the condition may prevent progression. Avoidance of extremes of

wrist flexions and extension can ease symptoms at any stage of the disorder, and this is most effectively achieved with the use of a wrist splint. A two-week trial of wrist splint usage is therefore always justified. The more advanced the problem is, the less likely simple conservative measures are to be effective. Injections of corticosteroid appear only to help in those with milder symptoms, that is those who have had the condition for less than a year with diffuse and intermittent symptoms and no weakness or atrophy. Those at a more advanced pathological stage with more severe symptoms only gain temporary relief and suffer a very high relapse rate following injections (Gelberman *et al.* 1980).

If a dysfunction is present, stretching five to ten times several times a day should be started. Show the patient how to lean onto a table keeping the elbow extended, then lean forward producing wrist extension sufficiently to produce their symptoms. As long as this does not worsen the condition and all symptoms go once the stretch is released, the exercises may be increased to every couple of hours.

If the stretch using wrist and elbow extension needs to be progressed, a movement that further tensions neural structures in the arm such as 'upper limb tension test one' (Butler 1991) could be considered. *It is important to use such manoeuvres that induce symptoms by stretching the nerve and causing ischaemia with great care.*

Photos 78, 79
Carpal tunnel syndrome dysfunction. Place the affected hand on a table and flatten with pressure from the other hand. Keeping the elbow in extension, lean forward over the table so that the wrist is fully extended, and then withdraw to the starting position. Stretching or paraesthesia should be provoked, which subsides as the starting position is resumed. This can be repeated up to ten times, as long as symptoms are not lastingly aggravated.



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Tenosynovitis – de Quervain's Syndrome ('trigger finger')

Various tendons of the hand and forearm are prone to developing inflammatory conditions. Cumulative micro-failure develops as a result of overuse and produces swelling of the tendon, thickening and stenosis of tendon sheaths, fibrocyte proliferation and adhesions These changes cause occlusion of blood flow and interruption of normal diffusion so that the tissues are deprived of nutrients required for repair and healing (Thorson and Szabo 1992). Tendons can develop a swelling or nodule which causes catching, triggering or locking of the digit, referred to as stenosing tenosynovitis or 'trigger finger' (Newport *et al.* 1990). These upper extremity tendonitises are frequently the result of cumulative trauma due to repetitive motion that overwhelms the normal recovery of tissues from activity – there is thus frequently an occupational element in the pathogenesis of these conditions (Thorson and Szabo 1992; Yassi 1997; Sola 1994) Tasks involving high levels of force and repetition of manual handwork constitute a considerable risk factor for developing hand–wrist tendonitis (Armstrong *et al.* 1987).

This problem may affect any of the tendons crossing the wrist, but is probably most commonly seen in the abductor pollicis longus and extensor pollicis brevis tendons – a condition known as de Quervain's Syndrome (Thorson and Szabo 1992). There is pain in the wrist and proximal thumb area, with marked tenderness over the radial styloid process. Resisted abduction and/or extension of the thumb are painful and weak. Ulnar deviation, especially with the thumb in flexion (Finkelstein's test), is painful and limited – this manoeuvre stretches the affected tendon (Thorson and Szabo 1992; Sola 1994; Witczak *et al.* 1990).

Management

Recommended management of this type of condition usually includes splinting, rest, NSAIDs and injection of corticosteroids (Sola 1994; Thorson and Szabo 1992; Witczak *et al.* 1990). In fact, splinting by itself is much less successful than injection, while a long history of the condition carries a poor prognosis even with injections (Weiss *et al.* 1994; Newport *et al.* 1990: Rhoades *et al.* 1984; Witt *et al.* 1991). Surgical release is recommended if conservative treatments fail.

There are no studies indicating that mechanical therapy may help with this problem. However, suitable advice concerns relative rest,

especially early on, followed by a functional rehabilitation approach involving graduated resisted exercises and stretches to attempt to normalise the impaired function of the tendon. This would involve resisted thumb abduction and /or extension at a load that generates symptoms without aggravating them. Stretches must be done with care and should start with separate ulnar deviation of the wrist and thumb flexion. Later these can be combined, which places considerable stretch on the affected tendon.

Photo 80
Self-applied isometric thumb extension with compression. To be performed at a rhythmical pace - pressure on, hold for a moment, relax, and then repeat up to ten times.

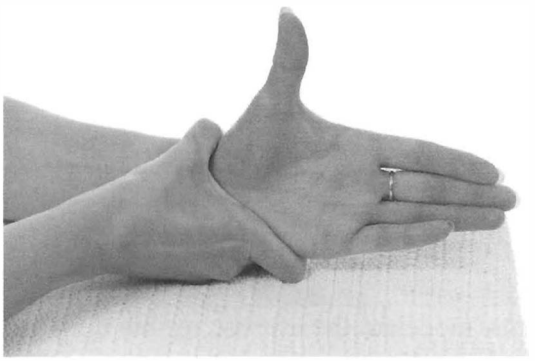

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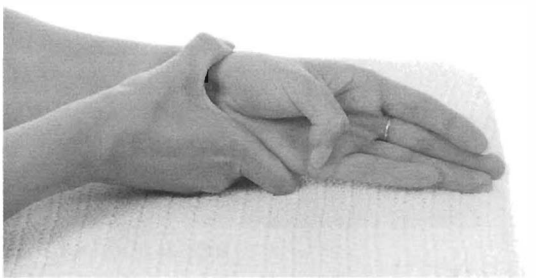
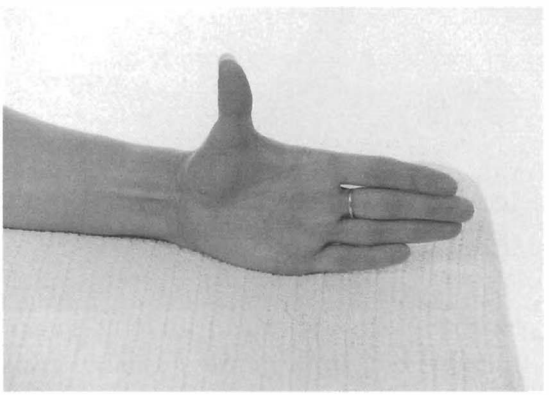
Photo 81
Self-applied isometric thumb abduction with compression. To be performed at a rhythmical pace - pressure on, hold for a moment, relax, and then repeat up to ten times.



81

Photos 82, 83
Early tension movement for thumb extensors, with wrist in neutral. The patient starts with the arm relaxed on a supporting surface with the thumb upright (82). The thumb is stretched in flexion as far as possible, then relaxed to the starting position. To be repeated up to ten times. Gradually the patient should increase the range of movement until able to touch the distal end of the fifth metatarsal (83).



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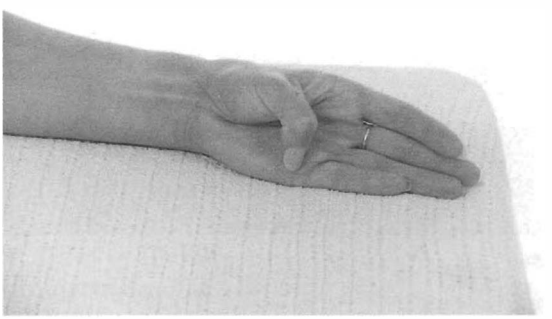

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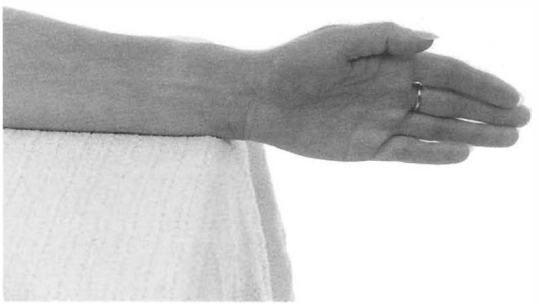
Photos 84, 85
Later tension movement for thumb extensors and abductors, including wrist ulnar deviation. The patient starts with the forearm relaxed on a supporting surface, with the wrist and hand over the edge and in neutral (84). Movements into ulnar deviation, with the thumb resting on the index finger, can be done in addition to regaining thumb flexion. Later the two movements can be combined, so that eventually with the thumb in flexion the patient is also able to move into ulnar deviation (85).



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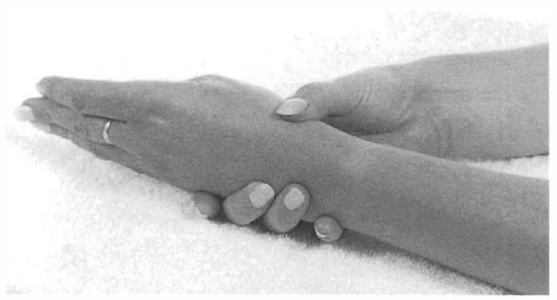
85

If a progression is needed the thumb of the other hand can be used to provide compression on the tender tendon while the patient actively flexes and extends the affected thumb. If this causes aggravation of symptoms, moderate the exercise regime until this has subsided.

Photos 86, 87
Compression during active movement in de Quervain's Syndrome. Apply pressure with opposite thumb on tender area of tendon. While maintaining compression, the patient flexes and extends the thumb ten times.



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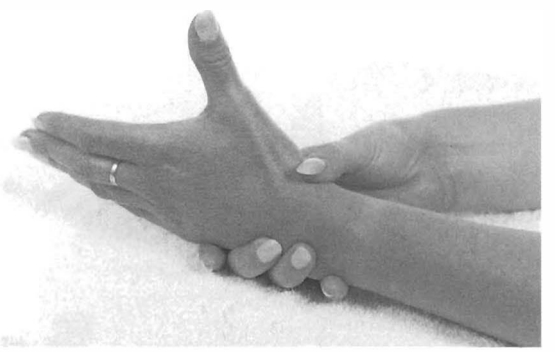
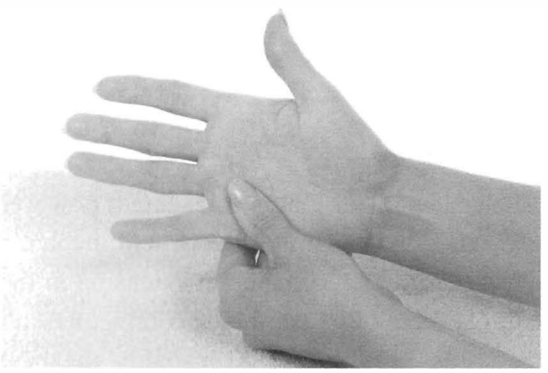


87

If a trigger finger is present, get the patient to place the other thumb as close to the nodule as possible and apply constant pressure. They should then repeatedly flex and extend the affected digit so that the nodule keeps passing beneath the pressure of the thumb. Once this has made movement easier, active movements should be done to maintain improvements.

Photos 88, 89
Compression during active movement in 'trigger finger' of fifth digit. Place the other thumb on the affected area as close to the nodule as practical. While maintaining constant compression, flex and extend all fingers, running the nodule under the pressure of the thumb ten times.



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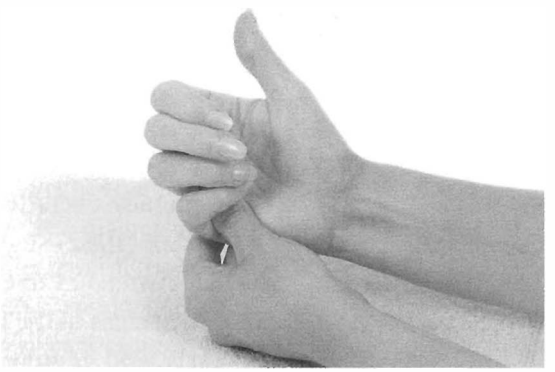

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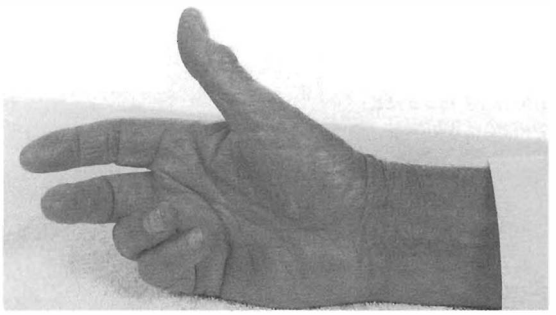
Dupuytren's Contracture

This condition involves a painless thickening and contracture of the palmar fascia due to fibrous proliferation, which results in flexion deformities and impaired function. Men are more commonly affected than women, with the incidence increasing progressively after 40 years of age. Similar conditions can occur in the plantar fascia.

The condition may occur unilaterally or bilaterally; the ring fingers and little fingers are most commonly involved, the middle and index fingers less often. The palmar fascia thickens and shrinks; nodules, adhesions and contractures develop; the palm is puckered and thick, and subcutaneous cords may hold the fingers in flexion. The disorder progresses, but at a variable and unpredictable rate. As it progresses flexion contractures limit the movement and function of the hand. Extension of the fingers becomes impossible, the grip is impaired, there is difficulty letting go of objects and the bent fingers become a nuisance (Apley 1982; Berkow 1987).

Photos 90, 91
Stretch for Dupuytren's Contracture. With the other thumb towards the end of the fingers, stretch into extension ten times strongly enough to provoke a burning sensation. 'No burn - no good.'



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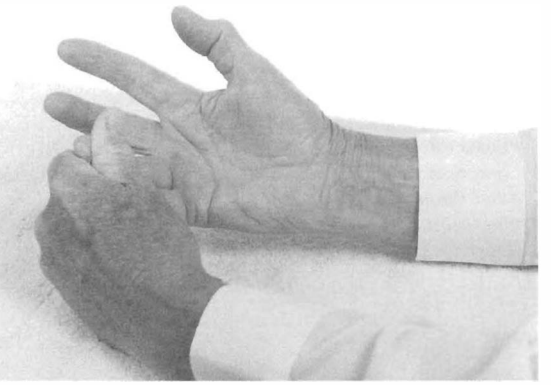

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Management

Generally operative management is all that is considered; however Cyriax (1982) suggested self-management by the patient involving daily stretches to elongate the fascia and maintain extension in the early stages of the condition. As in other soft tissue contractures, an early and regular stretching programme is probably the best management. The symptomatic response expected would be that of a dysfunction – pain produced on stretching, which abates when the stretch is relaxed.

Using the opposing thumb and index finger to grip the affected digits, the fingers should be stretched out into extension ten times, three or four times daily. This should be increased to every two hours after a few days. To be of value the *stretch needs to provoke a burning sensation* in the affected area. Commitment to the programme is vital if the patient is to prevent progress of the condition or reverse it.

Bursitis

This term refers to conditions involving a bursa, such as the trochanteric, subacromial or prepatellar, and denotes an inflammatory problem. At the shoulder acute and chronic subacromial bursitis is commonly diagnosed in about a third of patients visiting general practice (van der Windt *et al.* 1995). However, other authors do not refer to this diagnosis, and in physiotherapy clinics the prevalence

would seem far less (Liesdek *et al.* 1997; Pellecchia 1996). Trochanteric bursitis is said to be a common cause of lateral thigh pain (Little 1979), especially in patients with rheumatoid arthritis (RA) (Raman and Haslock 1982). Iliopsoas bursitis has been reported in patients with RA and OA, with an inguinal mass and leg swelling (Underwood *et al.* 1988). However, patients with putative 'trochanteric bursitis' are frequently misdiagnosed; their symptoms are in fact due to lumbar spine problems (Traycoff 1991). Pre-patellar bursitis, associated with considerable kneeling and consequently referred to as 'housemaid's knee', is more obviously recognised. Pain, tenderness and swelling are localised to the site of the bursa, and are nowadays most commonly found in carpet layers (Evans 1986). Generally treatment of bursitis involves injection or surgical incision, although the benefits have never been evaluated.

There is limited information about bursitis and its presentation is vague and unreliable. Its natural history is consequently unstudied, and the best management is unknown. It is frequently a diagnosis by exclusion of other possibilities. At its acute onset mechanical therapy is totally inappropriate – pain is constant and often extreme, and all movements are barely tolerated. Mechanical therapy cannot assist in these inflammatory conditions. However, if later the condition becomes chronic, and thus the problem is of tissue dysfunction and sensitisation, advice on an appropriate graded exercise problem may be appropriate.

