# THE CERVICAL & THORACIC SPINE MECHANICAL DIAGNOSIS & THERAPY

## VOLUME ONE


ROBIN MCKENZIE

CNZM, OBE, FCSP (HON), FNZSP (HON), DIP MDT, DIP MT

STEPHEN MAY

MA, MCSP, DIP MDT, MSC

# THE CERVICAL & THORACIC SPINE MECHANICAL DIAGNOSIS & THERAPY

## BY ROBIN McKENZIE AND STEPHEN MAY

This book is essential reading for any health professional involved in the management of patients with cervical or thoracic pain. Described within are the mechanical measures required for the diagnosis and treatment of these common problems. The precise identification and management of subgroups in the spectrum of mechanical cervical and thoracic disorders has been said to be a priority if we are to improve our methods of management of back and neck problems. This latest book in the series by McKenzie and May provides a system to identify subgroups and consequently provide better strategic solutions.

Robin McKenzie first published his landmark text outlining certain principles and concepts for the diagnosis and management of lumbar spine problems in 1981. Another volume addressing the cervical and thoracic spine was published in 1990. His first publications always stressed the importance of patient self-management and the relevance of this issue has been belatedly recognised by others. Since these publications considerable evidence has demonstrated the importance and relevance of those principles and concepts in the modern management of musculoskeletal problems.

This edition explains the centralisation and peripheralisation phenomena; the use of exercise to induce changes in pain location and intensity; the means of detecting the most effective direction in which to apply therapeutic exercise; differentiation
displacement, pain of contracture and pain arising from normal tissue; how to differentiate the pain of nerve root adherence from entrapment and sciatica.

This second edition of The Cervical & Thoracic Spine: Mechanical Diagnosis & Therapy parallels the changes in the updated Lumbar Spine text. It has been thoroughly revised and considerably expanded and explores in depth the literature relating to mechanical syndromes and neck and trunk pain in general. There are descriptions of the management of the three mechanical syndromes – derangement, dysfunction, and postural syndrome – as applied to neck, thoracic and headache problems. There is in-depth consideration of the literature relating to a number of issues, such as the epidemiology of neck pain, headaches, serious spinal pathology and whiplash. Operational definitions, descriptions and numerous tables provide clinical signs and symptoms to recognise or suspect mechanical syndromes or other diagnoses.

Robin McKenzie and Stephen May have produced another evidence-based and clinically relevant text for the new century, augmenting the other volumes available that relate to the lumbar spine and extremity problems. It provides a review of relevant general topics as well as the detail of how to evaluate and prescribe appropriate specific exercises and manual techniques. The system described in this book achieves a new benchmark for the non-surgical management of mechanical cervical and thoracic disorders.

# The Cervical & Thoracic Spine
# Mechanical Diagnosis & Therapy

## Volume One

Robin McKenzie
CNZM, OBE, FCSP (Hon), FNZSP (Hon), Dip MDT, Dip MT

Stephen May
MA, MCSP, Dip MDT, MSc

Spinal Publications New Zealand Ltd
Raumati Beach, New Zealand

The Cervical & Thoracic Spine: Mechanical Diagnosis & Therapy

First Edition first published in 1990 by Spinal Publications New Zealand Ltd

Second Edition first published in April 2006
Reprinted February 2007, March 2008 by
Spinal Publications New Zealand Ltd
PO Box 2026, Raumati Beach, New Zealand
Email: enquiries@spinalpublications.co.nz



ISBN 10 0-9583647-7-X
ISBN 13 978-0-9583647-7-5

Design by Next Communications
Edited by Autumnwood Editing and Jan McKenzie
Photography by John Cheese
Illustrations by Paul Pugh
Typeset and printed by Astra Print

## Dedication

To my patients who, from 1953 to 2003, taught me all I know.

# Foreword

The lumbar spine has two cousins: the cervical spine, which is the poor cousin, and the thoracic spine, which is the even poorer cousin. They are poor because they have so little science. Whereas the lumbar spine has been extensively studied, and some might venture that it is even reasonably well understood, this is not the case for the cervical and thoracic spines.

Some things are known about the cervical spine. We know its structure; we know how it works; we have some idea about how it can be injured. We have some understanding of the origins and causes of neck pain.

The same cannot be said about the thoracic spine. We have little insight into how the thoracic spine works. We have essentially no knowledge of the common causes of thoracic spinal pain. Various conjectures have been brought to bear, but none is accompanied by scientific data.

With respect to treatment, we have enough studies to show that, for acute neck pain, most interventions do not work. Keeping the neck active is the only intervention that has been shown to be effective. For chronic neck pain we have little data. For thoracic spinal pain, either acute or chronic, the literature is devoid of any scientific data.

It is into this environment that Robin McKenzie sends the second edition of his text. In this edition he reiterates his clinical protocol, but sets it in the context of what else is known about cervical and thoracic spinal pain.

The text provides a thorough and fairly comprehensive review of the background literature. Readers are apprised of what is known about the epidemiology and risk factors for neck pain and the little that is known about thoracic spinal pain. Particularly valuable for physical therapists are suitable accounts of the serious causes of cervical and thoracic spinal pain and an account of vertebral artery disorders and their recognition.

These accounts reflect what is available in the literature. Contributions to that literature have focussed on what might be construed as the easy

aspects of spine pain: counting its prevalence, describing its nature, and reporting serious causes. While valuable, these activities nonetheless fail to address the prevailing problems: what are the common causes, and what best should be done about them.

McKenzie does not provide a solution. For something to be a solution requires evidence of reliability, validity and efficacy. These remain lacking for neck pain and non-existent for thoracic spinal pain. The text recognises that, but does refer to the small handful of studies that have been undertaken.

What McKenzie does provide is an approach. In a field where there is no competition, where there is no proven method, one can argue that any approach is notionally valid. Its virtue is that it combats nihilism by giving readers a firm protocol to follow. This above all might be the critical therapeutic ingredient.

To date, no studies have shown that this protocol is valid, i.e. that the syndromes described really do exist and correspond to some sort of verified pathology, be that structural or physiological. Nor has it been established that the specifics of the protocol achieve unique and superior outcomes. We do not know if derangement and dysfunction mean anything more than idiopathic neck pain. We do not know if their specific detection and treatment is any more effective than comparatively arbitrary exercises to keep the neck moving. Especially we do not know the extent to which patients respond and benefit from confident, convincing explanations and concerted care, irrespective of content; yet it is that confidence that the McKenzie protocol provides.

As readers and practitioners explore the McKenzie protocol, they should remain open-minded. The protocol arms them with something pragmatic to do, i.e. to get on with managing patients. It is highly likely that they will meet with success sufficiently often to encourage them to continue using the protocol; yet they should not conclude that this success is due to the specifics of the protocol. Simply being a good, caring practitioner may be the active ingredient, not mastery of a particular catechism of activity and intervention. Practitioners should remain open to the possibility that it is the confidence and care that they express that generates their results.

This uncertainty should also be the prompt for McKenzie therapists to catch up with the research agenda. They have no grounds for complacency in this arena. They have produced a good deal of research on lumbar spinal pain. It has not been matched for neck pain or thoracic spinal pain. If the McKenzie protocol is to fill the therapeutic vacuum for neck pain and thoracic spinal pain, it needs the accompanying science to demonstrate reliability, validity and efficacy, which will promote it from a good idea to evidence-based practice.

Nikolai Bogduk BSc (Med), MB,BS, MD, DSc, Dip Anat, Dip Pain Med, FAFRM, FAFMM, FFPM (ANZCA)
Professor of Pain Medicine
University of Newcastle
Royal Newcastle Hospital
Newcastle, Australia

# Acknowledgments



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

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

I would also like to express my gratitude to Helen Clare, the Institute's Director of Education, Kathy Hoyt and Grant Watson, who gave so much of their time to read the manuscript and provide invaluable commentary and criticism.

Finally, to Jan, my daughter, who has so well managed and coordinated the various specialists required to successfully complete this major task, I give my grateful thanks.

*Robin McKenzie*
*Raumati Beach*
*New Zealand*
*April 2006*

# About the Authors

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

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

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

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

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

Robin McKenzie has authored four books: *Treat Your Own Back; Treat Your Own Neck; The Lumbar Spine: Mechanical Diagnosis & Therapy;* and *The Cervical & Thoracic Spine: Mechanical Diagnosis & Therapy* (1st edition) and *The Human Extremities: Mechanical Diagnosis & Therapy*.

Stephen May, MA, MCSP, Dip MDT, MSc, was born in Kent, England, in 1958. His first degree was in English literature from Oxford University. He trained to be a physiotherapist at Leeds and qualified in 1990. Stephen worked for many years in a primary care musculoskeletal physiotherapy clinic in the National Health Service in England. In 1995 he completed the McKenzie Institute International Diploma programme, and in 1998 he completed an MSc in Health Services Research and Technology Assessment at Sheffield University. In 2002 he became a Senior Lecturer in Physiotherapy at Sheffield Hallam University.

Stephen May is author or co-author of a number of articles published in international journals, and he has previously collaborated with Robin McKenzie on *The Human Extremities: Mechanical Diagnosis & Therapy* (2000) and *The Lumbar Spine: Mechanical Diagnosis & Therapy* (2nd edition 2003).

x

# Contents



## VOLUME ONE

# List of Figures

# List of Tables

# Introduction



It is twenty-five years since the first book on the McKenzie Method, *The Lumbar Spine: Mechanical Diagnosis & Therapy*, was published, and over fifteen since the first edition of this text described the system as it applied to the cervical and thoracic spine. Much has changed in the intervening quarter century in the world of spine care. It used to be that clinicians, whether physical therapists, chiropractors or osteopaths, dispensed treatments that included heat, electrotherapy modalities, massage and manipulative therapy to mainly passive patients for as many times as the clinician considered appropriate. Manual therapists worked to whichever specific model of joint dysfunction they adhered to, whether hypomobility, joint fixations or osteopathic lesions. The biopsychosocial model of pain had yet to be born; the importance of patient involvement in management had not yet been recognised and the term 'evidence-based health care' was unknown. Before these terms became familiar to all, the system of mechanical diagnosis and therapy provided a structured and logical means of controlling mechanical spine pain that allowed patients to be at the centre of management. This is still the case today, and in the interim the evidence to support and vindicate the approach continues to accumulate.

The second edition of *The Cervical & Thoracic Spine: Mechanical Diagnosis & Therapy* still presents the logical and structured approach to the assessment, classification and management of neck and trunk problems first described in 1990. The characteristics, assessment, management and clinical reasoning associated with the mechanical syndromes of derangement, dysfunction and postural syndrome are described. As the recognition of serious spinal pathology is important for safe practice, the limited literature available that describes 'red flags' in the cervical and thoracic spine is presented. The material has been thoroughly updated and expanded to include a general introduction to neck pain problems that will be of relevance to all who treat these patients. This includes a review of epidemiology, pain, and relevant biomechanics and pathophysiology. There are contemporary and detailed reviews of headaches and whiplash, detailing the usefulness and limitations of mechanical diagnosis and therapy in these areas.

Research regarding lumbar spine problems is far more abundant than that relating to the cervical spine, and this applies both to the general literature as well as the literature relevant to mechanical diagnosis and therapy. Nonetheless, there is increasing research into various aspects relevant to neck pain, and the present volumes use this abundantly. However, it is important neither to take all research at face value nor to slavishly accept all current research ideas.

There has been a recent trend to emphasise the psychosocial component of back and neck pain. The research into its evidence base is extensive, but often fails to account for other important prognostic factors such as centralisation; fails to account for studies in which pain gets better and the psychosocial factors dissipate; fails to determine if the psychosocial factors require treatment directed at them; and fails to distinguish between different degrees of psychosocial factors. Although psychological features of the spine pain experience are important, these have rarely been examined in conjunction with biomedical aspects. When they have, centralisation/non-centralisation categories have been found to be more important predictors of long-term outcome than fear-avoidance, depression, somatisation and non-organic signs. Few trials have been conducted in which classification system-based treatment has been compared to guideline-based treatment, but when this has been done targeted treatment has demonstrated better outcomes than guideline-led treatment. There is much further research to be conducted, especially pertaining to the cervical spine, but recent research continues to highlight the relevance of centralisation and classification-led management for all non-specific spinal patients.

Twenty-five years ago, when *The Lumbar Spine: Mechanical Diagnosis & Therapy* was published, it presented a logical system of assessment and management directed at mechanical syndromes with an accompanying conceptual model. The concepts were extended to include the cervical and thoracic spine in 1990 and the extremities in 2000. With the updating of *The Cervical & Thoracic Spine: Mechanical Diagnosis & Therapy*, the ongoing relevance of these concepts in the 21st century is apparent. It provides a structured system of examination and management of mechanical syndromes; one that is patient-centred and indirectly affects the psychological aspects of a spine pain episode; one that is alert to the epidemiological aspects of spine pain as it emphasises self-management; and a

system that permits early recognition of non-responders and 'red flags' when classification into a mechanical syndrome fails to be made. The astonishing prescience of the system of mechanical diagnosis and therapy is only truly recognisable in hindsight.

*Robin McKenzie*
*Stephen May*

# 1: The Problem of Neck Pain



## Introduction

This chapter provides background information about neck pain and its impact on the general population. Modern clinical epidemiology is concerned with the distribution, natural history and clinical course of a disease, risk and prognostic factors associated with it, the health needs it produces and the determination of the most effective methods of treatment and management (Streiner and Norman 1996). A brief overview of these dimensions as related to neck pain is provided.

Sections are as follows:

- prevalence
- natural history
- severity and disability
- health care-seeking
- risk factors
- onset
- prognostic factors
- cost
- treatment effectiveness.

## Prevalence

The epidemiology of neck pain in the adult population has been less thoroughly investigated than lumbar back pain, but there is still a reasonable amount of literature upon which to draw. Population-based studies give the best indication of the rates of a problem in the community, and their findings are displayed in Table 1.1. Sampling methods, response rates and definitions have varied between studies and may explain some of the differences in results. Nonetheless the surveys generally reveal the common nature of neck pain, although we cannot be sure of the exact prevalence rate in the population. The role that definition of pain site has in altering prevalence figures is illustrated by one study that gave year prevalence of neck pain as

31% and neck-shoulder-higher back as 44.5% (Picavet and Schouten 2003). In a postal survey it is not possible to confirm the anatomical origin of these symptoms.

Lifetime prevalence of neck pain was about 70% in two studies. Point, month and year prevalence range in a number of studies, between 12% and 41% of the general population (Table 1.1).

**Table 1.1 Prevalence of neck pain in general population studies**

| *Reference* | *Country* | *Point/month prevalence* | *6m-1y* prevalence* | *Lifetime prevalence* |
|---|---|---|---|---|
| Hasvold and Johnsen 1993 | Norway | 20% | | |
| Makela *et al.* 1991 | Finland | 41% | | 71% |
| Bovim *et al.* 1994 | Norway | | 34% | |
| Cote *et al.* 1998 | Canada | 22% | | 67% |
| Lock *et al.* 1999 | UK | | 21% | |
| Leclerc *et al.* 1999 | France | | 41% | |
| Takala *et al.* 1982 | Finland | | 17% | |
| Westerling and Jonssen 1980 | Sweden | 12% | 18% | |
| Urwin *et al.* 1998 | UK | 16% | | |
| Picavet and Schouten 2003 | Netherlands | 21% | 31% | |
| Bassols *et al.* 1999 | Spain | | 22% | |
| Linton *et al.* 1998 | Sweden | | 44% | |
| Hagen *et al.* 1997a | Norway | 15% | | |
| Cote *et al.* 2004 | Canada | | 53% | |
| **Mean** | | **21%** | **31%** | **69%** |

* six month or one year prevalence

The annual incidence of neck pain, defined as a new episode during a follow-up year in those free of neck pain at baseline, has been estimated to be 15% to 19.5% in three population studies (Leclerc *et al.* 1999; Croft *et al.* 2001; Cote *et al.* 2004). Although these studies reveal the common nature of these pain complaints in the general adult population, they do not tell us about persistence of symptoms, severity, or what impact neck pain has on people's lives.

## Natural history

A number of studies suggest that, like lumbar back pain, the natural history of neck pain is frequently protracted and episodic. In two long-term follow-ups of over 250 patients with neck pain, nearly 60% reported on-going or recurrent problems (Lees and Turner 1963; Gore *et al.* 1987). In those who had on-going symptoms, just over half reported them to be moderate or severe (Gore *et al.* 1987). Retrospectively, 42% of a general population sample of nearly four thousand reported an episodic history of neck-shoulder-brachial pain (Lawrence 1969). About one-third of patients with cervical radiculopathy have reported at least one previous episode (Radhakrishnan *et al.* 1994). In a study of nearly seven hundred individuals followed over a year, 40% reported neck pain on two occasions (Leclerc *et al.* 1999). A twelve-year follow-up study found only 4% of those initially sick-listed for neck pain to be pain-free, whereas 44% reported themselves to be the same or worse than they had been twelve years earlier (Kjellman *et al.* 2001). In a follow-up study of nearly eight hundred individuals who reported neck pain at baseline, 48% reported symptoms one year later (Hill *et al.* 2004). These reports all suggest that at least 40% of those who report neck pain will have a history of relapse and future episodes – very similar to the relapse rate reported in long-term studies of lumbar back pain patients (McKenzie and May 2003).

Equally, reports of persistent and long-term pain prolonged over many months are found amongst those with neck pain, just as in the lumbar back pain population (Table 1.2). Again, the difficulty of determining the origin of symptoms felt around the neck-shoulder-upper back region makes figures imprecise. Depending on whether the definition of neck pain is limited or inclusive, the average of these figures suggests between 16% and 23% of the adult general population suffer from persistent neck pain of at least three months' duration.

**Table 1.2 Prevalence of persistent neck pain in general population**

| *Reference* | *Country* | *Neck pain for > 3 months* |
|---|---|---|
| Andersson *et al.* 1993 | Sweden | 17% |
| | | 30% (Neck-shoulder area) |
| Brattberg *et al.* 1989 | Sweden | 19% |
| Bergman *et al.* 2001 | Sweden | 19% |
| Picavet and Schouten 2003 | Netherlands | 14%<br>36% (Neck, shoulders, higher back) |
| Makela *et al.* 1991 | Finland | 11% |
| Hill *et al.* 2004 | UK | 48%* |
| Cote *et al.* 2004 | Canada | 47% |
| Guez *et al.* 2003 | Sweden | 18% |
| **Mean** | | **26%** |

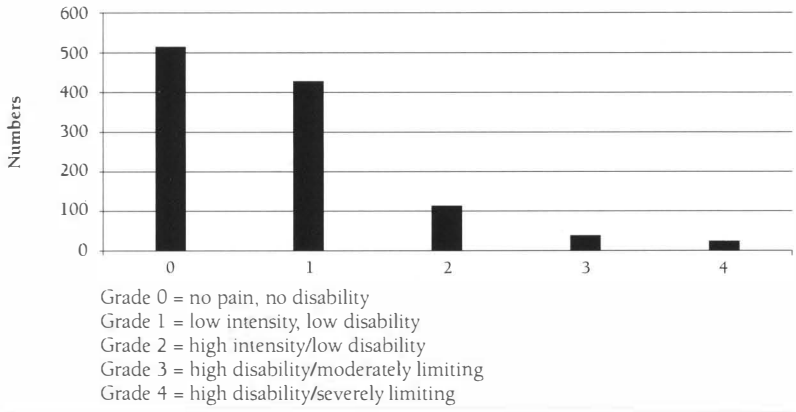
* persistent pain defined as chronic, recurrent or continuous

In over a thousand individuals, just over half of whom had neck pain at baseline, 15% developed new neck pain and 70% had persistent, recurrent or worse neck pain at one year (Cote *et al.* 2004). Amongst those who reported neck-shoulder-higher back pain, only 6% reported a single non-recurrent episode, 39% reported continuous pain and 55% reported recurrent symptoms (Picavet and Schouten 2003). It is clear that the natural history of neck pain is similar to that of back pain and is often persistent or recurrent.

## Severity and disability

The last study also reported on symptom severity. In those with continuous or recurrent pain (84%), 11% reported this to be severe, but a further 10% reported severe episodes against a background of mild continuous pain (Picavet and Schouten 2003). A minority (6%) reported partial disability from work and work leave greater than four weeks due to neck symptoms; however, while 29% reported some limitation of daily living, the majority (80%) reported no or minimal work loss (Picavet and Schouten 2003). High disability attributed to neck pain appears to affect the minority (<10%) of those with symptoms (Figure 1.1). Combined neck and arm pain have been reported as much more disabling than either symptom alone (Daffner *et al.* 2003).

**Figure 1.1 Severity and disability grading of neck pain (N = 1100)**




Source: Cote *et al.* 1998

## Health care-seeking

As with those who have lumbar back pain, not everyone with neck pain seeks health care. In the Netherlands, just over 50% of those with neck-shoulder-upper back pain had contact with a GP, specialist or physiotherapist (Picavet and Schouten 2003). In the US, in two studies with a mixed population of neck and/or back pain, 25% to 66% had sought health care from a complementary or conventional provider (Cote *et al.* 2001; Wolsko *et al.* 2003). In the UK, 69% consulted a health professional, mostly their GP or, less commonly, a physiotherapist (Lock *et al.* 1999).

Clearly not everyone with neck problems seeks treatment, but because of the high prevalence rate in the general population, neck pain patients feature prominently in health care services. Of 6,526 patients visiting GPs in Finland during a two-week period, 27% of those over the age of fifteen had musculoskeletal problems (Rékola *et al.* 1993). Twenty per cent of them had neck pain, compared to 18% with back pain, which represented over 4% of all GP consultations. In a survey of over 1,700 patients in primary care physiotherapy clinics in the UK, 22% had neck pain (May 2003).

## Risk factors

Risk factors are variables that are associated with a greater chance of acquiring the condition of interest; in this case, neck pain. There are numerous studies that have tried to identify risk factors that are

associated with neck pain, and several reviews are available (Bongers *et al.* 1993; Ariens *et al.* 1999; Vingard and Nachemson 2000). Studies tend to evaluate individual risk factors, physical and psychosocial work-related factors and non-work-related factors. Most studies address only a few risk factors, or only one type of risk factor, and do not account for other types of risk factors. This over-inflates the role of variables being considered and ignores variables that are not included in the analysis. Most studies are cross-sectional in nature, recording risk factor and outcome (neck pain) at the same time. This may reveal an association between the factor and pain, but does not confirm a causal link. Prospective study designs are more costly and complicated, but can more clearly establish a causal relationship as they are conducted in a cohort followed over time.

Individual factors associated with neck pain are female sex, increasing age up to about 50 when the risk declines, and history of previous neck pain. As already noted in the section on natural history, previous neck pain is a potent risk factor for further symptoms, especially for persistent neck pain (Leclerc *et al.* 1999; Croft *et al.* 2001). Most studies report higher prevalence rates of neck pain in women than men (Webb *et al.* 2003; Croft *et al.* 2001; Leclerc *et al.* 1999; Cote *et al.* 1998; Makela *et al.* 1991; Hasvold and Johnsen 1993; Andersson *et al.* 1993; Westerling and Jonsson 1980). Several studies found the prevalence of neck pain increases with age, at least until about 50 to 60 years of age, after which symptom reporting seems to decline (Hasvold and Johnsen 1993; Makela *et al.* 1991; Lock *et al.* 1999; Takala *et al.* 1982; Andersson *et al.* 1993; Kramer 1990). Weak associations have been found between smoking, obesity, low-pressure pain thresholds and neck pain (Makela *et al.* 1991; Cote *et al.* 2000; Andersen *et al.* 2002).

Comorbities have been associated with neck pain, namely other pain problems such as headache (Leclerc *et al.* 1999), lumbar back pain and previous neck injury (Andersen *et al.* 2002; Croft *et al.* 2001), but also digestive and cardiovascular problems (Cote *et al.* 2000). Other studies also suggest an association between neck pain and pain in other sites (Webb *et al.* 2003; Rekola *et al.* 1997; Kjellman *et al.* 2001).

Some studies identified psychosocial factors that are associated with neck pain, but many studies have found no association (Bongers *et al.* 1993; Ariens *et al.* 1999; Vingard and Nachemson 2000). Psychological distress has been associated with neck pain (Leclerc

*et al.* 1999; Makela *et al.* 1991; Croft *et al.* 2001). Barnekow-Bergkvist *et al.* (1998) found risk factors varied between men and women. Among men, self-employment and worry were associated with neck-shoulder symptoms; amongst women, monotony and control at work. Lower educational level, lower household income and raised material deprivation had some association with neck pain (Makela *et al.* 1991; Cote *et al.* 2000; Webb *et al.* 2003). There was no relationship between work satisfaction and neck pain, but high job satisfaction had a protective effect (Leclerc *et al.* 1999). High perceived job demands and low social support at work were associated with neck pain (Andersen *et al.* 2002; Ariens *et al.* 2001a). The latter was a prospective study design that adjusted for physical and individual characteristics, and thus had a strong study design (Ariens *et al.* 2001a). In a life-long prospective study, psychosocial factors in childhood were unimportant predictors of neck pain as an adult (Viikari-Juntura *et al.* 1991).

Physical work factors have also been shown to have a relationship with neck pain, although not all studies are consistent in their findings (Ariens *et al.* 1999). Reviews found various studies strongly correlated neck pain with work in static postures, such as typists, visual display workers and sewing machine operators (Grieco *et al.* 1998; Vingard and Nachemson 2000). Heavier work, repetitive work, force and neck flexion have been associated with neck pain (Makela *et al.* 1991; Andersen *et al.* 2002). Several other studies have found an association between neck flexion and neck pain (Dartigues *et al.* 1988; Kilbom *et al.* 1986; Ignatius *et al.* 1993). Seven studies looked at the association between sitting and neck pain: four found a weak association and three found no significant relationship (Ariens *et al.* 1999). However, in a study with a strong design, a positive independent association was found between sitting and neck pain, and between neck flexion and neck pain (Ariens *et al.* 2001b). This was a prospective study taking into account other confounding physical, psychosocial and individual factors.

Two studies have looked specifically at factors associated with prolapsed cervical intervertebral disc disease (Kelsey *et al.* 1984; Jensen *et al.* 1996). Frequent heavy lifting, cigarette smoking and diving were associated with the diagnosis in one study (Kelsey *et al.* 1984). Jensen *et al.* (1996) found that all men in occupations involving professional driving had an elevated risk of being hospitalised with prolapsed cervical intervertebral disc.

It is clear that there are a wide range of factors that may be potential risk factors for the onset of neck pain. The literature is generally poor quality, mostly consisting of cross-sectional studies from which a causal link between a factor and neck pain cannot be concluded. Some higher-quality studies with a prospective study design and adjusting for other potential risk factors have been conducted more recently. It is likely that physical load factors such as neck flexion, sitting and static postures and psychosocial factors at work are important predictors of neck pain. The present literature would support earlier suggestions that prolonged sitting and frequency of neck flexion are predisposing factors for neck pain (McKenzie 1981).

## Onset

Commonly, a sudden or insidious onset of neck pain is reported (McKenzie 1981; Kramer 1990). This would suggest that normal daily mechanical loading might frequently trigger neck pain symptoms. Kramer (1990) reports that symptoms are brought on by prolonged kyphotic posture during reading or deskwork, by rotational movements, or, so patients report, from sitting in a draft. Equally sustained loading during sleep may trigger symptoms. In a random population-based study, about 20% reported commonly waking with scapular, arm or neck pain, headache or neck stiffness (Gordan *et al.* 2002).

Neck pain can also be triggered by traumatic onset, most commonly whiplash-type injuries. See Chapter 25 for relevant material. However, it is important to bear in mind the insidious onset of much neck pain as this alerts the clinician to mechanical loading factors that may be implicated in predisposing, precipitating and prolonging a patient's neck pain.

## Prognostic factors

Prognostic factors are variables that affect the outcome of an episode of neck pain once it has started. The literature in this area is limited, and in reviews of the topic (Borghouts *et al.* 1998; Ariens *et al.* 1999) only six relevant studies, generally of poor quality, were found. Several reports were contradictory about the effect of age or gender on outcome, and arm pain and radiological findings were not associated with prognosis. However, severe initial pain and a history of previous episodes seemed to indicate a worse outcome.

Several recent cohort studies have identified items from the neck pain history and comorbidities as prognostic factors. High initial pain and functional disability scores, long duration of current episode, previous episodes of neck pain, lowered well-being and limited patient expectations of treatment have predicted poorer outcomes at twelve months (Kjellman *et al.* 2002). Older age (> 40) and concomitant low back pain have predicted a poorer outcome both short- and long-term, and trauma, long duration and previous history of neck pain have predicted poorer outcome long-term (Hoving *et al.* 2004). In a large population study, nearly eight hundred reported neck pain at baseline and were followed for a year when 48% reported chronic, recurrent or continuous neck pain (Hill *et al.* 2004). Significant baseline characteristics that predicted persistent neck pain were older age (> 45, especially 45 to 59), being off work at baseline, comorbid back pain and cycling as a regular activity.

## Cost

In the Netherlands the total cost of neck pain in 1996 was *estimated* to be US$686 million (Borghouts *et al.* 1999). Of this, 23% was spent on direct medical costs, mostly physical therapy, whereas 77% was absorbed by societal non-medical costs. This compared to an estimated cost of US$4,968 billion for back pain in the Netherlands in 1991 (van Tulder *et al.* 1995).

## Treatment effectiveness

As with lumbar back pain, a wide range of treatment interventions are offered to patients with neck pain. These interventions have not appeared to affect the underlying prevalence or recurrence rates. A number of systematic reviews have been undertaken to evaluate the treatment effectiveness of interventions for neck pain, and their conclusions are summarised here.

Evidence does not support the use of acupuncture for chronic neck pain; of eight high-quality trials, five were negative (Kjellman *et al.* 1999; White and Ernst 1999). Subsequent trials have demonstrated short-term changes in pain, but outcomes no better than sham treatment (Irnich *et al.* 2001, 2002) or not clinically significantly better than placebo (White *et al.* 2004). High-quality studies demonstrated lack of effect for traction (Kjellman *et al.* 1999; Philadelphia

Panel 2001). Data regarding the use of ultrasound, TENS, massage, electrical stimulation and other electrotherapy modalities or heat therapy is either lacking, limited or conflicting (Philadelphia Panel 2001; Kroeling *et al.* 2005).

At present there is little scientific evidence to support the effectiveness of multidisciplinary biopsychosocial rehabilitation programmes (Karjalainen *et al.* 2001).

Several reviews have provided limited to moderate support in favour of the short-term benefits of mobilisation and/or manipulation for some types of neck pain and/or headaches (Aker *et al.* 1996; Hurwitz *et al.* 1996; Kjellman *et al.* 1999; Bronfort *et al.* 2001, 2004). However, Di Fabio (1999) considered that the literature does not demonstrate that the benefits of manipulation outweigh the risks that are involved. Recent reviews (Gross *et al.* 2002, 2004) concluded that manipulation and/or mobilisation had no better effect than placebo or control groups and were equal when compared to each other, but done alone neither were beneficial. However, when manual therapy was combined with exercise, results were superior to control groups. To be of clear benefit, manual therapy, it seems, must be combined with exercise.

Several reviews have commented on the effectiveness of exercises for neck pain (Kjellman *et al.* 1999; Sarig-Bahat 2003). The Philadelphia Panel on Evidence-Based Clinical Practice Guidelines concluded that therapeutic exercise was the only intervention with clinically important benefits relative to a control (Philadelphia Panel 2001). When exercise has been compared to mobilisation or manipulation plus exercise, both groups showed similar improvements (Gross *et al.* 2004). This last review *"shows that it does not matter what kind of passive treatment one offers, it is what the patient does that really matters"* (Mailis-Gagnon and Tepperman 2004).

Hoving *et al.* (2001) identified and examined twenty-five reviews, of which twelve were systematic, but all these were from the 1990s. Conclusions lacked agreement about mobilisation, acupuncture and drug therapy, but agreed that the evidence was inconclusive on the effectiveness of manipulation and traction.

This brief summary of the literature, despite its limitations, would suggest certain conclusions about the management of neck pain that concur with management guidelines about low back pain. The range

of passive therapies offered to neck pain patients may provide some limited short-term pain relief at best, but most have failed to demonstrate any useful long- or even short-term benefit. For a wide range of passive therapies still being dispensed by clinicians on a regular basis, there is scant supportive evidence.

For more active treatments the evidence is more positive. Exercise appears to be effective. Manual therapy may be effective when combined with exercise, but has demonstrated equal or poorer outcomes when compared to exercise alone.

## Conclusions

Our understanding of the problem of neck pain must therefore be guided by certain irrefutable truths.

- Neck pain is so common it may be said to be 'normal', like the common cold. Resistance to the medicalisation of a normal experience should be allied to a self-management approach in which personal responsibility is engendered.
- The course of neck pain is frequently full of episodes, persistence, flare-ups, recurrences and chronicity. It is important to remember this in the clinical encounter; management must aim at long-term benefits, not simply short-term symptomatic relief.
- Many people with neck pain manage independently and do not seek health care.
- Management should be directed at trying to reduce the disability and need for care-seeking in this group by encouraging a self-reliant and coping attitude.
- Neck pain is not always a curable disorder, but for many a life-long health problem requiring on-going management. No intervention has been shown to alter the underlying prevalence, incidence or recurrence rates. Consequently management must, and should always, offer models of self-management and personal responsibility to the patient.
- Passive modalities appear to have no role in the management of neck pain. The evidence favours active interventions, primarily exercise.

Given the epidemiology of neck pain, the evidence about interventions and the role that psychosocial factors have in affecting chronic disability management, imperatives should be clear. Patients must be encouraged to avoid rest and return to normal activity. Advice should attempt to decrease anxiety about neck pain, affect attitudes and beliefs about pain and should address self-management of what may be an on-going or recurrent problem. Patients must be informed that their active participation is vital in restoring full function through self-management, exercise and activity. Patients should be provided with the means by which they can affect symptoms and thus gain some control over their problem.

A therapeutic encounter needs to equip the individual with long-term self-management strategies, which may be even more important than short-term measures of symptomatic improvement. To do otherwise and treat patients with short-term, passive modalities or manipulation, but not equip them with information and strategies for self-management, is ill-conceived and is not in the patients' best interest. If a condition is very common, persistent, often episodic and resistant to easy remedy, patients must be 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: Pain and Connective Tissue Properties

## Introduction

Pain is usually the prime concern of the patient. Thus, some means of understanding and interpreting pain is important. This chapter reviews certain aspects of pain that are relevant to the cervical spine. A distinction is made between nociception and the pain experience; common sources of pain in the cervical spine are identified; different types of pain are acknowledged, such as somatic, radicular, visceral and central, as well as local and referred pain, and pain of mechanical or chemical origin. The distinction between these two mechanisms of pain is an important determinant of the appropriateness of mechanical therapy (McKenzie 1981, 1990). In musculoskeletal problems a common cause of inflammation follows soft tissue trauma, such as during a whiplash injury, so the healing process of inflammation, repair and remodelling is briefly reviewed. Some consideration is also given to the issue of chronic pain.

Sections in this chapter are as follows:

- nociception and pain
- sources of neck pain and cervical radiculopathy
- types of pain
    - somatic pain
    - radicular pain
    - combined states
    - central pain
    - visceral pain
    - chest pain
- activation of nociceptors
- mechanical nociception
- chemical nociception
- trauma as a cause of pain
- distinguishing chemical and mechanical pain

- tissue repair process
  - inflammation
  - tissue repair
  - remodelling
- failure to remodel repair tissue
- chronic pain states.

## Nociception and pain

The means by which information concerning tissue damage is experienced and transmitted to the cortex is termed 'nociception'. This has several components (Bogduk 1993; Galea 2002):

- the detection of tissue damage (transduction)
- the transmission of nociceptive information along peripheral nerves
- its transmission up the spinal cord
- modulation of the nociceptive signals by descending pathways from higher centres in the central nervous system.