Overuse injuries

Occupational overuse syndrome is one term used to describe pain arising in various parts of the extremities and attributed to poor working environments. These disorders are also known by different terms in different countries: repetitive strain injuries, cumulative trauma disorders, repetitive motion disease, occupational cervicobrachial syndrome or work-related upper limb disorders. These terms, although developed to explain occupational aches and pains, are equally applied to recreational and domestic environments. Repetitive strain has been associated with playing musical instruments, electronic games, knitting, solving Rubik's Cube and marathon running, as well as a wide range of industrial and clerical jobs. They are umbrella terms describing a range of disorders that develop as a result of repetitive movements and awkward postures.

Such conditions are not new, and were first described by Ramzzini in the seventeenth century. However, there have been large increases in their reporting in Western countries in the last few decades. In the US there was a quadrupling of the rate of work-related repetitive motion disorders between 1977 and 1989, so that they constituted 60% of all occupational illnesses in 1990 with an estimated cost of compensation that exceeded $US20 billion per year (Louis 1987; Yassi 1997; Rempel *et al.* 1992). Whether this alarming rise is due to altered work practices that are causing an increase in pathological changes, or rather is due to greater accessibility to work compensation and altered patterns of socio-economic behaviour is unknown.

Ergonomic hazards that are said to pose a risk in the development of cumulative trauma disorders (CTD) include repetitive and forceful motions, static muscle loading and mechanical stress concentrations, extremes of vibration and temperature, and awkward and constrained joint postures. Risks are increased by certain organisational factors at work, such as excessive duration or speed, externally paced work (assembly line production), inadequate rest periods and monotonous tasks. Multiple risk factors increase the risk of CTD considerably (Viikari-Juntura 1998; Novak and Mackinnon 1998; Yassi 1997; Rempel *et al.* 1992; Silverstein *et al.* 1987). Women are affected with CTD almost twice as often as men (Doheny *et al.* 1995).

Low-load muscle activity may be involved in the aetiology – this does not induce tiredness as high-load muscle work does, which causes a natural constraint in the activity. Instead the activity can be maintained for prolonged periods without, initially, awareness of discomfort. However, at these low loads metabolic concentrations in the tissues are being altered, so that over prolonged periods the muscles become susceptible to necrotic changes (Sjogaard and Sjogaard 1998). The accumulative force, which is generated by repetitive motion and sustained postures over a prolonged period, is thought to cause micro-tears and trauma to the soft tissues. Inflammation, pain and swelling may ensue. Although a variety of soft tissues have been implicated, tendons are a prime source of symptoms with these disorders. Pathological changes and subsequent occlusion of blood flow combine to deprive the tendon and other affected tissues of the nutrients required for repair and healing. Degenerative changes in the tendon and immature repair tissue would appear to prevent full repair and encourage persistent symptoms

(Rempel *et al.* 1992; Thorson and Szabo 1992; Smith 1996; Leadbetter 1992). Nerves are also damaged in repetitive motion problems, presenting a broad spectrum of changes from perineural oedema to axonal degeneration – changes which might take many years to develop (Novak and Mackinnon 1998). Failure to rectify the excessive loading on the structures and to normalise the tissues may overwhelm the recovery capability of one or several of these structures (Allan 1998). The condition may become progressively worse, persistent and irreversible.

CTD can occur in the hands, wrists, elbows, shoulders, neck, back, hips, knees and ankles. Back problems are the most common, but upper-limb CTDs are the fastest growing group of occupational disorders (Yassi 1997). Specific clinical syndromes that are included within the umbrella term include de Quervain's tenosynovitis, carpal tunnel syndrome, tenosynovitis, lateral and medial epicondylitis, cervical syndrome and rotator cuff tendonitis (Yassi 1997; Rempel *et al.* 1992). Some of these have already been discussed in other sections where detailed descriptions, including suspected occupational risk factors, will be found. Such work-related upper limb disorders (WRULDs), which have relatively clear clinical characteristics, have been termed Type 1 conditions (Hutson 1997). The term Type 2 WRULDs is used to describe poorly defined regional pain syndromes, with a spread of symptoms from the neck to the hand that have a prime neurogenic component. It is thought that peripheral and central sensitisation creates this state of pain amplification producing refractory upper limb pain (Hutson 1997).

Since the terms 'repetitive strain injury', 'occupational overuse syndrome' or 'work- related upper limb disorders', etc. have been coined, pains of postural origin have been classified as injury and in some extreme cases patients have claimed permanent disability. The patient may go through a repeated cycle of rest, return to work, recurrence of symptoms and referral to various experts that ultimately may end in loss of work – ultimately only lawyers and health care professionals benefit (Yassi 1997; Louis 1987; Ireland 1998). The part that therapists and physicians have played in the increase of the complaint and its chronicity is unknown. Some authorities, however, berated their medical colleagues for the epidemic of repetitive strain injury which hit Australia in the early 1980s. One called it 'an iatrogenic epidemic of simulated injury' whose psychological basis was revealed by the rapid rise and equally rapid fall a few years later

in reporting of the condition (Bell 1989). It is said that the decline in the problem came about with awareness that it was a non-physical socio-political phenomenon and a corresponding loss of pecuniary benefits enjoyed by powerful vested interests and by sufferers (Ireland 1998). It is suggested that there are striking similarities between the Australian experience in the mid-eighties with repetitive strain injuries and the present experience in the US with CTD (Ireland 1998). Reports from both Australia and the US make clear that however genuine were the symptoms reported they tended to concentrate in certain areas, while workers with the same task in a different state or city were relatively unaffected. Patients who did particularly badly in one outbreak were those who received medical treatment outside their workplace, those who were treated surgically and those involved in litigation (Huskisson 1992; Hadler 1992a). Over-medicalisation of the disorder and failure to encourage patient responsibility for the appropriate behaviour is clearly a factor in the spread of this condition.

It is important to recognise that medical authorities are still very divided on this issue. Some categorise the more intangible conditions as purely psychosocial with the capacity to develop into epidemic proportions if so encouraged by lawyers and medics. Some find no evidence that links these disorders with occupational factors, although work may aggravate existing symptoms (Barton *et al.* 1992). Alternatively ergonomists argue that occupational factors have a causative role in many of these conditions. Although epidemiological data suggests association between certain occupations and the development of some of these conditions, it must be recognised that association is not causation, and that the true role of biomechanical factors has not yet been definitively answered. Other factors that may have a role in the development and reporting of symptoms such as leisure activities, individual factors, genetic predisposition, the degree to which individuals worry over normal aches and pains, job satisfaction and other sociocultural factors are rarely included in the analysis. Most studies analyse a point in time; disease causation is best examined by studying a cohort of asymptomatic individuals over time to see who develops the disease of interest. These studies are lengthy, complicated and considerably more costly. There has been no work that addresses differences in prevalence of these conditions between those working and not working – it is certainly not the case that those who are not in full-time employment are free of these musculoskeletal complaints.

An example of the importance of sociocultural factors was observed at McKenzie's Wellington Physiotherapy Clinic before and after 1973, which was the year of the introduction of the Accident Rehabilitation and Compensation Act in New Zealand. This Act covered the entire population 365 days a year (working or not) against injury by accident. Two years before this date, 63% of patients reported that their symptoms started for no apparent reason, whereas two years after this date, 69% attributed their problem to an accident. Throughout this period about 55% of patients were in paid employment (unpublished data, Wellington Physiotherapy Clinic patient records)

Once individuals develop pain these symptoms may be worsened by occupational activities, but this does not mean that these activities have caused them. Socioeconomic factors such as how people are paid when they are 'sick' and the influence of lawyers and trade unionists may encourage sufferers to attribute disease causation to occupational activities when there may be no absolute proof for this. The inability of 'sick pay' systems and workplaces to encourage a gradual return to normal activities does not help to clarify this conflict of interest. The role of health care professionals in this is ambiguous. Are disability, work avoidance and blame encouraged, however unwittingly, when rest from aggravating activities, which are seen as the cause of their problems, are condoned? Does denial of the patient's problem, as it lacks a clear pathological basis, only exaggerate conflict between 'the system' and the patient? This area is very complex and it is probably best, at this point in time, to recognise that the evidence concerning causation is unclear.

Management of overuse injuries

There is a general consensus that early diagnosis and intervention improves the prognosis of these problems (Yassi 1997). The more time symptoms have persisted, the more times the patient has been through the rest/return to work/recurrence cycle, then the more likely it is that the pathology will have progressed to being irreversible. The success rate over the years for either conservative or surgical treatment for these disorders has been, at best, only fair (Blair and Bear-Lehman 1987).

The ergonomic conditions at the workplace, which may generate these problems and probably aggravates them, needs to be addressed.

Failure to alter faulty tasks or postures will simply lead to the recurrence of problems as soon as the employee returns to work. Particular aspects of work that should be addressed by ergonomic interventions are repetitiveness, force/mechanical stress, posture, vibration and psychosocial stresses. Redesigning workstations and tools can be an effective means of reducing the risk factors for CTD (Yassi 1997). Work organisation ought to be addressed also so that the worker is able to take sufficient rest breaks and rotate various tasks. However, the responsibility is also on the worker to maintain appropriate postures, change positions frequently and perform tasks in the least stressful way. All patients will need to be given an understanding of these issues as they pertain to their individual circumstances. If working conditions and timetables cannot be ameliorated, the prognosis is frequently bleak.

Range of movement and stretching exercises should be introduced to prevent static postures and facilitate interruption of tasks. Postures need to be altered to prevent constrained, awkward loading that causes stress concentrations on certain tissues. If specific tendons are implicated then exercise programmes to strengthen and stretch should be used to try to normalise function. It is the clinician's responsibility to provide appropriate information that addresses these issues. However, it is only the patient who is actually able to put them into practice. The effectiveness of such exercise programmes is unclear. A one-year study found that two daily seven-minute exercise breaks had no effect on decreasing neck and upper limb symptoms (Silverstein *et al.* 1988).

Splints, NSAIDs, physiotherapy modalities and corticosteroid injections are recommended interventions (Yassi 1997). However, there is little evidence that these can successfully treat these conditions. For more detail see individual conditions discussed elsewhere in this chapter.

'Groin strain' or adductor strain

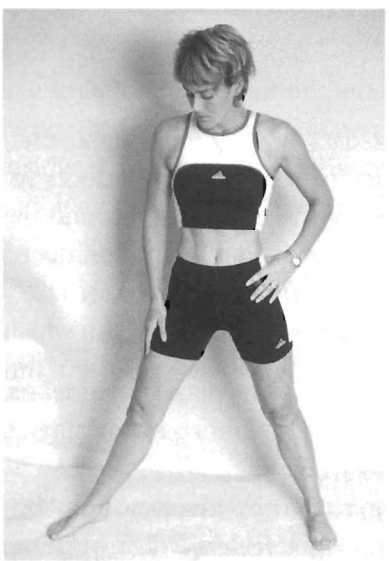
Muscle–tendon strains in the groin area are not uncommon sports injuries (Galasko *et al.* 1982). These most commonly involve adductor longus, rectus femoris, rectus abdominis or iliopsoas, and are often chronic by the time the patient seeks help (Renstrom 1992). Pain is localised to the groin, but pain in the groin can have many other

causes (Renstrom 1992; Muckle 1982). Adductor strains are probably seen most commonly, and their management will be described.

Pain will be elicited on resisted adduction and passive abduction or the FABER (flexion/abduction/external rotation) position – these stretches will be reduced in range compared to the non-injured side. There may or may not be tenderness to palpation. There may be pain at rest, depending on the stage or during or after activity, and stiffness may be reported (Renstrom and Peterson 1980). In the acute stage rest is followed by a gentle progressive exercise regime. If poorly managed it is easy for these injuries to become chronic, when they become extremely difficult to treat (Renstrom and Peterson 1980; Renstrom 1992). Once they have become chronic, return to full physical activity can be considerably delayed – many are not fully fit for five months (Renstrom and Peterson 1980)

Typically the patient will give a history of injury incurred playing football or while horse riding, or symptoms may begin insidiously. They may have rested initially and the injury appeared to resolve, but each time they return to the football pitch it is re-aggravated. The extra exertion provokes a constant ache for a few days, which then settles to an intermittent ache. Following each attempted return

Photos 92, 93
Adductor stretch in standing. Here the left leg is being worked on. The feet should be at right angles to each other, with legs astride (92). The patient then lunges onto the non-injured leg, stretching the adductor region (93).



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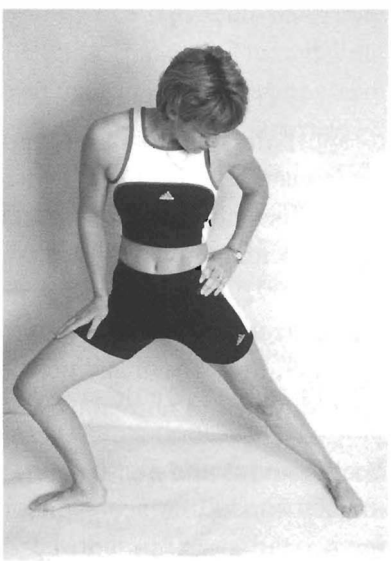

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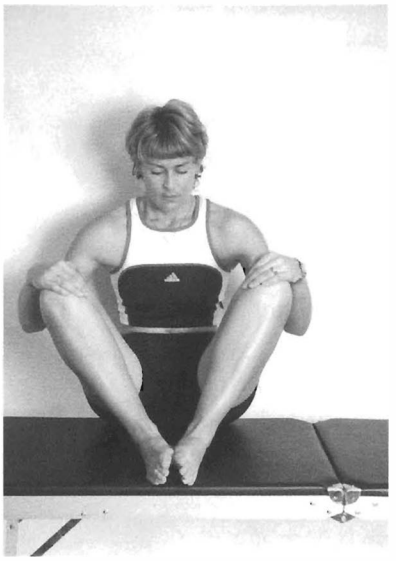
Photos 94, 95, 96
Adductor stretch in sitting, which generates more tension. The patient starts with the soles of their feet together, their hands resting on their knees, which are raised, and the legs relaxed (94). They push their knees towards the floor, generating tension in the groin region (95). Alternatively overpressure can be generated by the pressure of the elbows upon the knees (96). This stretch allows an easy comparison to be made between the injured and non-injured side.



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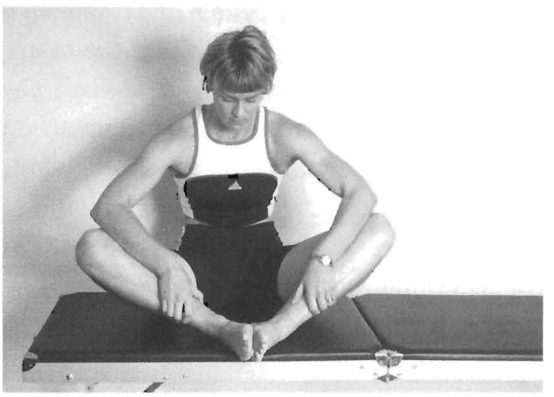

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to sport the same story is repeated. This history of rest, then inadequate rehabilitation, and return to activity that produces re-injury does not facilitate maturation of the injured tissue, and the self-perpetuating vicious cycle of a chronic condition is produced (Gross 1992; Renstrom and Peterson 1980). Groin strains are rarely seen as acute conditions; chronic problems are more likely, in which dysfunction is the main problem with the tissue sensitised and deconditioned to normal use.

Management and self-treatment exercises

If seen during the acute phase, management involves the RICE protocol followed by gradual progressive exercises involving loading into resistance and to encourage tensile strength. See *Treat Your Own*

Strains, Sprains and Bruises (Lindsay *et al.* 1994) for detailed early management.

If seen later the patient should be told of the lengthy period of rehabilitation required to return the tendon to full function. If sporting activities are causing a lasting aggravation of symptoms these should be temporarily avoided while rehabilitation takes place (Gross 1992). This should start with isometric contractions and stretches. As in all dysfunctions these exercises should reproduce the patient's pain, but not in a lasting way. Isometric contractions can be done by squeezing a pillow or ball between the knees. Adductor stretches can either be done in standing in a lunge position, sitting with the soles of the feet together and pushing the knees down with the elbows, or using the FABER position. The programme should progress into dynamic exercises, first without resistance then with resistance once the isometric exercises are easily tolerated (see Lindsay *et al.* 1994). Performing adductor stretches before sporting activities is an essential part of preventative training that the individual should maintain in the future (Renstrom 1992).

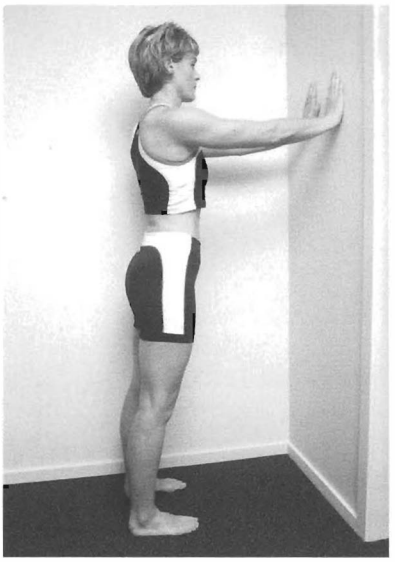
Achilles' tendon injuries

Achilles' tendon problems are common with recreational athletes (Allenmark 1992). They represent a range of tears, inflammation and degeneration of the tendon itself and/or the surrounding paratenon, which all benefit from a similar management approach (Allenmark 1992; Galloway *et al.* 1992). Although acute tears may go through the normal stages of inflammation, repair and remodelling in a straightforward manner, as with all tendon injuries healing may be retarded and the problem become chronic. The onset also may be insidious suggesting a degenerative or overuse component to the aetiology (Allenmark 1992; Galloway *et al.* 1992). The condition thus exemplifies many of the typical problems that complicate the healing responses of tendon injuries.

Typically the patient is able to localise the site of the problem; it is tender to the touch and sometimes swollen. Stretching the calf with dorsiflexion is painful and limited. This can be done against a wall with feet aligned at an equal distance from it, which allows comparison with the non-injured leg. The stretch should be done both with knees extended and flexed; this puts more tension on the gastrocnemius and soleus components of the muscle. Resisted movements will be

painful; getting the patient to perform heel raises first on both toes, then on the affected leg only, if pain permits, best tests this.

The pathology varies depending upon the stage of the disorder (Galloway *et al.* 1992). Early on, inflammation of the paratenon may be the key finding accompanied by granulation tissue, oedema and thickening. If this is allowed to heal the problem should be self-limiting, but working through the pain may cause scarring and structural disruption of the tendon. Degenerative changes can occur



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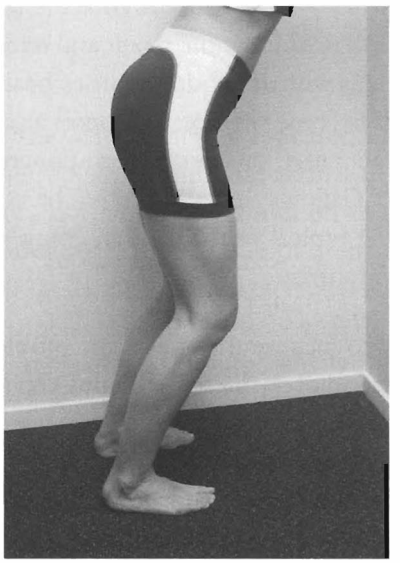


99

Photos 97, 98, 99
Calf stretch. The patient stands at arm's length from the wall, feet pointing forward and flat on the floor (97). Keeping the heels flat and the knees in extension they lean forward, stretching the gastrocnemeus (98). From this position, if the knees are allowed to bend by pushing the bottom out, still keeping the heels flat on the floor, the soleus is stretched (99). Each stretch should be repeated about ten times on each occasion. This stretch allows comparison between the injured and non-injured leg.

in the tendon – micro-tears, inflammation and fibrosis may promote weak and poorly organised scar tissue. These changes can accelerate further tendon degradation. There is an area of low vascularity two to six centimetres proximal to the calcaneus, and this may become vulnerable to ischaemic insult, which further weakens the structure. A complete rupture of the tendon represents the end point of the degenerative process (Galloway *et al.* 1992; Allenmark 1992). Such ruptures are always preceded by degenerative and structural changes, often those associated with hypoxia. Prior to spontaneous rupture many patients are completely asymptomatic and could recall no warning signal (Kannus and Jozsa 1991). This finding makes it clear that substantial degenerative changes can occur in the tendon which leave it seriously weakened without any previous symptomatic implications. While patients who incur other tendon ruptures are generally over 60, the average age of the patients with Achilles' tendon ruptures is 36 (Kannus and Jozsa 1991). There is still debate over whether the best treatment for Achilles' tendon ruptures is surgical repair or immobilisation in a plaster cast (Grisogono 1989).

Management and self-treatment exercises

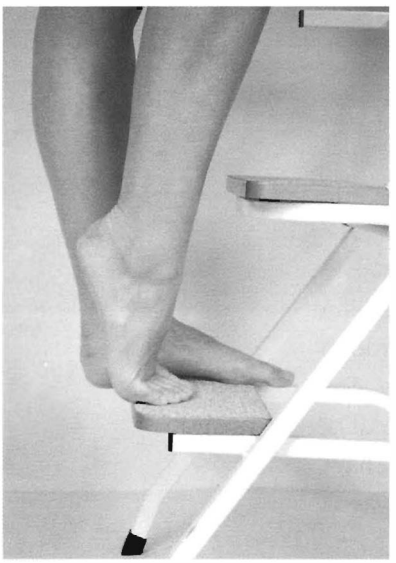
Acute injuries will need the **R**est, **I**ce, **C**ompression and **E**levation (**RICE**) protocol in the first few days and gentle range of movement, non-weight-bearing exercises from the second or third day. These should be progressed on to weight-bearing stretches and strengthening exercises. The timeframe for individual progression depends upon the severity of the damage, and is best gauged through assessment of symptomatic responses to loading strategies. See Lindsay *et al.* (1994) for more detail on acute injuries. NSAIDs, although widely prescribed for musculoskeletal overuse injuries, have not been conclusively shown to reduce recovery time (Galloway *et al.* 1992; Allenmark 1992).

One of the most important factors to promote healing in tendons is early functional activity; this accelerates parallel alignment of collagen fibrils and promotes tensile strength (Allenmark 1992). Thus the two key aspects of management are the stretching and strengthening components. Stretching the tendon with wall leans, knee flexion and extension should continue until the tendon feels the same as the non-injured leg. It is important to make clear to the patient that continuing stretching after the injury is a vital part of prevention of further problems (Galloway *et al.* 1992).

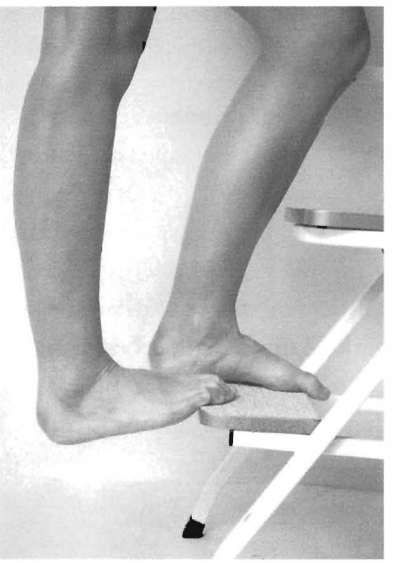
100

101

Photos 100, 101
Isotonic exercise for Achilles' tendon and calf; early weight-bearing. The patient starts with weight evenly spread on both feet, hands gently resting on the walls for support (100). They raise themselves onto their toes (101). To be repeated up to ten times.



102



103

Photos 102, 103
Single-leg eccentric loading of the Achilles' tendon during rehabilitation of a chronic problem. Start with the affected leg plantar-flexed and unloaded, here the right leg. The patient then transfers the weight onto the affected leg and lowers the heel over the back of the stair or chair, providing eccentric loading. This is repeated ten times. To apply only eccentric loading the other leg is used to return to the start position. The procedure is best applied on a staircase, but some patients prefer the stability of a high-backed dining chair. The same basic position can also be used for less loading by performing the exercise on both feet, or to do both concentric and eccentric loading by using the affected leg to return to the start position.

Early strengthening non-weight-bearing exercises in a sub-acute injury can be done using flexi-bands. These should progress to toe-ups when tolerable – that is, no lasting pain is provoked. Initially these are done on both feet, with support from an article of furniture if needed, and then progressed onto the injured leg only. This can be progressed by performing the same exercise with the heels over the side of a stair. In this way concentric and eccentric exercises can be done throughout the entire range. This can be done initially on both

legs, and then only one as tolerated. It is important that eccentric exercises are part of the rehabilitation process. These exercises are the optimal way to get strength gains (Fyfe and Stanish 1992), and as eccentric contractions are thought to have a prime role in causing tendon injuries it is important to specifically strengthen the muscle in that mode (Galloway *et al.* 1992).

Many patients are seen with chronic Achilles' tendon problems. These need to be addressed in the usual way with contractile dysfunctions. The ability of the tissue to stretch and contract needs to be restored. This can be done using the regime of stretching and strengthening as outlined above.

It has also been shown that a programme of eccentric training is highly efficacious in chronic Achilles' tendonosis which has not responded to other conservative treatments and when surgery was being considered (Alfredson *et al.* 1989). These patients, all recreational athletes, had symptoms for an average of a year and a half and had not benefited from rest, NSAIDs, orthotic devices, physiotherapy or ordinary training programmes. Exercises were performed twice daily, every day for twelve weeks, during which time running was permitted only if it provoked minimal or no discomfort. The programme consisted solely of eccentric exercises – the non-injured leg was used to return to the start position. They were done with heels over the edge of a stair progressing from both feet, to one foot, to increased loads (by adding weight in a backpack) as the previous exercise became pain-free. After the twelve-week training programme all fifteen patients were back at their pre-injury level of running activity, the calf muscle strength had returned to being similar to the non-injured leg, and pain was now minimal. A comparison group who also failed to improve with conventional treatment and came to surgery took twice as long to return to full running activity (Alfredson *et al.* 1989).

Patellofemoral joint pain

Knee pain is a common complaint among the general population, with prevalence across all age groups of between 10 – 15% (Badley and Tennant 1992; Cunningham and Kelsey 1984; Hadler 1992b). Osteoarthritis (OA) only accounts for a proportion of these knee pains; the rest are undiagnosed or non-specific. Within the older population

(over 55) isolated symptomatic patellofemoral joint OA has been estimated to affect 8% of women and 2% of men (McAlindon *et al.* 1992). The role of the patellofemoral joint in younger groups is unclear with the non-specific classification of 'knee pain' not differentiated into aetiological groups. Abnormalities of the joint have accounted for about 10% of knee assessments by arthroscopy (Lindberg *et al.* 1986). In a sports injury clinic 25% of all knee problems (N = 549) were related to the patellofemoral joint (Devereaux and Lachmann 1984). This limited epidemiological information suggests that the joint is frequently involved in knee problems, but that a more precise estimate of its role in 'knee pain' as a whole awaits further elucidation. The issue is further confused by the uncertainty surrounding validity and reliability in determining a specific diagnosis.

Patellofemoral joint problems or anterior knee pain is generally distinguished from chondromalacia patellae and osteoarthritis. Symptoms of anterior knee pain can arise without the pathological changes to the cartilage or bone which are characteristic of chondromalacia patellae or osteoarthritis (Insall 1979, 1982; Fulkerson and Shea 1990). These changes, though, are frequently incidental findings in the normal population (Stougard 1975; Insall 1982). Thus pain can exist in the absence of pathoanatomical changes, but equally morphological changes do not always predict symptoms. In those with anterior knee pain it is likely that there is a continuum from patellofemoral joint pain without pathoanatomical changes to chondromalacia with actual softening, fissuring and fasciculation of the cartilage.

It is widely stated that the symptoms of patellofemoral joint pain arise due to malalignment of the patella in the femoral groove (Insall *et al.* 1976; Insall 1979, 1982; McConnell 1986; Fulkerson and Shea 1990). The patella is a sesamoid bone within the quadriceps and its equilibrium within the groove is always open to the influence of static and dynamic factors at or about the knee (Schutzer *et al.* 1986; Kettelkamp 1981):

- the configuration of the patella may be abnormal
- there may be deficiencies in the supporting soft tissues about the patella
- there may be abnormalities in part of the lower limb

Three abnormal configurations of the patella have been identified. Symptomatic patellae have been seen to be laterally shifted, laterally tilted, or both laterally shifted and tilted (Schutzer *et al.* 1986; Fulkerson and Shea 1990). The height of the patella from the tibial tubercle may also be excessive, a finding known as patella alta, predisposing to excessive lateral shift (Insall 1982). It has been suggested that excessively tight lateral retinaculum may tilt the patella, and that insufficiency of vastus medialis obliquus may contribute to lateral shift (Fulkerson 1983; Fulkerson and Shea 1990; McConnell 1986; Insall 1982). Factors external to the knee have also been blamed for some of the symptomatology. Commonly stated abnormalities of the lower limb are increased Q angle, excessive pronation of the subtalar joint, and tightness of rectus femoris, the illiotibial tract, hamstrings or gastrocnemius (Kettelkamp 1981; Insall 1979; McConnell 1986; Hilyard 1990). Combinations of abnormalities are not unusual (Kettelkamp 1981).

Not all these factors have a proven role in the pathogenesis of anterior knee pain. For instance, the role of the illiotibial tract has been questioned (Rouse 1996). The aetiological significance of some of the other factors is by no means established. The variety of factors that different authors speculate are significant in the aetiology of patellofemoral problems undermines their clinical usefulness. The poor-to-fair reliability of four commonly used tests to judge for patellofemoral alignment further compromises clear recognition of a distinct disorder (Fitzgerald and McClure 1995).

However, the key factor in symptom production would appear to relate to abnormal mechanical forces at or about the knee that alter the posture of the patella statically or dynamically and consequently cause excessive loads through the joint. A thorough examination must include all the mechanical abnormalities that ***may*** have a role in symptom production. Not all these factors may be open to change, and of those that are a prolonged commitment from the patient is required to bring about a change. Perceived abnormalities may also be found in those without symptoms. Furthermore, abnormal mechanical forces are exacerbated during loaded flexion activities, which magnify the patellofemoral joint reaction force. For instance, knee-bending or climbing stairs causes force through the joint at 2–3 times body weight, while walking on a level surface the force is half body weight (Nordin and Frankel 1989b).

Where is the pain coming from? The patella cartilage is the thickest in the body (Evans 1986); cartilage is aneural and therefore incapable of being the source of symptoms. It is thought that pain might be caused by increased stresses on the subchondral bone (Goodfellow *et al.* 1976b; Gruber 1979). More specifically, it has also been proposed that patellar pain results from high intra-osseous levels of pressure which cause painful abnormalities in the vascular system in the bone (Arnoldi 1991). These intra-osseous pressures are magnified during flexion.

Although patients with severe stages of chondromalacia patella may show signs of inflammation involving the cartilage, the synovium or the plica (Arnoldi 1991), generally synovitis is a rare and inconsistent finding even in those with symptoms severe enough to seek surgery (Insall *et al.* 1976; Gruber 1979). Therefore in many patients pain is not the product of inflammatory chemicals but is due to abnormal postural mechanics. A combination of tight soft tissues and/or altered alignment of the patella generates points of excessive pressure. Ultimately this may cause the gross changes to the cartilage termed chondromalacia. When found, these are principally located roughly in the centre of the patella extending medially and laterally (Insall *et al.* 1976).

Contact areas and pressures between the patella and the femur alter with the flexion angle of the joint. As the joint moves from extension to flexion, the contact area moves from the inferior to superior aspect of the patella (Goodfellow *et al.* 1976a; Huberti and Hayes 1984). The area where damage tends to occur is in contact at about 60 to 90 degrees. Contact pressures are also highest between 60 and 90 degrees (Huberti and Hayes 1984). Thus the abnormal biomechanics are exacerbated during flexion loading activities to generate a focus of excessive pressure on the patella, which ultimately causes symptoms. The problem thus has a dynamic component. Vastus medialis obliquus is the only dynamic stabiliser of the patella, and thus insufficiency of this muscle will increase lateral shift (McConnell 1986).