The nerve endings that detect pain are not specialised receptors. Normally they are involved with other sensory functions, but as the stimulus becomes noxious, the graded response of the receptors crosses the threshold from normal mechanical or thermal sensation and triggers the nociceptive process (Bogduk 1993). After tissue damage is detected, this information is transmitted by way of the peripheral and central nervous system to the cortex. However, en route the nociceptive message is modulated; in this way the central nervous system can exert an inhibitory or excitatory influence on the nociceptive input (Wright 2002). Thus, the classical concept of pain being a straightforward reflection of specific tissue damage is outmoded, given the current understanding of pain. Especially with patients who have chronic pain, the factors that influence the clinical presentation are more than simple nociception (Unruh and Henriksson 2002).

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 and Bogduk 1994). This much-

quoted and widely accepted definition recognises that the experience of pain is a cortical phenomenon and is influenced by affective and cognitive factors as well as sensory ones (Bogduk 1993; Unruh and Henriksson 2002; Johnson 1997).

It is important to recognise that the experience of pain involves patients' emotional and cognitive reactions to the process of nociception. Patients' anxieties, fears and beliefs may strongly determine their response to injury, pain and treatment. Fear of pain and re-injury may lead to avoidance of activities that it is thought will do more harm. It may lead them to restrict their actions and movements and to withdraw from their normal lifestyle. An exaggerated fear of pain coupled with a hyper-vigilance to every minor discomfort can lead the patient into a perpetual circle of disuse, depression, disability and persistent pain (Vlaeyen and Linton 2000). Such lack of understanding of their condition causes inappropriate action in the face of pain and produces feelings of limited ability to control or affect the condition.

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

## Sources of neck pain and cervical radiculopathy

Any structure that is innervated is a ***potential*** source of symptoms. In and around the cervical spine the following structures meet this criteria: muscles, ligaments, zygapophyseal joints, intervertebral discs, anterior and posterior longitudinal ligaments, the atlanto-occipital and atlantoaxial joints and their ligaments, the blood vessels and the dura mater (Bogduk *et al.* 1988, 2002b; McLain 1994; Mendel *et al.* 1992; Groen *et al.* 1988, 1990). Posterior structures receive innervation from the dorsal rami of the cervical spine nerves, while the ventral rami and the sinuvertebral nerves innervate anterior structures (Bogduk 1982, 2002b). Regarding the intervertebral discs, no nerves have been found in the nucleus pulposus, and neural elements were most prevalent in the posterolateral region of the disc and penetrated to the

outer third of the annulus fibrosus (Mendel *et al.* 1992; Bogduk *et al.* 1988). The sinuvertebral nerves have been described as innervating the disc at their level of entry and the disc above (Bogduk *et al.* 1988); however, more variable patterns have also been demonstrated, with the nerve ascending or descending up to two segments (Groen *et al.* 1990). Nerves innervating the dura mater have been found to ramify over up to eight segments with considerable overlap between adjacent nerves (Groen *et al.* 1988). This distribution of innervating nerves provides an anatomical substrate for an understanding of extra-segmentally referred pain patterns. Cervical radiculopathy is the product of pathology affecting the cervical nerve root or dorsal root ganglion and is considered in more detail in the next section.

## Types of pain

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

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

Tissue injury pain relates to somatic structures, whilst nervous system injury pain includes neurogenic or radicular, as well as pain generated within the central nervous system. An example of transient pain is that produced in postural syndrome. The other source of pain that occasionally must be considered in the differential diagnosis is visceral pain from organs (Bogduk 1993).

**Table 2.1 Basic pain types**

| *Pain type* | *Structures involved* |
|---|---|
| Somatic pain | Musculoskeletal tissue |
| Radicular pain | Nerve root/dorsal root ganglion |
| Combined states | Equals both somatic and radicular pain |
| Central pain | Central nervous system |
| Visceral pain | Visceral organs |

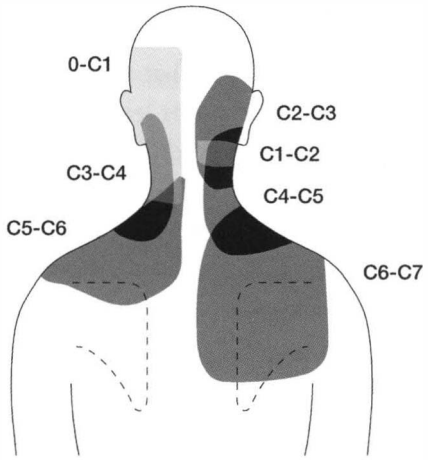
### Somatic pain

Somatic structures include the intervertebral discs, anterior and posterior longitudinal ligaments, zygapophyseal joint capsules, muscles, and so on. Only pain that originates from cutaneous tissue is felt localised to the area of tissue damage; all pain that stems from deep somatic structures is referred pain to a greater or lesser extent (Bogduk 1993). The deeper the structure, the more difficult it is to localise the pain source. Thus, most musculoskeletal pain is referred pain to a varying degree. The brain is simply aware of pain signals emanating from those structures that are supplied by a certain segment of the spinal cord. The most plausible mechanism for this is known as convergence. Neurons in the central nervous system receive afferents from structures in the cervical spine and the shoulder girdle, chest wall and upper limb. The brain is unable to determine the true source of nociceptor signals from the shared neuron (Oliver and Middleditch 1991; Bogduk 1997).

Referred pain simply reflects the lack of localising information available with nociceptor activity from deep structures. The quality of somatic referred pain is deep and aching in quality, vague and hard to localise. Experiments in the lumbar spine demonstrated that the stronger the noxious stimulus, the further the pain spreads down the limb (Kellgren 1939; Inman and Saunders 1947; Mooney and Robertson 1976). Similar experiments have not been conducted relative to the cervical spine.

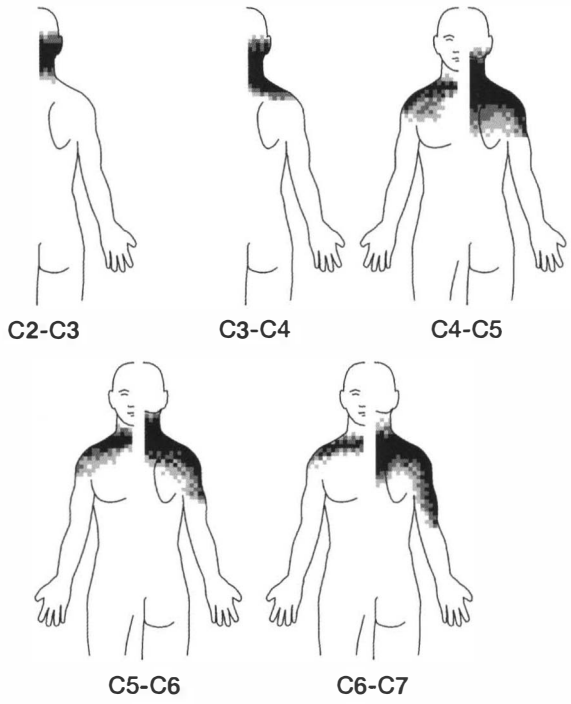
Stimulation of cervical muscles, ligaments, intervertebral discs and zygapophyseal joints with noxious injections have produced symptoms referred to the head, shoulder girdle, scapular, anterior and posterior chest wall, and upper limb depending on which levels are stimulated (Kellgren 1939; Feinstein *et al.* 1954; Dwyer *et al.* 1990; Cloward 1959; Schellhas *et al.* 1996; Grubb and Kelly 2000). Patterns of referred pain are very similar between different structures, and it is not possible to use patterns of pain referral to make diagnostic decisions (Figures 2.1, 2.2). Upper and mid-cervical segments tend to refer to the occiput, neck and upper shoulder; lower cervical segments refer to the shoulder, scapula and upper arm (Grubb and Kelly 2000; Schellhas *et al.* 1996; Dwyer *et al.* 1990). Painful intervertebral discs possibly are more likely to refer to the upper arm and anterior chest wall. The area of headache of cervicogenic origin is discussed more fully in Chapter 24.

**Figure 2.1 Patterns of referred pain produced by stimulating cervical zygapophyseal joints in normal individuals**




Source: Adapted from Dwyer *et al.* 1990 and Dreyfuss *et al.* 1994b with permission

**Figure 2.2 Patterns of referred pain produced by discography at symptomatic levels**




Source: Adapted from Slipman *et al.* 2005 and Grubb and Kelly 2000

### Radicular pain

An understanding of radicular pain is based largely on its presentation in the lumbar region. Nerve root compression by itself does not cause pain, only loss of neurological function; however, radiculopathy can be associated with pain and other signs and symptoms (Bogduk 2002b; Kramer 1990). The constellation of signs and symptoms that may indicate neurogenic pain include the following, although not all may be present:

- radicular pain pattern
- reduction or loss of sensation/paraesthesia/numbness in distal end of dermatome
- weakness or loss of power in specific muscles
- reduction or loss of specific reflexes.

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

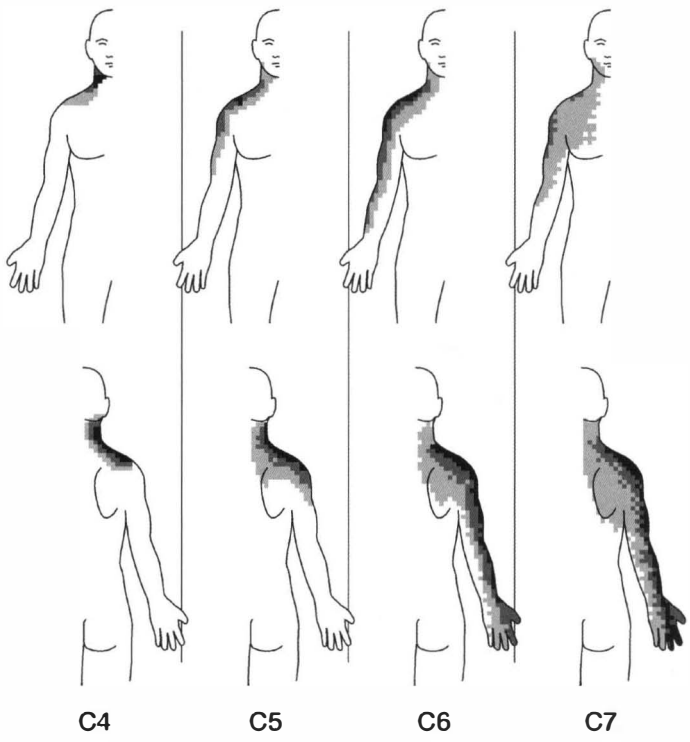
Radicular pain is different in quality from somatic pain and is frequently associated with other abnormalities of nerve conduction, such as weakness or numbness, and abnormal tension tests (Bogduk 2002b). Radicular pain is severe, lancinating or shooting in quality, felt along a narrow strip, and thus different in quality from the vague, dull aching associated with somatic-referred pain. All nerve root pain will be felt in the arm; it is always referred pain. Often the arm pain is worse than any neck pain that may be present. However, all arm pain is not nerve root pain as somatic structures can cause referred pain, at least into the upper arm.

With cervical radicular pain patterns, there is considerable variation between individuals, with no clear distinction between nerve roots in their proximal pain pattern (Slipman *et al.* 1998). Research using pain provocation has found common areas of referred pain (Figure 2.3):

- C4 around the lateral neck and top of the shoulder
- C5 is similar to C4, but extends more distally to the lateral arm

- C6 pain is distributed down the lateral arm and into the thumb and index finger
- C7 is similar to C6, but usually is more posterior and extends into the middle and ring fingers (Slipman *et al.* 1998).

**Figure 2.3 Cervical dermatomes derived by symptom provocation**




Source: Adapted from Slipman *et al.* 1998

Pain may be distributed anywhere in the dermatome in patches or in a continuous line. The distal pain is often worse. Motor and sensory abnormalities are not always present; root tension signs are earlier and more common than signs of root compression. Signs and symptoms of root compression present as muscle weakness or wasting, absent or reduced reflexes, and areas of paraesthesia, pins and needles or numbness. Sensory disturbance, when present, are found in the distal part of the dermatome – thus in the thumb and index finger for C6, middle fingers for C7 and little finger for C8.

Certain caveats have been suggested regarding the differential diagnosis between radicular and somatic pain (Bogduk 2002b). Because

there is considerable overlap between the pain from different nerve roots, segmental origin cannot be determined from the distribution of pain alone. Furthermore, the distribution of cervical radicular pain is somewhat similar to experimentally produced somatic referred pain (Kellgren 1939; Feinstein *et al.* 1954), and pain pattern alone cannot be used to distinguish between these different entities. However, more recent pain provocation studies involving the intervertebral disc and the zygapophyseal joints (Schellhas *et al.* 1996; Grubb and Kelly 2000; Dwyer *et al.* 1990; Barnsley *et al.* 1995; Lord *et al.* 1996a) suggest that somatic referred pain is most commonly felt in the shoulder girdle and upper arm and is rarely felt in the forearm or hand. Consequently, distinguishing between radicular and somatic referred pain is more likely if pain is felt in the distal part of the limb and especially if accompanied by neurological signs and symptoms.

### Combined states

Referred pain is thus either somatic or radicular in origin. These two states may be combined in one individual. For instance, a patient may have neck pain of somatic origin from pressure on the annulus fibrosus and arm pain of radicular origin caused by involvement of the nerve root.

### Central pain

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

A barrage of nociceptive input from a peripheral source, either somatic or radicular, can lead to sensitisation of central neurones. Following tissue damage, the response characteristics of certain cells may change so that normally non-nociceptive input generates pain perception (Wright 2002). This is characterised by reduced pain thresholds and increased responses to afferent input, heightened responses to repeated stimuli, expansion of receptive fields and spontaneous generation of neuronal activity.
highlights the plasticity of the nervous system. Afferent input may

not be modulated in a stable manner; recruitment of additional neurones, including normally non-nociceptive neurones, can lead to an up-regulation of the nociceptive system. Thus normal mechanical pressure can be interpreted as pain, and pain can be perceived without any appropriate peripheral input (Johnson 1997; Wright 2002). In effect, the pain generator has switched: the initial musculoskeletal problem has triggered increased sensitivity of the nociceptive system as a whole, which may now be the perpetuating problem.

### Visceral pain

Viscera may also refer pain – for example, renal pain may be felt in the loin and inguinal region, and cardiac pain may radiate down the arm (Bogduk 1993; Oliver and Middleditch 1991). Whilst the innervation of viscera is not constant and has not been definitively described, there is sufficient knowledge to formulate worthwhile clinical rules (Bogduk 1993). The heart, lungs and thoracic oesophagus are innervated by T1 – T4 and pain from these organs can be perceived over the chest or the upper lateral chest wall. The abdominal viscera is innervated by mid- and lower thoracic levels; for instance, the liver, gall bladder and pancreas T6 – T9, the stomach T6 – T10, the appendix T10, kidneys, ureter and bladder T10 – L2, and the colon T10 – T12 (Bogduk 1993). Consequently, pain from these structures may be referred to different segments of the trunk.

As pain patterns may be similar, there is capacity for confusion between pains of visceral or musculoskeletal origin. There have been reports of abdominal pain without concurrent thoracic spine pain that has been investigated for visceral causes, but has responded to intercostals blocks or spinal surgery confirming a thoracic spinal musculoskeletal cause (Ashby 1977; Whitcomb *et al.* 1995). Chest pain is an example of a symptom over which there has been much confusion between musculoskeletal and visceral problems.

### Chest pain

Chest pain is a frequent complaint in the general population and in primary medical care settings (Thurston *et al.* 2001; Mayou 1989). It is a symptom that frequently generates anxiety in both patients and clinicians, and thus referrals to cardiac clinics. However, whilst over 90% of individuals with coronary heart disease (CHD) experience angina (chest pain on exertion) as their primary complaint, a large proportion of individuals with chest pain do not have any underlying cardiac problem (Thurston *et al.* 2001; Brodsky 1985). Many patients

with chest pain in fact have cervical spine problems, and this may include even those who have been mistakenly treated as cardiac patients for many years (Brodsky 1985). In those with proven non-cardiac-related chest pain with on-going symptoms, for which they receive a poor explanation, continuing distress appears to generate continuing anxiety and health care usage, and it has been suggested a psychosocial approach needs to be used in management (Thurston *et al.* 2001; Mayou 1989; Mayou *et al.* 1994, 1999).

Chest pain per se is not evidence of cardiac failure – angina pectoris is defined as "*a clinical syndrome due to myocardial ischemia characterized by episodes of precordial discomfort or pressure, typically precipitated by exertion and relieved by rest*" (Berkow *et al.* 1992, p. 498). Generally angina pectoris is triggered by physical activity and subsides with rest. The pain is variable, often felt under the sternum as a dull ache that may become severe, or be felt as a crushing sensation. It may radiate to the left shoulder and down the left arm, but other symptom areas have been noted, such as the back and upper abdomen.

The prevalence of non-cardiac-related chest pain is variable; more musculoskeletal symptoms and other non-cardiac causes are generally reported in primary care than in some hospital-based surveys (Buntinx *et al.* 2001). In primary care about 30% of two large cohorts of patients attending with chest pain were diagnosed with a musculoskeletal problem (Buntinx *et al.* 2001; Nilsson *et al.* 2003). About 10% were diagnosed with angina pectoris or other serious cardiovascular disorder, while a further 9% required further investigation to reach a definitive conclusion (Buntinx *et al.* 2001; Nilsson *et al.* 2003). In patients attending a hospital emergency or medical department, cardiac problems were much more common (54% to 70%) and symptoms of musculoskeletal origin less frequent (7% to 13%) (Buntinx *et al.* 2001; Bechgaard 1981) or the same (30%) (Disla *et al.* 1994).

Differential diagnosis between pains of visceral or musculoskeletal origin begins with the history and identification of the aggravating and relieving factors. For instance, angina pectoris is often, but not exclusively, sited around the upper left lateral trunk region, and more importantly is associated with exertion and activity and eases with rest. Many musculoskeletal problems have opposite aggravating and relieving factors and symptoms are more likely to be provoked by sustained postures or certain movements. Physical examination should

be used to confirm exclusion/inclusion of musculoskeletal problems. This would be done using active movements and over-pressures – if these tests reproduce, increase, decrease or change symptoms in some way, a musculoskeletal problem is most likely. Over-pressure or spinal mobilisation has been suggested to improve diagnostic acumen of thoracic symptoms with musculoskeletal origin (Best 1999; Bechgaard 1981).

It is likely that the overlap between visceral and musculoskeletal symptoms around the thoracic region has led to confusion in differential diagnosis and subsequent management. Patients with an apparent visceral disorder have responded to thoracic mobilisation or manipulation and consequently a musculoskeletal technique has been thought to be effective for a visceral disorder. In such cases it is much more likely that a musculoskeletal problem has been mistakenly diagnosed as a visceral disorder because of the referral pattern. It is highly unlikely that mobilisation of spinal structures, whether through exercise or therapist techniques, would actually affect symptoms that were genuinely visceral in origin.

## Activation of nociceptors

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

## Mechanical nociception

Pain may be produced in the absence of actual tissue damage by excessive mechanical strain or tension upon collagen fibres. This is thought to be the result of the deformation of collagen networks so that nerve endings are squeezed between the collagen fibres, with the excessive pressure perceived as pain (Bogduk 1993). No damage to the tissues need have occurred, and when the stress is removed the pain abates. Me
weakened, damaged or abnormal tissues. If the excessive strain is so great as to produce actual tissue damage, the inflammatory process is provoked.

A simple example of mechanical articular pain is readily at hand. Using your right index finger, bend your left index finger backwards to apply overpressure as far as you can. Keep applying the pressure

and discomfort begins to be felt; however, the discomfort abates as soon as the pressure is released. This is simple mechanical deformation of pain-sensitive structures. Continuing or repetitive overpressure stimulates discomfort and pain more easily, but as long as tissue trauma is not provoked, symptoms dissipate rapidly upon release of the end-range stress. If you bend the finger backwards further, the intensity of the pain increases, and if you maintain the painful position longer, the pain becomes more diffuse, widespread and difficult to define. Thus, pain alters with increasing and prolonged mechanical deformation. If you now slowly return the finger to its normal resting position, the pain disappears.

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

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

## Chemical nociception

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

## Trauma as a cause of pain

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

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

## Distinguishing chemical and mechanical pain

As the cause of pain is an important determinant of the appropriateness of mechanical therapy, it is vital to distinguish between mechanical and chemical sources of nociception (McKenzie 1981, 1990). We can begin to distinguish between these types of pain by certain factors gained during the history-taking and largely confirm this impression during the physical examination. A key characteristic that indicates the possibility of pain of chemical origin is constant pain. Not all constant pain is inflammatory in nature, but chemical pain is always constant. The term 'constant pain' indicates that the patient is never without an ache or discomfort from the moment they wake until the moment they fall asleep. The ache may be exacerbated by movements and be less at times, but the dull, relentless ache never goes entirely. Constant pain may result from chemical or mechanical causes, or be due to the changes associated with chronic pain.

**Table 2.2: Key factors in pain identification**

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

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

*Key factors in identifying constant pain of mechanical origin:*

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

Intermittent pain is almost certainly mechanical in origin, and is generally easier to treat than constant pain. During normal daily activities the patient is causing sufficient mechanical stresses to trigger nociceptive signals, which may persist after that activity has ceased. They may also be performing certain activities or sustaining certain postures that reduce mechanical deformation sufficiently to abolish their symptoms temporarily. This sensitivity to mechanical forces, in which different activities and postures both aggravate and reduce symptoms, is a notable characteristic of most neck pain.

## Tissue repair process

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

**Table 2.3 Stages of healing – approximate timeframe**

| | |
|---|---|
| 1. Inflammation | < 5 days |
| 2. Tissue repair | < 7 weeks |
| 3. Remodelling | > 7 weeks |

### Inflammation

Inflammation is an umbrella term that encompasses findings at a molecular, cellular, physiological and clinical level (Scott *et al.* 2004) at which there is an extraordinary complexity of responses and inter-reactions between multiple variables. At a clinical level there may be heat, swelling, redness and pain. *"Dogma has suggested that the level of pain correlates highly with the underlying presence of inflammatory cells, but this diagnostic relation has not been borne out by biopsy or biochemical studies, particularly in chronic musculoskeletal disorders"* (Scott *et al.* 2004, p. 377). In other words, in normal musculoskeletal problems inflammatory problems are only a consideration in the first few days.

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 are 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.

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

between chemical and mechanical pain. Repeated movements cause a lasting worsening of symptoms (McKenzie 1981).

Because of this heightened sensitivity, there is a lack of correlation between mechanical stimuli and the intensity of the pain response – it hurts much more than it should (Woolf 1991; Wright 2002). When acute, this response is normal and 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, the repair process is retarded, remodelling does not occur, normal function is not restored and persistent symptoms are likely.

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

In 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 the repair. Ice, if applied in the first few days following injury, can reduce pain and oedema. Ice is of little value after the fifth day as the inflammatory cells are replaced by fibroblasts. These soon begin to lay down fibrils of collagen.

### Tissue repair

The fibroplastic or repair stage commences as the acute inflammatory stage subsides and lasts about three weeks (Enwemeka 1989). It is during this phase that the collagen and glycosaminoglycans that 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 laid down, 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 promotes greater tensile strength in the long-term. From the first week a progressive increase in movement should be encouraged so that full range is possible by the third or fourth week. *It is within this period that appropriate education and movement provide the optimal climate for uncomplicated repair.* An experimental animal model showed that the application of stress during this repair phase was able to change the length of scar tissue and thus remodel it according to function. The same stresses applied to scar tissue that was three months old had little effect on its length (Arem and Madden 1976).

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

If lasting pain is provoked it is likely that re-injury has occurred, the inflammatory phase has been re-triggered and resolution of the problem will be further delayed.

### Remodelling

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

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

Several factors can operate to promote a less than optimal repair. The granulation tissue, which repaired the damage, can now act as glue to prevent movement between tissue interfaces. During the period when collagen turnover is accelerated, there is also increased molecular cross-linkage – these processes may produce adhesion formation and impair collagen gliding (Hunter 1994; Donatelli and Owens-Burkhart 1981). Newly synthesised collagen tends to contract after three weeks; this naturally occurring shrinkage is said to continue

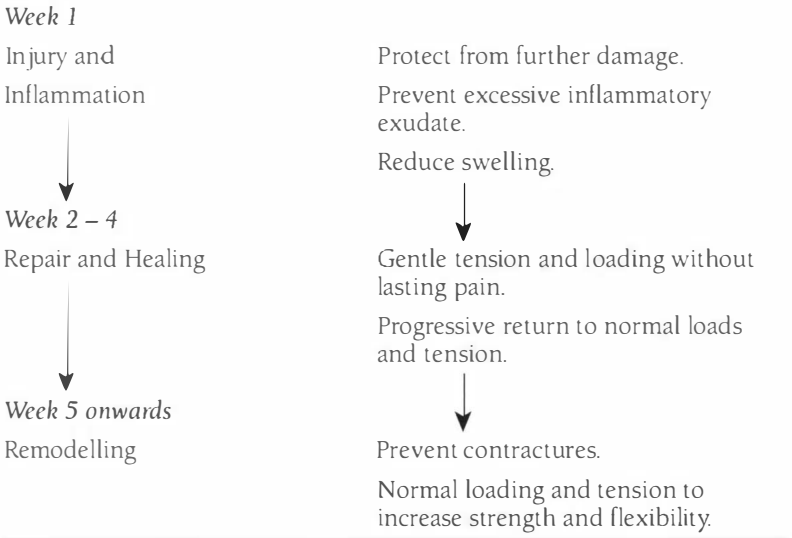
for at least six months, if not forever (Evans 1980). Thus, recently formed scar tissue begins shortening unless it is repeatedly stretched. Provided the stretching process is commenced in the early stages following injury and continued well after full recovery, no soft tissue shortening is likely to develop. Low load regular application of stress also helps to increase the tensile strength of the repair tissue (Hardy 1989). Failure to perform the appropriate tissue loading leaves the repair process complete, but the remodelling stage incomplete – the individual may still be bothered by pain and limited function, and the tissue left weak and prone to re-injury. The nerves that infiltrated the tissue during repair can now be sources of pain each time the scar is stretched or loaded. This is a common cause of persistent symptoms in many patients.

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

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

applied; however, of equal importance is the use of a progressive, controlled programme of loading the tissues at the appropriate time during the repair process in order to promote a fully functional structure *that the patient is confident to use*.

**Figure 2.4 Matching the stage of the condition to management**




## Failure to remodel repair tissue

Following tissue damage, an important factor in the physiology of repair is the phenomenon of contracture of connective tissues. A characteristic of collagen repair is that it contracts over time. Recently formed scar tissue always shortens 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).

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 around the time of the injury. They present later with restricted range of movement and pain provoked by stressing the scar tissue. The tissue becomes progressively more

sensitised and deconditioned for normal function with lack of use. In the cervical spine this situation occurs most commonly in patients with chronic whiplash who have not been instructed to recover full range of movement during the sub-acute repair stage.

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

## Chronic pain states

Chronic pain is different in quality, as well as time, from acute pain. In the latter, biomechanical and biochemical factors may be the dominant influences on the pain experience and there is a more straightforward relationship between pain and nociception. With the passage of time neurophysiological, psychological and social factors may come to dominate the maintenance of pain and the link to the original tissue damage may become minimal (Unruh *et al.* 2002).

This section briefly considers some of the reasons why a straightforward mechanical response may not be forthcoming in those who have developed 'chronic pain states'. However, it is emphasised that simply because patients have chronic or persistent pain does not necessarily mean they have a chronic pain state (Strong 2002). Many patients who have had long-term problems with neck pain benefit from a mechanical evaluation and respond positively; there should be no time limit after which a mechanical evaluation is refused. Many patients with persistent symptoms display a certain degree of poor coping with the problem – this should not, however, be taken to

mean that they have overwhelming psychosocial issues. Often with the right listening, rapport, advice and management such patients can learn to treat themselves and demonstrate self-efficacy – an example is provided in Chapter 15. In this text, criteria for chronic pain state are listed below and are determined by an analysis of the history as well as a thorough evaluation of the assessment process over several days (Table 2.3).

Strong (2002) distinguishes between chronic pain, which has lasted for a certain length of time, and chronic pain syndrome, in which pain is coupled with reduced functionality, mood changes and multiple failed treatments (see Table 2.4).

**Table 2.4 Chronic pain states**

- persistent widespread symptoms
- all activity increases symptoms
- exaggerated pain behaviour
- mistaken beliefs and attitudes about pain and movement.

**Table 2.5 Characteristics of chronic pain syndrome**

- multiple interventions
- poor response to analgesics
- increased feelings of helplessness and hopelessness
- mood changes
- psychosocial withdrawal
- loss of self-esteem
- withdrawal from work role
- decreased physical functioning
- increase in interpersonal conflicts
- conflicts with health care providers.

Source: Strong 2002

The plasticity of the central nervous system following a barrage of peripheral input can cause pathological changes that maintain the pain state in the absence of peripheral pathology (Johnson 1997; Siddall and Cousins 1997). Furthermore, psychological and behavioural attitudes and responses, as well as the process of nociception, shape the individual's experience of pain (Unruh *et al.* 2002).

The acute and sub-acute model of tissue injury and healing described earlier is not an appropriate model for an understanding of chronic

pain states. If pain persists beyond the normal healing time, other factors may exist that complicate the picture (Johnson 1997). Before suspecting non-mechanical factors, a thorough mechanical assessment should be made, as many patients with chronic symptoms have an undiagnosed derangement. However, persistent peripheral nociceptive input can induce changes in the peripheral and central nervous system (Woolf 1991; Melzack and Wall 1988). This may lead to the sensitisation of neurones in the dorsal horn, a state characterised by reduced thresholds and increased responses to afferent input so 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 response, known as central sensitisation, means that pain-related central nervous system neurones are in a state of increased excitability and painful output is more easily triggered (Wright 2002).

Thus, nociceptive signals can 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). Pain may radiate to be felt in uninjured areas adjacent to the original problem (secondary hyperalgesia), normal movement can be painful (allodynia), repeated movements can exaggerate pain responses, and pain signals may fire off without any appropriate stimulus (ectopic pain signals). The pain-generating mechanism now has little to do with the original problem and is more to do with a disturbed nociceptive system. A straightforward mechanical response is not forthcoming in such cases.

Psychosocial factors certainly have a role in people's 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, neurotocism, lack of control of events, passive coping style and focus on the pain (Cousins 1994). A recent systematic review of psychological risk factors in back and neck pain concluded that such factors play a significant role in the transition to chronic problems, and also may have a role in the aetiology of acute problems (Linton 2000). Psychosocial and cognitive factors are closely related to the development of chronic back disability. Depression, anxiety, passive coping and attitudes about pain are related to pain and disability. Catastrophising,

hyper-vigilance about symptoms and fear-avoidance behaviour are attitudes and beliefs that have been highlighted as being particularly significant in this context. These psychosocial factors, which may have prognostic significance, are termed 'yellow flags'.

It is assumed that these factors are prevalent in chronic neck pain as much as chronic back pain, although most of the research relates to the latter; however, there maybe subtle differences between these groups. In a cohort of patients entering a multidisciplinary centre, cervical patients were more likely to have greater chronicity, but significantly less disability and less fear-avoidance beliefs about physical activity than lumbar patients with similar pain intensity scores (George *et al.* 2001).

There are neurophysiological and psychological conditions that are capable of maintaining painful states beyond the normal timescale that have little or nothing to do with a biomechanical problem. The patient with a chronic condition may not only be experiencing persistent pain, but also be distressed, inactive, deconditioned and have unhelpful beliefs about pain. They may be overly passive and reliant on others, and possibly suffering economic and social deprivations due to the impact of the condition on their lifestyle. Such a state may cloud the diagnostic and therapeutic usefulness of mechanically produced symptom responses (Zusman 1994). Therefore, there exist in some patients with chronic pain conditions various factors that may confound attempts to resolve the problem and may confuse the diagnosis and symptom response.

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 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 reduction of normal activities and produce disability, in the acute stage this may be proportionate and appropriate, whereas in the chronic stage this is inappropriate and irrelevant.

Clinicians' behaviour towards patients at all stages of a condition should guard against encouraging any passive responses to pain – especially so in the chronic patient. It is hardly surprising that patients

get depressed, anxious, fearful and focussed on their persistent pain. Often health professionals seem unable to deal with it; some imply it is primarily 'in their heads' as the pain is "apparently discordant with discernible abnormalities" (Awerbuch 1995). Maladaptive or inappropriate behaviour in the face of ongoing pain states, 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 1994a, 1994b).

Although these complicating factors may undermine treatment attempts in some patients with chronic symptoms, these factors must be seen in perspective. Although 'yellow flag' variables have been quite commonly identified in back pain patients, the true prevalence of neurophysiological and psychological changes has not been consistently mapped. Because of the cross-sectional nature of much of the research in this area, although an association between psychological distress and pain has been demonstrated, this does not indicate a causal mechanism (Unruh and Henriksson 2002). There is in fact little convincing evidence that psychological factors have a causal role in most chronic pain problems (Gamsa 1994a, 1994b). The more likely explanation is that patients are distressed by their persistent pain and disability (Unruh and Henriksson 2002). When patients are followed over time, what often emerges is that psychological distress accompanies failure to resolve symptoms, whereas resolution of symptoms is accompanied by resolution of distress (Wallis *et al.* 1997; Radanov *et al.* 1996; Sterling *et al.* 2003a). Therefore, it is probably not appropriate to regard 'yellow flag' features as a separate sub-group of the pain population – if symptoms are made better, the distress will go. Many patients with persistent symptoms respond to mechanical therapy, and a mechanical assessment should never be denied patients according to the duration of their symptoms. With some patients in this group, attention must be paid to their coping strategies and their beliefs and attitudes about pain. Only a very small proportion of patients will ultimately be classified as having a chronic pain state. Although only a very small proportion of neck pain patients develop chronic intractable pain, given the complexity of the pain experience treatment in the acute stage should defend against chronic disability and in the chronic stage should be cognisant of psychological and behavioural dysfunction.

## Conclusions

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

In terms of pathology, the source of most neck and radiating pain is one of the various innervated structures in or around the cervical spine. Less frequently, radicular pain is the product of nerve root involvement also. Nociceptors are activated by mechanical and/or chemical mechanisms, a differentiation between which is crucial in the use of mechanical diagnosis and therapy. An understanding of the stages of the repair process that follows tissue trauma is essential. When patients present with painful musculoskeletal problems, this may be due to different conditions in peripheral or central structures, with the pain maintained by different mechanisms (Table 2.6). Within several states a distinction may be made between pains of somatic or radicular origin.

**Table 2.6 Pain-generating mechanisms**

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

# 3: Cervical Anatomy, Ageing and Degeneration



## Introduction

Cervical motion segments are not just smaller versions of the lumbar motion segment. A range of anatomical differences exists that may have clinical implications. Furthermore, changes related to ageing and degeneration occur differently in the cervical spine compared to the lumbar spine, which also has different potential pathophysiological outcomes. The possible clinical effects and consequent syndromes are discussed in other chapters. The aim of this chapter is an introduction to the clinical anatomy, ageing and degenerative changes of the cervical spine. The implications that the cervical disc morphology have on the McKenzie conceptual model are also discussed.

Definitions:

- upper cervical spine: occiput – C2
- mid-cervical spine: C3 – C5
- lower cervical spine: C6 – T1.

Sections in the chapter are as follows:

- cervical anatomy
- vertebrobasilar artery
- ageing and degeneration
- morphology, function and pathology
- cervical anatomy and the McKenzie conceptual model.

## Cervical anatomy

Cervical anatomy is described in detail elsewhere (for instance, Taylor and Twomey 2002; Oliver and Middleditch 1991; Bland 1998), and it is not the intention to replicate that material in this text; however, it is important to note certain key features. Between the occiput and first thoracic vertebra there are eight cervical motion segments. Cervical motion segments are not simply smaller versions of lumbar motion segments. Anatomical differences include the absence of intervertebral discs at occiput – C1 (atlas) and C1 – C2 (axis); atypical or absent

vertebral bodies at the atlas and axis; uncinate processes on the vertebral bodies of C3 – C7; and lacunae (foramen transversarium) in the transverse process of C1 – C6 through which the vertebral arteries pass (Taylor and Twomey 2002; Oliver and Middleditch 1991; Bland 1998). The uncinate processes arise on the lateral borders of the vertebral bodies. There is a close anatomical relationship between the uncinate process, the vertebral artery and the spinal nerves.