Clinical presentation

The typical patellofemoral joint patient is a young person, from teenager to twenties, without a history of injury, and more likely to be female. Symptoms may have been present for many months or

even years, and although usually felt around the patellar, they are sometimes referred to the posterior of the knee also. A large minority of patients have bilateral symptoms. Activities of flexion are typically painful. This may present as immediate pain on loaded activities, such as squatting or ascending/descending stairs, or as pain brought on by sustained loading, such as maintaining knee flexion at 90 degrees while in the cinema. Patients may describe the gradual onset of pain after sustained activity. Only sometimes is trauma involved in onset. Pain may be fairly constant or clearly related to activities.

On examination findings are often minimal. Normally active range of unloaded movement is full, although there is often pain on squatting. When it comes to determining the presence of shift, tilt and rotatory malalignment, reliability between clinicians is only poor to fair (Kappa = 0.1 – 0.36) (Fitzgerald and McClure 1995). Other suggested findings include increased pronation, increased Q angle, illiotibial tract tightness, squinting patellae, patella alta, hamstring tightness, or patella shift or tilt. Resisted extension at *some* point in the range is normally but not always painful; compression of the patella onto the femur may provoke pain at certain angles; and there may be tenderness on palpation of the underside of the patella. Often there is a paucity of abnormal findings on physical examination, with the information gained from the history-taking most clearly indicating anterior knee pain (McConnell 1986; Insall *et al.* 1976; Insall 1979; Gerrard 1989; Bentley and Dowd 1984).

Natural history

The natural history of this condition has not been much studied, but evidence suggests a prolonged history is not uncommon in many patients. Karlson (1939) studied a group of patients for up to twenty years who had received operative and non-operative treatment, with a diagnosis of chondromalacia patellae. Over a third of the surgical group and 86% of the conservatively treated group continued to have symptoms and functional loss. Only a few deteriorated, but 22% of the non-surgical group had severe symptoms. Hvid *et al.* (1981) also studied those with a diagnosis of chondromalacia patellae of varying grades for three to eight years. The majority were worsened or unchanged, with a poor prognosis being associated with clinical or radiographic markers of abnormal patellofemoral biomechanics. Robinson and Darracott (1970) found that 71% of their group had moderate to severe disability from knee pain two to six years after

the condition had started. Devereux and Lachmann (1984) followed up a group of athletes with patellofemoral pain for about a year after conservative therapy and found that only 29% were entirely free of symptoms at that time. In summary, from these long-term studies only about a quarter of patients become completely pain-free in this condition.

The natural history of a specific sub-group, adolescent girls, has been followed over two to eight years (Sandow and Goodfellow 1985). The majority still had symptoms, but in over 80% this was weekly or less frequent; in the majority restriction of sporting activity was occasional or not at all. Nearly half reported themselves better over the follow-up period, about 40% the same, and only 13% reported a worsening of pain severity.

From these reports it would seem that patients could have very persistent symptoms, but that the majority do not progress. Female adolescents in particular can have a reasonably good prognosis in the long term, with little functional restriction and only infrequent pain. However, it would appear that in other groups the prognosis is not so good. Most will have persistent symptoms, and unless the abnormal biomechanics of the joint can be altered these are unlikely to change. Indeed, many patients are described who have had symptoms that have persisted for years (McConnell 1986; Gerrard 1989; Sandow and Goodfellow 1985).

Management

Conservative management has traditionally been based on rest during acute phases, avoidance of flexed, loaded knee positions, straight leg-raising quadriceps strengthening exercises, NSAIDs, and occasional use of bracing or immobilisation (Fisher 1986; Kettelkamp 1981; Insall 1982; Fulkerson and Shea 1990). The value of NSAIDs is unproven – one small trial comparing aspirin to placebo found no significant differences between the two groups (Bentley *et al.* 1981). The effectiveness of exercises is said to be unquestionable, but the mechanism for their effect is unclear. Exercises should not be painful and must emphasise vastus medialis obliquus (Insall 1982). More recently stretching of soft tissues around the knee has also been thought to be important (Fulkerson and Shea 1990). Controlled trials of these interventions have not been done, so their true worth is impossible to substantiate.

McConnell (1986) proposed a similar, but more focused management approach. This involved stretching any tight structures, such as the illiotibial tract, lateral patella retinaculum or the hamstrings; correction of any malalignment of the patella (shift, tilt or rotatory component) using tape; and then progressive training of the vastus medialis obliquus to maintain improved control over the patella. Exercises are done in a loaded position, must only generate minimal discomfort, if any, and must include eccentric muscle work. Two uncontrolled studies of a large series of patients report good results in over 80% of the patients (McConnell 1986; Gerrard 1989). Improvement persisted at one year as long as patients maintained the exercises. Response was particularly good and rapid in those whose symptoms could be provoked by stairs, squatting or resisted quadriceps, which was improved after taping. By the end of treatment symptomatic responses to a range of tests became pain-free.

Mechanical diagnosis and therapy

Patients with patellofemoral pain problems do not fit neatly into one of the three mechanical syndromes; neither do these patients generally have inflammatory pain. There is some confusion over aetiological factors and problems with tests used to make this diagnosis. It is unclear if non-specific knee pain is unwittingly being included in this group, as the criteria for inclusion are so broad and often there is so little to see. As in most conditions the history frequently plays the most important role in implicating the patellofemoral joint, with exacerbation of symptoms on activities of flexion being the most consistent feature. Lack of clear physical findings obscures the direction management should take. There is no certainty concerning various issues that relate to this condition.

However, frequently these patients have a persistent postural abnormality which comes to generate an area of sensitised tissue on the under surface of the patella. The abnormality may be tight lateral structures and shift or tilt of the patella, or some combination of the factors outlined above. Often this will only produce symptoms during or even after activity. In other words, minor biomechanical abnormalities cause symptoms in the dynamic phase of knee function – this is a dynamic postural syndrome. Ultimately this may progress to constant pain, when the prognosis is poor. The abnormal postural mechanics that come to generate symptoms needs a lasting change to improve the condition. This can only happen over time. Tight

structures will have to be stretched and vastus medialis obliquus function will have to be improved. The sensitised area of the patella may need time to return to normal.

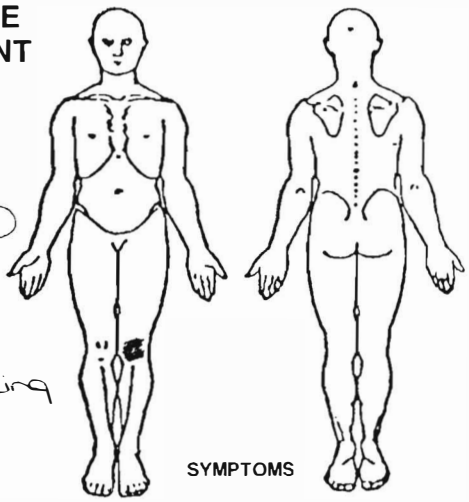
Throughout this process patient commitment is essential. This can only be based on excellent information provision by the clinician and avoidance of any passive modalities, the use of which denies the pivotal role that the patient plays in rehabilitation.

Self-treatment exercises

Management of anterior knee pain essentially involves the normalisation of any tight local and lower limb structures, and secondarily optimisation of vastus medialis function. Stretches may need to be applied to lower limb muscle groups, the iliotibial tract or the lateral retinaculum of the patella, as is applicable following the physical examination. Training of the quadriceps must be done in weight-bearing and dynamically, without aggravation of symptoms. Mini-squats and lunges onto the affected leg achieve this; if tolerated, squatting can be performed with minimal support from the other leg. Start exercises several times daily. If these are being performed without problems, progression involves the use of a stair – stepping up and down can be performed with the affected leg doing the main work each time; stepping down is usually more difficult. Lasting pain on any exercise is a contraindication to that particular loading, as pain inhibits strength gain.

Figure 10.5 Assessment sheet – anterior knee pain

THE McKENZIE INSTITUTE PERIPHERAL ASSESSMENT



Date ..... / ..... / .....

Name ..... M / F

Address .....

Date of birth ..... / ..... / ..... Age 16

Work / Leisure ..... school girl .....

Postures / Stresses ... sitting alot

sports : hockey, basketball, dancing

Functional disability from present episode .....

sometimes missing sports, not regularly .....

HISTORY

Present symptoms ..... left anterior knee pain .....

Present since ..... / ..... / ..... one year, coming & going Improving / Unchanging / Worsening

Commenced as a result of ..... or no apparent reason

Symptoms at onset ..... same .....

Constant symptoms ..... Intermittent symptoms most days 0% – 50%

What produces or worsens ..... sitting with knee flexion (30 minutes); squatting; sometimes after sports; stairs (down > up); walking (> 1 hour); running (> 10 minutes)

What stops or reduces ..... holidays; knee extension .....

Continued use makes the pain Better Worse No Effect

Pain at rest Yes / No

Disturbed night Yes / No .....

Other questions ..... swelling / locking / giving way — none .....

Treatment this episode ..... none .....

Previous episodes ..... none .....

Previous treatment .....

Spinal history ..... none .....

Paraesthesia Yes / No

Medications: tried ..... NSAID, 2 weeks Effect ..... no effect .....

Present medication ..... none .....

General health ..... good .....

Imaging ..... none .....

Summary: Acute / Sub-acute / Chronic Trauma / Insidious onset

Sites for physical examination .....

knee – patello-femoral joint .....

PERIPHERAL Knee

Observation NAD

Active movement Loss PDM ERP

Knee - flexion / extension - nil loss

Stretches - hamstrings, calf, quads, lateral thigh - Right = Left

Passive movement Loss PDM ERP
(+/-over-pressure)

Knee - flex, ext, lat & med rotation, valgus & varus -
no loss, no pain

Resisted tests Response Knee extension - 60° - 90° - pain & weak
Hamstrings - NE

Effect of repeated tests on pain: produces, abolishes, increases, decreases,
radiates, localises, better, worse, no better, no worse, no effect

SLR - NE; mini-squats - produce no worse
Lunge - NE; Step-ups - produce, no worse; step-downs - produce worse

Effect of repeated tests on movement: Better Worse (No Effect)

Static Positioning Effects patella compression - pain

Loaded / Unloaded squatting - moderate loss R.O.M
Unloaded flexion - nil loss

SPINE

Movement Loss

Effect of repeated movements:

Effect of static positioning:

Spine testing: (Not relevant) / Relevant / Secondary problem

CONCLUSION (Peripheral) anterior knee pain Spine

Dysfunction : Articular Contractile

Derangement Postural

(Other) Uncertain

PLAN

Exercise Frequency

SLR / mini-squats / lunge / step-ups : < 10 x 3x daily

Avoidance of sustained knee flexion

Degenerative joint conditions / osteoarthritis

The prevalence of osteoarthritis increases linearly with age from about 30, before which the disease hardly exists, with the average age of onset being 55. Before 50 men are affected more than women, but after this age the prevalence in women is two or three times greater than men (Felson and Zhang 1998; McCarthy *et al.* 1994). Symptoms are varied – pain, aching, swelling, bony tenderness and enlargement, activity-related pain, morning and inactivity stiffness, crepitus and painful limitation of movements (Oliveria *et al.* 1995; Huskisson and Hart 1987; Altman *et al.* 1986). The presence of osteoarthritis (OA) is confirmed by radiography; however, large numbers of individuals with the radiographic changes of OA do not have symptoms.

Visible changes on x-ray increase with age and are almost universal after 55, but it is estimated that only about 50% of individuals with these changes have symptoms (Huskisson and Hart 1987). For instance, whereas 80 – 90% of the population over 70 have radiographic changes in the hands, only 9% of males and 26% of females have pain (Petersson 1996). Radiographic knee OA was found in 20% of a large population survey, while only 5% had these changes and knee pain at the time of the survey (Claessens *et al.* 1990). Painful knees accompanied by x-ray changes occur in 6% of adults over 30, while radiographic changes are found in about 14 – 30% of those over 45 (Felson and Zhang 1998; Petersson 1996). Symptomatic hip OA occurs in 0.7 – 4.4% of adults, while about 20 – 30% of white Europeans over 55 display radiographic hip OA – the prevalence is much lower (1 – 3%) in non-Caucasian populations (Felson 1988; Felson and Zhang 1998).

Most studies show that the proportion of radiological disease that is symptomatic is between 40 and 80%. This depends partly upon the severity of the signs and the site – severely damaged joints are more likely to be painful than mild changes, knees and hands are more likely to be painful than hips. Many people with signs of OA do not have pain or have episodic or intermittent pain; many without signs of OA can have painful joints. The key message to patients is that radiographic evidence of joint changes does not correlate closely with symptoms (Hochberg *et al.* 1989; Spector and Hart 1992; Dieppe 1989; Forman *et al.* 1983; Acheson 1982).

Myths abound concerning OA, one of which is the inherently progressive nature of the disease. In fact not all patients progress

radiographically or symptomatically, although many do. In a substantial proportion of those with confirmed OA the symptoms either get no worse or actually improve over time (Hochberg 1996). Several studies have followed up patients with knee OA for about a decade or more. In one study twenty patients with OA knee got worse, seven remained the same and four got better (Massardo *et al.* 1989). In a long-term study, 57% of patients reported a worsening of symptoms, but 25% were unchanged and 18% were improved (Hernborg and Nilsson 1977). In another study with an eleven-year follow-up, less than half the patients showed a worsening of radiographic findings, while about 10% showed improvement. Although half thought their pain had worsened, their visual analogue pain scales were the same, and those reporting knee pain declined from 69% to 52% (Spector *et al.* 1992). These authors concluded, *"It appears that the long-term prognosis in a large proportion of patients with osteoarthritis is good. Only a minority are likely to progress rapidly and have a deterioration in their knee symptoms."* OA is neither an inevitable part of the ageing process nor necessarily progressive. While about 50% of those over 60 have symptoms of OA, this proportion remains reasonably stable through the 70s, 80s and 90s (Forman *et al.* 1983)

The hands, feet, knees and hips are the peripheral joints most commonly affected by degenerative changes. Ankles and shoulders are involved less frequently, and elbows and wrists only occasionally (Petersson 1996; Huskisson and Hart 1987). The cause of this predilection for certain joints is unknown. It is common for individuals to be affected in several sites.

OA is characterised by areas of local destruction of the articular cartilage and remodelling of the subchondral bone. If the joint damage is extensive enough these changes are seen on x-ray as narrowing of the joint, sclerosis of the bone and growth of osteophytes. Much of the pathology of OA is seen as an attempt at repair, or an aberrant version of the same. The articular cartilage, which is the main target tissue of OA, is devoid of nerve endings which may explain the lack of pain in many patients, especially those with only mild disease (McCarthy *et al.* 1994; Huskisson and Hart 1987).

Pain is thought to arise from the subchondral bone, periosteum, synovium, capsule and ligaments resulting from abnormal mechanical stresses on the peri-articular structures. Initially there is aching of

joints at rest often relieved by movement. As the disorder develops pain becomes related to use, being especially noticeable on initiation of movement or weight-bearing. There is fibrosis, thickening and contracture of the capsule causing stiffness, reduced movement and pain at the end of range. Restricted venous outflow causing stasis and congestion can also be a factor in pain production. Aberrant loading can cause excessive mechanical strain on ligaments, possibly leading to micro-trauma.

At the more extreme end of the continuum of pathological changes fragmentation of the cartilage or bone can lead to the formation of intra-articular bodies that may cause mechanical or inflammatory symptoms due to irritation of the synovium. There may be episodes of inflammation due to synovitis, causing a constant chemical pain. Noxious stimuli from diseased joints can cause inhibition of motor neurones leading to weakness and muscle atrophy. Muscle weakness and damage to ligaments may induce unstable joints that are vulnerable to trauma, and thus prone to episodes of acute injury, inflammation and post-traumatic pain. Persistent pain can lead to sensitisation at local and central levels resulting in a lowering of the pain threshold (McCarthy *et al.* 1994; Huskisson and Hart 1987; Preidler *et al.* 1996; Akeson *et al.* 1989; Threlkeld and Currier 1988; Marks 1992).

The cause of symptoms from degenerative conditions is therefore multifactorial, and represents a spectrum involving mechanical, inflammatory, ischaemic and other factors. It is suggested that OA is not in fact a single disease, but is an umbrella term for a range of similar conditions whose aetiology may be primarily systemic or biomechanical (Dieppe and Kirwan 1994). It is quite likely under these circumstances that different presentations may respond to different management strategies. Some patients having symptoms from OA may present with purely mechanical problems – for instance, end-range capsular stretching reveals limitation and pain. In other patients non-mechanical factors may be paramount. As in all persistent tissue abnormality the role of sensitisation and deconditioning from lack of normal use should be considered.

Management of degenerative joint conditions

Management guidelines for OA frequently stress the value of education and exercises (McCarthy *et al.* 1994; Dieppe 1993, 1995; Hochberg 1995a, 1995b). Exercise is the only physical modality that

has consistently been found to be valuable in the management of OA (Clarke 1999). A 'pyramid' approach to the management of OA has been proposed, in which all patients should have access to interventions at the base of the pyramid, whereas only the few with the severest symptom will need those at the apex (Dieppe 1995; Hochberg 1995a, 1995b). Management strategies to be used with the majority of patients include education, counselling, empowerment, exercises, sticks, physiotherapy and analgesics. A minority of patients may need NSAIDs, hydrotherapy and more complex aids, while a few will need surgery (Dieppe 1995).

The therapeutic value of NSAIDs for OA is not clear despite their routine use. OA frequently has only a minor inflammatory component (Dieppe *et al.* 1993). It appears that simple analgesics are as good as NSAIDs (Towheed *et al.* 1999; Dieppe 1993; McCarthy *et al.* 1994). Bradley *et al.* (1991) compared ibuprofen at an anti-inflammatory and purely analgesic dosage, and acetaminophen (purely an analgesic) and found that all three groups had improvements in major outcomes, which were non-significant between the groups. Comparisons between NSAIDs and placebos or analgesics are considerably less researched than comparisons between different NSAIDs. It would appear that the drug companies are happier funding comparative studies between different NSAIDs than comparing them to much less costly analgesics, let alone non-pharmacological interventions (Dieppe *et al.* 1993). There is also concern that there is biased interpretation of results in favour of a certain product in trials associated with drug manufacturers (Rochon *et al.* 1994).

NSAIDs are also associated with considerable morbidity and mortality due to gastrointestinal damage. A meta-analysis concluded that NSAID users are three times more likely to develop serious adverse gastrointestinal events than non-users (Gabriel *et al.* 1991). It is estimated that 20 – 30% of all hospitalisations and deaths in the over-65s are due to peptic ulcer disease attributable to NSAID therapy (Hochberg 1995a; Bakowsky and Hanley 1999). It is estimated that there are about 200 deaths a year just in the UK attributable to their use (Somerville *et al.* 1986), and that they account for 25% of all drug side effects (Rochon *et al.* 1994). Because of the numbers of people using NSAIDs worldwide – 30 million people are thought to use them every day – adverse events arising from their use are more frequent than with any other form of drug treatment (Gabriel and Bombadier 1990; Haslock 1990). As a consequence of this debate it

is suggested that drugs, often of the wrong sort, are overused, whereas biomechanical interventions for the management of OA are underused (Dieppe 1993).

A positive attitude towards patients with this condition is warranted as symptoms often stabilise or improve, and there is not necessarily an inevitable progression with increasing morbidity and disability. Another of the myths of OA – that cartilage cannot repair itself – is disproved by contemporary evidence (Bland 1993; McCarthy *et al.* 1994). It is important to try to 'de-medicalise' the condition, reassure about the prognosis, and encourage control and empowerment (Dieppe 1995). As in all chronic diseases the patient has certain responsibilities – to learn about the condition, to take certain responsibilities, and evaluate the health care experience (Brady 1998). Current meta-analysis of studies suggests that patient education for rheumatological conditions can affect knowledge, pain, joint counts, depression, exercise and coping behaviours, with self-efficacy and social support appearing to be strong causal mechanisms for these benefits (Lorish and Boutagh 1997). Increased self-efficacy, which is the patient's sense of ability to affect the consequences of disease, improve other outcomes such as pain and disability (Lorig *et al.* 1989). For instance, individualised programmes involving exercise, relaxation, appropriate use of affected joints and problem-solving resulted in better functional preservation, improved pain, fewer health care visits and less depression (Lorig and Holman 1989; Lorig *et al.* 1993; Mazzuca *et al.* 1997). The clinician's role is obviously to offer information about the condition and self-management strategies that will affect its consequences.

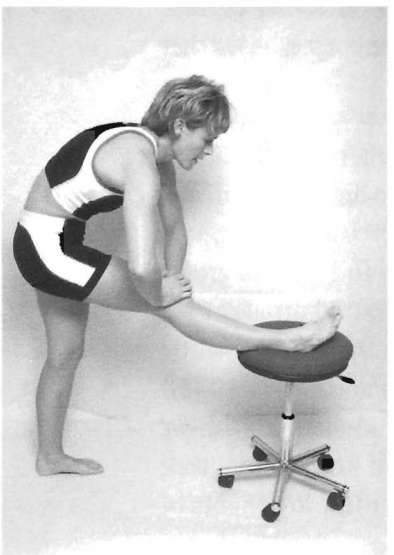
A systematic review of non-invasive studies into therapies for OA of the hip and knee found that exercise reduces pain and improves function in patients with knee OA, but generally failed to support passive treatment modalities. No studies were found by this group that evaluated exercise for OA of the hip. Of the 600 articles they initially reviewed over 60% related to treatment by NSAIDs, while only 5% looked at non-medical evaluations (Puett and Griffin 1994). Since then a trial comparing medication and education with or without supplementary exercises for OA hips and knees found that the exercise group, after twelve weeks, had greater improvements in reduction of pain and disability (van Baar *et al.* 1998b). Home exercises for the hip are as effective as outpatient hydrotherapy (Green

et al. 1993). Another wide-ranging review also found little evidence in favour of passive treatment modalities, but consistent value in strengthening exercises for the knee (Clarke 1999). An example is a supervised fitness walking and education programme that improved functional status and pain, and reduced medication use compared to a control group (Kovar *et al.* 1992). Participation in either an aerobic or resistance exercises programme brought about improvements in measures of disability, physical performance and pain compared to an education programme (Ettinger *et al.* 1997). Maintaining a full range of movement and muscle strength at an affected joint may reduce disease progression as well as greatly decreasing pain and disability (Dieppe 1995; McCarthy *et al.* 1994; Fisher *et al.* 1991).

Self-management exercises

Patients with degenerative joint conditions may present with restricted movements with pain at the end of range. This is due to capsular thickening and contracture of the joint capsule (McCarthy *et al.* 1994). This contraction and loss of elasticity causes dysfunction, and in many cases even small improvement in the range of motion can result in reduction of pain. Improvements are especially possible when the end-range feels plastic rather than like concrete.

Photos 104, 105
Different methods of self-mobilisation into knee extension, with or without overpressure. Pressure on, pressure off, ten times.



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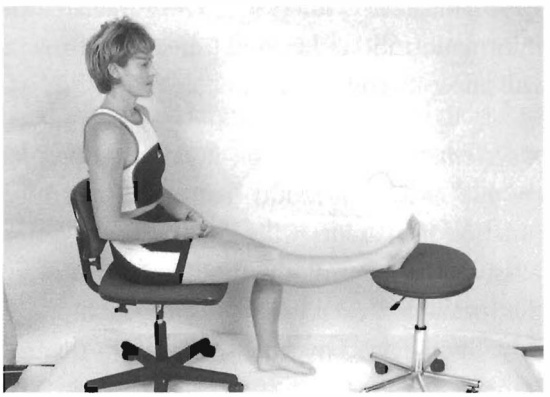

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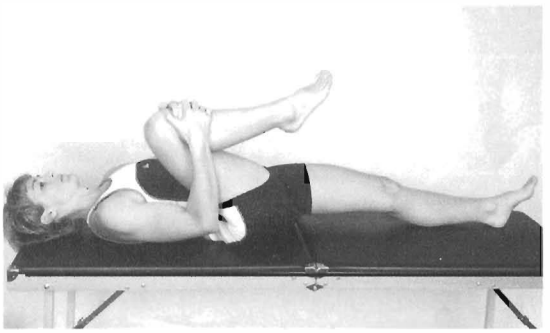
Photos 106, 107, 108
Different methods of self-mobilisation into hip flexion with overpressure. Pressure on, pressure off, ten times.



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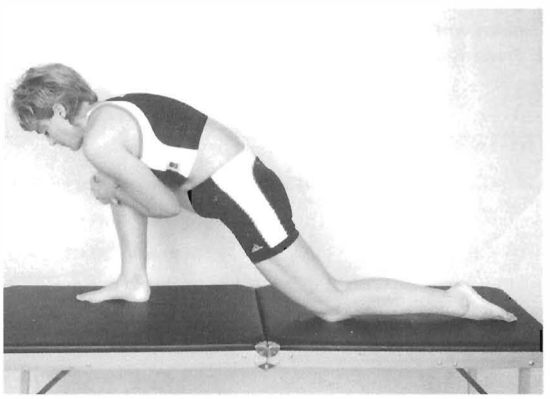

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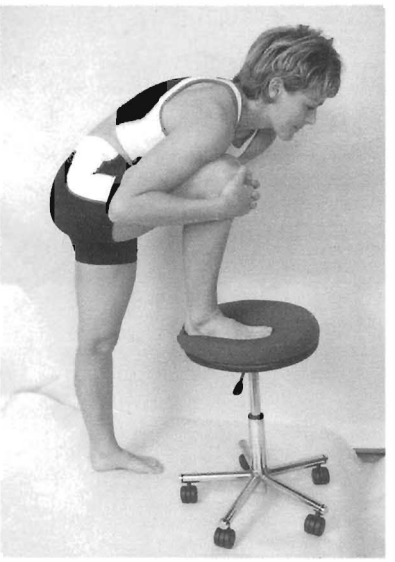

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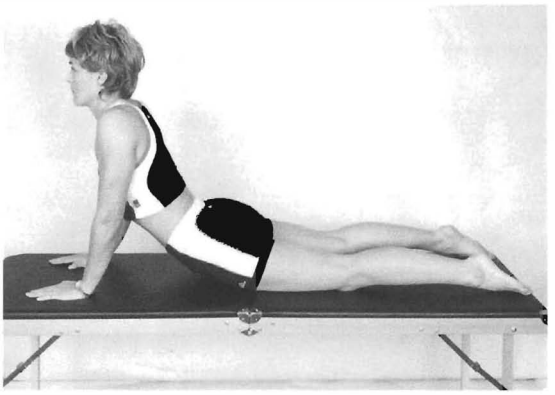
These problems are most commonly seen in patients with knee and hip OA. In hip OA different patterns of radiological and pathological changes have been observed (Cameron and Macnab 1975). They found that 40% of patients had early and marked capsular restriction, while in the rest capsular restriction was minimal until there were gross degenerative changes. It is suggested that the capsular changes are significant in the progression of the condition (Cameron and Macnab 1975). At the knee patients must be encouraged to regain and maintain extension, hyperextension and flexion. At the hip the key movements are again flexion and extension, but also full range of medial rotation and abduction are commonly lost (Dieppe 1995).

Self-management exercises can be supplemented by a short course of clinician mobilisations, with medial rotation at the hip sometimes being difficult for the patient to do adequately. Individual patients



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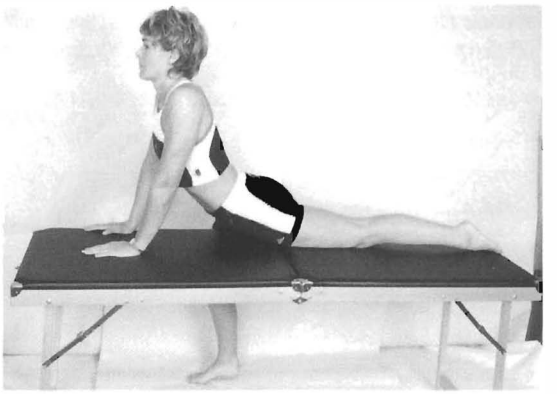

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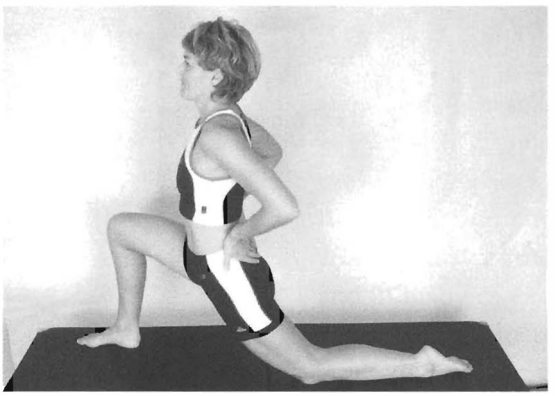

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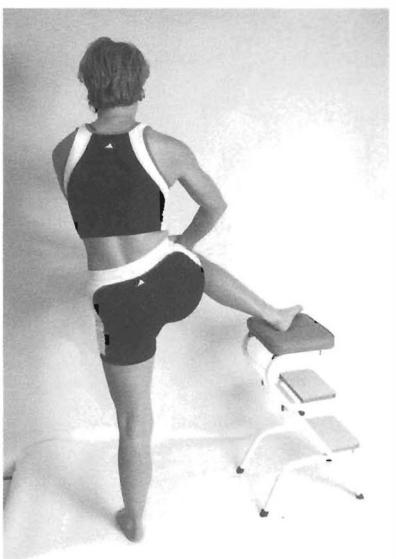
Photos 109, 110, 111
Different methods of self-mobilisation into hip extension with overpressure, bilateral and unilateral. Pressure on, pressure off, ten times.

Photo 112
Internal rotation of the hip, with or without overpressure. Pressure on, pressure off, ten times.



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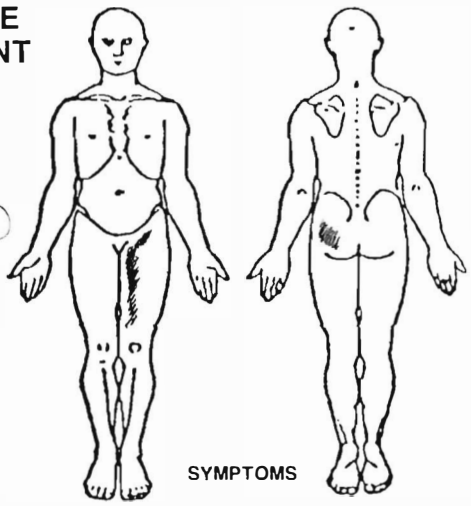
present with variations of movement loss and the particular movements that must be practised should be decided by physical examination rather than theoretical prescription. Patients will need to do the exercises with overpressure several times daily until improved mobility produces an improvement in symptoms. After this, exercises should be done on a daily basis to maintain the change

The previous analysis of the evidence makes it clear that a general strengthening or exercise programme is a vital part of the ongoing self-management of this condition. All patients should be advised on the value of performing such a programme, and clinicians should advise and discuss with patients the practicalities and problems of so doing.

Figure 10.6 Assessment sheet – hip joint OA

THE McKENZIE INSTITUTE PERIPHERAL ASSESSMENT



SYMPTOMS

Date ...../...../...../.....

Name ..... M / F

Address .....

Date of birth ...../...../..... Age 48

Work / Leisure .... Health visitor ; Sitting

Postures / Stresses ..... Driving

..... walks, swims

Functional disability from present episode .....

..... At work .....

..... Reduced walking .....

HISTORY

Present symptoms ... Left groin, buttock, anterior thigh

Present since ...../...../..... 10 months Improving / Unchanging / Worsening

Commenced as a result of ..... or no apparent reason

Symptoms at onset ... groin

Constant symptoms ..... Intermittent symptoms all intermittent

What produces or worsens ... walking (> few hundred yards) morning (½ hour)

... After sitting ; ... initial weight bearing ; squatting

What stops or reduces ... Sitting, lying, swimming

Continued use makes the pain Better Worse No Effect

Pain at rest Yes / No

Disturbed night Yes / No

Other questions ...