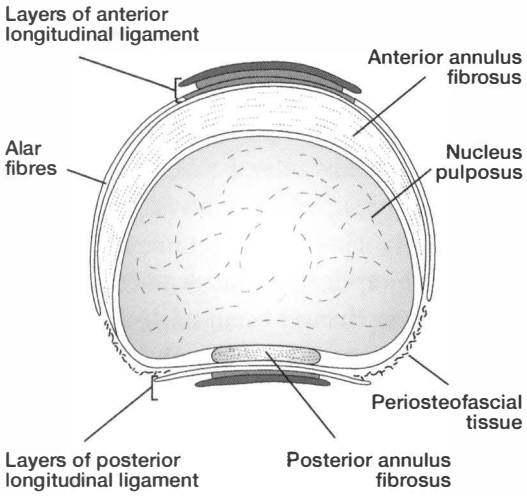
Upper cervical motion segments are unique – the atlanto-occipital and atlantoaxial joints are devoid of intervertebral discs, and the atlas lacks a body, instead receiving the odontoid peg of the axis, about which the head rotates. The upper cervical motion segments can be the source of cervicogenic headaches (see Chapter 24). In-depth knowledge of the anatomy and biomechanics in this area is essential prior to any manual therapy aimed at this section of the spine.

The adult cervical disc is different from the lumbar intervertebral disc and comprises four distinct structures (Mercer and Bogduk 1999): 1) a crescent-shaped anterior annulus fibrosus, thick anteriorly and tapered laterally toward the uncinate process; 2) the central fibrocartilaginous core of the nucleus pulposus; 3) periosteofascial tissue overlying the uncovertebral area; 4) a thin posterior annulus fibrosus. This is bordered anteriorly by the median anterior longitudinal ligament and posteriorly by the broad posterior longitudinal ligament with median fibres running inferior-superior, and alar fibres running at 45 degrees covering the postero-lateral aspect of the disc (Figure 3.1).

The nerve roots from C1 – C7 are named after the vertebral body below, whilst the C8 nerve root exits below C7 vertebral body, and nerve roots below this are named after the vertebral body above. There have been reports that from C3 – C4 caudally the anterior and posterior nerve roots exit four to eight millimetres below the intervertebral disc, and therefore disc compression of nerve roots is unlikely to occur (Bland 1994). Because of the close proximity of the zygapophyseal and uncovertebral joints and the large cervical dorsal root ganglion that almost fills the intervertebral foramen, it is suggested that cervical nerve roots are most in danger of entrapment by osteophytes from these joints (Taylor and Twomey 2002). However, understanding relations within the intervertebral foramen should not be based on these assumptions. An anatomical study found that all of 108 C5 – C7 nerve roots exited adjacent to the intervertebral disc,

whilst only the minority (22%) of thirty-six C8 nerve roots did so (Tanaka *et al.* 2000). Different aspects of the nerve root were closest to the disc in different individuals. The study also noted that out of 144 nerve roots, thirty were compressed in some way. A third were compressed by protruded discs and another third by osteophytes from the uncovertebral joints; the rest were mostly affected by the ligamentum flavum, and a few by the superior articular process of the zygapophyseal joint or periradicular fibrous tissue (Tanaka *et al.* 2000). Nerve roots most commonly affected clinically are C6 – C8. Clearly it is unwise to make assumptions about compressive factors that may be involved clinically.

**Figure 3.1 Sketch of the adult cervical disc**




Source: Mercer and Bogduk 1999, with permission

## Vertebrobasilar artery

The right and left vertebral arteries arise from the subclavian arteries; they then pass up through the foramen transversarium of C6 – C1. After the foramen transversarium of C1, they turn from a vertical to a nearly horizontal direction. Finally, the right and left vertebral arteries enter the foramen magnum, merge and become the basilar artery, which joins the Circle of Willis (Grant 2002). The vertebral arteries contribute about 11% of total blood supplied to the brain; asymmetry between right and left arteries is common, and a congenitally narrowed lumen is generally accommodated uneventfully. Equally, a degree of narrowing of the lumen by arteriosclerosis or osteophyte impingement can be tolerated if the system overall is able to

compensate for these compromises. Symptoms are produced only when the blood supply to an area is significantly compromised (Grant 2002). If present, symptoms reflect brainstem dysfunction – confusion, vertigo, diplopia, dysarthria, bilateral weakness or paraesthesia in the extremities and drop attacks may occur (Berkow *et al.* 1992).

Degenerative changes may affect the vertebral arteries in the foramen transversarium due to the proximity of the uncovertebral joints, which are subject to osteophyte growth in later years. However, the major vascular complication is thought to occur where the artery bends around C1 and is associated with cervical manipulation in patients in their late 30s (Di Fabio 1999). In the atlantoaxial component of the vertebral artery, it is thought damage may occur when excessive rotation causes the artery to be stretched, and thus is more likely to happen in younger populations when the range is still available (Grant 2002). This issue is considered more fully in Chapter 8.

## Ageing and degeneration

Certain changes occur within the cervical motion segment and are deemed to be 'normal'; some of these changes occur relatively early in life (Taylor and Twomey 2002; Oliver and Middleditch 1991; Bland 1998). Changes in childhood include the development of uncinate processes on the vertebral bodies and the formation of uncovertebral clefts from the uncinate processes. These clefts spread medially across the posterior disc during adolescence and early adulthood and commonly produce horizontal fissuring of the posterior annulus fibrosus during adulthood. The existence of an enclosed nucleus pulposus contained by an intact annulus fibrosus is limited, and by early adulthood a separate nucleus has generally ceased to exist. Also by early adulthood the gelatinous nucleus pulposus has been replaced by fibrocartilage and fibre components (Mercer and Jull 1996). By late adulthood the disc is fibrous and fragmented, containing islands of cartilage, and the posterior annulus is often completely bisected (Bland 1998). The adult cervical annulus fibrosus has been little studied, but appears to be thick anteriorly, but only a narrow central portion posteriorly (Taylor and Twomey 2002; Mercer and Jull 1996; Mercer and Bogduk 1999), whilst the posterior longitudinal ligament is four to five times thicker than in the thoracic and lumbar regions (Bland 1998).

Uncovertebral joints, or the joints of Luschka, develop between the uncinate processes and a part of the vertebral body above, known as the echancrure, separated by loose connective tissue (Hall 1965). These joints are best developed at C3 – C4 and least developed or absent at C5 – C7. They are clearly visible in the first or second decades of life and become more pronounced thereafter (Penning 1988, 1998; Bogduk 2002a).

Ageing changes differ at upper and lower cervical motion segments due to the presence or not of the uncovertebral joints. At upper cervical levels fissuring dissects the disc from lateral to medial from the uncinate processes, whereas at lower cervical levels fissuring starts from the centre and radiates in all directions (Penning 1998). Disc thinning and posterior bulging of the disc into the epidural space commonly occur. This leads to osteophyte formation at both the zygapophyseal and uncovertebral joints. Arthrosis of zygapophyseal and uncovertebral joints occurs most severely and frequently at upper and middle levels, whilst spondylosis of intervertebral disc occurs most severely and frequently at lower cervical levels, especially C5 – C7 (Penning 1998). Such changes are common radiographic findings in the asymptomatic population and are not necessarily a source of symptoms.

## Morphology, function and pathology

Unsurprisingly, the structure of the cervical vertebral column affects function. The unique anatomy of the axis, with its cranially projecting odontoid peg or dens around which the atlas rotates, permits the greatest range of movement – frontal rotation – of any motion segment in the spine (Oliver and Middleditch 1991). The development of the uncovertebral joints and the posterior cleft may facilitate rotation, but the cranially projecting uncinate processes as well as the facets of the zygapophyseal joints preclude pure lateral flexion (Oliver and Middleditch 1991; Bogduk 2002). The uncovertebral clefts create a bipartite disc with a gliding joint between the upper and lower parts that allows for increased translation and thus increased mobility for the cervical spine (Taylor and Twomey 2002).

There has been considerable debate about whether the uncovertebral joints are actually synovial joints or are a degenerative phenomenon (Oliver and Middleditch 1991; Mercer and Jull 1996). It has been

convincingly argued that the uncovertebral joints and the fissure across the posterior aspect of the intervertebral disc that runs between them are functional adaptations of the motion unit to gain improved flexibility for the cervical spine (Penning 1988, 1998; Bogduk 2002).

As mentioned already, many of the developments described above are part of 'normal' ageing and are considered to be usual morphological changes. However, certain aspects of the 'degenerative' process can have pathological consequences. Osteophytes from zygapophyseal and uncovertebral joints can encroach on the intervertebral foramen and impinge on the nerve root. Osteophytes from uncovertebral joints can encroach on the foramen transversarium and impinge on the vertebral artery. Posterior bars formed by bulging discs, osteophytes and buckling ligamentum flavum can reduce the area of the spinal canal and impinge on the spinal cord. Radicular and/or myelopathy symptoms can be caused by 'hard' stenotic changes or by 'soft' disc protrusions. The symptomatic presentation of these pathologies is discussed later.

## Cervical anatomy and the McKenzie conceptual model

The anatomy of cervical morphology has been used to disparage certain pathological concepts regarding the cervical spine. With knowledge of the fibrosed state of the adult cervical intervertebral disc, it is stated that it is generally impossible to herniate the nucleus as there is none (Bland 1998). This statement is misleading, suggesting that disc herniations are always of nucleus material, whereas in the lumbar spine they are clearly not, being nucleus, annulus and end-plate, or some mixture of these tissues (McKenzie and May 2003). One article appeared specifically challenging the McKenzie conceptual model of internal disc derangement given the fuller understanding of the normal morphology of cervical discs (Mercer and Jull 1996). These authors are certainly correct in their concerns about the misattribution of the cervical disc as if it were just a smaller version of the lumbar disc; however, they appear to assume that by undermining the conceptual model that was proposed to explain certain symptomatic and mechanical responses (McKenzie 1990) this somehow invalidates those responses. Furthermore, it should be remembered that the conclusions from both Bland (1998) and Mercer and Jull (1996) are based on anatomical studies, and the

morphological model that has been constructed arises from the post-mortem examination of cadaveric cervical spines.

To ensure a full understanding of the pathological cervical spine, it is important also to be aware of studies involving live and symptomatic individuals. Whatever the morphology of the adult cervical disc, it has been demonstrated to be a source of symptoms with pain provocation and pain abolition using cervical discography (Cloward 1959; Roth 1976; Parfenchuck and Janssen 1994; Schellhas *et al.* 1996). These studies also demonstrated that anatomical disruption of the inner and outer posterior annulus fibrosus and leakage of contrast material through the disc wall commonly occurred in asymptomatic volunteers (Parfenchuck and Janssen 1994; Schellhas *et al.* 1996). In other words, many cervical discs are morphologically abnormal, but this is common and not necessarily a source of symptoms. These findings are concordant with what is commonly observed throughout musculoskeletal medicine – symptoms cannot simply be predicted by abnormal morphology.

A number of studies have described symptomatology ascribed to disc protrusions or herniations that has resolved following surgical intervention (Nakajima and Hirayama 1995; Odom *et al.* 1958; Lunsford *et al.* 1980; Isu *et al.* 1986; Perneczky *et al.* 1992; Chen 2000). One report discusses the nature of the herniated material and classifies the protrusion as either annulus fibrosis or nucleus pulposus, which either lies behind or perforates the posterior longitudinal ligament (Isu *et al.* 1986). Other studies have described a correlation between the presence/absence of disc protrusions as found on imaging studies and the presence/absence of symptoms (Maigne and Deligne 1994; Bush *et al.* 1997; Mochida *et al.* 1998; Kobayashi *et al.* 2003). As symptoms resolved with conservative management, the disc protrusions often visibly regressed.

The nature of the herniated material in cervical disc protrusions has been little studied. Kokobun *et al.* (1996) noted fragments of hyaline cartilage alone or with fibrocartilage derived from the endplate, and nucleus pulposus or annulus fibrosus in twenty-one herniations removed at surgery. As in the lumbar spine, it would appear that the herniated material is heterogeneous. These authors also conducted an examination on a number of cadaveric cervical discs. Horizontal clefts were commonly observed, as well as horizontal clefts extending to the endplate and fragments of endplate. Abnormalities increased

with age and were more common in the lower motion segments. They suggest that in the cervical spine the cartilaginous endplate-type herniation is the predominant type and results from the clefts and fragments (Kokobun *et al.* 1996). As a consequence, cervical disc herniations are said by some authors to occur later in life than lumbar herniations, being most common in the fifth and sixth decades and comparatively rare under 30 years of age (Kokobun *et al.* 1996; Yamazaki *et al.* 2003).

Not surprisingly, given the lateral boundary provided by the uncinate process, most penetrations of the posterior longitudinal ligament occur in the median and paramedian plane (Yamazaki *et al.* 2003). In two hundred patients with myelopthay and radiculopathy, penetration only occurred laterally in two patients at C7 – T1 where the uncinate processes are absent. Upon penetrating the ligament, the herniations tend to track laterally. The site of herniation was median (40%) and paramedian (60%) in 150 patients with myelopathy, and paramedian (20%) and lateral (80%) in fifty patients with radiculopathy (Yamazaki *et al.* 2003). The study, using computed tomographic discograms, identifies differences between cervical and lumbar herniations, but confirms that a piece of the cervical disc can become a herniated mass just as in the lumbar spine. Very similar figures for the sites of herniated masses were recorded in an earlier smaller study (Kokobun and Tanaka 1995).

It is clear that the cervical intervertebral disc ages very differently from the lumbar disc and that the pathophysiology of disc herniation is different in the two areas of the spine. It is also true that symptoms of myelopathy and radiculopathy in the cervical spine can often be the product of osteophytes and other degenerative changes. However, there is enough evidence to be clear that the cervical intervertebral disc can both be the source of somatic discogenic pain, as well as herniated masses that can also produce symptoms of myelopathy and radiculopathy. As in the lumbar spine, these herniations comprise mixed tissue, cartilaginous endplate with annulus fibrosus or nucleus pulposus, and tend to penetrate the posterior longitudinal ligament centrally and then take a lateral course.

## Conclusions

In this chapter some of the key characteristics of cervical anatomy have been mentioned. It is important for the clinician to be aware of some of the unique aspects of the cervical spine, especially if manual therapy is being contemplated. The unique structure of the upper cervical spine, the uncinate processes, the vertebral arteries, and the particular way in which the cervical spine goes through the ageing and degenerative process are all important pieces of background knowledge that the treating clinician should hold. Cervical morphology and pathophysiology are in some ways distinct and different from the lumbar spine, and these differences have been used to denigrate certain conceptual models. Despite the differences, it would appear that all regions of the spine share certain pathophysiological concepts. Ultimately, it must be remembered that management strategies are devised in line with symptom and mechanical responses, and not simply to follow a patho-anatomical concept.

# 4: Movement and Biomechanics of the Cervical Spine

## Introduction

This chapter is an introduction to some of the key elements of cervical movement and biomechanics; it is not a thorough analysis of the topic. For a fuller discussion, consult other texts (for instance, Oliver and Middleditch 1991; Penning 1998; Bogduk 2002). The aim of this chapter is to draw attention to certain aspects of clinical anatomy that are relevant to an understanding of the cervical spine and are relevant to mechanical diagnosis and therapy as applied to the cervical spine.

Sections in the chapter are as follows:

- range of movement
- factors that affect the range of movement
- effect of posture on cervical spine
- role of uncinate processes
- effect of movement on structures
- upper cervical biomechanics
- sustained loading and creep.

## Range of movement

Sagittal plane movements are available at all segments and are a combination of sagittal translation and sagittal rotation. Normal ranges of translation in individuals without symptoms maybe as high as 4-5mm, depending on the segmental level, although there is considerable individual variation (Reitman *et al.* 2004). Flexion and extension are initiated in the lower cervical spine (C4 – C7). Most rotation occurs at C1 – C2 motion segment, with much less rotation available at all other segments (Bogduk 2002a; Iai *et al.* 1993). Rotation and lateral flexion are obligatorily coupled; thus rotation always accompanies lateral flexion; lateral flexion always accompanies rotation (Ishii *et al.* 2004). Biomechanically these movements are not in fact distinct and separate movements, as discussed below (Penning 1988, 1998). Rotation is coupled with extension in the middle cervical spine and with flexion in the lower cervical spine (Ishii *et al.* 2004).

Paradoxical motion may occur at some segments – as the whole spine moves into physiological flexion, some segments actually extend (van Mameren *et al.* 1990). This behaviour is particularly apparent at the two uppermost cervical segments. The range differs depending on whether movement is performed from flexion to extension or from extension to flexion. Furthermore, it is normal for the range to vary over time in the same individual (van Mameren *et al.* 1990). Bogduk (2002a) described the high-speed cineradiographic technique employed in this study as exquisite, producing top-quality images with shattering and definitive results that put paid to all previous studies of cervical range of movement. The implications of the findings are that single observations of range must be interpreted with care, variation is normal and claims of therapeutic success in restoring movement must be based on ranges greater than the 15 degrees range of uncertainty (Bogduk 2002).

Another surprising and important movement paradox is that during retraction and protrusion, the upper and lower cervical spine segments move in opposite directions (Ordway *et al.* 1999). During retraction there is more upper cervical flexion than with physiological flexion and some lower cervical extension. During protrusion there is more upper cervical extension than with physiological extension and some lower cervical flexion. Thus end-range for the upper cervical spine involves retraction with flexion and protrusion with extension. Average head translation is 45mm, with a spread of 2mm to 75mm. Parts of the cervical spine can move independently, for instance nodding the head is movement principally at upper cervical segments only.

## Factors that affect the range of movement

Various factors can affect the cervical range of movement. Some of these are temporary whilst others can be structural and lead to permanent changes in range if no effort is made to prevent this from happening. Age, degenerative changes, posture and the advent of neck pain may all produce a temporary or permanent change in available range of movement.

During life there is an overall reduction in cervical range of movement (Trott *et al.* 1996; Chen *et al.* 1999). Movement decreases first from childhood and adolescence to adulthood, and then in adulthood itself there is decreasing range with increasing age (Penning 1998). It is hypothesised that the tissue changes associated with ageing, such

as the desiccation of the disc and the growth of osteophytes, play a significant role in the loss of movement (Dalton and Coutts 1994). Quite significant losses of range can be seen in some individuals with marked degenerative changes. Usually movement loss is symmetrical; however, such changes are not necessarily symptomatic. The decline in range that occurs with age affects the anterior-posterior mobility as well as the physiological movements. There is a significant loss of retraction, particularly in the fourth and sixth decade, and individuals come to adopt a more forward natural head posture, especially in the sixth decade (Dalton and Coutts 1994). However, there is considerable variance at different ages, and in part major movement loss may be related to years of poor posture and lack of use.

In the thoracic spine there is also a progressive increase in the kyphotic angulation with age, and this is particularly marked in women from the sixth decade (Singer 2000). When this affects particularly the cervicothoracic junction, a deformity known as 'dowager's hump' may develop; sometimes this may occur earlier in life (Oliver and Middleditch 1991). When present, this may have a pronounced effect on the posture and the range of movement the individual can attain.

Some studies report greater range of movement in women than men, but the difference is only a few degrees and not consistent across all studies (Trott *et al.* 1996; Chen *et al.* 1999). A review of normative cervical motion studies also stated that passive motion is greater than active (Chen *et al.* 1999). For instance, it was found that there was significantly less range in active flexion and extension than flexion and extension with passive overpressure (Dvorak *et al.* 1988).

As well as long-term changes, cervical range of movement is not stable in the short-term. As already mentioned, range differs according to whether executed from flexion to extension or vice versa and also varies normally over time (van Mameren *et al.* 1990). Another source of short-term variability is the starting position (McKenzie 1990; Haughie *et al.* 1995; Walmsley *et al.* 1996). In a relaxed slumped posture, with kyphosed lumbar, thoracic and lower cervical spine, there is a reduced range of sagittal and frontal plane movements. For instance, there is a difference of about 10 degrees of extension between the two starting positions (Haughie *et al.* 1995). Range of axial rotation was markedly reduced when performed from a start position of fully retracted or protruded compared to a neutral start position (Walmsley *et al.* 1996). The clinical implication of this fact is

the importance of assessing range of movement in an upright neutral position on every occasion.

Another cause of temporary loss of range of movement is an episode of neck pain. Several studies have compared neck pain subjects with asymptomatic controls and found significant differences in range of movement, as well as the presence of pain on movement (Hagen *et al.* 1997b; Jordan *et al.* 1997; Hanten et al; 2000, Lee *et al.* 2003, 2005; Norlander and Nordgren 1998). One study found that in particular limited flexion and rotation, and pain on flexion, extension and rotation, were correlated with severity of pain and significantly different between symptomatic and asymptomatic groups (Hagen *et al.* 1997b). Also, significant reductions in range of extension have been found between patients and controls in women, but only in men in the third and fourth decades (Jordan *et al.* 1997), and significant differences in retraction-protraction range (Hanten *et al.* 2000).

## Effect of posture on cervical spine

In slumped, relaxed sitting with lumbar flexion, the thoracic spine is also fully flexed; this causes the lower cervical spine to be flexed and the head protruded (McKenzie 1990). Conversely, in a more upright posture the head is more retracted (McKenzie 1990; Lee *et al.* 2005). If the individual is looking forward, then the upper cervical spine is in extension. Weak but positive correlations have been found between forward head posture and increased cervicothoracic kyphosis and upper cervical extension (Raine and Twomey 1994). It is important that the patient realises the link between the position of the lumbar and the cervical spine. As mentioned above, the starting position has an effect on the available range of movement (McKenzie 1990; Haughie *et al.* 1995; Walmsley *et al.* 1996). For instance, there is 10 degrees more extension in upright sitting (Haughie *et al.* 1995) and less rotation in extreme retraction and protraction compared to neutral (Walmsley *et al.* 1996). It is easy to demonstrate this to your patients by instructing them to sit in a slumped posture and then look up at the ceiling and over their shoulders and see how far they can see; next instruct them to sit upright and repeat the same movements and again note how far they can see. There is usually a dramatic difference in range of vision. Understanding the effect of posture is important to ensure a consistent neutral start position for examination; it is also essential that the patient understands the link

between posture and head position, particularly when postures are sustained (see later section).

When examining cervical posture, it is unwise to assume that the visible surface curve is directly related to the underlying vertebral curve, however counter-intuitive this seems. Because of the length of the spinous processes and the depth of overlying soft tissue, there are differences between the curves of the vertebral bodies and the skin (Refshauge *et al.* 1994). Differences included surface markers overestimating forward head position and cervical lordosis, and reversing the direction of the concavity at the cervicothoracic junction compared to measurements of the vertebral bodies.

## Role of uncinate processes

The uncinate processes are bony protrusions on either side of the vertebral body. Uncovertebral joints, sometimes known as the joints of Luschka, develop from these uncinate processes. They are best developed at C2 – C4 and least developed or absent at C5 – C7 (Penning 1988, 1998; Bogduk 2002). The uncovertebral joints facilitate sagittal translation and frontal rotation (Milne 1993; Penning and Wilmink 1987). The uncinate process makes the cervical interbody joint a saddle-shaped joint – upwards concavity in the frontal plane, upwards convexity in the sagittal plane. Saddle-shaped joints have two axes of motion that are perpendicular to each other. Thus, just two planes of movement are permitted by these structures: sagittal plane movements (retraction, protrusion, flexion and extension) and axial rotation in the plane of the zygapophyseal joints cradled by the uncinate processes. This explains the obligatory coupling of rotation and lateral flexion. The uncinate processes in fact restrict pure lateral flexion. Such uncovertebral joints are present in bipedal animals that have to look about in an upright position, but are absent in quadrupeds that do this by bending the neck laterally (Penning 1988, 1998; Bogduk 2002).

## Effect of movement on structures

Movements of the head and neck have effects on both the soft and bony tissue in and around the cervical spine (Edwards *et al.* 2003; Farmer and Wisneski 1994; Kramer 1990; Magnaes 1982; Nuckley *et al.* 2002; Butler 2000; Yoo *et al.* 1992; Ordway *et al.* 1999; Lentell *et al.* 2002; Kitagawa *et al.* 2004).

Protrusion has the following effects:

- maximal upper cervical extension
- flexion of the lower cervical spine.

Flexion has the following effects:

- displacement of intradiscal matter posteriorly
- enlargement of intervertebral foramen
- enlargement of spinal canal
- tensioning effect on nerve roots, dura and spinal cord.

Retraction has the following effects:

- maximal upper cervical flexion
- extension of the lower cervical spine
- slight enlargement of cervical intervertebral foramina in mid- and lower cervical spine.

Extension has the following effects:

- displacement of intradiscal matter anteriorly
- narrowing of intervertebral foramen
- narrowing of spinal canal
- slackening effect on nerve roots, dura and spinal cord.

Rotation/lateral flexion:

- displacement of intradiscal matter contralaterally
- narrowing of intervertebral foramen ipsilaterally
- lateral flexion tensioning effect on contralateral nerve roots and dura.

Arm movements:

- combination of shoulder depression and abduction, elbow extension, supination, and wrist and finger extension has a tensioning effect on the brachial plexus.

## Upper cervical biomechanics

At the occiput – C1, maximum movement occurs in the sagittal plane – nodding the head. During flexion the occipital condyles roll forward and translate backwards on the lateral masses of the atlas and the atlas translates backwards relative to the occiput (Oliver and Middleditch 1991). The atlas tilts upwards at the same time so that the posterior arch of the atlas and the occiput are approximated. During extension the reverse occurs. There is controversy about whether axial rotation occurs at the joint, but this requires the occipital condyles to rise out of the sockets of the atlas, essentially distracting the joint (Bogduk 2002).

The C1 – C2 joint is the most mobile motion segment in the whole of the spine, with a total of 60 to 70 degrees of axial rotation. During rotation the atlas and the occiput move as one unit, pivoting on the odontoid process of the axis. Towards the end of C1 – C2 rotation, a small amount of rotation may occur at occiput – C1 (Oliver and Middleditch 1991). As the atlas is not bound to the axis by any substantive ligament and few muscles act directly on it, the atlas is essentially a passive washer between the axis and the occiput. This explains some of the paradoxical movement of the atlas (Bogduk 2002). Whether the atlas flexes or extends during flexion-extension is dependent on where the occiput rests on the atlas. If with flexion the chin is protruded, the centre of gravity of the head lies relatively anterior to the C1 – C2 joint; consequently the atlas is tilted into flexion by the weight of the head. However, if the head is retracted, the centre of gravity lies posterior to the joint and consequently the atlas is forced into extension by the weight of the head even though the rest of the neck is going into flexion (Bogduk 2002).

## Sustained loading and creep

Mechanical diagnosis and therapy makes use of the concept that different sustained postures (and movements) cause symptoms to decrease, abolish, centralise, produce, worsen or peripheralise. Certain therapeutic loading has a favourable effect on symptoms and should be encouraged, whilst other loading has an unfavourable effect on symptoms and should be temporarily avoided. Clues about this are gained during the history-taking, and these provide important pointers to management. The biomechanical substrate for the effect of sustained loading is *creep*.

### Creep, hysteresis and set

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

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

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

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

The study of Harms-Ringdahl (1986) has shown in the cervical spine the effect of sustained loading in healthy volunteers. They maintained a protruded head posture and began to feel pain within two to fifteen minutes, which increased with time until they were eventually forced to discontinue the posture. Abdulwahab and Sabbahi (2000) looked at the effect of sustained neck flexion for twenty minutes in patients with cervical radiculopathy and in controls. This had the effect of significantly increasing the radicular pain in the patient group, but also producing discomfort in some of the control group who were without prior neck symptoms. Gooch *et al.* (1991) studied ***in vivo*** creep of the cervical spine in sustained flexion in a mixed group of patients and controls. Over the ten-minute period, creep occurred in those who were able to sustain the position, with the effect of increasing the angle of cervical flexion. A third of the forty-seven individuals were unable to sustain the original position due to discomfort, but were able to remain in a less flexed posture. Twelve withdrew before completing the full ten minutes due to pain; most of these were in the 'neck pain' group, but one was in the control group. These studies all demonstrate that a sustained posture of cervical flexion can lead to an increase or production of neck pain or radicular symptoms and highlight the importance of addressing this component in patient management.

## Conclusions

This chapter has considered some of the aspects of cervical biomechanics that have significant clinical implications. It is important to be aware of normal ranges of cervical movement and how these can be affected by age and temporarily by neck pain. Different postures allow for different ranges of movement, and those movements affect the structures of the cervical spine in different ways. Sustained loading can generate creep distortion in soft tissues and such loading can have a role in the production and aggravation of neck symptoms.

# 5: Diagnosis and Classification



## Introduction

This chapter considers the problems in identification of specific pathology, the classification of neck pain and the value of broad non-specific classifications. The identification of specific pathology in the cervical spine is a problem as pathological terms are commonly used, but reliable and valid means of identifying the source of neck pain are largely unavailable. Imaging studies have problems of poor sensitivity and specificity. They can identify abnormal morphology but, without targeted injections, are unable to determine if this is the symptom generator. Classification systems for the lumbar spine can be used to make initial categorisation of patients who are suitable or unsuitable for a mechanical evaluation. Except for serious spinal pathology and nerve root problems, it is generally suggested that most spine pain is a non-specific condition (Spitzer *et al.* 1987; CSAG 1994; AHCPR 1994). McKenzie (1981, 1990), in an attempt to identify like patients in the non-specific spine pain population, proposed three non-specific mechanical syndromes: derangement, dysfunction and postural, which are now widely used in musculoskeletal care.

Sections in this chapter are as follows:

- seeking patho-anatomical diagnoses
- classification systems
- diagnostic triage
    - serious spinal pathology
    - nerve root problems
    - mechanical neck pain
- sub-group identification – indications and contraindications for MDT.

## Seeking patho-anatomical diagnoses

Several methods have been used to make patho-anatomical diagnoses and thus establish the site of patients' pain. The more traditional one is through imaging studies, initially through radiography, whilst in

more recent years this has been superseded in accuracy by magnetic resonance imaging (MRI) or computer assisted tomography (CAT). A more direct way of sourcing the pain generator is using radiographically guided injections to provoke and/or abolish pain. Even with recent technological advances, these tools have limitations in their ability to identify the site of pain. With imaging studies, poor sensitivity and poor specificity bedevil their capacity to identify pathology. With injections, problems exist of false positive responses, technical difficulty and availability.

Imaging studies are good at identifying morphological changes to spinal tissues, but the changes do not necessarily relate to the symptoms. Consequently these technologies frequently cannot distinguish the true positives (those whose symptoms do relate to an imaging abnormality) from the true negatives (abnormalities in an asymptomatic population) as the following examples demonstrate.

In a group of patients increasing levels of spinal degeneration shown on x-ray were related to increasing chronicity of complaint; however, there was no simple relationship between degeneration and pain (Marchiori and Henderson 1996). Findings of degeneration on x-ray lack sensitivity, as degenerative changes are common in the asymptomatic population (Gore *et al.* 1986; Teresi *et al.* 1987; Matsumoto *et al.* 1998). Radiological changes increase with age (Friedenberg and Miller 1963; van der Donk *et al.* 1991; Matsumoto *et al.* 1998; Gore *et al.* 1986) as does neck pain; it could be speculated whether this is causal or merely incidental. The increase in neck symptoms stabilises around the fifth to sixth decade. However, the prevalence of degenerative changes continues to increase. By age 60 to 65, 95% of men and 70% of women in a sample of two hundred without neck pain had at least one degenerative change on x-ray (Gore *et al.* 1986). Significant disc space narrowing was reported by magnetic resonance imaging (MRI) in 24% of individuals 45 to 54 and 67% of those older than 64, and osteophytes in 16% of those younger than 64 and 37% of those older than 64 (Teresi *et al.* 1987). Matsumoto *et al.* (1998) investigated nearly five hundred pain-free individuals with MRI and found signs of disc degeneration present in about 14% of those in their 20s and nearly 90% of those over 60 years old.

Disc herniation and bulge have also been found in the asymptomatic population. Protrusions were visible in 20% of those aged 45 to 54 and 57% of those older than 64 (Teresi *et al.* 1987). Even cord compression

due to disc protrusion or osteophytes is found in the asymptomatic population (Teresi *et al.* 1987; Matsumoto *et al.* 1998; Bednarik *et al.* 2004). These findings make clear that the use of imaging studies by themselves may determine morphological changes, but cannot determine symptomatic pathology. To use such findings to suggest management lacks validity and is fraught with error.

Joint injections to abolish or provoke pain are a way of proving the existence of certain pathological entities, but using these as a common diagnostic tool may be neither desirable nor practical. Analgesic injections into cervical zygapophyseal joints have been shown to abolish or substantially reduce patients' neck pain or headache (Bogduk and Marsland 1988; Aprill *et al.* 1990; Hove and Gyldensted 1990; April and Bogduk 1992; Bogduk and Aprill 1993; Barnsley *et al.* 1995; Lord *et al.* 1994, 1996a; Aprill *et al.* 2002) and provocation discography has been used to confirm discogenic neck pain (Aprill and Bogduk 1992; Bogduk and Aprill 1993; Cloward 1959; Grubb and Kelly 2000; Schellhas *et al.* 1996). However, these techniques are invasive, need skilled practitioners for their safe performance, and are not widely available even if it was thought they should be commonly used. Furthermore, cervical zygapophyseal joint blocks are accompanied by a rate of false positive responses to single blocks of between 21% and 27% (Barnsley *et al.* 1993a, 1993b, 1995), which has necessitated the use of double injections to definitively prove the diagnosis. Clearly this is not a practical way to reach a diagnosis for the majority of neck pain patients, especially as such identification does not necessarily result in an effective management strategy (Barnsley *et al.* 1994b).

Manual therapists advocate the use of palpation techniques in order to establish a diagnosis (Jull *et al.* 1988). Much is made of a single study, in which the validity of manual diagnosis to establish a diagnosis of cervical zygapophyseal joint pain was investigated in a small group of twenty consecutive patients (Jull *et al.* 1988). Findings from manual palpation were compared to radiologically guided diagnostic joint blocks. Fifteen of the twenty were diagnosed with zygapophyseal joint pain and the manual therapist was 100% sensitive and 100% specific in diagnosis and segmental level. However, only one manual therapist was evaluated, the study has not been replicated and inter-tester reliability needs to be established to vindicate manual therapy palpation techniques in general.

The ability of clinicians using palpation to detect joint dysfunctions, 'fixations', stiffness or other passive intervertebral motion abnormalities in a reliable and consistent way is unproven (Table 9.1). If clinicians so commonly disagree about the presence or absence of such clinical phenomena, their validity is open to doubt. Poor rates of intertester reliability mean that the existence of such phenomena is unsubstantiated, and therefore these are not valid clinical tests. Palpation is thus not fully vindicated as a tool for making a diagnosis.

## Classification systems

Thus, methods of identifying specific pathology have problems with reliability, validity, availability or acceptability, and generally the link between diagnosis, management options and improved outcomes is unsubstantiated. It has been suggested that non-specific classification systems have several advantages (McKenzie and May 2003; Childs *et al.* 2004a). The chief advantages of a classification system are in improved clinical decision-making and establishing a prognosis; hopefully this will lead to a more effective management if treated with regard to classification. Furthermore, classification systems aid communication between clinicians and can allow increased understanding of the different sub-groups.

The proposal that matching sub-groups of non-specific spinal pain to specific interventions will lead to improved outcomes, although logical, has until lately been hypothetical only. However, two recent studies, which both use the concept of mechanically determined directional preference either wholly or as part of the classification system, have demonstrated that patients treated according to classification do better than if treated in a non-specific, even if best practice, way (Long *et al.* 2004; Fritz *et al.* 2003). Further studies also suggest that sub-groups respond better to one type of intervention than another (Childs *et al.* 2003, 2004b; Haldorsen *et al.* 2002). These studies involve lumbar spine patients; the same evidence is not available relating to cervical spine patients. However, there is every reason to believe that management could equally be improved using a classification system for patients with neck pain (Childs *et al.* 2004a).