Treatment this episode ... Tablets

Previous episodes ... none

Previous treatment .....

Spinal history ... Previous episodes of back pain ; this is different.

... no back pain in last year Paraesthesia Yes / No

Medications: tried NSAID Effect ... no effect

Present medication ... none

General health ... good

Imaging ... slight degenerative changes - Lumbar spine & both hips

Summary: Acute / Sub-acute / Chronic Trauma / Insidious onset

Sites for physical examination .....

..... Hip (spine)

PERIPHERAL Hip

Observation

Active movement Loss PDM ERP

Passive movement Loss (+/-over-pressure) PDM ERP

Flexion - minimal loss ERP ; medial rotation 10° ERP
Abduction - 15° ERP ; Adduction flexion - mod loss

Resisted tests Response

NAD

Effect of repeated tests on pain: produces, abolishes, increases, decreases, radiates, localises, better, worse, no better, no worse, no effect

Flexion produced not worse
medial rotation produced not worse

Effect of repeated tests on movement: Better Worse (No Effect)

Static Positioning Effects

Loaded / Unloaded

SPINE

Movement Loss extension, flexion, side-gliding - nil loss

Effect of repeated movements: NE

Effect of static positioning:

Spine testing: (Not relevant) / Relevant / Secondary problem

CONCLUSION (Peripheral) Hip Spine

(Dysfunction) (Articular) early OA ? Contractile

Derangement Postural

Other Uncertain

PLAN

Exercise Frequency

Flexion + OP x 10 x 3/4
medial rotation + OP x 10 x 3/4

Lateral ankle sprains

Ankle injuries are common occurrences; 5,000 are estimated to happen every day in the UK and 23,000 every day in the US. Lateral sprains constitute over 90% of trauma to ankles. Individuals who sprain their ankles frequently attend hospital accident and emergency departments. They constitute 7 – 10% of those examined in emergency departments in Scandinavian hospitals, and over 3% of those visiting an emergency department in a UK hospital. Ankle sprains always have a traumatic onset, they are often sports-related injuries, and as a consequence the most common age group for this condition is under 35. They constitute over 2% of patients seen by Dutch physiotherapists, 6% of musculoskeletal problems attending GP clinics in the UK, and 20% of those attending an orthopaedic outpatient department. Recurrent injuries to the ankle are common (Hackett *et al.* 1993; Hockin and Bannister 1994; Simpson 1991; Kannus and Renstrom 1991; Roebroeck *et al.* 1998; Dunlop *et al.* 1986).

Of those attending accident and emergency departments, between 16% and 30% have sustained a fracture, either in the malleolar region, around the fifth metatarsal, or avulsion fractures. Pain and bone tenderness at certain sites and inability to bear weight are important clinical variables in predicting important fractures, and have been formulated as guidelines for selective radiographic examination of these patients (Dunlop *et al.* 1986; Stiell *et al.* 1995). Only a small proportion of patients had fractures that were not detected on their initial assessment at the hospital.

The most common sites of soft tissue sprains of the ankle are at the anterior talofibular ligament, with more severe trauma also involving the calcaneofibular ligament (Kannus and Renstrom 1991; Moller-Larsen *et al.* 1988). The severity of the injury can vary from slight tearing to complete rupture and gross instability at the ankle joint. The prevalence rate of the more extreme injuries appears to be unknown.

Management of ligament ruptures and sprains

The evidence provided by past trials and reviews of these studies is very clear in its recommendations. Even for complete rupture of the ligament a review of the literature recommends conservative treatment with early functional rehabilitation (Kannus and Renstrom 1991).

In the trials reviewed, 75 – 100% of patients had good outcomes at one year whether treated by repair and cast, cast alone or early controlled mobilisation. A short period of protection, early weight-bearing and range of movement exercises, followed by neuromuscular training of the ankle provides the quickest recovery to full function, and is no more associated with compromise of ankle stability than other interventions (Kannus and Renstrom 1991; Boruta *et al.* 1990; Ogilvie-Harris and Gilbart 1995; Renstrom and Konradson 1997).

Patients with less severe tears of the ligament recover quickly with a short period of immobilisation, RICE protocol and protection (see Lindsay *et al.* 1994), early active range of movement exercises, followed by weight-bearing strengthening and proprioceptive training (Kannus and Renstrom 1991; Ogilvie-Harris and Gilbart 1995; Renstrom and Konradson 1997; Shrier 1995). Using this management strategy with injured army cadets produced periods of disability of one to two weeks only (Jackson *et al.* 1974). Early mobilisation, compared to immobilisation in plaster, offers the most rapid return to normal functional activity (Brooks *et al.* 1981). Ice in the first few days is as effective as NSAID to reduce pain and oedema and promote early recovery, while there is limited evidence to support the use of ultrasound (Ogilvie-Harris and Gilbart 1995). In an acute group of patients with lateral ligament sprains, ultrasound was no better than placebo at reducing pain and swelling, and improving function (Nyanzi *et al.* 1999). It is widely agreed that with this early active management recovery is fast and the prognosis is good (Renstrom and Konradson 1997).

Medical and physiotherapy management of this condition does not always offer this optimal strategy to recovery. Pre-physiotherapy many patients are given no advice at all, or if they are this is often inappropriate, involving rest or non-weight-bearing exercise (Simpson 1991). In the US when surveyed about their management of this problem, doctors sometimes neglected advice on rest, ice, compression and elevation, and frequently failed to provide rehabilitation exercises (Kay 1985). Although Dutch physiotherapists use progressively more exercise therapy over the duration of treatment, ultrasound and short-wave therapy are still used in the last phase of treatment, and advice on activities and home exercises were at no point given to more than 15% of patients (Roebroeck *et al.* 1998).

Self-management

If patients are seen early, management progresses through the following stages:

Day 1 – Rest, ice, compression, elevation (RICE).

Day 2 – As Day 1, plus non-weight-bearing range of movement exercises, walking.

Week 1 – Non-weight-bearing range of movement and isometric exercises; possibly early weight-bearing exercises.

Week 2 – Weight-bearing exercises, progress stretching exercises, early balance exercises.

Week 3 – Progress weight-bearing exercises, stretching exercises, balance exercises, strengthening exercises.

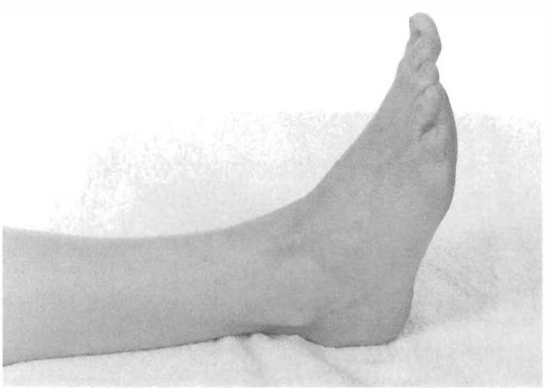
Week 4 – Functional rehabilitation.

The exact time scale that patients progress at depends on the severity of the injury and their response to exercise. These times provide a rough guide only (see also Lindsay *et al.* 1994).

Exercises need to stress the damaged ligament using a progression of forces. These can be done first actively and unloaded.

For a stronger stretch in the chronic stage, get the patient to use both hands to stretch into plantar-flexion and inversion, which should reproduce the patient's pain. Progressions involve weight-bearing

Photos 113, 114
Early unloaded active movements in lateral ankle sprain. Starting from a neutral position the patient moves into inversion ten times. They should stretch further and more forcefully each day.



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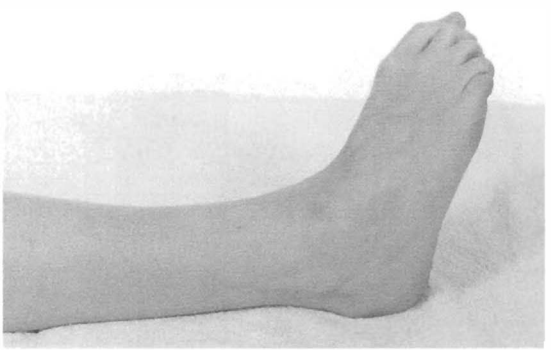
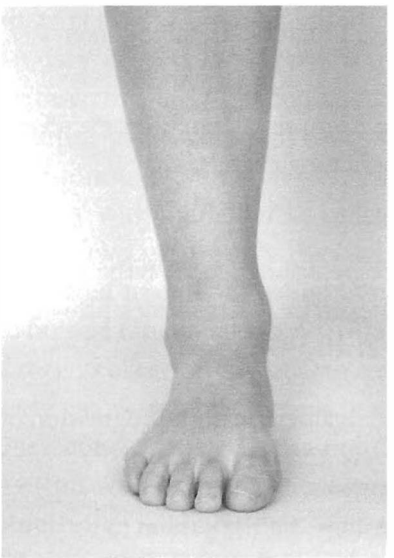


114

exercises, initially in bare feet and then with shoes, which in a controlled way stress the lateral ligament. Weight-bearing inversion should first be performed on the affected foot only, with the sound leg providing support. Later this can be performed on both feet, and then in shoes.

Photos 115, 116, 117, 118
Progression of loaded inversion stress for lateral ligament strain at the ankle. Start with foot flat, then the patient repeatedly inverts the foot ten times, causing eccentric and concentric loading. Performing the exercise bilaterally can be easier. To progress the exercises, perform the same exercise in shoes.



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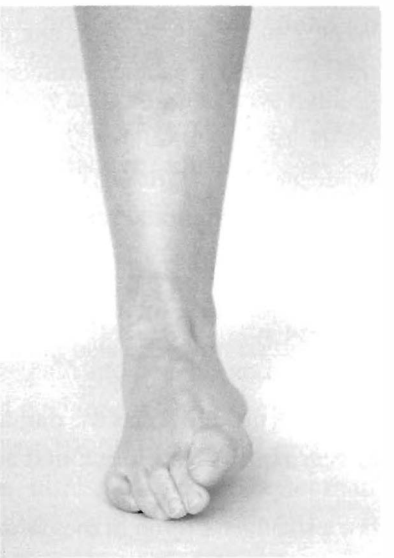

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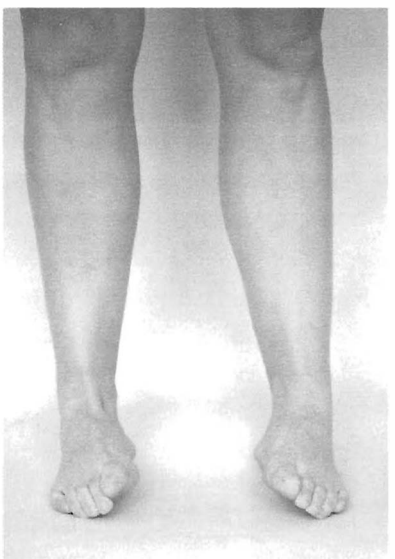

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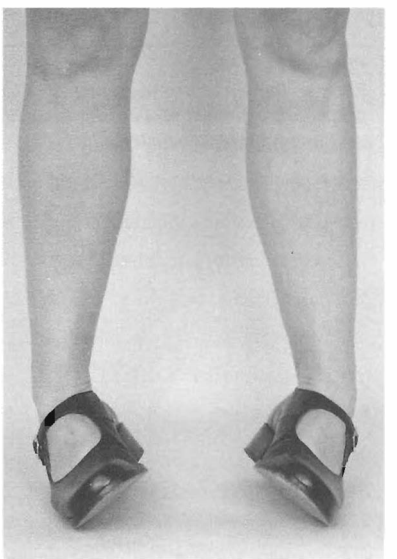

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Patients who present at a late stage may well display signs of dysfunction of the affected ligament. No histological changes have been found in chronic ligament problems except scarring (Renstrom and Konradson 1997). Following the acute inflammatory stage, adhesions may have been allowed to develop and now prevent full range plantar-flexion, inversion, and medial rotation of the foot. The appropriate self-stretches and loading exercises as outlined above should be performed.

Recurrent ankle sprains are common (Simpson 1991). Those who have had previous ankle sprains are twice as likely to have another injury compared to those with no previous history (Tropp *et al.* 1985). This may be due to decreased proprioception and/or perineal weakness (Boyle and Negus 1998; Boruta *et al.* 1990). Balance exercises, wobble-board work and general strengthening of muscles around the ankle are therefore an essential part of rehabilitation and prophylaxis against future sprains. Patients can perform appropriate exercises at home by standing on one leg with eyes closed, walking on tiptoes and heels, etc. Persistent problems of functional instability will be avoided in many patients if they are properly rehabilitated (Renstrom and Konradson 1997). In athletes, several months of coordination training or use of an orthosis had the effect of reducing recurrences dramatically, so that the incidence of recurrences was reduced to that of primary injuries (Tropp *et al.* 1985). Such treatment should be instigated for all patients, whether they are seen with an acute or chronic problem.

Figure 10.7 Assessment sheet – ankle inversion sprain

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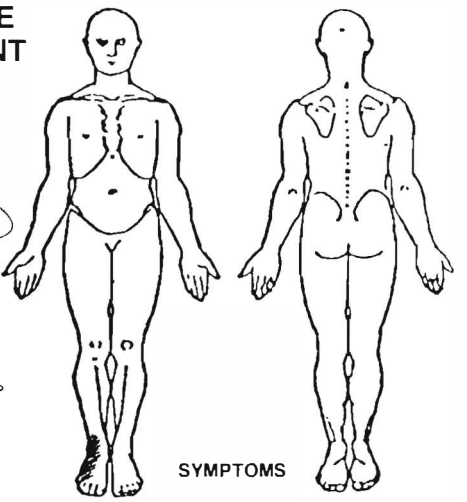

Date ...../...../.....

Name ..... M (F)

Address .....

Date of birth ...../...../..... Age 40

Work / Leisure Post Office; office work

Postures / Stresses Sitting mostly

walking., step aerobics

Functional disability from present episode

off work

off sport

HISTORY

Present symptoms Right lateral ankle / foot

Present since ...../...../ 5 weeks (Improving) Unchanging / Worsening

Commenced as a result of slipped off steps & inverted or no apparent reason

Symptoms at onset Immediate pain - ankle / foot; unable to weight bear, swell

Constant symptoms ..... Intermittent symptoms 90% of day

What produces or worsens all weight bearing - worsens

non-weight bearing movements. - painful, not worsened.

What stops or reduces A.M., rest

Continued use makes the pain Better (Worse) No Effect

Pain at rest (Yes / No) - mostly yes

Disturbed night Yes (No)

Other questions

Treatment this episode Tablets

Previous episodes x 2; both much less painful, for < 2 weeks

Previous treatment none

Spinal history none

Paraesthesia Yes (No)

Medications: tried NSAID Effect uncertain

Present medication NSAID

General health good

Imaging X-ray initially - no bony injury

Summary: Acute / (Sub-acute) Chronic (Trauma) / Insidious onset

Sites for physical examination

Ankle

PERIPHERAL Ankle - gait: abnormal, flat-footed; reduced dorsi-flexion

Observation minimal swelling lateral aspect (3pm)

Active movement Loss PDM ERP

Plantarflexion, dorsiflexion, inversion, eversion — all major loss ERP

Passive movement Loss PDM ERP
(+/-over-pressure)

All limited due to pain
Plantar flexion & inversion most painful

Resisted tests Response

All painful

Effect of repeated tests on pain: produces, abolishes, increases, decreases, radiates, localises, better, worse, no better, no worse, no effect

non-weight bearing active movements: increase no worse
weight-bearing - heel/toe raise increase no worse

Effect of repeated tests on movement: Better Worse No Effect

Static Positioning Effects

Loaded / Unloaded

SPINE

Movement Loss

Effect of repeated movements:

Effect of static positioning:

Spine testing: (Not relevant) / Relevant / Secondary problem

CONCLUSION (Peripheral) Ankle Spine

Dysfunction : Articular Contractile

Derangement Postural

Other lateral ligament & sub-acute Uncertain

PLAN

Exercise	Frequency	
NWB - active movements	x 10	x 3/4
Early WB active movements	x 10	x 3/4

11: Clinical Reasoning

Introduction

Clinical reasoning is the cognitive and decision-making process involved in clinical practice, which is used in the diagnosis and management of patients' problems (Terry and Higgs 1993; Jones *et al.* 1994). It is the process by which health care providers respond by logic and thought, rather than by routine and habit. Studies of clinical reasoning in the medical field have shown that it tends to be a process of hypothetico-deductive reasoning (Terry and Higgs 1993). This describes a process of hypothesis generation based on information gathered from the patient. The hypothesis is then tested out or further ones generated until a management pathway is clearly defined. Because hypotheses must be confirmed by responses to treatment, the process involves continual reassessment. In effect, every treatment is a form of hypothesis testing (Jones 1992).

An alternative model of clinical reasoning is based on pattern recognition (Jones 1992). Equipped with a well organised knowledge base, certain features in a clinical presentation remind the clinician of a previous pattern of presentation. Thus management strategies are derived from previous experience rather than an experimental 'try it and see' model. There are thus two models of clinical reasoning: hypothetico-deductive logic and pattern recognition. The latter is only possible with a well organised knowledge base and is only available to experts, while the former approach can be used by novices (Jones 1992). In the face of atypical problems, when pattern recognition is not possible, the expert reverts to hypothesis testing. As a means of determining management strategies, pattern recognition can have drawbacks. Failure to fully explore all options and bias for a favourite diagnosis can encourage premature foreclosure on alternative hypotheses. Pattern recognition, on its own, may be insufficient if it ignores certain complicating factors such as exaggerated fear–avoidance.

Clinical reasoning is informed by certain factors. These include data-gathering skills, aspects of the knowledge base available to the clinician, clinical experience and meta-cognition skills (Terry and Higgs 1993; Jones 1992; Jones *et al.* 1994). It is thus a complex and

cyclical process as suggested by the model of clinical reasoning for physiotherapy proposed by Jones (1992; Jones *et al.* 1994; Jones and Butler 1991). At every stage in this process errors may occur that could affect the reliability or validity of the reasoning process (Jones 1992).

On a practical level clinical reasoning is the process of deciding what problem the patient has and, from your knowledge base, deciding what can be done about it. First this involves the discovery of the 'character' of that problem – the patient is the best witness to this, and data-gathering skills must be capable of determining a clear 'big picture'. Second, a rounded knowledge base is needed to provide practitioners with an understanding of diverse factors such as a variety of clinical presentations, the natural history of a condition, pathophysiological changes, management strategies, the evidence base, the effect of an intervention, etc. The third and vital element is the ability to reason between the practical reality of the patient's problem and the available knowledge base. This involves constant interplay between theoretical concerns and clinical issues and a logical analysis of the effect of intervention strategies on the problem. Clinical reasoning is thus an essential element in the translation of clinical theory into clinical practice, and an exploration of its key elements is vital to make full use of any approach to a patient's problem.

Elements that inform the process of clinical reasoning

Data-gathering

This is the process of discovery about the patient's problem undertaken during the history-taking and the physical examination. The patient has available the essence of the problem; the skill is in accessing it. Certain aspects of questioning will be routine. Other elements may need developing for the individual patient and the evolving hypothesis. Questioning needs to be done in an open manner, rather than using leading questions. Communication skills are vital in this area; clinicians must provide an empathetic and listening approach to access the full information. Useful enquiry strategies include open-ended questions, forcing choices, minimal encouragement, repetition of the patient's language, summarising and silence. Closed questions may be used secondarily to probe in more detail or to clarify specific points. Research has shown that

manipulative physiotherapists overuse closed enquiry techniques, which limit patient responses, at the expense of open enquiry methods, which encourage patients to voice their concerns (Haswell and Gilmour 1997). From the history-taking an overall picture of the patient's condition should have been gained, and the direction that the examination is to proceed in should be suggested.

The physical examination is not a routine series of tests performed uniformly on every patient, but follows on directly from the data-gathering and hypothesis-testing of the history.
knee test on a patient who has knee pain is as unhelpful and as illogical as gathering every scrap of information about the patient's medical history. Data collection continues until a decision can be made about management strategies. The decision may be tentative, in which case further data will be gained at the next session and from the patient's response to the proposed management strategy (Jones 1992; Jones *et al.* 1994; Terry and Higgs 1993).

Knowledge base

Clinical practice requires a wide-ranging breadth of knowledge from different areas. However, for the knowledge to be of value in the elucidation of the patient's problem, it must have clinical relevance. Very often what is learned in training may have only a slight bearing on what is done in the clinic. What is significant and what is irrelevant is only learned through clinical experience. Theoretical knowledge must have practical significance for it to be of worth.

It is suggested (Jones *et al.* 1994) that the following topics are of relevance to the knowledge base of physiotherapy – anatomy, physiology, pathophysiology, procedures, patterns of clinical presentation and concepts. Also, hypothesis-making happens in six key areas – the mechanism of symptoms, differential diagnosis, predisposing or contributing factors, precautions and contraindications, management and prognosis (Jones and Butler 1991; Jones 1992; Jones *et al.* 1994). It is suggested that this is not a complete list; clinical practice also needs to be informed by knowledge about epidemiology, the research evidence, psychological issues and communication with patients.

Any of these factors, and more, may provide useful clinical information on different occasions. However, their value rests on

their clinical application rather than their theoretical strength. The value of some of the more important aspects of the knowledge base will be presented to exemplify their practical worth.

Epidemiology This area provides background knowledge about the prevalence of a condition and its likely natural history and clinical course. This information is limited in many musculoskeletal complaints. However, what is known makes clear that frequently such problems are very widespread and can have a persistent or recurrent history. This needs to be translated into an appropriate management strategy that recognises that such persistence may arise despite or even because of previous treatment. In effect, the outcome needs reformulating from how to 'cure' this patient or abolish their pain to the best strategies available for this person to manage their chronic disorder.

Evidence-based physiotherapy It has been said that because physiotherapy practice has been so poorly evaluated and rests largely upon theoretical hypotheses, spontaneous improvement due to the passage of time, and subjective impressions that *"today 'physiotherapy science' is still an oxymoron"* (Sala 1997). Research into current practice is certainly inadequate and often inappropriate. However, as a profession, physiotherapists must recognise that the future funding and allocation of scarce healthcare resources will ultimately only be endorsed if the evidence is available to support such use. Partial as it is, the evidence already available gives clear indications of the way we should be approaching musculoskeletal problems.

From a wide range of randomised controlled trials and systematic reviews quoted widely in this text, it is apparent that many commonly used treatments provide only short-term relief, if any. Passive -therapies such as ultrasound and other electrotherapy modalities appear to be so widely associated with lack of treatment effect, apart from placebo, that such approaches may need to be abandoned completely. Active, exercise-based approaches are consistently, across a variety of disorders, able to generate a positive treatment effect, so that the general direction of physiotherapy management strategies should be clear. When this evidence is considered in the light of other sources of evidence such as epidemiology and the role of psychosocial factors, any other approach appears to be based on habit and training rather than logic. The theory provided by research gives

practical guidelines for the development of the science of physiotherapy.

Differential diagnosis Awareness of possible causes of a symptom complaint is necessary to direct treatment towards the appropriate structure. Groin pain may be due to problems in the lumbar spine, the hip, the sacroiliac joint, the adductors, or other non-musculoskeletal conditions. Effective management must be directed towards the source, which is accessible through taking a thorough history and mechanical testing of the different structures. However, within this context it is important to be aware of certain issues that obscure the clarity of a precise diagnosis. In chronic problems mechanically produced symptom responses may be confusing and unclear (Zusman 1994), the validity of musculoskeletal tests is largely unproven, and they frequently lack reliability. In particular, intertester reliability of observation, palpation and identification of anatomical landmarks has been shown to be poor. These problems should not deter clinicians from offering treatment; however, there should be caution about dogmatic diagnoses. There should instead be acceptance of proposing management with a non-specific diagnosis or with a syndrome classification.

Concepts Using the McKenzie approach requires an understanding of the mechanical syndrome classification. The postural, dysfunction and derangement syndromes describe clinical presentations and responses to specific mechanical loading strategies – in other words, they describe what happens. Their clinical value, gained once the clinician is able to recognise them (using the tools of data-gathering and cognition), is their use in proposing management guidelines. Confirmation of a proposed syndrome hypothesis is gained by expected responses to the applied loading strategy.

Absence of a purely mechanical response to loading suggests the need for further hypothesis generation. Is an acute or sub-acute problem still in an inflammatory state? Is a chronic problem, which is easily aggravated by little movement just like an acute problem, actually showing the hypersensitivity and deconditioning characteristic of many chronic problems? Is failure to respond due to non-mechanical factors that the clinician has not been aware of and which have not been sufficiently addressed? Failure to detect a purely mechanical problem suggests the consideration of non-physiological factors, such as psychosocial issues.

Is intermittent pain due to postural factors, a dysfunction or a derangement? As above, a logical analysis of the symptomatic and mechanical response to different end-range loading strategies helps to classify the problem and leads to the formulation of a management plan. If the response does not fit with the classification, has a non-mechanical factor been omitted from the analysis, or is further testing appropriate? Use of the standardised terms when assessing the symptom response and the Traffic Light Guide give a safe and logical format for developing and progressing a management programme.

Communication with the patient This includes data-gathering techniques, mentioned above, as well as the provision of information. Data-gathering requires empathy and active listening by the clinician. Unless the situation is relaxed, friendly, respectful and non-judgemental, the patient is unlikely to tell the whole true story. Unless questions are open and inviting all responses, even the ones you might not want to hear, the clinician may be incapable of hearing the true story and may not obtain commitment from the patient to the treatment.

The specifics of information provision vary between patients. Find out what they want to know as well as what they should know, and what their particular concerns and worries are – make sure these issues are addressed. Self-management requires support, encouragement, explanation and education to provide the means for the patient to analyse their problem. Individual patients will vary in their particular requirements, but successful self-management is unlikely without this input.

Psychosocial factors Knowledge of the role of psychosocial factors in the development of chronic musculoskeletal problems should be translated into clinical practice by seeking to address these issues from the outset. Key factors such as fear–avoidance behaviour, low self-efficacy, external health locus of control and passive coping need to be addressed by an approach that encourages activity, provides reassurance and explanation, and offers some control in managing their problem. Equally it must be recognised that sometimes these factors are so dominant that a multi-disciplinary approach may be necessary.

Mechanism of symptoms Pain may be predominantly mechanical in origin or predominantly chemical. Symptoms may be modulated

strongly by pain behaviours dictated by the higher centres. Neurophysiological changes in the central nervous system may be the cause of persistent symptoms when peripheral tissue damage is resolved. Patients with these different problems will describe different presentations and will respond differently to mechanical loading. The clinical value of knowing about mechanical, inflammatory and chronic pain states is the ability to perceive these different clinical presentations, and thus the different management guidelines that these different presentations need.

Pathophysiology It is important to know the normal healing process that follows tissue injury and the factors that may retard healing. This needs to be translated into an active, exercise-based approach to patient's rehabilitation. The knowledge that joint capsules, contractile structures and other soft tissues can respond to disorders by the development of contractures, adhesions and scarring is vital to our understanding of chronic musculoskeletal problems. Knowledge of typical areas of pain that emanate from pathology at certain sites gives a focus for the physical examination.

Some pathophysiological concepts are essential to an understanding of musculoskeletal conditions. The normal healing process, the necessity of remodelling to regain full function, the problematical nature of tendon healing, and the recognition of non-mechanical factors in chronic pain are examples of such concepts that have wide relevance. Specific pathophysiological problems relevant to individual conditions are addressed in Chapter Ten. Management of these conditions is improved with an understanding of these issues.

Contributing factors Various factors may have a role in causation or prolonging the patient's problems. These may be static or dynamic postural forces that can be interrupted, such as occupational or domestic stresses. Sometimes workers are unable to interrupt work routines for fear of endangering their jobs. Pre-existing overuse or degenerative changes may also be contributory factors that are less amenable to alteration.

Specific contributing factors to different conditions have been discussed previously, especially in Chapter Ten. In those musculoskeletal problems that do have a clear occupational aetiology, an inability to break from those aggravating factors is likely to augur a poor outcome. However, sometimes the patient assumes a causative

role to exercise or activity, which on close questioning is not apparent. Careful assessment and monitoring of possible causative and contributing factors would seem obligatory.

Procedures This text has concentrated almost entirely on procedures performed by patients rather than clinicians. Various exercises have been mentioned, described, or photographed in the text – it should be noted that not all exercises that could be used have been photographed or described. This is not a 'recipe book' of procedures to apply in all situations. Exercises described under one condition may be used for another, if appropriate. Not all procedures that may be used in all circumstances have been described. At times it will be appropriate to find different procedures that suit the patient in front of you. As long as the reasoning and assessment process is gone through, exploratory exercises may be safely introduced to address the particular problem of the patient.

Patients may present as 'classical' examples of conditions described in the chapter on common disorders, and respond to the guidelines in a 'textbook' way. Frequently they will not. Nonetheless, an understanding of the concepts of mechanical diagnosis and assessment of symptomatic and mechanical presentations should allow the safe introduction of exercise management.

Clinical experience

Clinical reasoning requires clinical experience. It is only having seen hundreds of patient presentations that patterns will be recognised, and skills of data-gathering will be mastered and focused into hypothesis generation and confirmation. However, clinical experience can also lead to rigid thinking and failure to countenance unfamiliar presentations. Pattern recognition is not about squeezing square pegs into round holes, but continually re-evaluating data to confirm or deny a proposed hypothesis. Clinical experience of itself does not necessarily lead to improved clinical reasoning.

Cognition and meta-cognition

Cognition refers to the thinking processes involved in data-gathering, the application of a knowledge base and clinical experience. It is this thinking process that guides clinical decision-making, and thus proficiency in this area should lead to better patient management. Meta-cognition refers to reflection during the clinical process and monitoring of thinking processes (Terry and Higgs 1993; Jones *et al.*

1994). It is not enough to know things in theory; their translation into clinical practice only comes with reflective reaction.

Errors in clinical reasoning

Errors in the thinking process may occur at any stage during data-gathering, analysis, hypothesis-generation and testing. These may be errors of perception, enquiry, interpretation, synthesis, planning or reflection (Jones 1992)

Examples of typical errors are making assumptions without further checking, prematurely limiting hypotheses under consideration, failure to gather enough information, attending to those features that accord with a favoured hypothesis while ignoring contradictory information, and gathering redundant information (Jones 1992) Failure to listen carefully to a patient may mean ignoring a key piece of information and a false trail of hypothesis-generation. Focusing on the traumatic onset of a condition may lead to the assumption that the stages of inflammation, repair and remodelling must be gone through, ignoring the fact that a derangement may be the cause of symptoms. Doing every available test is a common way of gathering redundant information that the clinician is unable to use to fashion a treatment direction. It is important to question openly and listen without making assumptions. The use of the form focuses the data-gathering on certain key areas, which should be sufficient in most cases, and avoids the gathering of redundant information that will not help in decision-making.

Data-gathering skills vary with different presentations. Sometimes close questioning concerning symptomatic responses to loading strategies is necessary to determine the correct management strategy. At other times, in chronic patients, a close focus on pain is less relevant, and the attention should be on function. The failure to find a favoured presentation should not lead to trying to squeeze patients into diagnostic boxes that they do not fit. Do not make clinical decisions without sufficient information. If pattern recognition is not immediately available, revert to hypothesis-generation tactics. Use repeated movements and progressive loading to determine the appropriate management strategy. Equally, do not begin to change a management strategy without having first fully explored the patient's response

An example of clinical reasoning

In the following example a description of the findings from the data-gathering is given in normal type. After each section some instances of the clinical reasoning that these issues may provoke is given in italics.

History-taking

A 55-year-old man is referred by his GP with neck and arm pain. He had worked in metal engineering for fifteen years in jobs that involved sustained postures, with his arms frequently in raised and overhead positions. He had two weeks off work near the beginning of the episode on the 'doctor's orders', after which he was a bit better. He had remained at work on a different job that involved less overhead work. He usually played golf once a week, but had stopped since the onset of the problem; he hadn't been given any clear advice on whether to play again or not. He had played a few rounds a month previously and although it had hurt more for a couple of days it then felt better for about a week. He was keen to resume playing golf. On the assessment tools his pain visual analogue score was 4, on a 0 to 10 scale, and on the Croft Shoulder Scale (Croft *et al.* 1994) for measuring functional problems he had marked six out of twenty-two activities.