Work on cervical classification systems is limited (Childs *et al.* 2004a). The Quebec Task Force (QTF) classification system (Spitzer *et al.* 1987) was written to apply to all activity-related spinal disorders,

and the more recent triage classification systems for back pain (CSAG 1994; AHCPR 1994) are just as relevant to the cervical spine. The QTF group have also produced a review and classification system for whiplash associated disorders (Spitzer *et al.* 1995), which is detailed in the chapter on whiplash, and a development of this system has been suggested by another group (Sterling 2004). Other classification systems for neck pain have been proposed (Childs *et al.* 2004a; Wang *et al.* 2003; Schenk *et al.* 2002). The issues that are relevant in the lumbar spine apply equally in the cervical spine:

- the value of the triage system to rule out serious spinal pathology
- the difficulty of identifying anatomically specific sources of neck pain
- the limitations of classifying by pain pattern as in the QTF (Spitzer *et al.* 1987)
- the role conservative evaluation should have in both somatic and radicular pain
- the value of using non-specific mechanical syndromes based on symptom and mechanical responses (McKenzie and May 2003; Childs *et al.* 2004a).

For a classification system to be of clinical value, certain characteristics must be demonstrated. Appropriately trained and experienced clinicians need to be able to differentiate the different sub-groups in a reliable way. When applied to a broad range of neck pain patients, the classification system must be shown to have a high prevalence of application. Finally, the value of the classification system needs to be evaluated by undertaking clinical trials after sub-classification to determine that the specific intervention recommended produces better outcomes than a non-specific intervention. Regarding the McKenzie classification system in the lumbar spine, all of these characteristics have been demonstrated (McKenzie and May 2003; May 2004a, 2004b; Long *et al.* 2004). Currently work in these areas is less advanced as far as the cervical spine is concerned, although obviously this is on-going. Initial studies have demonstrated moderate to good levels of reliability for the McKenzie classification system in the cervical spine (Dionne and Bybee 2003; Clare *et al.* 2004a, 2004c). Centralisation has been demonstrated as commonly in the cervical as in the lumbar spine (Werneke *et al.* 1999). In a survey

involving over eight hundred patients, of whom 178 had neck pain, 88% of these were classified in one of the mechanical syndromes (May 2004b). There is more detail concerning these studies in Chapter 7. In both the lumbar and cervical spine, evidence for the McKenzie classification system is more developed than for most comparable classification systems.

## Diagnostic triage

The aim of triage is to exclude patients who are unsuitable for mechanical evaluation because of suspicion of serious spinal pathology. The triage concept is familiar in the field of back pain assessment (CSAG 1994; AHCPR 1994) and suggests three groups that are easily transposed to the neck:

- serious spinal pathology – tumour, inflammatory joint disease, myelopathy, upper cervical instability, vertebral artery insufficiency, etc.
- nerve root problems – cervical radiculopathy
- mechanical neck pain – non-specific neck pain with/without radiation in which symptoms vary with activity and time.

The majority of all patients with neck pain fit in the last category. A much smaller percentage have nerve root problems, and both of these categories are suitable for mechanical evaluation. The smallest category, probably about 1% of all patients with neck pain, is unsuitable for mechanical evaluation and should be referred for further investigation.

### Serious spinal pathology

It is imperative that clinicians managing musculoskeletal patients have an awareness of the 'red flags' that might indicate serious spinal pathology. The first task of the assessment is to screen out those patients, very few in number, who must then be referred for further investigations. As in back pain patients with serious spinal pathology, the key clues are usually found in the history. These pathologies and their presentations are considered in more depth in Chapter 8.

If suspicion of serious spinal pathology is not clear from the history, it should become apparent quickly that loading strategies produce no lasting symptom reduction. Worsening of symptoms in response to all loading strategies is likely.

### *Management*

Any patients in whom 'red flags' feature must be referred for further investigation. *If serious spinal pathology is suspected, mechanical therapy is contraindicated.* Such patients in normal practice are rarely encountered, but unless a suspicion is maintained they will be missed.

## Nerve root problems

Cervical radiculopathy is suspected from the history, with characteristic pain patterns and possibly the patient reporting numbness or pins and needles. Testing reflexes and myotomes may confirm initial clues (see Table 5.1).

**Table 5.1 Typical signs and symptoms associated with nerve root involvement**

| *Root level* | *Typical area of sensory loss* | *Common motor weakness* | *Reflex* |
|---|---|---|---|
| C4 | Top of shoulder | Shoulder elevation | |
| C5 | Lateral arm/deltoid area | Shoulder abduction | Biceps |
| C6 | Thumb/index finger(s) | Elbow flexion | Biceps |
| C7 | Middle finger(s) | Elbow extension | Triceps |
| C8 | Little finger(s)/ring finger(s) | Thumb extension | |
| T1 | Medial border forearm | Finger abduction/ adduction | |

Source: Kramer 1990; Slipman *et al.* 1998; Butler 2000; Bland 1994; Lestini and Wiesel 1989

Most commonly C6, C7 or C8 nerve roots are affected causing loss of sensation in and pain down to the thumb, middle finger(s) and little finger(s) respectively (Kramer 1990). Less commonly C5 and C4 are involved, affecting the lateral arm and the shoulder respectively (Slipman *et al.* 1998; Butler 2000).

Although rare, it should be remembered that cervical radiculopathy can be the product of serious spinal pathology, such as sarcoidosis or giant cell arteritis (Atkinson *et al.* 1982; Sanchez *et al.* 1983). Be aware of symptoms that may alert to the presence of more sinister pathology, such as neurological signs/symptoms at multiple or non-adjacent levels, or *atypical responses to mechanical testing*.

*Management*

Patients with cervical radiculopathy should be given a mechanical evaluation; within this group will be derangement responders, but also patients with irreducible derangements. A small minority of this group who fail conservative therapy may be referred for and undergo surgery. Those with less severe symptomatology are more likely to respond, and those with constant pain and neurological signs and symptoms less likely. Those patients with intermittent symptoms have a very good chance of responding.

**Mechanical neck pain**

This group represents the majority of individuals with neck pain and may include those with somatic referred pain into the arm. As it is mechanical in nature symptoms will vary with time and activities, and neck pain is often accompanied by a limited range of movement. These patients are otherwise well, but suffering from a temporary and local musculoskeletal problem.

*Management*

Patients with mechanical neck pain will all be given a mechanical evaluation, which is described later. Most of these patients will be classified under one of the mechanical syndromes. After five sessions and failure to classify in a mechanical syndrome, one of the 'Other' categories may be considered.

## Sub-group identification – indications and contraindications for MDT

The majority of neck pain patients, including those with neurological signs and symptoms indicating cervical radiculopathy, are thus suitable for a mechanical evaluation using repetitive end-range motion and/or static loading. Most of these will be classified in one of the mechanical syndromes, predominantly derangement, and a few with dysfunction and postural syndrome. The effect of repeated or static end-range loading on pain patterns can determine, often on day one, the potential of that patient to respond to mechanical therapy. Treatment response indicators can also be observed during the mechanical evaluation when a mechanically determined directional preference or other consistent mechanical response is sought – thus indicating the presence of one of the three mechanical syndromes (derangement most commonly, followed by dysfunction and then postural syndrome). The majority of patients with non-specific spinal pain can be classified

into one of these three sub-groups of mechanical spinal disorders described in the next chapter. Thus we are able to identify those patients who may be helped and, just as importantly, those who are unlikely to respond to mechanical therapy. Some at the severe end of the continuum will be classified as irreducible derangement, but this classification should only follow a failure to respond to several sessions. If after five sessions there is a lack of response that indicates a mechanical syndrome, one of the 'Other' categories, described in Chapter 9, may be considered. *Secondary classifications should only be considered once an extended mechanical evaluation has ruled out a consistent mechanical response.*

*Patients whose history suggests serious pathology are absolutely unsuitable for mechanical therapy.* Patients in whom there is suspicion of myelopathy, cancer, fracture or instability, systemic disease, or progressive neurological disease should be immediately referred for further investigations.

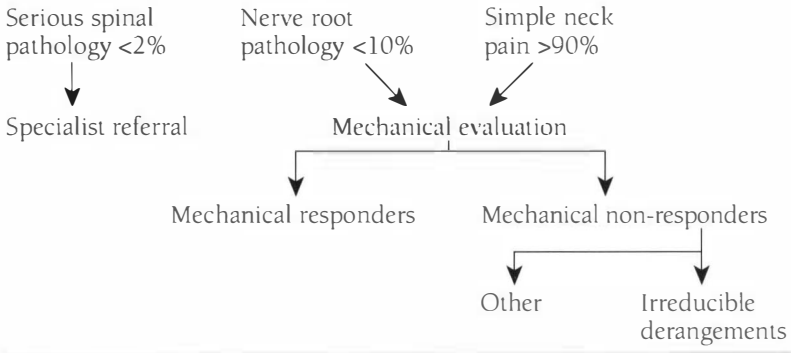
## Conclusions

This chapter has described the initial classification algorithm for evaluation of those with neck pain. In very general terms patients either present with mechanical neck pain, nerve root pathology or serious spinal pathology. The latter, if detected, is unsuitable for mechanical diagnosis and therapy and any patient with the features outlined above should be referred to a specialist – these are considered in more detail in the chapter on serious spinal pathology (Chapter 8).

Ninety-eight per cent or more of patients with neck pain are suitable for a mechanical evaluation including those with signs of nerve root involvement. The full mechanical assessment, which will be described later, seeks to identify those patients whose conditions are mechanically responsive and fit into one of the mechanical syndromes. These are described in the chapters on derangement, dysfunction and posture syndromes, and briefly described in the next chapter. Testing for them should be carried out over several days.

Not all patients will fit neatly into one of the mechanical syndromes. During the period of mechanical evaluation, atypical or inconclusive responses may arise. In that event one of the specific or non-specific categories described in Chapter 9 should be considered. Figure 5.1 gives an outline of initial clinical categories.

**Figure 5.1 Initial management pathway – key categories, estimated prevalence in neck pain population**



# 6: Mechanical Diagnosis



## Introduction

A syndrome is a characteristic group of symptoms and pattern of happenings typical of a particular problem (*The Chambers Dictionary*). Thus, syndrome describes a condition that is recognisable by a characteristic pattern of symptoms, which can be used to guide management as syndrome also describes a distinguishing pattern of responses.

The three mechanical syndromes described by McKenzie (1981, 1990) are recognised by features of the clinical presentation and responses elicited when applying a structured sequence of loading strategies. The characteristic response of each syndrome in response to repeated and/or sustained end-range loading is completely different. Correct syndrome identification allows the application of the appropriate mechanical therapy. The vast majority of non-specific spinal problems fall into these syndromes. Their clinical presentation and management are outlined in more detail in the chapters relevant to each syndrome. This chapter briefly defines the three mechanical syndromes and their accompanying conceptual models.

A minority of patients will not be classified in one of the mechanical syndromes. One per cent or less may be serious spinal pathology, hopefully recognised during history-taking. There may also be a minority of patients who after five sessions cannot be classified under one of the mechanical syndromes and whose clinical presentation equates to one of the 'Other' categories discussed elsewhere.

Sections in this chapter are as follows:

- derangement syndrome
- dysfunction syndrome
- postural syndrome.

## Derangement syndrome

This is the most common of the three mechanical syndromes encountered in spinal problems. The clinical pattern in derangement is much more variable than in the other two syndromes. Pain from

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

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

The conceptual model that has been used to explain derangement syndrome relates the presentation to internal intervertebral disc displacements (McKenzie 1981, 1990; McKenzie and May 2003). These may present in a variety of different ways, as derangements are a continuum. At its embryonic stage individuals may suffer from brief bouts of neck pain and stiffness and minor limitations of function, which last only a few days and resolve spontaneously. At its most extreme disc tissue may extrude into the spinal or intervertebral canal, causing predominantly radicular signs and symptoms.

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

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

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

## Dysfunction syndrome

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

It is relatively straightforward to distinguish these separate types of dysfunction in extremity problems; in the spine the distinction is not so clear. In the spine the syndrome presents as articular dysfunction, with pain at limited end-range. Most cervical dysfunctions cause local spinal pain only; the single exception is the adherent nerve root.

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

Dysfunction syndrome arises from a past history of some kind, such as trauma or a previous episode of neck pain, or it can arise insidiously, resulting from years of poor posture or degenerative changes. A common cause of multi-directional dysfunction in the neck is a previous history of a whiplash associated disorder. The original neck pain has resolved, but at least six to eight weeks later the individual is left with persistent symptoms each time they stretch the affected tissue. Now pain is produced at limited end-range flexion, extension, lateral flexion and rotation.

Another common cause of cervical dysfunction is cervical spondylosis. With degeneration of the motion segments there is a loss of movement. Often this may be quite painless and the patient demonstrates stiff and limited movements only, but at times the individual also presents with painful movements at end-range. Alternatively, persisting poor postural habit could have had the effect of over-stretching ligamentous and capsular structures, causing minor but recurrent micro-trauma and repair. Eventually this may lead to a loss of elasticity, a restricted range of movement and pain when the affected tissues are stretched.

Whatever the initial cause, structurally impaired soft tissues now cause a painful restricted end of range; pain is produced each time the affected tissue is stretched or compressed, but abates as soon as the position is released. In each instance tissues have gone through the repair process but have not been adequately remodelled to return to full function. Generally the exact tissue at fault in dysfunction syndrome is not known. In spinal problems pain is always produced at end-range, when tissues are stretched and/or compressed. Thus in the spine dysfunction presents as articular, but involvement of contractile structures cannot be ruled out.

In one instance, adherent nerve root, the source of symptoms is known. In this form of dysfunction a past derangement causing an episode of cervical radiculopathy has resolved, but the repair process has left some tethering or adherence that now inhibits full movement of the nerve root/dural complex. In the case of an adherent nerve root, flexion and contralateral side-flexion are restricted and each attempt to flex fully reproduces the patient's pain, which is felt in the arm. This is the only dysfunction that produces peripheral pain.

Pain from dysfunction will not go away by itself, but persists as long as the structural impairment exists, and is consistently reproduced

every time the affected tissue is stressed. The only way to resolve dysfunction is a regular remodelling programme that repeatedly stresses the tissue in order to return it to full function.

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

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

## Postural syndrome

The postural syndrome is characterised by intermittent pain brought on only by prolonged static loading of normal tissues. Time is an essential causative component, with pain only occurring following *prolonged loading*. However, the loading period required to induce symptoms may decrease with repeated exposure over time. Patients with the postural syndrome experience *no pain with movement or activity*. Neither do they suffer restriction of movement. No pathological changes occur in this syndrome. Once the aggravating posture is changed the symptoms cease. The most common posture to provoke pain in this syndrome is slumped sitting, which produces a protruded head posture. In this position the upper cervical spine can be maximally extended and the lower cervical spine is in a position of flexion.

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

Clinically, patients with pain of postural syndrome rarely present for treatment, as they learn how to abolish symptoms by changing their position. Occasionally concerned parents accompany their teenage

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

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

### Management

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

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

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

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

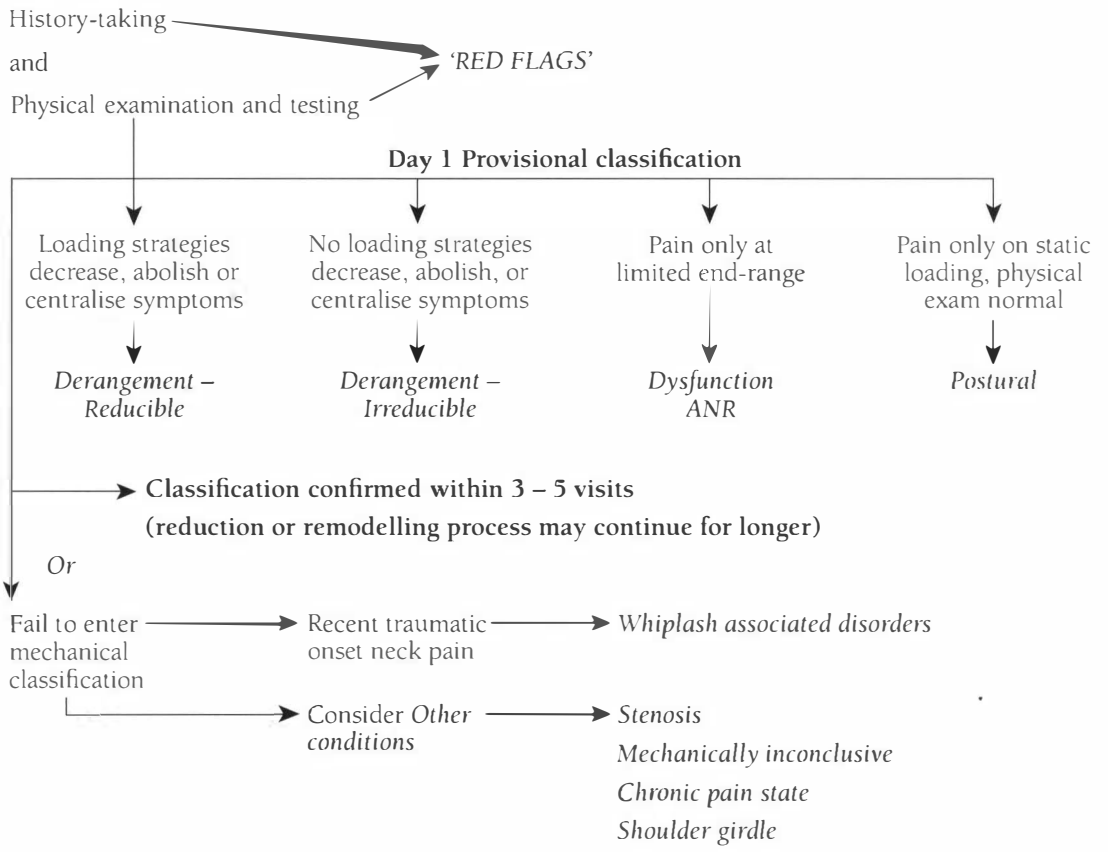
## Conclusions

These non-specific mechanical syndromes include the majority of patients with spinal pain. Failure to clearly identify a mechanical response or an atypical response may require further classification in a limited number of patients. In these instances various non-mechanical or specific categories of neck pain may need to be considered. These are described elsewhere in the book. *Other categories should never be considered without first conducting a thorough mechanical evaluation over several days.* Recognition of these other categories is based on factors in the history-taking, failure to respond in a typical manner to a mechanical loading evaluation pursued over several days and certain responses to mechanical testing.

Figure 6.1 displays the clinical reasoning process for determining the mechanical or non-mechanical diagnosis. Suspicion of 'red flag' pathology should mostly be determined by history-taking. Everyone else, about 99%, should been given a thorough physical examination as described later. From this most patients can be classified by a

mechanical diagnosis, although initially in some this will be provisional. By five visits the mechanical diagnosis will be confirmed, or, due to an atypical response, one of the 'Other' categories may be considered. The patient can display no symptom response that suggests a mechanical diagnosis, as well as displaying signs and symptoms appropriate for that 'Other' diagnosis. *The algorithm must be used in conjunction with the criteria and operational definitions in the appendix.*

**Figure 6.1 Classification algorithm for cervical spine**



# 7: Literature Review



## Introduction

Most of the research into mechanical diagnosis and therapy that has been conducted to date has involved patients with back pain. There is a considerable body of evidence that has been published regarding efficacy, reliability, mechanically determined directional preference and centralisation relating to the lumbar spine (McKenzie and May 2003, Chapter 11). Since the publication of the second edition of *The Lumbar Spine: Mechanical Diagnosis & Therapy*, more literature has appeared. Of particular importance are a systematic review about centralisation (Aina *et al.* 2004), a systematic review about the efficacy of the McKenzie approach (Clare *et al.* 2004b), and an efficacy study that established mechanically determined directional preference prior to randomisation (Long *et al.* 2004). This study is briefly described below. However, it is still the case that most of this literature relates to back pain, not neck pain, patients.

This chapter covers the literature that is directly relevant to the practice of mechanical diagnosis and therapy in the cervical spine, which at this point is limited. More literature, however, continues to emerge and the following website, which is regularly updated, is recommended to maintain an up-to-date knowledge of the available evidence base: www.mckenziemdt.org/research.

This chapter contains the following sections:

- efficacy studies
- mechanically determined directional preference
- centralisation
- reliability
- prevalence of mechanical syndromes in neck pain patients.

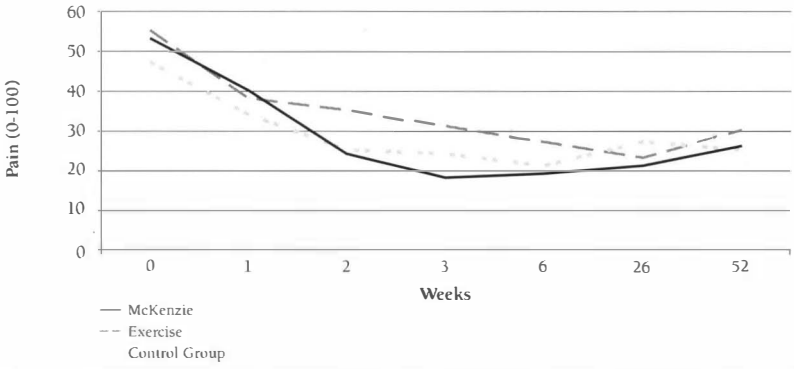
## Efficacy studies

A systematic review (Clare *et al.* 2004b) into the efficacy of McKenzie therapy for spinal pain, with strict inclusion and exclusion criteria, included six randomised controlled studies, one of which involved

patients with neck pain (Kjellman and Oberg 2002). In this trial the McKenzie group had less pain and disability in the short- and medium-term; however, the effect sizes were small and not statistically significant. The effects on pain were a difference of minus eight and minus two at the different outcome points and the effects on the Neck Disability Index were a difference of minus five and minus two respectively on 100-point scales, favouring the McKenzie group. As there was only one study with data on cervical spine patients, the review (Clare *et al.* 2004b) concluded there is insufficient data to determine efficacy for cervical pain.

Kjellman and Oberg (2002) randomly allocated seventy-seven patients to general exercise, McKenzie therapy or a control group, 91% of whom were followed-up at twelve months. Pain intensity and frequency and neck-related disability improved in all groups with no significant differences in a three-group analysis (Figure 7.1). However, in a two-group analysis there was significantly greater improvement in McKenzie compared to the control group in pain intensity and Neck Disability Index, and after treatment the McKenzie group had improved by thirty-four points compared to twenty-nine and twenty-six in the exercise and control groups respectively. Significant improvements were noted for The Distress and Risk Assessment Method in the McKenzie group only, and whilst 70% of the two active groups were normal according to this measure, only 42% of the control group were normal. With a definition of clinically important change as five or more points on the Neck Disability Index, 60 – 63% of patients in the exercise and McKenzie groups achieved this compared to 37% in the control group. The exercise group had considerably more treatment during the intervention period (mean number of sessions thirteen compared to seven or eight for McKenzie and control groups), and during the follow-up year (102 visits to a health care professional compared to 46 and 140 respectively).

**Figure 7.1 Pain intensity changes: 0 – 100 scale over weeks**



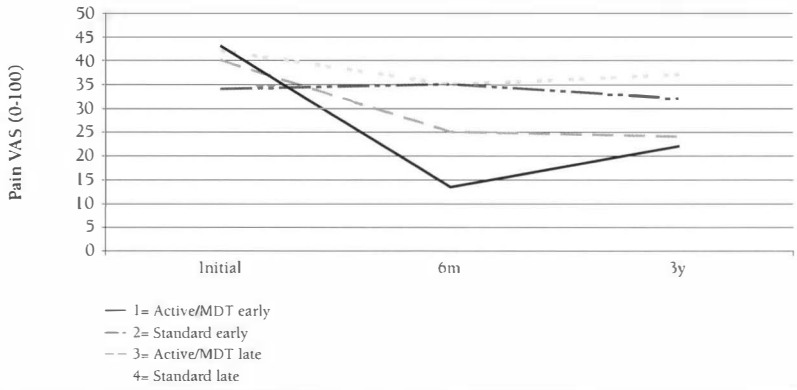

Source: Kjellman and Oberg (2002)

Secondary analysis of this trial involved objective measures of range of movement and muscle endurance and strength (Kjellman and Oberg 2004). Although there were improvements in the other groups, only the McKenzie group improved on all objective measures.

Rasmussen *et al.* (2001) reported on an uncontrolled cohort of sixty patients with neck and arm pain, with many demonstrating signs and symptoms of cervical radiculopathy that were followed-up at one year after McKenzie evaluation and management. Of the forty-five not receiving compensation, thirty were much better, eleven somewhat better, two were unchanged and two were lost to follow-up. Of the fifteen receiving compensation, two were much better, one somewhat better, nine were unchanged and three were worse. The differences were highly significant, and the authors concluded that with a low level of intervention after careful instruction, the McKenzie method was effective for treatment of cervical radiculopathy in patients not receiving compensation.

Regarding patients with symptoms from whiplash, early active movement augmented by mechanical diagnosis and therapy has been shown to be effective (Rosenfeld *et al.* 2000, 2003). This study is described in more detail in Chapter 25, but the results are presented below (Figure 7.2).

**Figure 7.2 Outcomes of whiplash: MDT versus standard intervention**




Source: Rosenfeld *et al.* 2003

The single efficacy study for non-specific neck pain published to date, although it suggests superiority of mechanical diagnosis and therapy over a control group, does not provide definitive evidence of treatment efficacy. Clearly more studies are needed in this area before firm conclusions can be drawn.

## Mechanically determined directional preference

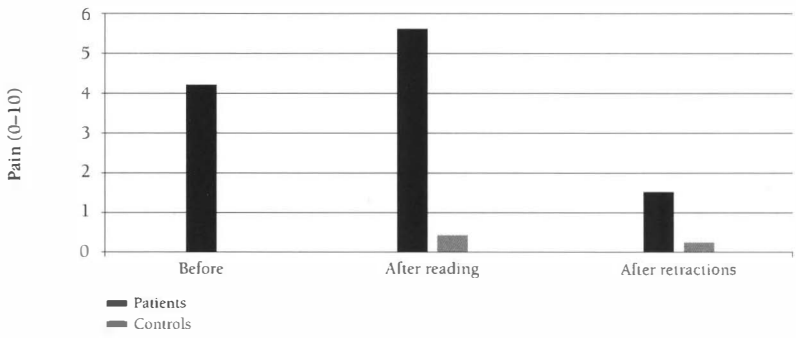
Mechanically determined directional preference describes the situation when postures or movements in one direction centralise, abolish or decrease symptoms and lead to an improvement in mechanical presentation. Very often postures or movements in the opposite direction cause symptoms and signs to worsen, although in part this is a response to the length of exposure to the provocative loading. The phenomenon of mechanically determined directional preference is characteristic of derangement syndrome and helps to identify the specific directional exercise that will lead to the best management strategy (Long *et al.* 2004). This study only involved patients with back pain, but it is a key study in demonstrating the importance of mechanically determined directional preference. It is proposed that mechanically determined directional preference will present in a similar way in patients with neck pain, although currently the evidence is limited. At randomisation patients were allocated to exercises that matched their mechanically determined directional preference (extension responder did extension exercises, for instance), were opposite to their mechanically determined directional preference (extension responder did flexion exercises), or general exercises

and evidence-based active care. At two weeks in nearly all outcome measures there were significant differences favouring the matched group. Over 90% reported themselves to be resolved or better, compared to 24% in the opposite group and 42% in the evidence-based group (Long *et al.* 2004).

Donelson *et al.* (1997) examined the pain response to repeated end-range testing of sagittal plane movements in eighty-six patients with neck and referred pain. Patients were randomised to perform the movements in different orders, which did not affect responses. In 45% of subjects, sagittal plane movements had consistent and opposite effects. Of these, 67% improved with retraction and extension and worsened with protrusion and flexion, and 33% improved with protrusion and flexion and worsened with retraction and extension. In another ten subjects (12%), both flexion and protrusion caused peripheralisation of pain, but either decreased pain intensity, or centralisation only occurred with retraction or extension rather than both. Thus, in total 57% of this sample displayed mechanically determined directional preference – in a single mechanical evaluation limited to four sets of ten repetitions, which did not use overpressure, mobilisation or frontal plane forces. In 43% of subjects there was an increase in pain intensity or peripheralisation with lower cervical flexion (flexion and protrusion) and a decrease in pain intensity or centralisation with extension and/or retraction.

Abdulwahab and Sabbahi (2000) investigated the effect of twenty minutes of sustained flexion and twenty repeated retraction movements in thirteen patients with cervical radiculopathy and ten control subjects. Flexion was mid-range as participants were simply asked to read a magazine in their own relaxed style. Outcomes evaluated were radicular pain intensity and the H-reflex amplitude as a measure of compression of the nerve, with a decrease representing compression. The H-reflex amplitude was significantly decreased after flexion and significantly increased after the retraction exercises. There was a significant increase in symptoms following sustained flexion and a significant decrease following retraction exercises in the radiculopathy group (Figure 7.3). Even the asymptomatic control group felt some discomfort after the period of sustained flexion.

**Figure 7.3 Effects of sustained flexion and retraction exercises on cervical radiculopathy and controls (N = 23)**



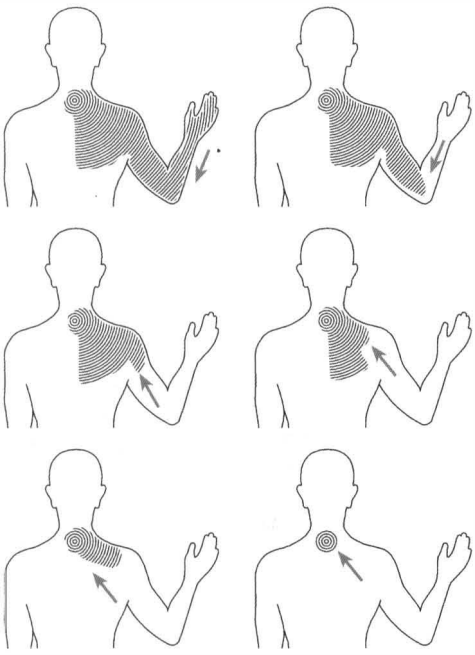

Source: Abdulwahab and Sabbahi 2000

## Centralisation

Centralisation refers to the phenomenon by which distal limb pain emanating from the spine is abolished in response to the deliberate application of loading strategies (Figure 7.4). The phenomenon is characteristic of derangement syndrome, and its high prevalence rate, reliability of assessment and value as a prognostic indicator has been established in a review (Aina *et al.* 2004). The review highlights the limited documented evidence about centralisation in the cervical spine.

Werneke *et al.* (1999) described the symptomatic responses of 289 patients, of whom 66 (23%) had neck pain. Centralisation was strictly defined as clear-cut abolition during mechanical evaluation that remained better and progressively improved at each session. Another group, classified as 'partial reduction', displayed gradual improvement over time, but this was not necessarily progressive or directly related to the treatment session. Similar percentages in the neck and back pain patients demonstrated centralisation (25% and 31% respectively) and partial reduction (46% and 44% respectively). There were no significant differences in outcome by pain site, so back and neck pain patients were analysed together. Centralisers averaged significantly fewer visits (four) than the partial reduction and non-centralisation groups (eight). However, there was no significant difference in pain or functional outcome between centralisation and partial reduction groups, which were both significantly better than the non-centralisation group.

**Figure 7.4 Centralisation of distal pain in response to repeated movements**



## Reliability

When an examination procedure is being used to determine management strategies, it is important that it has good intertester reliability to ensure that the procedure is consistently interpreted between clinicians. If a procedure has poor reliability, it demonstrates that clinicians cannot agree on how to interpret a particular finding. Unstable interpretations of physical examination findings are likely to lead to unsound and random clinical decisions about management. Although reliability is widely considered an important aspect of any examination process, deciding 'how much' reliability is enough is unclear and controversial. Kappa values of 0.4 have been accepted (Seffinger *et al.* 2004), but values below 0.5 have been said to indicate poor levels of agreement (Altman 1991), and 0.75 has been deemed a 'minimal requirement' (Streiner and Norman 2003).

Clare *et al.* (2004a) examined the reliability of fifty McKenzie-credentialed therapists in classifying fifty patients, twenty-five each cervical and lumbar, from McKenzie assessment forms. Results were not separated for neck and back paper-based cases. Kappa value for syndromes was 0.56 and for sub-syndromes was 0.68.

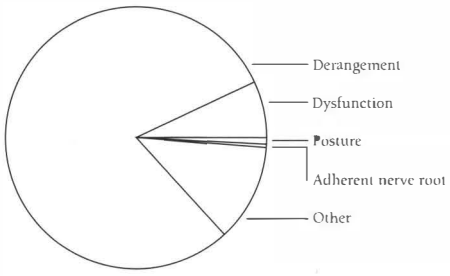
Clare *et al.* (2004c) examined reliability of assessment of fifty patients by pairs of therapists, fourteen in total; half of the patients had neck pain and half back pain. Prevalence of derangement was 88% / 84%, dysfunction 0% / 4%, posture 0% / 0% and 'Other' 12% / 12% for the two therapists. Kappa values for lumbar syndromes and sub-syndromes were 1.0 and 0.89 and for cervical syndromes and sub-syndromes 0.63 and 0.84 respectively.

Dionne and Bybee (2003) videotaped twenty patients with neck pain during a mechanical evaluation and then had fifty-four therapists at varying levels of the mechanical diagnosis and therapy educational programme view the videos and classify the patients. Reliability on agreement for diagnosis was kappa 0.55, for sub-syndrome classification was kappa 0.48 and for mechanically determined directional preference was 0.45.

## Prevalence of mechanical syndromes in neck pain patients

Two surveys have been conducted of consecutive patients seen by McKenzie educational faculty (May 2004a, 2004b). In total, details of over one thousand patients were included in the two surveys from nearly eighty contributing faculty members, which included 256 patients with neck pain. The results were similar in the two studies, with most neck pain patients being classified as derangement (80%), fewer numbers in other mechanical syndromes (8%, mostly dysfunction) and some classified as non-mechanical syndrome (12%). The minority of patients not receiving mechanical classification were mostly classified as mechanically inconclusive (4%), trauma (4%) and chronic pain state (3%).

**Figure 7.5 Classification of 256 consecutive neck pain patients**




Source: May 2004a, 2004b

Of those patients classified as derangement, the most common reductive force was extension (66%), but 25% used some element of the lateral treatment principle and 6% used flexion as the treatment principle.

## Conclusions

This chapter has outlined the available evidence that is directly relevant to the practice of mechanical diagnosis and therapy in the cervical spine. The main point is that the evidence is limited and so definitive conclusions about any aspect of the approach should be made with caution. In general, only one or two studies are available regarding any particular aspect; furthermore, a number of these studies are only available as abstracts or articles that have not been published in peer-reviewed journals. The evidence to date gives some support for efficacy, reliability, the existence of centralisation and mechanically determined directional preference and a high prevalence rate of mechanical syndromes in neck pain patients. However, further research is needed to reach definitive conclusions about all these aspects of mechanical diagnosis and therapy in the cervical spine and nothing at all has been published relevant to the thoracic spine.

# 8: Serious Spinal Pathology



## Introduction

The mechanical syndromes (McKenzie 1981, 1990) that encompass the majority of patients are described in other chapters. Most will be classified as derangement, with some dysfunction, and postural syndrome classification only occasionally. Only a small proportion of patients are not classified in one of the mechanical syndromes (May 2004b). This includes a very few patients who have serious spinal pathology, which is the subject of this chapter.

*Within specific conditions that must be considered are the serious spinal conditions that need early identification and referral to an appropriate specialist*. A brief description is given in the chapter of cervical and thoracic myelopathy, fractures, tumours, spinal infection and other conditions as examples of serious spinal pathology, which are *absolute contraindications* for mechanical diagnosis and therapy. Mention is also made here of inflammatory arthropathies and osteoporosis, conditions whose management may involve physiotherapy, but require special consideration and appropriate diagnosis. The contentious issues of vertebrobasilar artery insufficiency and cervical manipulative therapy are also considered in this chapter.

During the initial assessment, an index of suspicion for serious spinal pathology should be maintained, with the triage system (after Spitzer *et al*. 1987; CSAG 1994) being used:

- serious spinal pathology
- cervical radiculopathy
- mechanical neck pain.

The incidence of serious spinal pathology may be less in the neck than the back, where an incidence of 1.4% has been reported (McGuirk *et al*. 2001). In general, carcinomas and spinal infections affect the cervical spine less than the other two spinal regions (Durr *et al*. 2002; Narlawar *et al*. 2002). In a survey of McKenzie Institute faculty of over eight hundred patients, no 'red flag' conditions were reported for neck pain patients, while there was a prevalence of 1.3% for back and 6% for thoracic problems (May 2004b).

Sections in the chapter are as follows:

- identification of serious spinal pathology
- cancer/tumour
- Horner's syndrome
- spinal cord
    - cervical
    - natural history and treatment options
    - thoracic
- fractures and dislocations
- osteoporosis
- spinal infection
- rheumatoid arthritis (RA)
- ankylosing spondylitis
- upper cervical instability
- extreme dizziness/vertigo
- cervical spine and vertebrobasilar artery insufficiency (VBI)
    - background
    - testing protocol
    - dizziness
    - VBI test protocol
    - problems with the tests
    - legal situation
    - implications for mechanical diagnosis and therapy
    - end-range sustained testing
- carotid artery pathology.

## Identification of serious spinal pathology

It is recommended that the same 'red flags' used to provide clues as to the existence of serious spinal pathology in patients with back problems be applied to patients with neck pain (Nachemson and Vingard 2000; Honet and Ellenberg, 2003). The recommendation

exists because there is a lack of evidence with regard to 'red flags' for the cervical spine (Nachemson and Vingard 2000; Honet and Ellenberg, 2003). Minimal work has been done to evaluate the diagnostic accuracy, incidence or comprehensiveness of these 'red flags' in large groups of neck (or thoracic) pain patients. Indeed, most reports on 'red flags' have been based on case studies or series that are present in the literature, but are not helpful in addressing these issues.

Rather than seeking to identify specific pathology through individual items of history or physical examination, an alternative approach has been to predict serious spinal pathology in general from these items (Waddell 2004). When diagnostic triaging is involved, the key distinction is between a patient with serious spinal pathology, who should be referred for further investigations, and a patient with mechanical neck pain, who should be treated. Determining exactly which pathology is involved is less important at this stage. Bisschop (2003) and Ombregt (2003) produced a list of warning signs for the cervical and thoracic spine without indicating specific pathologies. This included items such as progressively increasing pain, unaffected by movements and postures, and involvement of two or three nerve root levels. It should be noted that a patient with an internal carotid artery dissection brought on by a violent sneeze presented first of all with signs and symptoms of mechanical neck pain before the development of more severe symptoms (Taylor and Kerry 2005).

**Table 8.1 'Red flags' that may indicate serious pathology in neck and thoracic pain patients***

| *Condition* | *Symptom or sign* |
|---|---|
| Spinal tumours | Age > 50 years |
| | Previous history of cancer |
| | Unexplained weight loss |
| | Constant progressive pain at night |
| | Pain lasting more than one month |
| | No improvement after one month of conservative management |
| | Elderly person with neck pain for first time |
| | Elderly person with rapidly increasing pain and/or stiffness in the neck |
| | Dysphagia |
| | Multiradicular weakness. |

Continued next page

| *Condition* | *Symptom or sign* |
|---|---|
| Spinal infection | Age > 50 years |
| | Cause for infection – urinary tract, skin or respiratory infection, intravenous drug use, tuberculosis, surgery |
| | Fever/systemic illness. |
| Fracture | History of major trauma |
| | Age > 70 years |
| | Corticosteroid use. |
| Spinal cord lesion** | Bladder or bowel dysfunction |
| | Widespread progressive motor weakness, disturbed gait, clumsiness, loss of dexterity |
| | Widespread paraesthesia |
| | Increased tone/spasticity/hyperreflexes/clonus |
| | Positive Babinski sign (extensor plantar response). |
| Inflammatory arthropathy | Gradual onset < 40 years of age |
| | Marked morning stiffness |
| | Persisting limitation of movement |
| | Peripheral joint involvement |
| | Iritis, skin rashes, colitis, urethral discharge |
| | Family history. |
| Vascular/neurological | Extreme dizziness |
| | Abnormal speech, sight or swallowing |
| | Blackouts or falls |
| | Positive cranial nerve signs. |

Source: Nachemson and Vingard 2000; Barnett *et al.* 1987; Bland 1994; Ombregt 2003; Bisschop 2003

* If suspicion of serious spinal pathology is not clear from the history, it should quickly become apparent that loading strategies produce no lasting symptom reduction. Worsening of symptoms in response to all loading strategies is likely.

**more detail in Table 8.2.

## Cancer/tumour

Tumours, whether benign or malignant, are a rare occurrence in the cervical spine (Bland 1994) and more common but still rare in the thoracic spine. The incidence of metastases or secondary malignant tumours is greater than primary spinal tumours (Slipman *et al.* 2003). Bland (1994) gives a 3:1 ratio of benign versus malignant tumours in the cervical spine.

Most primary or secondary spinal tumours occur in the thoracic (about 50%) or lumbar region (about 30% to 45%), with only 6% to 19% occurring in the cervical region (Weinstein and McLain 1987; Bernat *et al.* 1983; Schaberg and Gainor 1985; Durr *et al.* 2002; Rao and Davis 1998). Metastases in the spine may pass unnoticed for a considerable time and sometimes are only discovered during routine radiography. Especially in the cervical spine, where presentation of the disease is less dramatic, many patients do not present with neurologic deficits and symptoms until later stages (Rao and Davis 1998).

When symptomatic, pain is the earliest and most prominent feature in over 90% of cases (Portenoy *et al.* 1987; Portenoy 1993; McCallister and Kaufman 1994; Daw and Markman 2000) with nerve root involvement and/or spinal cord compression symptoms and signs usually occurring only at later stages, although this can depend on the type of tumour (Posner 1987; Auld and Buerman 1966; Portenoy 1993). The pain tends to be unremitting and often more intense at night or during times of inactivity (Clark 1991; Bisschop 2003).

The suggested 'red flag' warning indicators for tumours in the cervical and thoracic spine are the same as suggested for the lumbar spine (Table 8.1), although their diagnostic accuracy has not been evaluated exclusively in the context of cervical and thoracic tumours. Diagnostic studies have tended to focus on the identification of signs and symptoms for spinal tumours regardless of the anatomic site. For instance, Slipman *et al.* (2003) reported an incidence of 0.69% in academic spine centres, compared to 0.12% in private practice spine centres for all spine tumours. Patients had an average age of 65 years and reported night pain (48%), spontaneous onset of symptoms (94%), history of cancer (55%) and unexplained weight loss (23%) commonly, but not universally.

## Horner's syndrome

Horner's syndrome occurs as a result of interference to the sympathetic nerve supply to the eye in the central or peripheral nervous system (Berkow *et al.* 1992; Walton and Buono 2003). It comprises the following symptoms:

- variable drooping of upper eyelid – ptosis
- constriction of the pupil – miosis

- ipsilateral loss of sweating – anhydrosis
- recession of eyeball into the socket – enopthalmos.

This may result from central nervous system lesions or damage to the cervical sympathetic chain or ganglion, and includes some conditions that may initially mimic thoracic or cervical problems. This includes Pancoast tumours or cord lesions, such as a disc herniation, at C8 – T1 (Clark 1991; Mellion and Ladeira 2001) (see Spinal Cord section).

Pancoast tumours develop in the apical parietal pleura and comprise less than 5% of all lung tumours, are primarily found in men, and are most commonly diagnosed in patients between 50 and 60 years of age (Kovach and Huslig 1984). The tumour may invade the stellate ganglia of the sympathetic chain as well as the brachial plexus to produce the constellation of symptoms known as Horner's syndrome, but not all symptoms may be present (Clark 1991; Spengler *et al.* 1973). Musculoskeletal complaints are frequently the initial symptoms, with similar pain patterns to cervical problems, sometimes intermittent, and demonstrating response to thoracic, neck or shoulder movements or limitation of shoulder movements. Sleep may be disturbed. The early presentation may be pain around the neck, scapula and shoulder, often with radiation into the arm and paraesthesia. Individuals are generally 50 years of age or older and are frequently smokers, often with an associated smoker's cough, but the tumour has been reported in non-smokers and patients as young as 30 (Yacoub and Hupert 1980; Downs 1990; Spengler *et al.* 1973; Kovach and Huslig 1984). Because of the diffuse nature of complaints, diagnosis is often delayed for many months. When Horner's syndrome is present, ptosis and miosis are the most usual elements. Late clinical findings may include weight loss, hoarseness, weakness in the upper limb, and upper motor neurone signs and symptoms (Kovach and Huslig 1984).

Since Horner's syndrome can result from any pathology affecting the spinal cord, brain stem or sympathetic nervous system in the thoracic or cervical spine, its presence is a contraindication for mechanical therapy until the cause is identified. Horner's syndrome has been reported as a rare complication following manipulation (Grayson 1987).

## Spinal cord

Spinal cord lesions may result from different lesions in the cervical and thoracic spines; most commonly these are:

- degenerative changes producing stenosis in the cervical spinal canal
- disc lesions in the thoracic spinal canal.

In the cervical spine some authors make a distinction between mechanical and vascular causes of myelopathy, and whether it is combined with radiculopathy (Ferguson and Caplan 1985). Signs and symptoms vary due to different spinal levels being involved.

### Cervical

At surgery in the cervical spine, two causes of myelopathy and radiculopathy have been noted: soft or hard disc herniations. Hard discs refer to bony growths, such as foraminal spurs, transverse bony ridges on the vertebral body, uncovertebral exostosis and other degenerative changes that occur with cervical spondylosis (Odom *et al.* 1958; Henderson *et al.* 1983; Mosdal and Overgaard 1984; Allen 1952). Spondylotic bars or ridges encroach into the spinal canal, whilst hypertrophying bony tissue from the zygapophyseal or the uncovertebral joints encroach into the foramen (Parke 1988). Soft disc herniation, that is cervical disc prolapse, has also been reported to be the cause of radiculopathy and myelopathy (Bertalanffy and Eggert 1988; Young and O'Laoire 1987; O'Laoire and Thomas 1983).

Cervical spondylotic myelopathy is reported to be the most common cord lesion after middle age (Young 2000), but cervical spondylotic radiculopathy is more prevalent (Bland 1994; Yu *et al.* 1987).

Patients may present with predominantly an upper motor lesion or predominantly a lower motor lesion, but a mixed pattern does occur (Gregorious *et al.* 1976; Bertalanffy and Eggert 1988). There may be lower motor involvement at the level of the lesion and upper motor neurone below this level (Clark 1991). In general, cervical myelopathic symptoms are dependent on the etiologic process and the pathophysiology that is present.

Five categories of cervical spondylotic myelopathy based on neurological findings have been described in order of decreasing frequency (Crandal and Batzdorf 1966; Clark 1991):

- transverse lesion syndrome – corticospinal, spinothalamic and posterior column involvement
- motor syndrome – corticospinal or anterior horn cell
- central cord syndrome – motor and sensory involvement of upper extremities more than lower extremities
- Brown-Sequard syndrome – unilateral cord lesion, with ipsilateral corticospinal tract involvement and contralateral analgesia below the level of the lesion
- brachialgia and cord syndrome – predominant upper limb pain, with some long tract involvement.

Cord involvement may or may not be symmetrical, so symptoms may be bilateral or unilateral. The tracts most commonly affected are the pyramidal, spinothalamic and posterior column (Yu *et al.* 1987). Cord involvement may be characterised by a deep aching pain and burning sensation (Clark 1991). Signs and symptoms are those of an upper motor lesion and spinal pain is not always present.

More commonly myelopathy arises from lesions in the lower cervical spine causing lower limb involvement, with non-dermatomal patterns of muscle weakness and/or sensory disturbance, hyperreflexes, and bladder or bowel dysfunction. Less commonly upper cervical segments are involved and symptoms mainly affect the upper limbs (Clark 1991). There are no or minimal symptoms in the legs, but paraesthesia and proprioceptive loss in the hands. Typically the patient complains of numb, clumsy hands and loss of dexterity.

Cases have been reported of mid-cervical lesions causing predominantly upper extremity signs and symptoms (Shinomiya *et al.* 1994; Nakajima and Hirayama 1995). Motor loss at deltoid, possibly associated with sensory loss or localised pain, or numbness in the fingers and clumsiness of the hands were predominant symptoms, but examination reveals changes in the lower limbs also. As in other areas, a mixture of lower and upper motor neurone changes may be present.

Onset tends to be insidious, unless trauma is involved, and diagnosis may not be made for several years. The neurological signs and symptoms vary, considerably reflecting the nature and site of the lesion, the rate of progression and the extent of degenerative changes (see Table 8.2).

In the cervical spine, as myelopathy is associated with degenerative changes, the patient is likely to be 50 years old or more.

As in other areas of the spine, stenotic symptoms have a static and dynamic, and possibly an ischaemic component (Zeidman and Ducker 1998; Young 2000). The static factors relate to congenitally narrow canals as well as the degenerative changes of cervical spondylosis, whilst the dynamic component involves the narrowing effect extension has on the spinal canal and intervertebral foramen, and the translation of vertebrae on each other (Nurick 1972a; Edwards *et al.* 2003; Magnaes 1982). Thus flexion may temporarily relieve symptoms and extension temporarily increase them.

### Natural history and treatment options

These patients should not be treated with mechanical forces; they should be referred for further investigations. However, it may be helpful to have an understanding of the natural history and the treatment options available.

Few studies of the natural history of spondylotic myelopathy have been performed (Lees and Turner 1963; Nurick 1972b; Philips 1973) and those that have were small, with dissimilar disability grading systems and outcomes that were not comparable (Zeidman and Ducker 1998). It has been reported that myelopathy is nearly always a progressive condition (Bohlman and Emery 1988). In some cases rapid deterioration can occur; once the spinal cord has reached a critical level of compression, trivial trauma may produce sudden and severe symptomatology and neurological deficit (Bohlman and Emery 1988). Chronic myelopathy secondary to cervical spondylosis can also occur (Zeidman and Ducker 1998).

However, although the evidence is of poor quality, it seems that the prognosis for those with cervical myelopathy can be variable. From a review of several early studies, Edwards *et al.* (2003) concluded that although the majority of patients experienced gradual deterioration in their neurological status, some improved with conservative treatment. A conservative approach to management of cervical myelopathy has been demonstrated to be a viable option, with outcomes as good as surgery, in patients with mild to moderate symptoms and shorter disease duration (Yoshimatsu *et al.* 2001; Kadanka *et al.* 2002). (See Surgery for cervical and thoracic problems section in Chapter 9.)

### Thoracic

Thoracic spinal stenosis is rarely mentioned in the literature. It is much less common compared to its incidence in the cervical and lumbar spine, with no information about its natural history (Errico *et al.* 1997; Kalfas 2000). One author reports six cases of thoracic myelopathy associated with thoracic spinal stenosis (Barnett *et al.* 1987). Thoracic stenosis is usually the result of degenerative changes in the three-joint complex of the spine (disc and zygapophyseal joints), with hypertrophy of the zygapophyseal joints and osteophyte narrowing of the spinal canal resulting in pain and neurologic symptoms (Errico *et al.* 1997; Barnett *et al.* 1987).

Reports of thoracic spinal cord lesions due to disc herniations are more common. The anatomy of the thoracic spinal canal as well as the blood supply to the thoracic cord predispose to a spinal cord impingement from even a relatively small disc herniation (Errico *et al.* 1997).

Thoracic disc herniations are said to make up only 1 – 2% of all spinal disc herniations (Kramer 1990; Arce and Dohrmann 1985; Mellion and Ladeira 2001). Given the comparatively low proportion of thoracic problems, the large number of these reports suggests the need for greater alertness for this 'red flag' in this region. Disc protrusion in a central posterior direction may affect the spinal cord, which includes about 70% of herniations (Arce and Dohrmann 1985; Mellion and Ladeira 2001). Unusual presentations with diffuse symptoms have been reported, such as chronic shoulder pain or buttock and thigh symptoms, which sometimes make it a diagnostic challenge (Wilke *et al.* 2000; Singer and Edmondston 2000).

Synopses of the literature have been performed at different dates (Arce and Dohrmann 1985; Mellion and Ladeira 2001). Herniations may occur at any segmental level, but the majority occur in the lower thoracic spine and reports of upper thoracic disc herniations are extremely rare (Arce and Dohrmann 1985; Mellion and Ladeira 2001; Singounas *et al.* 1992; Arseni and Nash 1960; Russell 1989). As the narrowest section of the spinal canal is between T4 – T9, thoracic disc herniations may affect the cord in a disproportionate way (Kramer 1990; Logue 1952; Ravichandran and Frankel 1981).

Presentation will be extremely variable, but three patterns are common (Mellion and Ladeira 2001):

- somatic – local/radiating pain
- radicular pain – band-like chest pain/lower extremity pain
- cord compression signs – motor weakness/sensory disturbances/ bladder and bowel disturbance.

Thoracic disc herniations presenting with somatic and/or radicular symptoms can be given a mechanical evaluation in the normal way, although failure to respond in the second group may lead to classification as an irreducible derangement. The third group is the concern of this section.

Onset has been reported to be insidious in at least 70% of cases (Arce and Dohrmann 1985; Mellion and Ladeira 2001; Russell 1989). Only in a minority of cases is there a history of acute onset cord signs and symptoms. Most commonly symptoms start as back pain and spread to include more serious sensory and motor symptoms over time, possibly in an episodic way. However, back pain may not even be present. This means that long delays between onset of symptoms and diagnosis may occur, especially as there maybe confusion with visceral disease because of the site of symptoms.

Patients with thoracic disc herniations have been misdiagnosed with cardiac, pulmonary, gastrointestinal, renal, cardiac neurosis or psychiatric disorders (Mellion and Ladeira 2001). Prolonged misdiagnosis as a non-specific complaint may not only lead to inappropriate treatment, but to spinal cord compression and permanent upper motor neurone lesion. There will be a variety of presentations that may or may not include pain, with a mixture of neurological signs and symptoms related to an upper motor lesion (Table 8.2).

As symptoms are due to a disc herniation rather than degenerative changes, as in the cervical spine, the age is usually younger with reported mean ages in the 40s (Mellion and Ladeira 2001).

Disc herniations at upper thoracic levels are rare (Mellion and Ladeira 2001) and are usually manifested by symptoms in the ulnar distribution in the arm and sensory or motor deficit in the hand. A lesion at T1 may be accompanied by the signs of Horner's syndrome (see above). Pain may involve the neck, scapular and anterior upper chest, and there may be reduced reflexes in the upper limb. It has been suggested that as mechanical T1 lesions are so rare, clinicians

should always be aware of non-mechanical and serious pathology (Mellion and Ladeira 2001).

**Table 8.2 Signs and symptoms associated with spinal cord lesions in the cervical and thoracic spine**

| | |
|---|---|
| Cervical spine | • non-myotomal weakness/wasting in the hands |
| | • clumsiness of the hands/diminished dexterity |
| | • non-myotomal weakness and atrophy upper limb |
| | • non-dermatomal paraesthesia/numbness upper limb |
| | • Lhermitte's sign – electric shock-type sensation down spine or legs on neck flexion. |
| Cervical and thoracic spine | • non-dermatomal paraesthesia/numbness in lower limbs |
| | • non-myotomal muscle weakness in lower limbs; may present initially as stiffness, clumsiness and unsteadiness in the limbs and progress to gait disturbance/broad-based gait |
| | • decreased co-ordination |
| | • flaccid/spastic paraplegia |
| | • increased tone/spasticity |
| | • hyperreflexes |
| | • extensor plantar reflex/positive Babinski sign |
| | • clonus |
| | • bladder, bowel or genital dysfunction – retention/incontinence/sphincter disturbance. |

Source: Yu *et al.* 1987; Clark 1991; Lestini and Wiesel 1989; Connell and Wiesel 1992; Arce and Dohrmann 1985; Mellion and Ladeira 2001; Edwards *et al.* 2003

## Fractures and dislocations

Fractures of the cervical or thoracic spine or ligamentous instabilities of the upper cervical spine may be caused by a variety of traumatic events, such as motor vehicle accidents, diving into shallow water, falling from a high place or a number of athletic activities. The thoracolumbar junction is reported as the most common site for non-osteoporosis-related spinal column fractures (Huler 1997). Fractures of the ribs may be caused by repeated muscular contractions. There are more reports of stress fractures of the first rib than any other single rib (Gregory *et al.* 2002).

Fractures and dislocations may also occur secondary to existing systemic bone-weakening pathology, such as rheumatoid arthritis or ankylosing spondylitis in the cervical spine or osteoporosis in the

thoracic spine (Bland 1994). The earliest osteoporotic fracture events are typically seen in the upper thoracic spine, but surprisingly up to 50% of these fractures are asymptomatic (Kanis and Pitt 1992). Vertebral collapse is often insidious, progressing over weeks or months without any radiographic evidence of fracture (Kostuik and Heggeness 1997). Progressive collapse of multiple vertebrae in this anatomic region, however, can lead to a significant upper thoracic kyphosis, often referred to as 'dowager's hump' (Kostuik and Heggeness 1997).

Not everybody involved in an accident to the neck needs an x-ray; for instance, patients at the minor end of the traumatic continuum. Following a whiplash-type injury, it is suggested that certain factors indicate the need for radiological investigation (see Table 8.3; Figure 8.1).

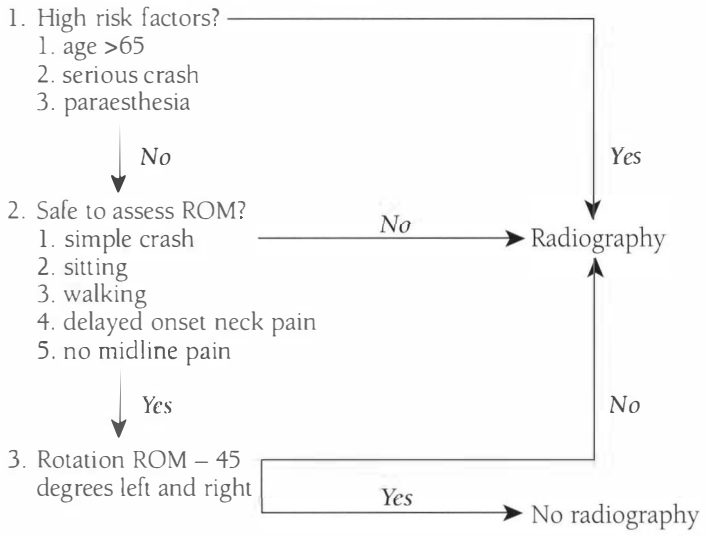
**Table 8.3 Suggested indicators for investigation following trauma**

- loss of consciousness
- death of another occupant of the vehicle
- high speed or high impact injury
- QTF WAD Grade III*
- bilateral extremity symptoms.

Source: Bidese *et al.* 2001; Banerjee *et al.* 2004

* Neck complaint and neurological signs

**Figure 8.1 Canadian C-spine rules for radiography in alert and stable patients**




Source: Stiell *et al.* 2001, 2003

These are not absolute variables, however, and clinicians' clinical reasoning must be used to determine the potential value of an x-ray. Obviously the greater the traumatic impact, the more useful radiography is to reassure both clinician and patient. Most of those who need an x-ray will have received the investigation at the time of the accident. However, if concern persists in the presence of one or more of these criteria, further investigation may be warranted. Initially clear x-rays do not always guarantee individuals have avoided significant damage. Six patients with normal radiographs, including flexion-extension views and normal neurology, were found to have zygapophyseal joint dislocation or subluxation and neurological deficits at two-week follow-up (Herkowitz and Rothman 1984). Another case study describes an individual with chronic neck pain two months post-injury, with normal x-rays, who appeared to have alar ligament laxity on later radiography (Derrick and Chesworth 1992). If any of the systemic diseases mentioned above are present, relatively minor trauma may be an indicator for an x-ray.

When radiographic investigation is indicated this should include: anterior-posterior, lateral and open-mouthed views (Bidese *et al.* 2001). The latter visualises the odontoid, axis body, atlas lateral masses and periodontal interspaces in the coronal plane (Deltoff 2001). As 5 – 10% of patients with spinal injury have multiple rather than single-level spinal injuries (Huler 1997), it is suggested that the entire spine should be evaluated radiographically if a spinal injury is identified (Bisschop 2003; Huler 1997).

## Osteoporosis

Osteoporosis is the most common metabolic disorder affecting the spine. The suggested World Health Organisation definition is bone mineral density more than 2.5 standard deviations below the mean of normal young people (Melton 1997). According to this definition, approximately 30% of post-menopausal white women in the USA have the condition, and 16% have osteoporosis of the spine. Prevalence is less in non-white populations. Bone density decline begins in both sexes around 40 years of age, but accelerates after 50, especially in women (Bennell *et al.* 2000).

Low bone density leads to increased risk of fracture with no or minimal trauma. The most common fracture sites are the spine, femur and radius. Vertebral fractures affect at least 25% of post-menopausal

women; however, a substantial proportion of fractures are asymptomatic and never diagnosed, and therefore the true rate could be higher (Cummings and Melton 2002). Fractures of the thoracic spine are common and can lead to an exaggerated thoracic kyphosis. Despite widespread belief that osteoporosis primarily affects women, recent data shows that in fact vertebral fractures are as common in men as women. Because women live longer, the lifetime risk of a vertebral fracture from 50 onwards is 16% in white women and only 5% in white men (Melton 1997; Andersson *et al.* 1997).

Although it occurs predominantly in the elderly and in post-menopausal women, there are important secondary causes of osteoporosis not related to age. These include history of anorexia nervosa, smoking, corticosteroid use, inadequate intake or absorption of calcium and vitamin D, amenorrhea, low levels of exercise, lack of oestrogen, and coeliac disease (Smith 2000; Bennell *et al.* 2000).

Low bone mass (osteopenia) is in itself asymptomatic and individuals may be unaware that they have the condition until a fracture occurs. Although pain can be absent, it can be severe, localised and difficult to treat and take many weeks to settle; the fractures also cause a loss of height (Smith 2000). Specific aspects of history-taking and physical examination appropriate to this group have been detailed elsewhere (Bennell and Larsen 2004).

*The condition, or suspicion of it, is an absolute contraindication to manipulation and mobilisation techniques*. However, exercise is not only *not* contraindicated, it should be included as part of the management strategy for primary and secondary prevention. The effects of exercise on skeletal strength vary at different ages (Bennell *et al.* 2000). Gains in bone mass are much greater in childhood and adolescence than in adulthood. The adult skeleton is very responsive to the adverse effects of stress deprivation and lack of exercise, which tend to exacerbate the natural decline in bone density that occurs with ageing. Trials of exercise have consistently shown that loss of bone mass is reduced, prevented or reversed in the lumbar spine and femur (Bennell *et al.* 2000; Wolff *et al.* 1999; Bennell and Larsen 2004).

Exercise that has a higher ground impact is most effective at bone strengthening. Non-weight-bearing exercises such as cycling or swimming will not strengthen bones, whatever other benefits they may provide (Bennell *et al.* 2000). Exercise programmes have

included stair-climbing, aerobics, skipping, jumping, dancing and jogging. More impact and loading is appropriate in primary prevention, but a less vigorous programme should be used in frailer groups. Programmes should be progressed in terms of intensity and impact, and maintained indefinitely, as the positive effects are reversed when regular exercise is stopped. Physiotherapy management and exercise guidelines have been reviewed in considerable detail (Bennell *et al.* 2000; Mitchell *et al.* 1999). Exercise therapy is complementary to but not a substitute for medical management, which includes hormone replacement therapy, calcium, vitamin D, calcitonin, biphosphonates and fluoride (Lane *et al.* 1996).

Posture is an important factor in osteoporosis. Flexion should be minimised as this can trigger damage to the vertebra; extension exercises and an extended posture should be encouraged. A group of fifty-nine women with post-menopausal osteoporosis were allocated to different exercise groups, performing extension, flexion, a combination of both or a no-exercise group. At follow-up at least sixteen months later, the extent of further fractures in the different groups was compared. Further deterioration was significantly less in the extension group (16%) than the flexion group (89%); the combined group (53%) and the no-exercise group (67%) (Sinaki and Mikkelsen 1984).

Established osteoporosis is difficult to treat, and optimal management is about maximising bone mass in early adult life and preventing excessive bone loss in later life (Turner 2000); in other words, the answer lies in prophylaxis. *"Osteoporosis is preventable, and its prevention is a priority for all health professionals"* (Turner 2000).

## Spinal infection

Infection in the cervical spine is a very rare occurrence, being the most uncommon spinal region affected, and in the thoracic spine still rare but less so. Although the cervical region is not affected as frequently as the thoracic and lumbar spine, it is suggested that cervical infections have the highest rate of neurologic compromise and the greatest potential for causing disability (Currier *et al.* 1998). The proportion of spinal infections diagnosed in each region of the spine has been reported as follows: cervical 0% – 12%, thoracic 33% – 48%, lumbar 39% – 59% (Carragee 1997; Krogsgaard *et al.* 1998; Narlawar *et al.* 2002). One report suggests an annual incidence of only 0.0005%

(Krogsgaard *et al.* 1998). The most at-risk groups seem to be the young and the old. Patients may present with spinal pain, neurological signs and symptoms, and/or bladder or bowel symptoms. Symptoms are likely to be progressive and of a non-mechanical nature, and can be accompanied by severe restrictions of movement.

Spinal infection is mostly associated with another source of infection, commonly urinary tract, skin or respiratory infections or tuberculosis, although the origin is not always obvious and fever is not always present. Another cause may be infection resulting from surgery. Usually patients are unwell, suffering a general malaise, night pain and sweats, with a raised temperature (Bland 1994; Narlawar *et al.* 2002; Carragee 1997; Krogsgaard *et al.* 1998).

The suggested 'red flag' warning indicators for infection in the cervical and thoracic spine are the same as suggested for the lumbar spine (Table 8.1), although their diagnostic accuracy has not been evaluated in this context.

## Rheumatoid arthritis (RA)

If patients develop neck pain as a result of one of the systemic arthropathies such as RA or AS, they will generally have had symptoms for many years (Maghraoui *et al.* 2003). The cervical spine is an unlikely site for onset of symptoms, and the patient will generally be aware of the diagnosis. In patients with RA, neck pain has been reported in 40 – 88%; cervical subluxations have been observed in 43% – 86%; and neurologic deficit has been reported in only 7% – 34% (Pellicci *et al.* 1981).

The disease process involved with RA may produce significant instability of the cervical spine that ***might*** threaten or result in neurological compromise (Clark 1991; Bisschop 2003). Common patterns involve subluxation or impaction involving the atlantoaxial or subaxial joints (Kauppi and Hakala 1994; Fujiwara *et al.* 2000).

There may be minimal neck symptoms, but usually chronic and multiple involvement at other joints. Symptoms may include or progress to referred pain and neurological deficit associated with upper and lower motor neurone involvement. The reported rate of neural impairment due to cervical instability varies considerably (Conaty and Mongan 1981; Sherk 1978; Fujiwara *et al.* 2000). This discrepancy may be

attributed to variability in neurologic classification systems as well as to difficulty in detection of subtle neurologic deterioration in patients who have muscle weakness and atrophy secondary to chronic RA (Boden and Clark 1998). Given that the condition is both progressive and associated with significant instability at the upper cervical spine, any involvement with RA patients should always be conducted with considerable caution.

## Ankylosing spondylitis

Ankylosing spondylitis is an inflammatory systemic disease that can affect the whole spine. It usually commences with sacral and lumbar pain, but may involve the thoracolumbar spine early on (Singer 2000). Once the thoracic spine is involved, due to involvement of the costal joints, respiration may become impaired. Later on the disease causes ankylosis of joints and ossification of ligaments leading to an immobile, fused spine and structural deformity, such as a fixed thoracic kyphosis. Although not a contraindication to physiotherapy, it is important that ankylosing spondylitis (AS) is recognised and the patient is seen by a rheumatologist and receives specialist advice. Management is by rheumatology and physiotherapy and involves regular exercise programmes to limit the effect the disease has on postural deformity. Prior to diagnosis, which involves radiographic changes at the sacroiliac joints, patients may present with thoracolumbar pain that is caused by unrecognised AS. The patient will likely be young and male, have had persistent symptoms for some months, not be relieved by rest, have associated early morning stiffness and improvement with exercise; however, these items lack sensitivity in the general population. Pain is caused by the inflammatory process, and such patients present with an atypical non-mechanical response, although they may demonstrate improvements with a general exercise regime. For a fuller review, see McKenzie and May (2003).

## Upper cervical instability

A generally accepted definition of instability does not exist (Swinkels and Oostendorp 1996). Concerns about instability at upper cervical levels relate to systemic conditions, such as rheumatoid arthritis (RA) or cervical trauma (Aspinall 1990; Bland 1994). There may be a discrepancy between the degree of destruction or instability and the symptoms. Patients with slight instability may have major neurological

problems, whereas others may have significant laxity without neurological symptoms (Meijers *et al.* 1974; Shaw and Cartlidge 1976).

In RA the upper cervical spine may become involved through erosion or stretching of ligaments and/or erosion of bone leading to subluxation or longitudinal settling of occiput – C1, or C1 – C2. Compression of the cervical spinal cord and/or brainstem may result either from direct compression by synovial pannus or indirect compression due to cervical subluxations (Boden and Clark 1998). Mechanical therapy is ***absolutely*** contraindicated in anyone with moderate to severe RA. Cervical spine involvement may be predicted by the presence of severe deformity of the metacarpophalangeal joints, steroid use for more than ten years, and possibly seropositivity (Bland 1994). The lifetime risk of cervical deformity in RA patients has been estimated at 33% to 80%, with the lower values underestimating the true risk (Bland 1994).

Major trauma is the other possible cause of cervical instability. Radiography or imaging studies are not routinely needed for patients following whiplash or trauma, but it should be noted that a plain x-ray might miss significant bony injury (Barnsley *et al.* 2002). Serious injuries do occur during motor vehicle accidents, but these are rare and should be detected at the time of the accident (Robertson *et al.* 2002). Currently the most common way of investigating subluxation instability in the upper cervical region is by x-ray, with measurements taken of the atlas-dens interval in flexion, neutral and extension (Cattrysse *et al.* 1997).

Any indication that the patient has an upper motor neurone lesion or major instability in the neck requires urgent specialist referral and immobilisation of the neck prior to the patient leaving the clinic. An anecdotal clue to instability is if the patient enters the clinic cradling the chin between the hands to prevent any movement. Various tests have been described, whose validity and reliability has not been proven, the purpose of which is to determine if upper cervical instability is present – an undertaking that seems potentially dangerous in itself.

One of these is the Sharp-Purser test, the predictive value and specificity of which has been evaluated in one study as 85% and 95% respectively (Uitvlught and Indenbaum 1988). Sensitivity varied depending on the criteria: if greater than 3mm or greater than 4mm of movement, from 69% to 88% respectively. However, an earlier study gave much

poorer diagnostic accuracy, with sensitivity of 32% and specificity of 56% (Matthews 1969). The reliability of three tests has also been investigated in eleven children with Down's syndrome, two of whom had radiographic atlantoaxial instability (Cattrysse *et al.* 1997). For the upper cervical flexion test, intratester reliability was greater than kappa 0.64 for three out of four testers, and intertester reliability was greater than kappa 0.64 in four out of six comparisons, with the other two being kappa 0.50. For the lateral displacement and Sharp-Purser tests, intratester reliability is questionable and intertester reliability is poor, and so these tests are not recommended.

## Extreme dizziness/vertigo

Although patients with cervicogenic disorders can sometimes present with associated dizziness, extreme dizziness, especially if associated with other neurological symptoms, can indicate pathology of the central nervous system, and treatment is contraindicated. Dizziness can also be associated with other pathologies.

For dizziness to be deemed cervicogenic in origin, the onset and duration must parallel the neck pain and must be associated with neck movements. If with further questioning any of the symptoms listed in Table 8.4 are reported, pathology of the central nervous system should be suspected, further treatment is contraindicated and the patient should be referred to the appropriate specialist. See next section for fuller discussion about dizziness.

**Table 8.4 Unexplained or new onset symptoms that may require immediate medical attention**

- constant dizziness/vertigo
- feeling of being pushed to one side
- facial asymmetry
- dysarthria
- dysphagia
- oculomotor impairment (cranial nerves III, IV, VI)
- ptosis
- vertical nystagmus
- loss of consciousness
- repeated, unexplained falls
- severe headache
- upper motor neuron signs and symptoms.