Psychosocial factors *His reluctance to be off work and brevity of sick leave when it was encouraged, despite ongoing symptoms, suggests that issues about 'yellow flags' can be discounted. This is endorsed by the correlation between his pain visual analogue scale and functional disability score. He is keen to have advice about returning to his favourite hobby. These aspects suggest he will be well motivated to perform an exercise programme.*

His present symptoms were two separate areas of pain, local right-sided neck pain and also in the top of his right arm, in the area of his deltoid muscle. He felt his arm pain was his main complaint; this had developed insidiously six months previously and remained at the same site, with occasional referral of pain to the elbow. After an initial improvement in the first few weeks, the condition has been overall unchanging. In the last three or four weeks he developed neck pain, which he felt was not related, and which was in fact now improving. Initially his arm symptoms had been constant, but had been intermittent in recent months. He estimated he felt it about

30% of the day. Shoulder movements brought on his arm pain, but he could move the limb fully.

The arm pain was brought on by raised-arm activities, such as putting on sweatshirts or jackets, reaching into the back of the car, or pulling the bathroom light switch, which he did by lifting his arm sideways. Some lifting activities at work also provoked it. Generally the pain stopped when he ceased the activity, but if he persisted sometimes it ached for an hour or so afterwards. Also sometimes it ached in the evening after work. Although generally it eased when he rested the arm, pain always returned with the same activities. There was no clear diurnal pattern, although sometimes it worsened as the day went on and some days it got easier. One or two nights a week he was woken by it, which he thought was due to lying on the right side.

The neck pain he felt was a separate problem, which now only troubled him intermittently on neck movements. Moving the spine had never affected the pain in the top of his arm.

Pathophysiology The patient's age and occupation and the insidious onset of symptoms suggest an underlying degenerative component to his problem. This may have implications for prognosis.

Mechanism of symptoms *A predominantly mechanical problem can be suspected, with the intermittent pain produced by consistent activities.*

Contributing factors His work is likely to be a contributory factor in causation and maintenance of his problem. However, he is not in a position that allows him to easily change careers, so rehabilitation has to take place alongside his continuing occupation.

Primary site Where he is feeling the pain suggests that his primary problem is at his shoulder. Shoulder complaints are commonly felt in the top of the arm. The neck pain seems secondary, and to him a separate, lesser and resolving problem. The aggravating factors for his primary complaint also focus attention on the shoulder.

Stage of condition *This is in the chronic stage. This may have implications for prognosis and the timescale of any improvement.*

Status of condition *This is unchanging. The stage and status of the condition mean that a reasonably vigorous approach should be used in this man's rehabilitation. He may experience a temporary worsening prior to improvement as occurred when he tried to play golf.*

Loading strategies and target zone *His list of aggravating factors has given a clear indication of the area in which his problem can be loaded and targeted.*

Symptomatic presentation *His pain visual analogue scale, functional disability score, daily duration of symptoms, and aggravating activities give a range of baseline measurements against which improvement or lack of it can be judged.*

State of the tissues *Although in the chronic stage, this man seems to be suffering a primarily mechanical problem, possibly related to contractile dysfunction. Non-mechanical factors appear to be irrelevant.*

A reasonable 'big picture' of this man and his problem has been gained from the initial part of the history-taking, and at this stage no further questions pertaining to divergent hypothesis generation need to be made.

He had attended a private physiotherapist for a few weeks which had involved ultrasound and mobilisation. This made his arm feel better for a brief period, but he stopped going as overall it wasn't really improving. He had had no previous history of arm or neck pain apart from this present episode. His doctor had provided him with two courses of NSAIDs. The first course in the first month, accompanying his two weeks off work, seemed to have helped somewhat; it turned from an 'ache to a pain'. A more recent course of anti-inflammatory medication made no alteration to his symptoms, but he had stopped as he started to develop indigestion and heartburn.

He is taking no medication at the moment. He considers his general health to be good. Apart from his recent visits to the doctor about his shoulder he had hardly been at all in the last ten years. There had been no x-rays or investigations.

Previous treatments *It is unclear if the initial improvement was due to the rest from work or the NSAIDs, but either way this medication*

had not resolved his problem. When taken later it had no affect on his symptoms. This confirms that the problem has no inflammatory component, as would be expected at this stage. Passive physiotherapy treatment had also been of no benefit. This again confirms earlier thinking that a reasonably vigorous rehabilitation approach should be instigated.

Summary *This is a chronic condition with an insidious onset, which is at present unchanging. The prime site for physical examination is the shoulder, but the cervical spine will have to be definitely ruled out and therefore needs to be examined also.*

Physical examination

There is minimal loss of cervical extension and right rotation, and both movements produce his neck pain. All other cervical movements are full and pain-free. Upon repeated extension movements the right-sided neck pain is produced, but his range increases. The pain progressively lessens and subsequently right rotation is easier. At no time while testing out his cervical spine is the arm pain felt, and following the repeated extension the pain on moving his arm is unchanged.

Spine testing *This has revealed an unrelated secondary problem, namely a resolving cervical derangement. Advice needs to be given on the appropriate extension exercises, but the neck pain is clearly not relevant to his primary problem, which is at the shoulder.*

Active movements of his shoulder (flexion, abduction, hand up the back and adduction are tested) reveal a full, or nearly full, range in all planes. This is confirmed by passively testing lateral rotation and gleno-humeral abduction, which are also the same as the left arm. There is pain during movement with flexion and abduction, and at end-range lateral rotation.

Differential diagnosis *The full range active and passive movements discount restricted shoulder conditions, namely capsulitis. The site of pain and that adduction is pain-free discounts the acromioclavicular joint. Findings so far, such as these negative findings and the painful arc, begin to focus on subacromion structures.*

Epidemiology *Probably the most common disorder at the shoulder involves the rotator cuff. These problems are frequently persistent and resistant to usual therapy.*

Resisted tests produce the patient's pain strongly with abduction, and less so with lateral rotation. All other resisted tests are painless. Upon repeating resisted abduction ten times the arm pain is produced each time but fades away within a couple of minutes of stopping. Abduction through the arc of pain, or target zone, was also repeated ten times. Again each movement was painful, but after a few minutes he did not remain worse.

Mechanical presentation *His response to different loading strategies, namely active flexion and abduction, resisted abduction and lateral rotation provides movements against which any changes in his condition can be compared.*

Differential diagnosis *A supraspinatus condition is confirmed with the primary pain being reproduced with resisted abduction. The response to loading this tissue was that of a dysfunction (pain produced and not worsened), which confirms earlier hypothesis generation. No further testing need be done at this stage, as no contradictory information suggests otherwise.*

Repeated movements *The movements that were selected to be repeated were those that loaded the abnormal tissues, which were the source of his pain, maximally. These had been garnered from the history-taking and physical examination.*

Self-management strategy *These movements provide the starting point for the patient's exercise programme.*

Education and active mechanical therapy

The patient is informed that it looks like a tendon problem, which is part of the muscle, and that it hasn't healed up properly. It needs to be worked regularly but not excessively to stimulate the healing and blood flow and to try to strengthen it again for normal use. As the problem has been present for some time and because it involves a tendon whose healing properties are not very good, a quick resolution is not to be expected. As happened when he played golf, an initial temporary worsening of his symptoms may occur, but there is no cause to worry about this as long as it settles down after a day or two. He is keen to resume playing golf and that will be the target for his rehabilitation.

The patient is given active abduction exercises through the arc of pain. He is told to repeat this ten times every two hours. He practices

this so he is clear about what he should do. He is given the opportunity to ask any questions. Due to work commitments he is not able to attend for review until the following week.

Information provision *It is important that the patient is told not only what to do but why, which includes some information about the problem itself. He needs to be given guidelines as to how often he should perform the procedure and what likely response he may get. He must practice it and be given an opportunity to ask any other questions. He must be warned that if there is progressive increase and worsening of his symptoms over the next few days, he must cease exercising until reviewed again.*

Review 1

On his return the patient says he has managed to perform the exercises about six times on most days. He is asked to demonstrate what he has been doing, and shows that he has been doing it correctly. The first few days his arm felt more sore and ached a bit more frequently, but then the exercise became easier. Now it still aches about 20% of the day, but it is less severe than it was. Pain is produced by the same activities. On physical examination flexion is now pain-free, but there is still a painful arc on abduction.

No rapid change would be expected in this condition, so the symptomatic and mechanical presentation is not likely to have changed much. Initially there was a temporary aggravation of his symptoms, a common occurrence in chronic tendon problems. He feels the exercises are helping. Overall diagnosis and management is confirmed as appropriate and the same strategies should be continued. At this stage the only consideration is whether to increase the force by adding a weight, but this is deemed unnecessary as the symptoms are still being easily provoked by the present exercise. He will continue with the same plan and be reviewed in one week.

Review 2

At the second review the patient reports that the exercise got progressively easier and now hardly hurts at all. He still feels the pain in his arm occasionally (less than 10% of the day), but several activities that were painful no longer cause any problem. Active flexion and abduction no longer provoke his pain. There is slight pain on resisted abduction, none on resisted lateral rotation. He has found the most painful movement to be lateral rotation performed at 90

degrees abduction. When abducting through the target zone with a small weight in his hand some pain is also produced.

He is continuing to improve – symptomatic and mechanical presentations, and his analysis of his problem all attest to this. At this point the exercises are not sufficiently loading the tissues to provoke pain; consequently they need to be progressed. It must also be seen if he can return to golf.

New exercises are given – abduction through the target zone with a small weight, and lateral rotation in 90 degrees abduction. He is unable to return for two weeks, and so is also encouraged to play a few rounds of golf before returning.

Review 3

He reports that when he first started to do the new exercises it was initially a bit more sore, but this didn't concern him as he was forewarned and it had happened before. As he continued over the following week it got easier, and now when exercising the arm only hurts slightly. Apart from when doing the exercise he hardly felt the arm pain at all. He had not been woken by it in the last week. He had played a few rounds of golf the previous weekend and only had occasional twinges. Upon repeating the assessment tools his pain visual analogue score was now 1, and on the functional disability questionnaire no activities were still a problem. He was happy to be discharged at this point.

Conclusion

Clinical reasoning is about the decision-making that occurs in a clinical situation. It is not a static, one-off event, but a dynamic process involving proposed management strategies and re-assessment of responses to these proposals. It is underpinned by a sound knowledge base. It requires good data-gathering skills, both in history-taking and in physical examination. It requires the ability to recognise common patterns of clinical presentation such as dysfunction or derangement, but also the ability to generate alternate hypotheses if the presentation is equivocal. This needs good re-assessment skills and an ability to closely analyse patient's mechanical and symptomatic presentations. Cognitive skills are an essential tool in the interplay between theoretical concerns and concepts and the practical interpretation of patient's problems and proposed management strategies.

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Glossary of Terms

Derangement syndrome

Internal dislocation of articular tissue, of whatever origin, that causes a disturbance in the normal resting position of the affected joint surfaces. This deforms the capsule and periarticular supportive ligaments resulting in pain, which will remain until such time as the displacement is reduced or adaptive changes have remodelled the displaced tissues. Internal dislocation of articular tissue obstructs movement attempted towards the direction of displacement.

Directional preference

Used to describe the phenomenon of preference for movement in one direction, which is a characteristic of the derangement syndrome. It describes the situation when movements in one direction will improve pain and the limitation of range, whereas movements in the opposite direction cause signs and symptoms to worsen.

Dysfunction syndrome

Normal mechanical deformation of structurally impaired soft tissues that results in pain. This abnormal tissue may be the product of previous trauma, or inflammatory or degenerative processes. These events cause contraction, scarring, adherence or adaptive shortening. Pain is felt when the abnormal tissue is loaded. Dysfunctions may be located in articular or contractile tissue.

Iatrogenic disability

Symptoms or disability induced in patients by the treatment or comments of a clinician (originally a physician).

Loading strategies

Describes the applied movements, positions or loads required to stress particular structures; may be dynamic or static. For example, in the case of articular structures this may be end-range active movement, or for contractile structures this may be a resisted test.

Mechanical presentation

Outward manifestations of a musculoskeletal problem, such as loss of movement range or velocity of movement, and functional disability resulting from condition. Very important in re-assessment of treatment efficacy.

Mechanical response

Behaviour of mechanical presentation, for instance movement loss, in response to a particular loading strategy.

Non-mechanical factors

In the acute phase of a problem the pain-generating mechanism may be primarily inflammatory. In the chronic stage various non-mechanical factors, such as central or peripheral sensitisation or psychosocial factors, may be maintaining the pain.

Pain

Acute pain

Pain of recent onset, less than seven days, and thus predominantly inflammatory.

Sub-acute

Pain that has lasted between seven days and seven weeks, and may represent an interface between inflammatory and mechanical pain.

Chronic pain

Pain that has lasted for longer than seven weeks; this may be mechanical or non-mechanical in nature.

Chronic pain states

Pain of long duration in which non-mechanical factors are important in pain maintenance. These factors may relate to peripheral or central sensitisation or psychosocial factors, such as fear–avoidance, etc.

Chemical or inflammatory pain

Pain mediated by the inflammatory chemicals released following tissue damage.

Mechanical pain

Pain that results from mechanical deformation of tissues. This occurs with abnormal stresses on normal tissues, and normal stresses on abnormal tissues, such as occurs in derangement and dysfunction.

Constant pain

Constant pain describes symptoms that are present throughout the patient's waking day, without any respite. This may be chemical or mechanical in origin, and also exists in chronic pain states.

Intermittent pain

This describes pain that comes and goes during the course of the day. Commonly this relates to intermittent mechanical deformation and pain.

Localisation of pain
This describes the abolition of distal, widespread or diffuse pain and its movement or localisation towards the site of the problem as it resolves.

Postural syndrome
Mechanical deformation of normal soft tissues or vascular insufficiency arising from prolonged positional or postural stresses, affecting any articular or contractile structures and resulting in pain.

Stage of condition
All musculoskeletal conditions can be anywhere on the continuum from acute to sub-acute to chronic. These stages are often of more significance to management than a structural diagnosis.

Standardised terms
These are used to make consistent descriptions of symptomatic responses to different loading strategies to judge their value for self-treatment.

Standardised terms used to describe symptom behaviour:

During loading:

Increase	Symptoms already present are increased in intensity.
Decrease	Symptoms already present are decreased in intensity.
Produce	Movement or loading creates symptoms that were not present prior to the test.
Abolish	Movement or loading abolishes symptoms that were present prior to the test.

After loading:

Worse	Symptoms produced or increased with movement or loading remain worse following the test.
Not Worse	Symptoms produced or increased with movement or loading do not remain worse following the test.
Better	Symptoms decreased or abolished with movement or loading and remain better after testing.
Not Better	Symptoms decreased or abolished with movement or loading and do not remain better after testing.
No Effect	Movements or loading have no effect on symptoms during or after testing.

State of tissues

This describes the different conditions that tissues could be in. They may be normal or abnormal. Abnormal tissues may be injured, healing, scarred or contracted, with healing suspended, hypersensitive to normal loading due to changes in the nervous system, degenerated or painful due to articular derangements.

Status of condition

This describes the direction the condition is going relative to recovery. It may either be improving, worsening or unchanging. Its status is significant to decisions about management.

Symptomatic presentation

This describes the details of the patient's complaints and can be assessed and re-assessed regarding site, temporal component, severity and consequent analgesic/NSAID consumption. This is very important in re-assessment of treatment efficacy.

Symptomatic response

The behaviour of pain in response to a particular loading strategy.

Target zone

The part of the range of movement where maximal pain is provoked. This is a characteristic of some contractile dysfunctions and a target for loading strategies for remodelling.

Traffic Light Guide

Identification of patient's responses to loading strategies using standardised terminology determines the appropriateness of a management direction. If the patient remains worse afterwards this is a red light; if the patient remains better this is a green light for that exercise; if there is no change more force *may* be required.

Yellow flags

Term used to describe psychosocial risk factors for developing or perpetuating long-term disability or sick leave as a consequence of musculoskeletal symptoms. They include factors such as the attitudes and beliefs of the patient about their problem, their behavioural responses to it, inappropriate health care advice, and relations with family and work.

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