Source: Wrisley *et al.* 2000

## Cervical spine and vertebrobasilar insufficiency (VBI)

### Background

Traditionally a series of movements or positions thought to test the integrity of the vertebrobasilar arteries have been advocated prior to manipulation or mobilisation of the cervical spine (Maitland 1986; Grant 1994a; McKenzie 1990). Such cervical procedures have sometimes been associated with complications, very rarely of a serious nature, such as death or cerebrovascular accident. The aim of the test movements and certain direct questions is to try to identify patients for whom this type of treatment may be contraindicated. The topic is a controversial one. Some authorities are of the opinion that the risks of manipulation outweigh the benefits (Di Fabio 1999; Refshauge *et al*. 2002), and many consider the screening procedures unreliable and invalid (Dunne 2001; Rivett 2001; Gross and Kay 2001).

It has also been argued that the proof of a link between cervical manipulation and stroke is missing, as multiple case studies do not prove causation and a valid study to determine the true risk of manipulation would be impossible to conduct, as it would require millions of subjects (Chestnut 2004). In addition, several studies have postulated the incidence of stroke after neck manipulation; however, these estimates are hypothetical due to the lack of epidemiological evidence on the incidence rate of stroke in a representative population (Cote *et al*. 1996). The risk of neurovascular complication arising from cervical manipulation has been compared to the risk of serious gastrointestinal complication from NSAIDs for osteoarthritis (Dabbs and Lauretti 1995). The latter is more risky: 0.4% compared to 0.001%.

Di Fabio (1999) reviewed 177 reports of injuries associated with cervical manipulation published between 1925 and 1997. The most common were arterial dissection, injury to the brain stem, cerebellar or spinal cord and Wallenberg syndrome. Death occurred in 18%. The majority of incidents were attributed to chiropractors; 41% of patients had received at least one other manipulation before the incident; rotation manipulations were the most common type of intervention; and the mean age of patients was 40.

Terrett (1998) reviewed a similar number of incidents with similar findings, but suggested that the younger age group and attribution to chiropractors simply reflected those most commonly attending for treatment and those most commonly providing manipulative

treatment. Most disturbingly, some clinicians continued with further manipulation after the advent of symptoms associated with vertebroartery insufficiency (VBI). Ernst (2004) summarised the literature from 1995 to 2003 – in total more than three hundred patients had been reported, most commonly suffering stroke due to arterial dissection after cervical spinal manipulation.

Surveys of neurologists conducted in New Zealand and the UK demonstrate that complications following cervical manipulation, including strokes, have occurred without documentation (Rivett and Milburn 1997; Stevinson *et al.* 2001). The documented evidence thus probably underestimates the true extent of complications following manipulation (Terrett 1998), and complications appear as likely in the hands of physiotherapists as chiropractors (Rivett and Reid 1998).

The mechanism of injury is generally believed to be trauma to the vertebral artery around the level of the atlantoaxial joint (Grant 1994a; Terrett 1998; Mann and Refshauge 2001; Rivett 2004). This section of the artery can be subject to excessive tension with the large range of rotation available at the C1 – C2 level and where the vertebral arteries are relatively fixed at the transverse foramina (Grant 1994b; Terrett 1998; Rivett 2004). A number of cadaveric studies demonstrated that certain movements caused a narrowing of the vertebral artery: cervical rotation in particular, extension with rotation, although not always extension only, and additional traction (Grant 1994b). It was this clinical and anatomical background that led to the establishment of test procedures to try to identify patients unsuitable for cervical manipulation. The vertebral arteries feed into the circle of Willis, providing less than 20% of cerebral blood supply, whereas the carotid arteries provide more than 80% (Grant 2002; Kerry 2005; Rivett 2004). This latter can also be affected by movement, especially extension (Rivett *et al.* 1999; Kerry 2005) (see carotid artery pathology section later).

Wallenberg's syndrome (Shelokov 1991) has been reported occurring as a result of a severely diminished flow in one vertebral artery; the decrease in flow can lead to the occlusion of the posterior inferior cerebellar artery on that side, resulting in a lateral medullary infraction. The most prominent clinical features are:

- dysphagia and ipsilateral palatal weakness (involvement of the nucleus ambiguous)

- impairment of sensation to pain and temperature on the same side of the face (involvement of descending root of the fifth cranial nerve)
- Horner's syndrome in the ipsilateral eye (involvement of the descending sympathetic fibres)
- nystagmus (involvement of the vestibular nuclei)
- cerebellar dysfunction in the ipsilateral arm and leg (involvement of the restiform body and cerebellum)
- impairment of sensation to pain and temperature over the opposite half of the body (involvement of the spinothalamic tract).

### Testing protocol

Attempting to identify potential problems with vertebrobasilar insufficiency involves several components:

- items from history (Table 8.5)
- physical examination tests (Table 8.8)
- awareness during treatment
- awareness following treatment.

Given the potential risky nature of the tests themselves, it is obviously better to try to identify at-risk patients before any examination or intervention is undertaken. Awareness of possible clues in the patient's history is therefore critical to safe management. If manual therapy is going to be performed, it is imperative to monitor the patient's response both during and after procedures, even when tests have been performed uneventfully.

Certain signs and symptoms have been associated with vertebrobasilar insufficiency (VBI); these are listed in Table 8.5. It is important to remember that none of these is diagnostic of the condition, their diagnostic accuracy has not been tested, and VBI may not be the only cause of such a symptom. A useful tool to enhance memory of these signs and symptoms are the five *Ds* (dizziness, drop attacks, diplopia, dysarthria, dysphagia) and the three *Ns* (nausea, numbness, nystagmus).

**Table 8.5 Clinical features associated with vertebrobasilar insufficiency or vertebral artery dissection**

| | |
|---|---|
| History | • pain in head or neck<br>• sudden head/neck pain that has not been experienced before<br>• pain severe and sharp<br>• time delay between symptoms and features of brainstem ischaemia can be up to fourteen days. |
| Clinical features – most common, in order of frequency | • dizziness/vertigo – most common<br>• nausea/vomiting<br>• facial paraesthesia – less commonly can involve trunk and limbs<br>• unsteadiness of gait/uncoordination<br>• diplopia<br>• extremity weakness – uncommon. |
| Other signs and symptoms | • hearing loss<br>• dysarthria<br>• dysphagia<br>• blackouts/fainting/drop attacks<br>• blurred vision/transient hemianopia<br>• tinnitus<br>• pallor and sweating. |

Source: Grant 2002; Furman and Whitney 2000; Terrett 1998; Thiel amd Rix 2005

### Dizziness

Dizziness is a symptom with multiple causes. It is a common symptom in older populations, reported by 30% of people aged over 65 years (Colledge *et al.* 1996). Dizziness maybe caused by benign paroxysmal positional vertigo (BPPV), postural hypotension, a vestibular condition, labyrinthine concussion, a perilymphatic fistula, a mechanical cervicogenic condition as well as VBI (Furman and Whitney 2000; Wrisley *et al.* 2000). Possible causes of dizziness (Aspinall 1989):

- central (eg, demyelinating disease, tumour of the eighth cranial nerve, VBI)
- peripheral (eg, benign paroxismal positional vertigo, vestibulopathy, meniere, cervical reflex vertigo)
- systemic (eg, drugs/alcohol, hypotension, endocrine disease).

In the older population, the most common causes of dizziness are central vascular disease and cervical spondylosis, with postural

hypotension and benign paroxysmal positional vertigo being relatively unusual (Colledge *et al.* 1996).

Wrisley *et al.* (2000) present clues for the different causes and a clinical reasoning algorithm. Cervicogenic dizziness is a diagnosis of exclusion, based on the exclusion of competing diagnoses, with the development of a robust test to demonstrate the cervical origin of dizziness being elusive (Wrisley *et al.* 2000). There is a test in which the head is stabilised and the body rotated, theoretically stimulating the neck proprioceptors and not the inner ear structures. However, this test has demonstrated poor specificity and sensitivity (Wrisley *et al.* 2000). Consequently, to establish a relationship between dizziness and a cervical problem, the following points are recommended (Wrisley *et al.* 2000):

- close temporal relationship between neck pain and dizziness both regarding onset and severity
- previous neck problems, possibly also with accompanying dizziness
- elimination of other causes of dizziness.

Again, many clues are to be found during the patient's history-taking.

**Table 8.6 Differentiation between dizziness of cervical or other origin**

| *Possibly cervical in origin* | *Non-cervical in origin* |
|---|---|
| Transient dizziness | Constant dizziness/vertigo |
| Neck pain | Feelings of being pushed to one side |
| Neck pain associated with dizziness | Speech problems |
| Limited cervical movement | Upper motor neurone signs and symptoms |
| Headache/upper limb symptoms | Severe headache |
| Nausea | Sight problems |
| | Hearing problems |
| | Blackouts/falls. |

Source: Wrisley *et al.* 2000

If the dizziness is associated with the neck pain in terms of onset, frequency and severity, and there are no other related features (Table 8.6), cervical origin is possible. Transient dizziness or spinning

associated with changes in head position without neck movement, such as sitting up or turning over in bed, especially in the morning, may be due to benign paroxysmal vertigo. If a person answers yes to the following two questions, the Dix-Hallpike test should be used to rule out benign paroxysmal positional vertigo (BPPV) (Furman and Whitney 2000):

- Do you always have dizziness when you rise from lying to sitting?
- Do you always have dizziness when rolling over in bed?

The Dix-Hallpike manoeuvre can be used to exclude dizziness from BPPV; however, for this the patient needs an adequate range of cervical movement. The patient is positioned in long sitting and the clinician rotates the head to 45 degrees and then brings the patient into supine quickly as they extend the head 30 degrees. If the patient cannot tolerate this manoeuvre because of pain, an alternative method is to have the patient in side-lying with the head rotated so their nose is pointing up and the back of the head is on the surface of the treatment table. The extension component is gained by lowering the end of the treatment table so that the patient's head falls into extension. Symptoms of spinning or signs of nystagmus indicate a positive test for benign paroxysmal positional vertigo (Wrisley *et al.* 2000; Colledge *et al.* 1996; Lempert *et al.* 1995). Another test that has not been formally tested, but is said to indicate BPPV if it provokes severe dizziness, uses rapid head movements in different planes.

### VBI test protocol

Various test protocols have been described, all with the aim of detecting patients who may have symptoms related to VBI (Aspinall 1989; Cote *et al.* 1996; Terrett 1998; Carey 1995; Barker *et al.* 2000; APA 1988; Grant 2002; Magarey *et al.* 2004). Although there are minor variations to these pre-manipulation clinical tests, essentially they use the same manoeuvres, with end-range positional tests in rotation, extension, a combination of rotation and extension, and sometimes a position that mimics the manipulation position. The length of time that positions are sustained varies in different protocols, but in line with Australian Physiotherapy Association (APA 1988) test protocol, ten seconds is commonly given as the time. This is less if symptoms are evoked, and a gap of ten seconds should be included following each movement to allow for any latent response.

### Problems with the tests

Concerns about the pre-manipulative cervical tests have been raised on several issues. By their very nature the tests are provocative. They attempt to provoke the symptoms one wishes to avoid, and therefore obviously may be dangerous in themselves (Di Fabio 1999); neurological complications due to testing have been reported (Rivett 2004). There have been reports of stroke induced by merely placing the head into the rotated position (Terrett 1998). The APA (1988) test protocol is time-consuming and not strictly adhered to even by manipulative therapists (Magarey *et al.* 2004). Perhaps most fundamentally is the research underpinning the test protocols, which is contradictory and ambiguous, whilst the reliability and validity of the test protocol remains uncertain (Assendelft *et al.* 1996; Kunnasmaa and Thiel 1994; Cote *et al.* 1996; Thiel and Rix 2005).

First, although it is likely that VBI test positions alter the flow parameters of the vertebral artery in some individuals, from a number of studies the evidence is contradictory and blood flow reduction does not appear to be a universal phenomenon (Magarey *et al.* 2004; Zaina *et al.* 2003; Rivett 2004; Thiel and Rix 2005). Whilst studies that use ultrasonography, Doppler ultrasound with real-time imaging or angiography have demonstrated reduced flow in some volunteers, almost an equal number of studies have found no differences in test positions (Rivett 2004; Magarey *et al.* 2004).

The link between flow parameters and symptoms has not been established. When significant reductions in blood flow have been demonstrated on contralateral rotation using Doppler sonography, no warning symptoms were elicited (Mitchell *et al.* 2004; Thiel and Rix 2005). Furthermore, case reports have identified individuals with false-negative symptom response to tests in the presence of occluded arteries (Bolton *et al.* 1989; Westaway *et al.* 2003). In a review of 321 studies in which blood flow reduction was matched with provoked symptoms, only 35 out of 274 'positive' tests induced symptoms, and conversely only 11 out of 47 'negative' tests were asymptomatic. Thus, the sensitivity and specificity of the tests in terms of correlation between blood flow response and symptom response were 13% and 23% respectively, making the test mathematically and clinically useless (Kerry 2005).

Cases have been reported of patients suffering serious complications after negative test results and after previously uneventful manipulations

(Terrett 1998; Rivett and Reid 1998). Apart from cadaveric studies, there is no evidence to suggest that if these tests were positive this indicates an underlying predisposition for VBI if a manipulation was performed (Terrettt 1998). Groups of patients reporting positive pre-manipulative tests have shown no decrease in blood flow in the vertebral arteries using Doppler ultrasonography (Licht *et al.* 2000, 2002; Thiel *et al.* 1994; Cote *et al.* 1996).

In a review of sixty-four patients who suffered cerebrovascular ischaemia shortly following cervical manipulation, no features in the history or examination, including the screening tests, allowed identification of an at-risk profile (Haldeman *et al.* 2002). Furthermore, a spontaneous onset vertebrobasilar artery dissection is more common than onset following manipulation (Haldeman *et al.* 1999).

The aim of the test protocol is to detect patients at risk of VBI prior to cervical manipulation or end-range mobilisation. The rationale is based on assumptions that (Rivett 2004):

1. positions of rotation and rotation/extension cause stenosis or occlusion of the contralateral vertebral artery
2. this causes reduction in blood flow through the vertebral artery
3. this will manifest itself in transient ischaemic signs and symptoms
4. the patient is unsuitable for vigorous manual therapy techniques as this might trigger VBI.

As the review has demonstrated, most of these assumptions are untenable (Thiel and Rix 2005). Stenosis or occlusion and disturbance of blood flow are not universal in the test positions; when blood flow is decreased this is rarely associated with symptoms, and people who have had previous manipulation or a negative response to the test have had a neurological event.

Thus, it is not currently possible to predict an at-risk patient prior to manipulation; even a negative test or previous uneventful manipulation is no guarantee that the procedure will be perfectly safe. The screening procedure may only indicate the patient's likelihood of survival if manipulation injures the vertebral artery (Mann and Refshauge 2001). Vertebrobasilar complications can occur spontaneously or with trivial force, but are also associated with cervical manipulation. To minimise

risk it is prudent to employ the minimum force required to achieve the therapeutic ends using a progression of forces, and remembering that manipulation is not consistently better than other forms of treatment, including mobilisation or exercise (Refshauge *et al.* 2002). At-risk patient groups, who are more likely to suffer vascular incidents, may be better identified by history items, such as raised blood pressure, family history, smokers, overweight and so on (Kerry 2005). If anyone has clinical features that suggest VBI or vertebral artery dissection, provocative testing should not be performed and the patient should be referred appropriately.

*"It is apparent that the validity of pre-manipulative testing is at best questionable, and its clinical value is limited; the capacity of the VA to withstand thrusting forces is not tested, although it may test the adequacy of the collateral circulation to maintain hindbrain perfusion"* (Rivett 2004, p. 269).

*"Provocative testing is very unlikely to provide any useful information in assessing the probability of manipulation induced vertebral artery injury"* (Thiel and Rix 2005, p. 157).

### Legal situation

Surprisingly, a large proportion of therapists who used manipulative therapy regularly did not provide information about the risks of the procedures, nor did they formally gain consent on every occasion (Magarey *et al.* 2004). Some even expressed strong opinions against the use of gaining consent, as it would likely put patients off if they knew the dangers involved. Such an attitude is an abandonment of legal and ethical duties that clinicians owe to patients. Legal situations vary around the world, but the contemporary philosophy of health care is much more consensual and based on informed patient participants than in the past. Provision of information and gaining consent for a technique that the patient cannot control and that involves a degree of risk is not simply recommended, but in many countries is a legal necessity (Magarey *et al.* 2004). The tests, *"although poorly validated, seem to carry an important weight in court cases involving cerebrovascular injury after cervical spine manipulation"* (Cote *et al.* 1996, p. 163).

*"Health professionals have both an ethical and legal obligation to provide information and gain consent for techniques such as cervical manipulation"* (Magarey *et al.* 2004, p. 103).

After the death of a patient in September 1996 from thrombosis of vertebral artery and cerebellar infarction following a cervical manipulation, the Canadian coroner's court jury made the following recommendations:

- practitioners should obtain written informed consent
- an information sheet outlining risk of stroke should be provided
- provocative testing has not been demonstrated to be of benefit and should not be performed.

Providing information and gaining consent is as much a duty of care as the treatment itself, and failure to do so is a breach of this duty for which, in the changing legal climate, clinicians may well be sued for negligence (Refshauge *et al.* 2002; Magarey *et al.* 2004).

### Implications for mechanical diagnosis and therapy

There are reports of vascular accidents associated with non-manipulation scenarios that involve cervical rotation or extension, such as turning the head whilst driving, rap dancing, wrestling, archery, star gazing, neck extension during overhead work, radiography or a bleeding nose (Terrett 1998). On the whole, normal daily activities cause variations in vertebral artery blood flow that do not provoke symptoms; however, these cases reiterate the need for clinicians to be aware of the symptoms associated with VBI (Table 8.5) at all times during repeated movement testing. If patients report symptoms that are suggestive of vertebrobasilar problems, movement testing should proceed with caution. However, it should also be remembered that dizziness of cervicogenic origin might occur during movement testing – this is likely to lessen or abate with further repeated movement testing. If improvement does not occur, force progression should not be undertaken. If movement testing repeatedly provokes symptoms associated with VBI that do not improve, then testing should be abandoned and the findings reported to the patient's physician.

The first clues to help identify an at-risk patient might be picked up during the history-taking. The McKenzie system uses a progression of forces that starts with mid- to end-range patient forces prior to progressing to end-range, before therapist overpressures or mobilisations are even considered. Clearly this has an in-built safety mechanism. If very rarely individuals are prone to vertebral artery damage with relatively trivial forces, progression of forces ensures that such a

response is likely to be recognised before major damage has occurred. Equally, this system of sequential force progressions allows time to establish the safety of one movement or level of force before progression to the next level.

Both overpressure and mobilisation techniques can generate high levels of force and are not completely free of risk, with a case of stroke reported after a vigorous rotatory mobilisation (Michaeli 1993). However, most of the alarming accidents reported earlier were all associated with cervical manipulation. Only the final stage in the progression of forces uses manipulative procedures. These are only recommended if previous procedures in the same treatment principle have decreased but not abolished symptoms. If overpressure or mobilisation techniques have abolished symptoms, then the focus should be on repetition or avoidance of provocative postures. Before manipulation is even considered, the patient must have passed through several sessions in which lesser forces have been used.

If finally it is thought that manipulation is the appropriate intervention, a full discussion should be held with the patient about benefits, risks and alternatives and their written informed consent gained. If the patient gives informed consent to the procedure, end-range sustained positions should precede the manipulation. Position the patient in the pre-manipulative test position, question them about response, and return to neutral before the actual manipulation is performed. Never perform more than one manipulation per session, and do not repeat unless there is clear evidence of improvement.

If manipulation is performed, it should be remembered that rotation manipulation is particularly associated with VBI-type symptoms, especially if force is directed at the upper cervical levels. Lateral flexion, which has not caused loss of Doppler sounds indicating reduced vertebral artery flow (Terrett 1998), does not appear to be associated with these symptoms.

**Table 8.7 Mechanical diagnosis and therapy and safeguards with VBI**

- all clinicians need awareness of VBI-associated symptoms (Table 8.5)
- undertake a thorough history with specific closed questions about appropriate symptoms if indicated
- always use the progression of forces – test safety of movement and degree of force before progressing to end-range, overpressures or mobilisation
- monitor symptom response at all times
- enquire about any new symptoms
- never progress forces if VBI-associated symptoms are provoked
- only progress forces if transient dizziness has improved (no longer provoked) with repeated movements
- only progress to manipulation if all previous level of forces has decreased, but not abolished symptoms
- ensure that patient is fully informed about benefits, risks and alternatives of manipulation
- give space for patient questions and ensure patient consent is obtained
- perform end-range sustained tests as outlined below
- position patient in pre-manipulative test position and check response
- use only one manipulation in a session – monitor response
- never instigate further manipulation if no benefit gained or any adverse reaction provoked
- if manipulation is to be repeated on a subsequent occasion, consent and testing must be performed each time.

### End-range sustained testing

This is to be performed prior to each manipulation. Test protocol has been described in both sitting and lying; it is described here in lying (McKenzie 1990), but equally can be performed in sitting. The intensity and location of any symptoms are recorded prior to the performance of the test. The patient lies prone on the treatment table, leaning on the elbows and resting the chin on the outstretched fingertips with head maximally protruded and extended. Encourage the patient to relax so that the passive overpressure from the fingers allows maximum extension. The patient maintains this position for up to ten seconds, during which time they are asked to report any adverse effects or alteration in symptoms. Any provocation of adverse effects should terminate testing and contraindicate manipulation. On return to the neutral position, the patient is asked, *"As a result of adopting that position, do you feel any nausea, dizziness or other effects?"*

If the patient is unaffected by this position the procedure is repeated, but with the addition of a rotation component, first to one side and then the other. The patient extends as before and then, whilst in extension, rotates as far as possible to one side and maintains this position for up to ten seconds. Again symptoms are monitored during and after the procedure. If the patient is unaffected by adverse symptoms the procedure is repeated to the opposite side, with symptom response again being monitored during and after the procedure. Finally, prior to the manipulation itself, the patient's head is positioned where the thrust would be performed; again this is maintained for up to ten seconds.

In the event that the patient becomes nauseous, dizzy or feels unwell during any part of the test procedure or afterwards, let the patient rest in the neutral position for several minutes until symptoms abate. The test movement may be repeated, but if the patient consistently reports adverse symptomatology, manipulation should be abandoned and the response and its possible implications reported to the patient's physician.

**Table 8.8 Physical examination screening tests for patients prior to manipulation**

- sustained extension
- sustained left/right rotation
- sustained extension and rotation
- simulated manipulation position
- positions are sustained for ten seconds (less if symptoms are evoked)
- ten seconds in neutral position before next sustained posture
- if during any sustained position any symptoms from Table 8.5 are provoked, position is abandoned and patient is contraindicated for manipulation.

## Carotid artery pathology

As has been mentioned already, it is the carotid arteries that provide the majority of cerebral blood supply: about 80% compared to 10 – 20% from the vertebral arteries (Grant 2002; Kerry 2005; Rivett 2004). Although discussed in the literature much less commonly, carotid artery pathology exists and is also relevant in cervical management. The arteries can be affected by vascular disease and dissection can occur spontaneously, following trauma, including manipulation or as the result of vomiting, prolonged telephone use, coughing or sneezing (Taylor and Kerry 2004, 2005). Internal carotid artery

dissection accounts for about 20% of strokes in young adults (Blunt and Galton 1997).

The carotid arteries can be affected by movement, especially extension (Rivett *et al.* 1999; Kerry 2005). Carotid pathology often presents with pain, which typically precedes neurological features by hours or weeks (Silbert *et al.* 1995; Taylor and Kerry 2005). Pain can be related to physical exertion and cervical movement and be felt as neck or facial pain and headache/migraine. The headache may be sudden and of a 'thunderclap' nature, and tinnitus may be present. When neurological features start to appear, signs may be facial palsy, ptosis or miotic pupils and other symptoms of Horner's syndrome (Taylor and Kerry 2004, 2005).

## Conclusions

This chapter has considered some of the most common serious pathologies that may affect the cervical spine. These conditions are only rarely encountered in clinical practice, but unless an index of suspicion is maintained they might be missed. Thus an awareness of the 'red flags' that may indicate the presence of serious pathology is essential to safe practice. Certain clues may be gained in the history and these are detailed above, but also such patients are unlikely to respond consistently in any normal mechanical fashion.

Many of the conditions mentioned above *are absolute contraindications* to mechanical therapy. It is not necessary to make a diagnosis with such patients. If there are suspicious factors in the history and physical examination, it is always better to be safe than sorry – get the patient to a specialist as soon as possible.

# 9: Other Diagnostic and Management Considerations

## Introduction

Most patients can be classified into one of the mechanical syndromes; the majority with derangement, and smaller groups of patients with dysfunction and postural syndrome. There are several scenarios when other diagnostic considerations should be made. First, if there are any 'red flags' suggestive of serious spinal pathology, further questioning should explore this possibility, and if necessary the patient referred for further investigation. Serious spinal pathologies that affect the cervical and thoracic spine are considered in Chapter 8. In general these conditions are absolute contraindications for instigating treatment, but their incidence is rare. Second, a patient may attend following involvement in a road traffic accident in the last few days, or with a primary complaint of headache. Whiplash injuries are considered in Chapter 25. Primary headaches are discussed in Chapter 24.

Third, if after three to five sessions the patient has persistently failed to demonstrate the symptomatic or mechanical responses that are described for the three mechanical syndromes, and none of the other two situations described above apply, other conditions might be considered. This chapter considers the evidence relating to the existence, recognition and management of some of these other conditions. It is emphasised again that these conditions are usually considered following the failure to classify in one of the mechanical syndromes. This chapter also contains sections about other diagnostic and management considerations that may pertain in certain situations, such as cervical radiculopathy and surgery.

Sections in this chapter are as follows:

- cervical and thoracic zygapophyseal joint pain
- shoulder pain
- mechanically inconclusive
- chronic pain
    - management of patients with chronic pain state
- cervical spondylosis/stenosis

    - symptoms and radiographic changes
    - symptomatic presentation
- cervical radiculopathy
    - tests
- surgery for cervical and thoracic problems
- post-surgery
- Thoracic Outlet Syndrome
    - tests.

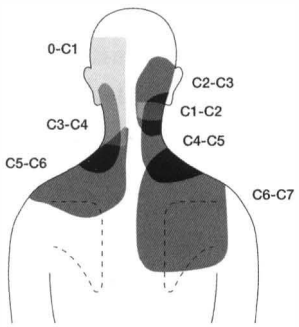
## Cervical and thoracic zygapophyseal joint pain

Zygapophyseal or facet joint pain/somatic dysfunction is a common diagnostic label used by manual therapists (Maitland 1986; Trott 2002; Gatterman 1998; McClune *et al.* 1998). This section explores what is documented, rather than speculated, about this syndrome in terms of diagnosis and treatment. Zygapophyseal joints are involved in the normal ageing process of cervical spondylosis, but changes such as anterior and posterior osteophytes, bony hypertrophy and foraminal stenosis are commonly found in the asymptomatic population and thus are not intrinsically a source of symptoms (Gore *et al.* 1986; Friedenberg and Miller 1963; Teresi *et al.* 1987; Matsumoto *et al.* 1998).

Much of the work done on this topic has been on individuals with chronic whiplash symptoms, and mostly by the same team of researchers. Pain patterns from different levels (C2 – 3 to C6 – 7) were determined in five asymptomatic volunteers by distending the joint capsules under fluoroscopic control (Dwyer *et al.* 1990). Distinguishable and characteristic patterns from each joint space allowed the construction of a pain chart (Figure 9.1). A similar study undertaken at the atlanto-occipital and lateral atlanto-axial joints revealed a consistent pattern for the latter, but more variability for the atlanto-occipital joint (Figure 9.1) (Dreyfuss *et al.* 1994b). The validity of the first pain chart was tested in a group of ten consecutive patients with chronic cervical pain (Aprill *et al.* 1990). There was virtually complete agreement between two observers regarding which segmental level was involved from the patients' pain patterns, and in nine out of ten cases this was validated by a positive response to a zygapophyseal joint block at the appropriate level. These studies

revealed the zygapophyseal joints could be a source of pain, and symptoms were in a consistent pattern of limited referral.

**Figure 9.1 Patterns of referred pain produced by stimulating cervical zygapophyseal joints in normal individuals**




Source: Adapted from Dwyer *et al.* 1990 and Dreyfuss *et al.* 1994b, with permission

A series of studies was undertaken to determine the prevalence of cervical zygapophyseal pain in consecutive chronic whiplash patients. These were done under image intensifier and either used two injections to provoke and then relieve the patient's pain or double blocks with different time-acting anaesthetics, or other strict inclusion criteria for deciding if symptoms were from the zygapophyseal joints (Barnsley *et al.* 1993a; Lord *et al.* 1996a). Such rigorous methodology and the use of strict criteria are necessary as single cervical zygapophyseal joint blocks have a false positive rate of 27% (95% confidence interval 15%, 38%), which disqualifies the validity of studies not using this methodology (Barnsley *et al.* 1993b).

In a total of over two hundred patients with chronic neck pain or headache in five separate studies (Bogduk and Marsland 1988; Bogduk and Aprill 1993; Lord *et al.* 1994; Barnsley *et al.* 1995; Lord *et al.* 1996a), the prevalence rate varied from 53% to 71%, with a net prevalence rate of 67%. Relief was achieved by injection at all levels between C2 – 3 and C6 – 7, but most commonly at C2 – 3 and C5 – 6, with C2 – 3 joint being the source of occipital headaches. There were no distinguishing features on history or examination that predicted a positive response to injection (Lord *et al.* 1994, 1996a). For patients with a headache emanating from C2 – 3, tenderness over the joint was common, with a sensitivity of 85%, a positive likelihood ratio of 1.7 and a negative likelihood ratio of 0.3 (Lord *et al.* 1994). In this population if headache was the predominant complaint and tenderness was present over C2 – 3, the positive likelihood ratio was 2:1.

In some of the studies carried out by this research group, discogenic pain was also identified. This was done by provocation discography and either subsequent relief on anaesthetic or no pain on provocation of two adjacent discs (Aprill and Bogduk 1992; Bogduk and Aprill 1993). In 310 patients who received discography (182), zygapophyseal joint blocks (52) or both (76), discography was positive in 53% and zygapophyseal joint block positive in 26%, of which 8% were positive to both diagnostic tests (Aprill and Bogduk 1992). In a sub-set of fifty-six patients from this population, 41% had both a symptomatic disc and zygapophyseal joint and only 17% had neither (Bogduk and Aprill 1993). The authors maintained that either method, if used in isolation, is inadequate in the diagnostic exploration of chronic post-traumatic neck pain (Bogduk and Aprill 1993). Clearly both intervertebral discs and zygapophyseal joints were commonly symptomatic, often simultaneously, in these groups of patients.

Thus, zygapophyseal joint pain has been found to be a very common source of symptoms in chronic neck pain patients involved in trauma, mostly post-whiplash, all attending tertiary care for further investigations. Full demographic data is not always given, but these are mostly patients with very long histories of neck pain, measured in years rather than months. There is some suggestion that they may have been involved in more severe crashes, involving higher impact speed (Gibson *et al.* 2000). Although it is clear that zygapophyseal joint pain is common in this type of patient, this refers to a very particular sub-group of neck pain patients and these findings cannot be extrapolated to all neck pain patients. It is worth recalling at this point the commonly dichotomous natural history of neck pain following whiplash – the majority get better in the first two to three months, and a minority have persistent and apparently irresolvable symptoms (Chapter 25). It is probably this latter group that are being investigated in these studies.

Another research group has investigated the prevalence of cervical and thoracic zygapophyseal joint pain in several studies (Manchikanti *et al.* 2002a, 2002b, 2002c, 2004). In five hundred consecutive patients presenting with chronic neck, thoracic or low back pain, diagnosis was made using controlled comparative local anaesthetic blocks (Manchikanti *et al.* 2004). The study confirmed high false-positive rates with single blocks of 63% in the cervical spine and 55% in the thoracic spine. Mean patient age was 47 years; mean duration of pain

was around eight years; nearly 50% had been involved in trauma; two hundred had pain in more than one area; and all patients had failed a range of conservative management. The double local anaesthetic block provided a prevalence rate of zygapophyseal joint pain of 55%, 42% and 31% respectively in the cervical, thoracic and lumbar spines. Their other studies confirmed high false-positive responses to single blocks, similar numbers reporting onset following trauma, extremely protracted episodes of neck pain, and similar prevalence levels of cervical zygapophyseal joint pain (Manchikanti *et al.* 2002a, 2002b). Similarly, in forty-six patients with chronic thoracic pain, symptoms had been present for an average of seven years, all patients had failed conservative management, onset was following trauma in 39%, and 48% reported pain relief with double joint blocks (Manchikanti *et al.* 2002c). A systematic review concluded that studies demonstrated prevalence of zygapophyseal joint pain of between 54% and 67% in the cervical spine and up to 48% in the thoracic spine (Boswell *et al.* 2003a).

Apart from the obvious component from their history of very persistent symptoms and a traumatic onset, the studies reviewed above did not identify any other distinguishing clinical features, except in one study when tenderness over C2 – C3 was associated with a positive zygapophyseal joint (Lord *et al.* 1994). The validity of manual diagnosis of cervical zygapophyseal joint pain has been investigated in a small group of twenty consecutive patients (Jull *et al.* 1988). Neck pain had been present for at least twelve months, but onset was not described, and findings from manual palpation were compared to radiologically controlled diagnostic nerve blocks. Fifteen of the twenty were diagnosed as zygapophyseal joint pain, and the manual therapist was 100% sensitive and 100% specific in diagnosis and segmental level. However, only one manual therapist was evaluated, the study has not been replicated, and intertester reliability needs to be established; therefore the study cannot be said to vindicate manual therapy palpation techniques in general. In a study (Jull *et al.* 1997) evaluating intertester reliability to detect painful upper cervical zygapophyseal joint problems, kappa values were consistently high for dichotomous decision-making, but much more varied when deciding on the symptomatic level, as detailed in Table 9.1. However, of twenty volunteers without symptoms, three were judged to have upper cervical joint dysfunction, indicating problems with specificity.

A number of studies have examined intertester reliability of a variety of palpation techniques using kappa values. These have examined localisation of tenderness or pain at segmental levels or nearby soft tissues, judgements on passive intervertebral motion, diagnosis of 'fixations', or stiffness or joint dysfunction at segmental levels (Table 9.1). There is considerable variability in kappa values, from negative values, which indicate reliability less than expected by chance, to 1.0, which indicates perfect agreement. The rate of an acceptable kappa value for clinical utility is still debated, but Altman (1991) suggests that a value less than 0.5 indicates poor reliability. Out of over 160 clinical judgements made in these studies, only just over a quarter have a kappa value greater than 0.5 – nearly half of these relate to identifying painful levels or soft tissue. The ability of clinicians to use palpation to detect joint dysfunctions, 'fixations', stiffness or other passive intervertebral motion abnormalities in a reliable and consistent is clearly unproven. If clinicians so commonly disagree about the presence or absence of such clinical phenomena, their validity must be open to doubt.

Finally, it is worthwhile to consider if there is clinical value in making the diagnosis of cervical zygapophyseal joint pain; will the knowledge of this diagnosis lead to a better outcome for the patient? Intra-articular corticosteroid injections have been found to be no more effective than intra-articular anaesthetic injection at providing lasting pain relief. Most patients' pain returned to near pre-injection levels within a few days, and by about three weeks less than 20% of both groups had pain less than 50% of pre-injection levels (Barnsley *et al.* 1994b). Percutaneous radio-frequency neurotomy produced relief of chronic zygapophyseal joint pain at six months in seven of twelve patients compared to one of twelve in the placebo-controlled local anaesthetic injection group (Lord *et al.* 1996b). In the short-term this intervention appeared to be effective for some, but its invasive and highly skilled nature do not make it readily available to most patients.

In summary, cervical zygapophyseal joint pain does exist; the only proven means of recognition requires a double joint injection block. Using this technology, cervical zygapophyseal joint pain has been found to exist in patients with very chronic neck pain of traumatic and non-traumatic origin that generally has failed to improve with conservative management. Reliable and valid identification by manual palpation may be feasible, but is unproven. To date recognition of this diagnostic category has not brought an effective means of treatment.

**Table 9.1 Intertester reliability of examination by palpation in the cervical and thoracic spine**

| *Item* | *Study* | *Kappa** | *Kappa =/> 0.5*** |
|---|---|---|---|
| Tenderness/pain | Hubka and Phelan 1994 | 0.68 | |
| | Strender *et al.* 1997 | 0.31-0.52 | 1/3 |
| | van Suijlekom *et al.* 2000 | 0.0-0.87 | 8/17 |
| | Bertilson *et al.* 2003 | 0.22-0.79 | 5/9 |
| | Levoska *et al.* 1993 | 0.15-0.62 | 4/8 |
| | Christensen *et al.* 2002 | 0.67-0.7 | 2/2 |
| | Horneij *et al.* 2002 | 0.12-0.49 | 0/4 |
| Passive general movement | Fjellner *et al.* 1999 | 0.26-0.66 | 5/8 |
| Fixations/stiffness/ joint dysfunction | DeBoer *et al.* 1985 | -0.03-0.45 | 0/8 |
| | Nansel *et als.* 1989 | 0.013 | |
| | Smedmark *et al.* 2000 | 0.28-0.43 | 0/4 |
| | Comeaux *et al.* 2001 | 0.12-0.56 | Detail not given |
| | Fjellner *et al.* 1999 | 0.17-0.5 | 1/8 |
| | Jull *et al.* 1997 | 0.25-1.0 | 15/22 |
| Passive intervertebral motion | Fjellner *et al.* 1999 | -0.17-0.49 | 0/58 |
| | Strender *et al.* 1997 | 0.06-0.15 | 0/3 |
| | Hanten *et al.* 2002 | -0.07-0.86 | 4/11 |
| | Christensen *et al.* 2002 | 0.22-0.24 | 0/2 |
| | Pool *et al.* 2004 | -0.09-0.63 | 1/9 |

* range of kappa values or pooled kappa values

**number of clinical judgements with kappa value =/> 0.5/total number of relevant clinical judgements

## Shoulder pain

Several studies have indicated that pain around the scapular and shoulder region commonly arises from cervical discogenic or zygapophyseal joint disorders (Cloward 1959; Smith 1959; Whitecloud and Seago 1987; Grubb and Kelly 2000; Dwyer *et al.* 1990; Aprill *et al.*1990). Stimulation of thoracic structures has also caused pain in the chest and scapular region (Bogduk 2002c). Irritation by hypertonic saline of the acromioclavicular joint (ACJ) and subacromial space suggests that these structures may refer proximally, but that

predominantly symptoms are felt either around the ACJ or around the shoulder respectively (Gerber *et al.* 1998). Differential diagnosis is an important consideration when examining patients with symptoms in the shoulder area. The link between cervical and shoulder problems and the ability of cervical problems to mimic shoulder problems (van der Windt *et al.* 1996; Hargreaves *et al.* 1989; Wells 1982; Schneider 1989) means that when pain is present at the shoulder, the source of the symptoms must be considered carefully. Three scenarios might exist:

- shoulder pain is entirely cervical referred pain and responds to neck management described elsewhere in the book
- shoulder pain is entirely local somatic pain and responds to shoulder management (McKenzie and May 2000)
- shoulder (and neck) pain is a combination of cervical and shoulder problems, both of which need addressing.

There can be problems identifying the source of pain in the shoulder area. Often it is reasonably obvious from the history that the source is either cervical or shoulder, and the site of the physical examination is clear. However, sometimes the history does not make this clear, and various clues may help in this differential diagnosis, although none is an absolute indicator (Table 9.2). Any combination of neck and scapular or shoulder pain is most likely to be referred pain from cervical structures.

**Table 9.2 Differential diagnosis of cervical and shoulder problems**

| *Item* | *Cervical* | *Shoulder* |
|---|---|---|
| Pain pattern | Neck, scapular, shoulder (chest, arm, forearm), hand, fingers | Acromion process, shoulder, deltoid (arm, forearm) |
| Other symptoms | Paraesthesia, weakness | |
| Mechanical presentation | Loss ROM neck, (shoulder) Pain on neck (shoulder) movement | Loss ROM shoulder Pain on shoulder movement |

ROM = range of movement

If it is suspected that the patient's problem has a mixed origin with components from both the shoulder and the neck, then in general it is best to direct management first to the neck, but at the same time monitor what happens at the shoulder to see if this improves. Likewise if it is suspected that the shoulder problem is cervical in origin,

monitor the shoulder as management is directed at the neck. To monitor the shoulder, take a baseline assessment of shoulder range of movement and pain responses during the physical examination. This should include active range of movement, including overpressure and resisted movements. Review the mechanical presentation at the shoulder later to see if this has improved in line with the neck response. If it has changed, obviously the neck was the source of both problems. If the neck has improved but the shoulder has remained completely the same, then further management needs to be directed at the shoulder itself (McKenzie and May 2000, Chapter 11).

Furthermore, if shoulder pain is present and management has been directed at this but to no avail, response to neck treatment should be evaluated. If both neck and shoulder symptoms are present, but the pain in the shoulder area is the most severe and causing the most functional disability, it is still important to evaluate responses to repeated movements of the neck. Unless there are rapid changes in response to these movements, management should very quickly be directed at the shoulder.

## Mechanically inconclusive

There is a small group of patients whose symptoms are influenced by postures and movements, and yet who do not fit one of the three mechanical syndromes. Symptoms are affected by loading strategies, but in an unrecognisable or inconsistent pattern. This group does not display a mechanical presentation – range of movement is preserved, and there is no obstruction to movement. Pain may be constant or intermittent and is frequently produced or increased at end-ranges. Repeated end-range movements in all planes may produce a worsening of symptoms, but no obstruction of extension or flexion by loading in the opposite direction. Thus, no mechanically determined directional preference is indicated.

There may be variations on a similar theme; for instance, catches of pain during movement, or initially there is a favourable response to repeated movement in one direction, which then becomes inconsistent or causes a worsening of symptoms if continued or if force progressions are included. The key to this mechanically inconclusive group, who nonetheless have symptoms that respond to loading strategies, is that a consistent mechanically determined directional preference cannot be found.

Criteria for mechanically inconclusive group are:

- symptoms affected by spinal movements
- no loading strategy consistently decreases, abolishes or centralises symptoms, nor increases or peripheralises symptoms
- inconsistent response to loading strategies.

This group sometimes responds to mid-range postures rather than end-range movements. Maintenance of posture correction, use of mid-range movements, especially slouch-overcorrect, avoidance of end-range postures and movements, and interruption of painful positions may be helpful for this group to decrease symptoms.

## Chronic pain

Chronic pain has traditionally been defined by pain duration; for instance, symptoms that have persisted for more than three to six months. However, time scale alone is generally now considered to be an inadequate definition for chronic pain. Other factors are considered important in the chronic pain experience. Psychosocial and behavioural factors complicate the clinical problem and pain is disassociated from tissue damage. Patients may experience widespread pain, and the problem is more likely to prove difficult to treat.

From the review of the epidemiology of neck pain in Chapter 1, it is apparent that many individuals have persistent symptoms, but that in this group severity and disability are often minimal. Categorisation of chronic patients should not be determined simply by pain duration. Within those who have persistent symptoms, many demonstrate mechanical responses, although sometimes response may be slower.

The length of time that symptoms have been present should never be seen as deciding factor in the application of therapy. Many of those with chronic symptoms benefit from a mechanical assessment. *Patients who have long-standing neck pain should not be denied a mechanical assessment*. Many patients with long-term problems display mechanically determined directional preference for certain repeated movements. Not all resolve their problems, but many patients with chronic symptoms improve their ability to manage their condition. Because of the length of time the problem has been present, a slower and more ambivalent response may occur. However, also within this

group it should be recognised that alternative approaches may be appropriate.

Symptoms may become complicated and persist due to non-mechanical problems. These are considered in more detail in Chapter 2, but in brief these consist of psychosocial or neurophysiological factors that act as barriers to resolution and obscure a mechanical problem. Psychosocial and cognitive factors are closely related to the development of chronic back disability. Depression, anxiety, passive coping, fear-avoidance and attitudes about pain are associated with chronic pain and disability (Linton 2000). The same issues are thought to be relevant for neck as back pain, but most of the literature in this area is for back pain.

Strong (2002) distinguishes between chronic pain, which has lasted for a certain length of time, and chronic pain syndrome, in which pain is coupled with reduced functionality, mood changes and multiple failed treatments (Table 9.3).

**Table 9.3 Characteristics of chronic pain syndrome**

- multiple interventions
- poor response to analgesics
- increased feelings of helplessness and hopelessness
- mood changes
- psychosocial withdrawal
- loss of self-esteem
- withdrawal from work role
- decreased physical functioning
- increase in interpersonal conflicts
- conflicts with health care providers.

Source: Strong 2002

Furthermore, persistent peripheral nociceptive input can induce changes in the central nervous system (Woolf 1991; Melzack and Wall 1988). This may lead to the sensitisation of neurones in the dorsal horn – a state characterised by reduced thresholds and increased responses to afferent input, such that normal mechanical stimuli is interpreted as pain. In this situation pain, aching and tenderness are likely to be widespread, and most normal activity is perceived as painful.

Thus, a chronic pain state is not simply related to the time that symptoms have been present. These are patients in whom a mechanical response to loading strategies is obscured by non-mechanical factors, which may be psychosocial or neurophysiological in origin. Symptoms are likely to have been present for a prolonged period, but this may not always be so. Interruption of their normal lifestyle has usually occurred. Multiple or widespread pain sites are common. All activity increases symptoms, at least initially. There is no obvious mechanically determined directional preference or clear mechanical response, again, at least not initially. Often these patients display exaggerated pain behaviours and vocalisation. They nearly always hold mistaken beliefs and attitudes about pain and movement, and in particular are fearful of movement. Depression, anxiety and distress are all commonly found.

**Table 9.4 Key factors in identification of chronic pain state**

- no lasting or consistent change in pain location or pain intensity in response to therapeutic loading strategies
- persistent widespread symptoms
- all activity increases symptoms
- exaggerated pain behaviour
- mistaken beliefs and attitudes about pain and movement.

### Management of patients with chronic pain state

This is a difficult group to treat, but it is apparent that the emphasis should be on improved function, coping and self-management, rather than resolution of pain. Foremost in the clinician's mind when assessing the patient should be the importance of focussing on functional changes rather than highlighting the effects of repeated movements on pain. The confounding effect that non-mechanical factors can have on the efficacy of purely mechanical interventions should be recognised.

For chronic musculoskeletal problems it has been recommended that a cognitive-behavioural framework be used for interaction with the patient (Turner 1996). This requires:

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

  - self-management principle for on-going health problems
  - activity for musculoskeletal conditions
  - reassurance that pain on movement does not mean an exacerbation of the problem
- encouragement of a graduated, systematic resumption of activities
- avoidance of over-treatment.

Common features of successful programmes for chronic back problems have been identified (Linton 1998), and it is unlikely these differ much from the needs of patients with neck chronic pain state:

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

## Cervical spondylosis/stenosis

The ageing process is associated with certain anatomical changes in the cervical spine. The nature of these changes is dealt with in more detail in Chapter 3; here we wish to consider the symptomatic presentation of these degenerative changes, often referred to as cervical spondylosis. In brief, these changes involve the early desiccation and transverse fissuring of the intervertebral disc. The associated thinning of the disc leads to greater load bearing at the zygapophyseal and uncovertebral joints, which may produce osteophytes and posterior bulging of the disc as a bony ridge (Taylor and Twomey 2002). These degenerative changes may produce lateral foraminal stenosis affecting the nerve root or spinal canal stenosis affecting the spinal cord.

### Symptoms and radiographic changes

An important clinical point is that these changes, demonstrated on x-ray, can exist in a symptom-free population. Narrowing of joint space, disc herniation, anterior and posterior osteophytes, bony hypertrophy, foraminal stenosis and even spinal cord compression are found in the asymptomatic population (Gore *et al.* 1986; Friedenberg and Miller 1963; Teresi *et al.* 1987; Matsumoto *et al.* 1998). Some of these changes are present in about 20% of individuals with no neck pain in their 30s, about 75% in their 50s and over 80% in their 60s (Gore *et al.* 1986; Matsumoto *et al.* 1998). No difference has been found in pain and disability levels between those with or without evidence of cervical spine degeneration (Peterson *et al.* 2003). Thus, the same radiographic presentation can be found in a symptomatic or asymptomatic individual – this issue is discussed at more length in Chapter 5.

### Symptomatic presentation

Some individuals who have these radiographic changes also present with symptoms. Although cervical spondylosis may be a source of somatic neck pain only, the reports in the literature are dominated by the more severe presentations of cervical radiculopathy and cervical myelopathy. The latter is considered a serious spinal pathology as the central nervous system is involved, and therefore such patients should be referred to a specialist – cervical myelopathy is discussed in the previous chapter. Signs and symptoms are those of an upper motor neurone lesion, but a combined presentation of radiculopathy and myelopathy occurs. Evidence of the link between degenerative changes and symptoms stem from multiple surgical reports (for instance: Odom *et al.* 1958; Henderson *et al.* 1983; Mosdal and Overgaard 1984; Allen 1952; Bertalanffy and Eggert 1988; Young and O'Laoire 1987; O'Laoire and Thomas 1983; Epstein *et al.* 1978; Gregorious *et al.* 1976; Mosdal 1984; Perneczky *et al.* 1992; Vassilouthis *et al.* 1989; Gore and Sepic 1984).

At surgery in the cervical spine, two causes of myelopathy and brachialgia or radiculopathy have been noted: soft or hard disc herniations. Hard discs in fact refer to bony growths, such as foraminal spurs, transverse bony ridges on the vertebral body, uncovertebral exostosis and other degenerative changes that occur with cervical spondylosis (Odom *et al.* 1958; Henderson *et al.* 1983; Mosdal and Overgaard 1984; Allen 1952; Epstein *et al.* 1978; Bertalanffy and Eggert 1988). Spondylotic bars or ridges encroach into the spinal

canal, whilst hypertrophying bony tissue from the zygapophyseal or the uncovertebral joints encroach into the foramen (Parke 1988). Soft disc herniation, that is cervical disc prolapse or bulging, has also been reported to be the cause of radiculopathy and myelopathy (Bertalanffy and Eggert 1988; Perneczky *et al.* 1992; Young and O'Laoire 1987; O'Laoire and Thomas 1983; Vassilouthis *et al.* 1989; Nakajima and Hirayama 1995).

Cervical spondylosis may thus produce the following patterns of symptomatology (Lestini and Wiesel 1989):

- somatic neck and referred pain, alone or in combination with:
    - radiculopathy – disc herniation
    - radiculopathy – foraminal stenosis
    - radiculopathy – combination foraminal stenosis and disc herniation
    - myelopathy – disc herniation
    - myelopathy – spinal canal stenosis
    - combination radiculopathy/myelopathy.

The concern in this section is with somatic symptoms; cervical radiculopathy is discussed in the next section, and myelopathy is considered in the previous chapter. Somatic symptoms that stem from degenerative changes may demonstrate a beneficial response to repeated movements, and many patients with cervical derangement also have cervical spondylosis on x-ray. A not uncommon clinical presentation in older patients with chronic symptoms is symmetrical loss of rotation and lateral flexion and major loss of extension; radiographs of such patients often display cervical spondylosis or osteoarthritis. On mechanical evaluation, multiple direction dysfunction is often demonstrated. Cervical dysfunction is considered in more detail in Chapter 21, but in brief a global restriction of movement is accompanied by end-range pain in multiple directions. Some such patients respond in a few weeks to regular repeated movements; however, some patients respond much more slowly. The response is often connected to the length of time symptoms have been present and takes the form of increased range of movement with decreased pain or increased range before pain or decrease of pain with same range of movement.

## Cervical radiculopathy

Cervical radiculopathy is a specific lesion affecting the cervical nerve roots in which neck pain is accompanied by upper limb pain and possibly neurological symptoms and signs (Radhakrishnan *et al.* 1994). This section presents some details about the epidemiology, pathology and recognition of cervical radiculopathy. For its management refer to Chapter 20, where it is included in the derangement category with referred arm pain below the elbow. The radiculopathy is most often attributed to cervical disc herniation or spondylosis, classified often as soft or hard discs respectively (Wainner and Gill 2000; Radhakrishnan *et al.* 1994). However, it should be noted that a number of case studies mention less common causes that include serious spinal pathology, such as tumour and arteritis (Vargo and Flood 1990; Sanchez *et al.* 1983; Wainner and Gill 2000).

The prevalence of cervical radiculopathy has been estimated at 3.3 cases per 1000 population (Salemi *et al.* 1996), and the peak incidence is most commonly reported to occur between the third and fifth decades of life (Wainner and Gill 2000; Radhakrishnan *et al.* 1994). The natural history of cervical radiculopathy appears to be generally benign, but no prognostic or risk factors have been firmly established (Wainner and Gill 2000). Over time the resolution of pain is often, although not always, accompanied by regression of the disc herniation (Bush *et al.* 1997; Maigne and Deligne 1994; Mochida *et al.* 1998).

The nerve root is either irritated by bony osteophytes at the zygapophyseal or uncovertebral joints that are acquired secondary to cervical spondylosis or by a cervical disc herniation. The disc herniation is composed of mixed annulus or nucleus material, always with a fragment of cartilaginous end-plate (Kokobun *et al.* 1996). In those coming to surgery, cervical disc herniations causing cervical radiculopathy tend to be predominantly lateral (80 – 88%) with a few that are paramedian (12 – 20%), compared to those causing myelopathy, which are all median (36 – 40%) and paramedian (60 – 64%) (Kokobun and Tanaka 1995; Yamazaki *et al.* 2003).

Distinction between bony stenosis and soft disc herniation as cause for radicular symptoms may be important for prognosis, and perhaps needs to be borne in mind for management. If foraminal stenosis is the cause, an immediate and lasting beneficial response is unlikely and radicular symptoms are likely to be aggravated by extension

that narrows the intervertebral foramen (Yoo *et al.* 1992; Nuckley *et al.* 2002; Farmer and Wisneski 1994). The same response may initially occur if disc herniation is the cause, but multiple repetitions of retraction and then extension may generate a positive response. The foramina are also narrowed by ipsilateral rotation and lateral flexion (Yoo *et al.* 1992; Nuckley *et al.* 2002), but again, with cervical disc herniation, multiple repetitions of these movements may produce a lasting change in symptoms.

Distinguishing bony stenosis from soft disc herniation as a cause for radicular symptoms may not be straightforward. Both can present with neck and arm pain in the dermatomal pattern, sensory and motor deficit, reflex changes and restriction of movement. Both occur predominantly at C5 – C6 and C6 – C7 segmental levels (between 70% and 90%). Patients with soft disc herniations tend to be younger and have shorter duration of symptoms, although these are relative rather than absolute differences (Bertalanffy and Eggert 1988; Odom *et al.* 1958; Lunsford *et al.* 1980). In patients with bony stenosis spontaneous improvement or resolution is less likely, aggravating factors will be more consistent (extension, ipsilateral rotation and lateral flexion), and flexion may temporarily decrease symptoms, whereas flexion is likely to aggravate symptoms from a disc herniation. Ultimately only a mechanical evaluation can determine if their symptoms will respond to mechanical therapy, and it is likely that a number will not respond. However, see section in Chapter 20 on 'Non-responders to mechanical therapy'.

### Tests

Clinical recognition of cervical radiculopathy has been made traditionally by pain pattern and accompanying paraesthesia or muscle weakness; some specific provocation tests have also been described. The radicular pain is severe and often obscures the neck pain, and sensory loss or myotomal weakness tends to be variable (Aldrich 1990). However, there have been reports of much overlap in pain pattern between those with clear neurological deficit and those without (Dalton and Jull 1989). Groups with one or more neurological signs were more likely to have pain in the forearm and hand, and more likely to report these as the worst sites of pain, but these were relative, not absolute differences.

The C6 and C7 nerve roots are most commonly affected, with pathology at C5 – C6 and C6 – C7 motion segments respectively; the

nerve roots of C8, C5 and C4 are less commonly affected (Aldrich 1990; Wainner and Gill 2000; Radhakrishnan *et al.* 1994). Regarding cervical radicular pain patterns, there is considerable variation between individuals, with no clear distinction between nerve roots in their proximal pain pattern (Slipman *et al.* 1998). Research using pain provocation has found common areas of referred pain distally: C4 is around the lateral neck and top of the shoulder; C5 is similar to C4 but extends more distally to the lateral arm; C6 pain is distributed down the lateral arm and into the thumb and index finger; C7 is similar to C6, but is usually more posterior and extends into the middle and ring fingers (Slipman *et al.* 1998).

A number of pain provocation tests have been described, with some reporting of validity and reliability (Viikari-Juntura 1987; Viikari-Juntura *et al.* 1989; Tong *et al.* 2002; Wainner *et al.* 2003). Spurling's test involves head compression in ipsilateral lateral flexion, or compression in rotation and extension that provokes the patient's radicular symptoms. The shoulder abduction sign involves relief of symptoms on placing the affected arm on the head, and the neck distraction test involves pain relief on axial traction of the applied though the occiput and chin. Tests for cervical radiculopathy have been reviewed for their clinical utility (Malanga *et al.* 2003; Wainner and Gill 2000).

Overall, reliability for many of the tests can be reasonable, but this is not consistently so. Most of the tests have poor sensitivity except the upper limb tension test, but this has low specificity and so will be positive in many patients without cervical radiculopathy. High sensitivity allows the diagnosis to be ruled out if the test is negative (Sackett *et al.* 1997). Most of the rest of the tests have good specificity. High specificity allows the diagnosis to be ruled in if the test is positive (Sackett *et al.* 1997).

The best non-operative management for cervical radiculopathy has not been determined. A plethora of treatments have been tried, and although improvements are frequently reported, it is unclear if this is a specific treatment effect or simply a benign natural history at work (Wainner and Gill 2000). A number of problems mean the evidence is very weak in this area; most importantly, randomised controlled trials using homogenous groups of patients are rare.

**Table 9.5 Reliability and validity of physical examination for cervical radiculopathy**

| *Physical examination item* | *Reliability*[1] | *Sensitivity* | *Specificity* |
|---|---|---|---|
| Spurling's Test | 0.40-0.77[2] | 40-60%[3] | 92-100%[3] |
| | 0.60-0.62[5] | 30%[4] | 93%[4] |
| | | 50%[5] | 74-86% |
| Shoulder Abduction Sign | 0.21-.40[2] | 43-50%[3] | 80-100%[3] |
| | 0.20[5] | 17%[5] | 92%[5] |
| Neck Distraction Test | 0.50[2] | 40-43%[3] | 100%[3] |
| | 0.88[5] | 44%[5] | 90%[5] |
| Dermatome sensation | 0.41-0.74[2] | 12-29%[5] | 66-86%[5] |
| | 0.16-0.67[5] | | |
| Myotome testing | 0.40-0.64[2] | 3-24%[5] | 84-94%[5] |
| | 0.23-0.69[5] | | |
| Reflex | 0.73[5] | 3-24%[5] | 93-95%[5] |
| ULTT | 0.35[2] | 72-97%[5] | 22-33%[5] |
| | 0.76-0.83[5] | | |

1 = kappa scores; ULTT = upper limb tension tests

Sources: 2 = Viikari-Juntura 1987; 3 = Viikari-Juntura *et al.* 1989; 4 = Tong *et al.* 2002; 5 = Wainner *et al.* 2003

Epidural steroid injections are sometimes advocated for radiculopathy, especially to avoid surgery (Boswell *et al.* 2003b). Again, positive results are frequently reported in uncontrolled trials, although benefits appear to be short-term and limited in the long-term. There appear to be a lack of placebo controlled trials in a homogenous population with established cervical radiculopathy (Wainner and Gill 2000; Boswell *et al.* 2003b; Derby *et al.* 2004). When non-randomised observational studies were considered as well, the conclusion was slightly more positive (Abdi *et al.* 2005).

According to mechanical diagnosis and therapy diagnostic criteria, cervical radiculopathy will mostly be classified as derangement. The value of posture correction and retraction exercises in cervical radiculopathy has been demonstrated (Abdulwahab and Sabbahi 2000). Some patients respond to extension exercises, but a proportion of these patients require lateral forces or unloaded forces to gain a response. There will also be a group, those with more severe constant radicular pain and constant neurological signs and symptoms, who

will be unresponsive to conservative treatment – those with irreducible derangements. Such patients will likely improve over time, but may wish to consider surgery – see the next section. If the patient presents with a two- to three-month history of cervical radiculopathy, which has improved but is now intermittent and unchanging, the alternative classification of adherent nerve root should be considered. For the differential diagnosis and management of reducible derangement, non-responders to mechanical therapy and adherent nerve root, see Chapters 20 and 22.

## Surgery for cervical and thoracic problems

Indications for cervical surgery are said to be instability, often secondary to rheumatoid arthritis or trauma, radiculopathy, myelopathy and tumour (Jones 1998). In the thoracic spine, thoracic disc herniations causing progressive myelopathy, trauma that may cause spinal cord lesions, and progressive deformity that fails to respond to conservative measures are said to be indications for surgery (Findlay and Eisenstein 2000). If treatment is considered for thoracic scoliosis deformity, this may be either conservative or surgical, with decisions for the latter based on severity of the curve, rate of curve progression and skeletal maturity of the patient (Findlay and Eisenstein 2000).

The scientific literature on surgery for neck pain and radiculopathy consists mostly of uncontrolled case series with varying periods of follow-up time (Carlsson and Nachemson 2000). Cervical radiculopathy caused by nerve root compression from disc herniation or spondylosis has been considered an indication for surgery; however, there is no clear validation for this assumption (Carlsson and Nachemson 2000). Several prospective studies have in fact demonstrated the resolution of cervical radiculopathy with time and/or conservative management (Bush *et al.* 1997; Mochida *et al.* 1998; Maigne and Deligne 1994; Saal *et al.* 1996).

Only one randomised controlled comparison of surgery and conservative management for chronic cervical radiculopathy was available in the literature up to 2000 (Carlsson and Nachemson 2000; Fouyas *et al.* 2002). Cervical radiculopathy had been present for at least three months and the diagnosis was confirmed by MRI; there were twenty-seven patients in each of three groups: surgery, physiotherapy and cervical collar (Persson *et al.*1997a, 1997b; Persson and Lilja 2001). Physiotherapy consisted of an eclectic range of modalities, manual

therapy and exercise distributed over fifteen sessions (Persson *et al.* 1997a). The surgery group demonstrated significant change after treatment, and surgery and physiotherapy groups were both significantly better than the collar group. At one year, however, there were no significant differences between any of the groups. In effect all three groups improved, but the surgery group had a quicker improvement in pain, function and other outcomes. However, all groups still had moderate levels of pain at one year, and 29% of the surgery group underwent additional surgery in the following year.

A prospective, multicentred study with independent review of patients with cervical radiculopathy failed to find significant differences between surgically and medically treated patients (Sampath *et al.* 1999). There was incomplete follow-up, but both groups showed significant improvements over time. A study in which surgical patients were matched by gender and age with untreated patients reported better outcomes in the surgery patients, but median pain rating was little changed in both groups at nine months or two years (Lofgren *et al.* 2003).

Regarding the value of surgery for other cervical spine conditions, there were no randomised controlled trials evaluating surgery for whiplash associated disorders (WAD) (Carlsson and Nachemson 2000). Even for patients with cervical myelopathy a conservative approach to management has been demonstrated to be a viable option, with outcomes as good as surgery in patients with mild to moderate symptoms and shorter disease duration (Yoshimatsu *et al.* 2001; Kadanka *et al.* 2002). In forty-nine patients with mild to moderate cervical myelopathy, randomised to conservative or surgical treatment, there was no significant difference between the two groups, but no improvement over time, whereas twelve patients with severe myelopathy showed significant improvements after surgery (Bednarik *et al.* 1999).

A Cochrane review on the role of surgery in cervical spondylotic radiculomyelopathy found that the evidence was inadequate to provide reliable conclusions on the balance of risk and benefit from cervical spine surgery (Fouyas *et al.* 2002). Evidence for the value of surgery in the treatment of neck pain and cervical radiculopathy is largely absent, and with a few exceptions surgery for mechanical disorders of the cervical spine is unnecessary (McKenzie 1990).

## Post-surgery

There is limited documented evidence about the best rehabilitation approach following cervical surgery. The emphasis of examination and management depends on when the patient is seen after surgery and what type of surgery has been performed. There is much greater variety of surgical procedures than at the lumbar spine. Cervical disc herniations are commonly treated by microdiscectomy with or without fusion, and instability or trauma by fusion and possibly fixation. Spinal surgeons may have specific protocols for post-surgery rehabilitation, but post-surgical rehabilitation is often not requested. The surgical approach is commonly anterior to reduce interference with muscles and maintain posterior structure stability.

Following microdiscectomy, the emphasis is on reassurance of the patient, posture correction, gradual restoration of all movements and progressive return to normal function. Movement should be regained in a graded progressive fashion and neural mobility could be included. For surgery that involves interbody fusion immobilisation is preferred and generally physiotherapy avoided; for instance, prescription of a cervical collar for two or three months post-operatively has been suggested (Kokobun and Tanaka 1995). Following fusion, end-range movements should be avoided for up to three months, but lots of mid-range movements may help to restore patient's confidence and strengthen the fusion. It is to be expected that such patients will have reduced range of movement.

## Thoracic Outlet Syndrome

There is still controversy about the existence of Thoracic Outlet Syndrome (TOS), mainly because of the lack of reliable and valid diagnostic criteria (Rayan 1998; Huang and Zager 2004). Part of the controversy lies in the fact that it is detected and treated by surgeons more often in some countries than in others (Lindgren 1993). Whilst not uncommon in the US, it is reported to be unknown in Australia and seldom diagnosed in England and Europe (Lindgren 1993; Schenker and Kay 2001). Reported incidence consequently varies between three and eighty cases per thousand population (Huang and Zager 2004). TOS is a diagnosis by exclusion of all else.

By definition TOS refers to the compression of the brachial plexus and sub-clavian blood vessels at the apex of the thoracic cage (McKenzie

*et al.* 2004). Although compression is generally considered to be the sole causative factor, in fact tension on the neurovascular bundle has also been identified as a common cause of symptoms, which obviously has implications for management (Ide *et al.* 2003). Symptoms are variable but include pain, numbness, tingling and/or weakness in the arm and hand. TOS is usually classified as either neurological or vascular depending on the site of compression. 'True' neurological TOS, about whose existence there is little controversy as signs and symptoms are clear, is sometimes distinguished from 'disputed' TOS because of the more subjective nature of the complaint in the latter (McKenzie *et al.* 2004; Schenker and Kay 2001). Vascular TOS is much less common and symptoms include Raynaud's phenomenon, limb ischaemia, cyanosis, oedema in the hand and arm, and pallor. With these symptoms there is less likelihood of diagnostic confusion with cervical disorders and this description focuses on the neurological category of TOS, over which diagnostic confusion is much more likely.

Compression of the brachial plexus or the vessels occurs as they pass out of the thorax between the first rib and the scalene muscles or the clavicle, or between the rib cage and pectoralis minor (McKenzie *et al.* 2004; Rayan 1998). Predisposition to TOS may result from congenital or acquired factors (Rayan 1998). Cervical ribs, which may occur in up to 1% of the population, and first rib, vertebral or soft tissue anomalies are all thought to be possible congenital predisposing factors. Acquired predisposing factors are said to be poor posture, repetitive upper limb occupational stresses requiring work above shoulder level, hypertrophic muscles in athletes and bony abnormalities. Sub-classification of TOS based on the hypothesised site of compression is sometimes recommended (McKenzie *et al.* 2004; Kreig 1993).

The signs and symptoms of TOS can be highly varied, with more extreme clinical presentations in those with more advanced disease (Rayan 1998; McKenzie *et al.* 2004; Balci *et al.* 2003; Ide *et al.* 2003; Schenker and Kay 2001; Huang and Zager 2004). Those with the more extreme presentation can show dramatic wasting of hand musculature and other neurological signs, but often pain is not a key feature. In the non-specific, more common type, pain is the main complaint; initially there are symptoms of pain and paraesthesia, and later there may be signs of numbness and muscle weakness as the condition progresses. Onset is usually between 20 and 40, with a cervical rib usually the

cause if incidence is younger; TOS is more common amongst women than men. The patient often has poor posture and weak musculature. Initially symptoms are provoked by raised arm activities, head rotation or even taking a deep breath, but later they may be present at rest. The symptoms are usually vague and diffuse, involving the whole of the upper limb, the scapular region and the lateral neck; they can be accompanied by headache and can be bilateral. The neurological symptoms can be sensory or motor: paraesthesia, numbness, weakness, clumsiness. Paraesthesia again may occur with raised arm activity but later may be present at rest, and the neurological symptoms can be radicular or diffuse in nature.

### Tests

Various provocative diagnostic tests have been described to monitor vascular integrity, but there is no absolutely reliable and accurate test to make a diagnosis of TOS (Rayan 1998; McKenzie *et al.* 2004; Mackinnon and Novak 2002). The various tests use provocative positions whilst the radial pulse is monitored; however, as this is less relevant to neurological TOS, it is accepted now that the provocative position may simply reproduce the patient's symptoms. In Adson's manoeuvre the arm is by the side, the head turned to the affected side, and the patient is instructed to inhale deeply. In the Halsted manoeuvre or costoclavicular test the patient braces the shoulder girdle down and backwards. In Wright's test the shoulders are abducted 180 degrees, and in the hyperabduction test deep inhalation is added to this. In Roo's or elevated arm stress test the patient elevates the arms over halfway for three minutes whilst they open and close the hands, and this is considered positive with reproduction of symptoms. In the Cyriax Release test the shoulder girdle is passively elevated for three minutes and a positive test occurs when paraesthesia, numbness or pain is provoked (Brismee *et al.* 2004). In obvious cases patients simply raise their arms above the head and paraesthesia, numbness or pain is rapidly provoked, and symptoms are unaffected by neck movements.

The validity of the tests has been questioned because of false-positive and false-negative responses (Brismee *et al.* 2004). The tests are commonly positive in the asymptomatic population (Plewa and Delinger 1998; Warrens and Heaton 1987; Rayan and Jensen 1995; Gergoudis and Barnes 1980; Brismee *et al.* 2004). For instance, an altered pulse has been found in 11% to 60% (mean 29%) of volunteers with different tests. Paraesthesia has been provoked in 2% to 36%

(mean 15%) of asymptomatic volunteers with different tests. Pain has been provoked in 21% of asymptomatic volunteers with Roo's test.

However, positive provocation tests, with elevated arms, have been found in 94% of fifty patients diagnosed with TOS (Novak *et al.* 1993). The hyperabduction tests have shown good sensitivity, but poor specificity; Adson's test has shown moderate sensitivity (79%) and specificity (76%) (Gillard *et al.* 2001). When using multiple tests there were modest gains in sensitivity and specificity (Gillard *et al.* 2001). Lowest false-positive rates were achieved with pain with Adson's, costoclavicular manoeuvre, or any two shoulder manoeuvres (Plewa and Delinger 1998).

Diagnosis of TOS therefore requires a staged process:

- diffuse neck/shoulder/arm symptoms of pain/paraesthesia
- provoked with raised arm activities
- failure to reach an MDT classification after extended mechanical evaluation including force progressions and force alternatives
- positive concordant pain response to at least two TOS provocation tests.

Regarding management of TOS, it is recommended that conservative management be tried before surgical intervention is considered, especially for neurological type of TOS (Rayan 1998; McKenzie *et al.* 2004). A range of treatment options have been recommended, but none have been adequately assessed (Lindgren 1997). Most conservative regimes emphasise exercise to improve patient's posture, which is usually of protruded head and depressed and rounded shoulder girdle, and strengthening exercises to maintain the improved posture. Aggravating postures should be avoided, and tight structures should be stretched to improve flexibility. Only if conservative management fails should surgery be considered. Although case series frequently report positive outcomes from surgery, repeat surgery is not uncommon, length of follow-up is often limited or unclear, an independent reviewer has been rarely used, and no attempt has been made to compare conservative and surgical treatment in a scientific way (Lindgren 1997). An eight-year follow-up of forty-five surgically treated patients found that 57% of operations failed to abolish symptoms (Lindgren and Oksala 1995).

## Conclusions

This chapter has discussed a few other considerations of diagnosis and management that might sometimes be encountered beyond the mechanical syndromes. If after a detailed and thorough mechanical evaluation conducted over several sessions there has been a failure to classify the patient into one of the mechanical syndromes, only then should other diagnoses be considered. This is the case in only a small minority of patients. Patients with some combination of shoulder and neck pain need assessment of both sites prior to initiating management, and then monitoring of response at both sites to determine the source of symptoms. Other categories include cervical stenosis, mechanically inconclusive, chronic pain state and thoracic outlet syndrome, which are all considered in this chapter. Headaches and whiplash are covered in separate chapters. This chapter has also considered a number of other diagnostic and management issues.

# 10: The History



## Introduction

*"Every patient contains a truth....The [clinician] must adopt a conscious humility, not towards the patient, but towards the truth concealed within the patient"* (Cyriax 1982, p. 45). 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. The patient knows the details of the history, onset, symptom pattern and behaviour since onset, and aggravating and relieving factors. Only from the patient is it possible to gain insights into various aspects of the clinical presentation, which are essential to inform issues such as the stage and nature of the disorder, the prognosis and the management. Very often the history-taking provides information that is at least as important as that gained from the physical examination, if not more so.

In mechanical diagnosis and therapy we wish to understand the effect that different movements and positions have on symptoms and use this understanding to shape an appropriate management strategy. This understanding comes through analysis of the history and physical examination.

The interview requires skills of questioning accurately and appropriately as well as listening. It is important that we make the patient as relaxed as possible, for instance by avoiding use of medical jargon that may be unfamiliar. The use of a structured, but flexible interview format so that all pertinent factors from the history and behaviour of the condition are collected will facilitate a good understanding of the patient's problem. The standardised assessment form includes the most important aspects of the history that need gathering; mostly it is unnecessary to add to this information.

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. Focussed questions may be used to follow up if particular aspects need more detailed information. The form should not prevent further specific questioning if this is thought to be

necessary. Thus, management decisions can be grounded in the particular patient's problem and their response to it.

Sections in this chapter are as follows:

- aims of history-taking
- interview
- patient demographics
    - age
    - occupation/leisure activities
    - functional disability
- symptoms this episode
- previous history
- specific questions
    - 'red flags'.

## Aims of history-taking

Using the form and the appropriate questioning technique at the end of the history-taking, ideally the following will have been obtained:

- an overall impression of the clinical presentation
- the functional limitations that the condition has imposed on the patient's quality of life
- location of 'neck' pain: central/symmetrical, or unilateral/asymmetrical; if unilateral, is the pain in the neck, scapular, shoulder and arm, or referred below the elbow
- determination if a neurological examination should be conducted
- the stage of the disorder – acute/sub-acute/chronic
- the status of the condition – improving/unchanging/worsening
- identification of 'red flags' or contraindications
- baseline measurements of the symptomatic (and mechanical presentations) against which improvements can be judged
- movements and positions that aggravate and relieve the problem, and the role of posture, which may help guide future management

- the severity of the problem, which may guide the vigour of the physical examination
- an impression about the way the patient is responding to their condition, and how much encouragement, information, reassurance or convincing they may need to be active participants in their own management
- a hypothetical diagnosis by syndrome.

## Interview

During the history-taking, seat the patient on the treatment table or a backless chair so that they reveal their true relaxed sitting posture.

## Patient demographics

### Age

Patients are more susceptible to certain problems at different times of life. Postural syndrome is more likely to be present in the young, whilst young to old adults have derangements and dysfunctions. Osteoporosis is generally only relevant in the elderly, especially post-menopausal women, although there are exceptions. With increasing age spinal degeneration is more likely to be present, the intervertebral disc becomes dehydrated and fibrosed, and osteophytes and other bony changes can occur around the zygapophyseal and uncovertebral joints and vertebral bodies (Taylor and Twomey 2002). Such changes may predispose to spinal stenosis affecting nerve roots or the spinal cord. Malignancies are also more common in the older age group. Completely new onset of headache or neck pain in older patients who have never experienced this before is also a possible warning symptom.

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

### Occupation/leisure activities

It is important to know the individual's occupation and the kind of postural stresses it entails. Do they spend their day mostly sitting, driving or bending forwards? Are they constantly changing activity?

Are they on their feet most of the time? We wish to know the predominant activities of their working hours so that detrimental daily loading factors can be eliminated or lessened. Also it is useful to know if there has been a recent change in occupation, from a sedentary to a more manual job, or vice versa. Either change may be a trigger to potential overloading.

We also want to know about their usual sporting or recreational activities outside of work. Do they exercise regularly, or do they lead a largely sedentary life? Hobbies might include largely sedentary activities, such as fishing or knitting, so questioning needs to ensure that all types of pastimes are included.

### Functional disability

We wish to know if the patient is off work at present, and/or not participating in any of their usual sporting or leisure activities because of their neck problem. The earliest possible return to full normal function is the suitable goal for management. The worker should be encouraged to remain at work or to return as soon as possible. The common misconception that they should be pain-free before returning to work should be addressed. Return to work should be a primary outcome of treatment.

Equally we should be aware of any normal sporting or recreational activities that they have stopped because of neck pain. An early return to such activities, possibly in a gradual way, should be encouraged as soon as possible. General fitness has a therapeutic as well as protective effect for spinal pain, and management of the patient should address these issues.

Knowledge of the activity limitations that neck pain has caused in the patient's normal lifestyle provides some understanding of their response to the problem in terms of their fear and anxiety. A brief and temporary interruption of normal activity may be necessary in some episodes; a long-term abstention from normal activity is unnecessary and disproportionate. Persistent avoidance of daily routines often indicates an exaggerated and inappropriate response to pain. Such patients need specific encouragement to return to normal activities and care must be made not to further exaggerate such inappropriate fear-avoidance behaviour.

## Symptoms this episode

*Where have you had symptoms this episode?*
*Where have you had pain or aching?*
*Have you had any pins and needles, tingling or numbness?*
*Have you had any weakness in the arm?*
*Where are you still having symptoms?*

All the symptoms that have occurred during the present episode should be accurately marked on the body chart. To ensure accuracy this can be shown to the patient and checked by them. The relevant symptoms are those that have been felt in the last few days and are still a problem – these are noted on the line below. Baseline symptoms, which are still troubling the patient, must be recorded in full so that any changes in pain pattern over time can be appreciated.

We wish to know if the present pain is central/symmetrical or unilateral/asymmetrical. If symptoms are unilateral or asymmetrical, is the pain felt in the neck and arm, or is it referred below the elbow? We wish to know the most distal extent of any pain. If the patient reports pain in the arm or forearm, they should be asked if 'pins and needles', tingling or numbness are present at any time, and exactly where; also if they have experienced any noticeable weakness in the arm. In later chapters management is described relative to different symptom patterns (Table 10.1).

**Table 10.1 Symptom patterns relevant to management decisions**

- symmetrical pain
- asymmetrical +/- pain to elbow
- asymmetrical pain or paraesthesia below elbow.

The location of pain provides various useful pieces of information. Central or bilateral symptoms invariably need sagittal plane procedures. Patients with unilateral symptoms may require lateral forces in their management, although their response to sagittal plane forces is generally tested first.

The extent and degree of referred or radiating pain and other symptoms gives some indication of the severity of the problem. More peripheral referral of symptoms, as well as the presence of paraesthesia or marked focal weakness, both of which may accompany symptoms referred below the elbow, tend to indicate a more severe problem.

If the location of pain has changed since onset, this may provide a clue to the status of the condition. Pain that was felt into the arm and is now felt only in the neck demonstrates an improving situation. Conversely, pain that began in the neck and has gradually spread down the arm demonstrates a worsening situation.

The location of pain gives some insight into mechanical syndrome classification. The pain experienced with the dysfunction and postural syndromes is almost always felt locally, with no radiation of pain. An exception to this is referred pain caused by an adherent nerve root, which is described later. If pain radiates into the arm or forearm, a derangement is likely.

Nerve root involvement is possible if pain is described in the typical pattern of a dermatome (Slipman *et al.*1998), especially when other neurological signs are present. Paraesthesia of diagnostic significance most commonly occupies the distal end of the dermatome; the patient reports tingling or numbness – C6 the thumb, C7 the middle finger, C8 the little finger. Less commonly C5 or T1 are involved – sensory loss occurring on the lateral border of the arm and the medial border of the forearm just below the elbow respectively (Kramer 1990; Butler 2000). It is not uncommon for there to be individual variation from the typical patterns.

There is ample room for confusion between symptoms that emanate from the cervical spine, the thoracic spine and shoulder problems – pain patterns may provide some clues. Several studies have indicated that pain around the scapular and shoulder region commonly arises from cervical discogenic or zygapophyseal joint disorders (Cloward 1959; Smith 1959; Whitecloud and Seago 1987; Grubb and Kelly 2000; Dwyer *et al.* 1990; Aprill *et al.*1990). However, stimulation of thoracic structures has also caused pain in the chest and scapular region (Bogduk 2002b). Irritation of the acromioclavicular joint (ACJ) and subacromial space suggests that these structures may refer proximally, but that predominantly symptoms are felt either over the ACJ or around the shoulder (Gerber *et al.* 1998). Any combination of neck and scapular or shoulder pain is thus most likely to be referred pain from cervical structures.

**When did this present episode start?**

This question is to determine when this particular episode started. If the patient has suffered recurrent problems, at this stage we are only

interested in the present attack. Very often the patient is aware of the time an episode started. If pain has been present for a long time, an acute exacerbation of a chronic problem may have caused them to seek help. In this case the episode has lasted since the original onset.

It is helpful to know if we are dealing with an acute, sub-acute or chronic problem. In this text these are defined in line with the Quebec Task Force definitions (Spitzer *et al.* 1987), which correspond with the known healing process.

**Table 10.2 Definitions of acute, sub-acute and chronic**

- acute – less than seven days
- sub-acute – between seven days' and seven weeks' duration
- chronic – more than seven weeks' duration.

The length of time that the condition has been present may give some indication of the stability of the problem. Acute problems can easily be worsened as well as improved, so care with movement testing may be necessary. Acute and sub-acute problems are most probably due to derangement, whilst any three of the mechanical syndromes could be the cause of chronic problems.

Knowing the length of time that the problem has been present allows us to determine the state of the tissues. Days after onset tissues may be damaged and inflamed, whereas a few weeks later tissues may be healing. If the symptoms have been present for a couple of months, adaptive changes may have occurred in the collagenous repair tissue indicating that dysfunction may be the cause of persisting symptoms. If the problem has been present for many months, although a straightforward mechanical condition may be present, the chance that the tissues are hypersensitive and deconditioned should be borne in mind (Johnson 1997). 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 if it does respond. Many patients have a long or recurrent history of their problem and therefore the educational component of management is particularly important to improve their future self-care. The duration of the episode thus provides diagnostic and prognostic information.

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

**Is it getting better or worse or is it staying the same?**

It is important to know if the patient thinks their problem is improving, worsening or unchanging. Judgements about the status of a condition may be based on five criteria (Table 10.3). A true understanding of the patient's condition comes from both the history and the physical examination. Only some of the information will be gained during the history-taking. If the patient volunteers that the condition is getting better or worse, it is important to confirm what they mean by this against some of the criteria outlined below.

**Table 10.3 Criteria for defining status of condition**

| *Criteria* | *Dimensions* |
|---|---|
| Time | Constant/intermittent |
| | Frequency increase/decrease |
| Intensity | Increase/decrease |
| Referral of pain | Centralising/peripheralising |
| Mechanical presentation | Movement increase or decrease |
| Activity limitation | Increase/decrease |

When the patient reports that their condition is improving, a review of the problem and its prognosis is often all that is required. Avoid the inclination to embark on a programme of passive therapies. If history and evaluation of repeated movements confirm the process of recovery is under way, continuing at a steady rate, and accompanied at the same time by improvement in function, there is no justification for any intervention other than education and assurance, unless or until progress comes to a halt. Provide guidelines for the progression of activity and exercise and give advice on posture where necessary, but such patients do not need to attend a clinic for regular 'treatment'.

If the pain is unchanging, a routine approach to the assessment can proceed. Stable and persistent symptoms generally permit a reasonably vigorous approach to assessment and management. Derangement or dysfunction may cause pain and functional impairment that may continue unabated for weeks or months, and may only be exposed using vigorous procedures.

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

In the event that the patient describes that his or her symptoms are worsening since onset, it is necessary to investigate the cause of deterioration. A rather gentle approach to the mechanical evaluation is always required if the patient describes that their pain is progressively increasing, and symptom response must be very carefully monitored. Under these circumstances a purely educational approach may be indicated, certainly for the first twenty-four to forty-eight hours. Sustained positions may be of more use than repeated movements in attempting to improve symptoms. Increasing pain intensity could indicate more serious pathology, but certainly indicates an unstable condition in which greater care should be taken.

Patients whose symptoms are worsening should be seen on a regular basis until stability or improvement occurs, or until it becomes obvious that referral for further investigation is necessary. If the patient describes any of the 'red flag' indicators of serious pathology, or if the reactions to mechanical evaluation are atypical or if they fail to affect the symptoms, referral for further investigation should be considered. For instance, an insidiously worsening neck pain in an older patient

who reports being unwell should be the cause of some concern. Appropriate blood tests or radiological assessment may shed light on the origin of the symptoms in such cases.

**How did the neck pain start?**

We want to know what the patient was doing when the pain started. In most instances there is no apparent reason and symptoms arise insidiously (McKenzie 1990; Kramer 1990). McKenzie (1990) reported from his records that in two-thirds of patients neck pain had come on for no apparent reason. Some patients relate the onset of symptoms to stress. Some patients report that the pain has gradually come on during a normal day's activity. In such instances it is important to ascertain what these activities are and what postures they entail, as adaptation of these positions might be important in management. Symptoms are commonly triggered by prolonged kyphotic posture of the cervical spine during reading, watching television or working at a desk (Kramer 1990). Commonly neck pain comes on during the night and the patient awakes with the symptoms. Often patients are reluctant to accept no obvious reason for the onset of symptoms, especially if compensation is an issue. They may ascribe a causative role to some recent event that might not relate to the onset of neck pain. Careful questioning may be needed to determine the true relationship between the event and the onset of symptoms. Trauma is an obvious cause of neck pain in some patients, especially those involved in vehicle collisions or head injuries. Although in most such instances soft tissue injury only is present, sometimes it is desirable to exclude bony injury with the use of further investigations.

Where symptoms have commenced for no apparent reason and are progressively worsening, it is possible that some more sinister cause may be present. The likelihood of the presence or otherwise of serious pathologies should be determined from further 'red flag' questioning.

**Where was the pain at onset?**

This question is to determine if, since onset, the location of the symptoms have changed. Specifically, we wish to know if pain and other symptoms are peripheralising – a worsening scenario; or if there has been a resolving of arm pain that was originally present – an improving scenario. When patients describe symptoms that change location, a derangement is always suspected.

**Is the pain constant or intermittent?**

This is one of the most important questions we must ask all patients with musculoskeletal disorders. Be sure that by 'constant pain' the patient does actually mean that their pain is there 100% of their waking day 'from the moment you get up to the moment you fall asleep'. Because some patients can always produce their pain with certain movements, they interpret this as constancy. Likewise, if pain has been present for a prolonged period and is there every day, patients might erroneously report constant pain. 'Is there any time day or night when you have no pain or discomfort?' Pain must be classified as intermittent even if there is only half an hour during the day when they are pain-free. Truly constant pain is present in about a third of spinal patients (McKenzie 1990).

Constant pain is most commonly the result of constant mechanical deformation, which is only present in derangement syndrome. Constant pain is also caused by inflammatory diseases and may be present when patients have suffered recent trauma causing an inflammatory response.

If the cause of the constant ache or pain is chemical, the symptoms will not be reduced or abolished by mechanical assessment procedures. Normally innocuous mechanical stresses can become painful under these circumstances. Movements may enhance existing chemical pain, but they never reduce or abolish it (McKenzie 1981, 1990). In general the inflammatory period following trauma does not exceed five days (Evans 1980; Hardy 1989). If an inflammatory disease or arthropathy is present, chemical pain will persist for longer periods.

If the cause is mechanical in origin, movements and positions can usually be found that reduce or stop the aching or pain. Constant mechanical pain is the result of internal derangement. The derangement syndrome can be associated with a constant ache, whereas patients with the postural and dysfunction syndromes do not experience constant pain. Constant pain is more difficult to treat than intermittent pain because the patient is usually unable to identify a directional or postural preference.

Chronic pain states usually present with constant pain, which is likely to worsen initially on mechanical assessment. The history will reveal the length of time symptoms have been present and also may relate previous failed treatments. Unremitting, constant pain that does not

abate even when lying down, which is worsening, may be caused by serious spinal pathology (see 'Red flags' section below).

If pain is truly intermittent, it must be mechanical in origin and is produced by intermittent mechanical deformation. Inflammatory pain is excluded. It could be postural, or result from dysfunction or derangement.

Intermittent pain is usually easier 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 one that is present most of the time to one that is present only sometimes both represent improvements in symptomatic presentation. Such improvements should help in the overall assessment of management strategies. It is therefore useful to ask about the proportion of the day that pain is present. This could be expressed as a percentage; for instance, 'On average, how much of the time are your symptoms present – for 80% of the day, 50% of the day or 20% of the day?' Alternatively, patients may report that their pain occurs intermittently, and increasing or decreasing frequency of pain occurrence can be used to evaluate management efficacy.

**What makes the pain worse? What makes the pain better?**
These questions allow the patient to provide us with information that helps determine 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 protocol. It is also important to record which movements or activities aggravate the symptoms. As part of the educational strategy, it may be necessary temporarily to avoid such causative factors, or alter the way an activity is performed so that stresses are lessened.

It is also useful to know which type of loading strategies, static or dynamic, most affects the symptoms. The patient may reveal that

sustaining a position reduces their symptoms, and so static forces would be explored in the physical examination. They may reveal that a prolonged position produces their pain after a certain time, and so interruption of aggravating postures before pain is created will be important in management.

Specifically, we must ask about the effects of sitting, bending, turning, lying and rising and any difference between stationary and dynamic positions on the patient's symptoms. Everyone is subjected to these forces every day, so questioning can generally be confined to universal daily activities. Furthermore, in these positions the anatomical alignment of the cervical joints is relatively well understood. In general, sitting, driving and bending are activities of cervical flexion. This allows us to determine which situations increase and which situations decrease mechanical deformation (McKenzie 1981, 1990). This information is vital for optimal management. In lying, the posture of the spine varies according to whether the individual adopts a supine, prone or side-lying position, and upon the number of pillows used.

We also wish to know if the symptoms are better when the patient is still or on the move. Very often patients feel less pain when they are moving regularly and worse when still. These patients often respond well to an exercise programme. Some patients with more severe conditions only gain some relief when they are lying down.

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. Rotation and extension are common provocative movements. Conversely, if activities are *sometimes* painful and *sometimes* not, derangement is usually implicated.

It is helpful to know if the movement that relieves or aggravates the pain brings about a lasting improvement or worsening condition. A lasting improvement following a particular loading strategy gives a useful idea about self-management procedures. If pain is produced by certain activities but quickly abates once the movement ceases, the disorder is at a reasonably stable stage. If pain remains worse for hours after a relatively trivial movement or sustained posture, the disorder is likely to be an unstable derangement.

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

Some patients who have had pain for a long time can have difficulty in determining what makes their symptoms better or worse. Further detailed questioning may provide the relevant information. However, at times patients may have become incapable of analysing their pain, or movements and positions may have no consistent effect upon their symptoms. In such instances the information obtained from the history is insufficient, and during the physical examination it is essential to produce a change of symptoms by utilising end-range repeated movements and sustained positions. An indeterminate effect of loading strategies on symptoms is common in chronic pain states when mechanical factors have become less relevant to pain persistence.

**Are you better or worse on waking in the morning? Are you better or worse as the day progresses?**

We wish to know if there is any consistent pattern to the symptoms during the day. Their answers provide some clues as to the effect of different activities over time and the effect of general activity compared to rest. The diurnal pattern of symptoms can give an indication of the effect of sustained postures. Patients who report that they wake with pain that was not present the night before or is worse in the morning may be using an unsuitable pillow or adopting an inappropriate position during the night.

**Does the pain wake you at night?**

If pain wakes the patient at night this may be the result of an unreduced derangement, unsuitable pillow or inappropriate sleeping posture causing a derangement. A cervical roll may be beneficial and should be tried for a few nights. Sleeping postures are difficult to alter, but prone sleepers are particularly susceptible to having their cervical spines in positions of prolonged end-range rotation, which may have a causative role in symptoms.

If neck pain is disturbing the patient's sleep, then supplementary questions should be asked about sleeping postures, the bed and the number of pillows used.

## Previous history

**Have you had neck pain before? If so, approximately how many episodes?**

Many individuals have an episodic history of neck pain. It can be useful to know if the episodes have been the same over time or if the patient feels they are getting worse gradually. If worse, describe in what way – longer, harder to get rid of, shorter between episodes, or more severe or more peripheral pain, for instance. A history detailing every episode and its outcome is unnecessary.

Sometimes it emerges that the patient has had mild symptoms for years, interspersed by episodes of more severe pain, one of which has made them seek treatment. In such instances the best that may be achieved is a return to the underlying mild symptoms. In any patient with multiple episodes the importance of achieving independent self-management and discussion of a realistic prognosis is vitally important. An episodic history usually indicates recurring derangement.

**Have you had any previous treatment that you found particularly helpful?**

As with the previous question, precise detail is not required here. Most importantly, we wish to know if the patient thinks anything has helped previous episodes, rather than a detailed history of every treatment received. However, if patient reports 'successful' treatment of an acute episode of neck pain, this should be viewed in light of the brief episodes that many experience. If previous treatment was a mixture of manual therapy and exercise, it may be difficult to interpret the therapeutic value of each component. If the patient thinks that exercises helped previously, it is important to know if they have they been doing them recently and with what effect.

## Specific questions

These questions serve three purposes. First, it gives us some idea about previous medical management and imaging studies for their neck problem. Second, this helps to bring to our attention any systemic or other conditions that may affect mechanical management or prognosis. Specific questions also raise questions about and explore any concerns we might have about serious spinal pathology as opposed to normal neck pain.

With general medical questions, we need to know what drugs the patient takes on a regular basis for any problem. We also wish to know what medication they have taken for this problem and with what effect. Ask what they are presently taking.

It may be reassuring to know that patients who have been involved in trauma have received x-rays or some other form of imaging. However, the validity of the relationship between imaging studies and symptoms in non-specific neck pain and even whiplash is unclear.

Questioning the patient about present medication and any other on-going medical problems can alert clinicians to contraindications or cautions to force progressions, as well as raising possible 'red flags' about serious spinal pathology.

**'Red flags'**

The aim of these questions is to help exclude the possibility of certain specific pathologies that contraindicate mechanical therapy and may need urgent onward referral. The questions do not allow confident diagnosis of these problems; rather, they should make clinicians suspicious of unsuitability for mechanical therapy, especially when response to mechanical therapy is atypical. Sometimes it may be clear from the history alone that sinister pathology is possible, in which case no further testing is carried out and referral is immediate. If you suspect something serious, it is always better to err on the side of caution and refer to a specialist.

However, it should be remembered that serious spinal pathology makes up only 1 – 2% of all spinal patients (AHCPR 1994). Relevant pathologies are cancer, infection, cord signs, fractures, upper cervical instabilities and other non-mechanical pathologies. All these specific pathologies are rare, but they will be missed if they are not at least considered. This is the basis of the triage system – to filter out those very few individuals who need further investigation. At this point exact diagnosis is not necessary, but suspicion of serious spinal pathology demands action.

Most of this screening is done during the history-taking, which generally is more instructive than any aspect of the physical examination. By this point certain features in the patient's history may already have made you suspicious and determined the need for more detailed questioning. However, in the majority of patients by this stage you

may be confident that this is a straightforward mechanical problem, and further special questions may be unnecessary. Special questions may include the following (see Table 10.4 for fuller explanation):

- *Are they walking normally? Is there any weakness or clumsiness in arms and/or legs?*
- *Are there any pins and needles or altered sensations in arms and/or legs?*
- *Is their bladder and bowel function normal for them?*
- *Is there any history of serious illness? (specifically cancer)*
- *Is the patient forced to leave the bed at night because of pain?*
- *Has there been any unexplained weight loss?*
- *Is there any systemic ill health or malaise?*
- *Has there been any major surgery?*
- *Have there been any major recent accidents?*

**Table 10.4 Features of history ('red flags') that may indicate serious spinal pathology**

- Age > 60. Serious spinal pathology, such as cancer, myelopathy or osteoporosis, is more likely in older patients.
- Upper motor neurone signs and symptoms. Variable range of findings may suggest spinal cord involvement: sensory disturbance upper and lower extremity, muscle weakness upper and lower extremity, diminished manual dexterity, gait disturbance, muscle wasting, increased muscle tone, hyper-reflexivity, positive Babinski, bladder and bowel disturbance (Edwards *et al.* 2003; Nachemson and Vingard 2000). This may occur with cervical radiculopathy or with no or minimal neck symptoms.
- History of cancer. Cervical spine is the least common site of benign or malignant tumours, but they do occasionally occur, whilst tumours in the thoracic spine are about as common as lumbar tumours (Weinstein and McLain 1987; Schaberg and Gainor 1985; Bernat *et al.* 1983). Previous history of cancer is a strong risk factor for cancer-related back pain (Deyo *et al.* 1992). Unexplained weight loss and night pain severe enough to drive a patient from their bed may be other findings.
- Systemic symptoms. Even with very severe neck pain, the patient is well. If the patient is generally unwell, with raised temperature or unintentional weight loss, suspicion of some systemic disease should be raised. Diseases such as osteomyelitis and tuberculosis occur much less frequently in the cervical spine compared to the lumbar and thoracic, but do rarely occur (Krogsgaard *et al.* 1998; Narlawar *et al.* 2002). Recent fever with accompanying neck pain could also be due to tonsillitis, upper respiratory tract, streptococcus throat or other local infection.
- Major recent trauma. Considerable force may cause a fracture. Postmenopausal women and those on long-term steroids may incur a fracture with relatively trivial injuries.

## Conclusions

At the end of the history-taking certain aspects of the patient's presentation have been clearly identified. The clinician must feel happy to continue to explore mechanical therapy and that serious spinal pathology has been discounted. A hypothetical mechanical diagnosis should be under consideration, or possibly a non-mechanical syndrome. It is important at this point to summarise briefly to the patient the main points in the history from your understanding; this ensures that your impression of their problem accords with their knowledge.

Various issues of prognostic significance will have been explored. This includes the stage and status of the condition, its apparent mechanical sensitivity, and any 'yellow flag' factors concerning the patient's response to the problem. The mechanical sensitivity, as in the aggravating and relieving factors, provides clues for management strategies.

A range of baseline measurements will have been taken against which to judge the effects of subsequent mechanical therapy. These include the site, spread and temporal component of the pain, and functional limitations.

Information gained during the history-taking should provide insights into what needs to be explored in the physical examination and how vigorous it can be; for instance, should a neurological examination be conducted? Is there a strong suggestion of a mechanically determined directional preference, or is a protracted period of exploring loading strategies more likely?

# 11: Physical Examination



## Introduction

The physical examination will relate closely to the findings from the initial interview. The history given by the patient should already have provided the clinician with sufficient information to be making tentative conclusions about certain aspects of the case. Very often it is clear from the history to which mechanical syndrome the patient belongs, whether the patient has simple neck pain or with nerve root involvement, and whether there exists a mechanically determined directional preference. Details gathered may suggest serious spinal pathology that needs further investigation.

The clinical examination is designed to confirm the initial findings and fully expose the mechanical nature and extent of the problem. The two parts of the first day's assessment should thus produce a good general picture of the patient's symptomatic and mechanical presentations. From these findings come the optimal management of the condition. The whole assessment provides baseline measures of pain, movement and function against which to judge 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 physical examination involves various observations and movements about which the clinician must make judgements. Such perceptual tests, in which a human being is the measuring device, are bedevilled by subjectivity with consequent variability of results. Intraobserver and interobserver variability is seen as the inevitable consequence of such perceptual tests (Gray 1997). Although we cannot totally prevent this phenomenon, we can limit its impact by conducting the examination in the same way each time it is done. Clinicians need to perform tests consistently on each occasion, and patients must always start from the same position – in this way we can be more certain that different test results reflect changes in the mechanical presentation rather than being the fault of inconsistent examination technique.

Given the problem of reliability, it is probably best to limit the information sought during the assessment. It is especially important not to overburden the physical examination with an excessive number of tests and movements. Multiple tests have a greater chance of generating unreliable information and may only serve to confuse rather than enlighten the examining clinician.

Sections in this chapter are as follows:

- aims of physical examination
- sitting posture and its effects on pain
- neurological examination
- examination of movement
    - protrusion
    - flexion
    - retraction
    - extension
    - rotation (right and left)
    - lateral flexion (right and left)
- repeated movements
    - repeated movements in derangement syndrome
    - repeated movements in dysfunction syndrome
    - repeated movements in postural syndrome
- selecting repeated movements
    - repeated test movements
    - protrusion (sitting)
    - retraction (sitting)
    - retraction and extension (sitting)
    - retraction and extension (lying)
- exploring frontal plane movements
    - lateral flexion (sitting)
    - rotation (sitting)
    - flexion (sitting)

- static mechanical evaluation
- testing inconclusive
- other examination procedures
- mechanical syndromes
    - derangement
    - dysfunction
    - postural
- inconclusive.

## Aims of physical examination

During the physical examination the following points should be exposed:

- usual posture
- symptomatic response to posture correction
- any obvious deformities or asymmetries that are related to this episode
- neurological examination
- baseline measures of mechanical presentation
- symptomatic and mechanical response to repeated movements.

The following conclusions should be made:

- syndrome classification
- appropriate therapeutic loading strategy, or
- appropriate testing loading strategy.

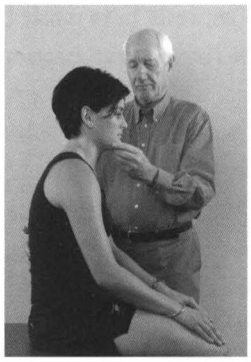
## Sitting posture and its effects on pain

If during the history-taking the patient is seated unsupported on a treatment table or examination couch, we are able to observe their natural unsupported seating posture. Posture is best observed without the patient being aware that you are doing so, such as during the history-taking. Often patients sit slouched, in a posture of lumbar and thoracic flexion, which produces a protruded head posture of lower cervical

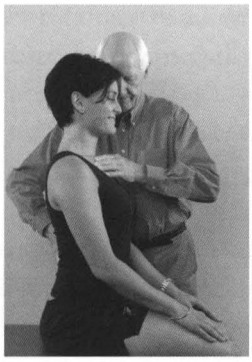
flexion and upper cervical extension. Some patients are more aware of the relationship between their posture and pain and make an attempt to sit upright as experience has told them this is more comfortable, but such patients are unusual. Regarding recognition of a protruded head posture, it may be helpful to imagine dropping a plumb line from the patient's chin. If this would fall in space some way in front of their trunk, then head posture is protruded; would this fall onto their chest, then head posture is reasonably upright. This model can also be helpful to explain to patients a better sitting posture.

Other points to be aware of are an exaggerated cervico-thoracic kyphosis and a lateral deviation of the head. When there is a fixed increased cervico-thoracic kyphosis, attaining full range cervical extension or retraction can be very difficult. If a lateral or rotational deviation is present, you need to know if it is fixed or if the patient can correct it. Sometimes patients assume this position out of habit or as a voluntary way of achieving a less painful posture; however, they are quite capable of correcting this and rotating or lateral flexing in the opposite direction. In a small number of patients, usually acute with severe onset, the neck is stuck in this laterally deviated position and any attempt to correct it is extremely painful and impossible. This deformity of wry neck is the cervical equivalent of the lateral shift in the lumbar spine.

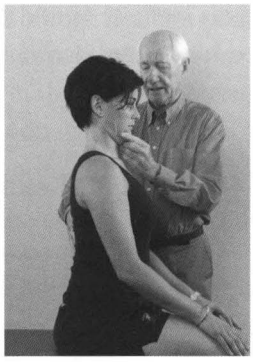
The patient will have been in sustained sitting for fifteen to twenty minutes while the history was taken – this is a good moment to investigate the effect of posture correction on neck and associated symptoms. Just as with any evaluation of symptom response, we must first determine the baseline symptoms. 'As you are sitting there now, do you have any of the symptoms that you have told me about?' It is, as always, especially important to determine the most distal symptoms, and may be useful to know if symptoms have worsened or come on whilst they have been sitting. Then the procedure of posture correction is performed, as detailed in Chapter 14, Procedure 3. The lumbar lordosis is restored, and then the head is retracted to a neutral position. Once in this position for a minute or two, the patient is again questioned about symptoms and symptom location. 'In that position, do symptoms feel better, worse or the same?'



1



2



3

*Photos 1, 2, 3: From slumped position (1), gentle pressure on the spine and sternum restores the lordosis (2) gentle pressure at chin and thoracic spine corrects the head posture (3) Symptom response is monitored before and after.*

Symptom response varies. Patients may report an easing or centralising of neck or referred symptoms. This might confirm a mechanically determined directional preference that has already been exposed during the history-taking and also is a useful teaching tool for the patient. Sometimes posture correction aggravates symptoms and less often has no effect. The response provides knowledge about their response to loading strategies that help to provide the appropriate classification and management. Sometimes posture correction may decrease arm symptoms, but increase or produce symptoms of headache. Variability of symptom response only occurs in derangement syndrome, when posture correction may reduce, abolish, centralise, increase, produce or peripheralise symptoms. In posture syndrome, posture correction abolishes symptoms immediately or within a minute or two, which will not return during the rest of the physical examination. In dysfunction syndrome, the effect of posture correction depends on the site and direction of dysfunction.

Posture in standing could be examined at this point if needed. Again, to gain a true impression of the patient's normal standing posture, it is best to get them to maintain that position for several minutes; meanwhile engage them in a conversation or ask other questions. Often cervical spine patients' symptoms are easier when standing, as the trunk and neck are more upright. If this proves to be the case, or if a patient's symptoms have deteriorated whilst sitting, this provides a clue to appropriate management strategies.

In those patients, few in number, who display a lateral deviation that is too painful to correct, the normal examination is usually abandoned after attempts to judge the range of movement are clearly pointless. These acute wry necks or torticollis sometimes occur in adolescents.

Examination should continue in the unloaded position where, with a combination of appropriate positioning and time, movement begins to return.

## Neurological examination

If a neurological examination is deemed necessary, this is a suitable time before the examination of repeated movements so that the effect of exercises on nerve root signs and symptoms can be monitored. A neurological examination should be conducted if nerve root involvement is suspected.

**Table 11.1 Conducting a neurological examination**

*Criteria:*

- paraesthesia in the upper limb
- weakness in the upper limb
- arm or forearm symptoms, especially in a radicular pattern.

*Neurological examination may involve four components:*

- sensation
- muscle power
- reflexes
- nerve tension tests.

Comparing the perception of light touch with the opposite limb tests sensation. More sophisticated testing can be done using sharp or blunt instruments in the affected dermatome. The distal end of the dermatome is the area that should be tested; this is the area that is most likely to be affected. Most commonly the patient reports tingling, pins and needles or actual numbness. It is valuable to note the area of paraesthesia and whether the symptoms are constant or intermittent; changes in these dimensions can be used to judge changes in status. Most commonly C6 and C7 are affected, causing loss of sensation in the thumb and middle fingers (Kramer 1990). Less commonly C8, C4 and C5 are involved affecting the little finger, lateral arm and the shoulder respectively (Slipman *et al.* 1998; Butler 2000).

Weakness, when present, is usually reasonably obvious, with a clear loss of power as the patient tries to resist. Pain on resisted movement is not relevant, but may be a confounding factor in the patient's ability to perform the test. Reflexes in some individuals are not easily elicited, especially by those with limited experience of reflex testing.

However, if there is a clearly diminished reflex in the affected limb when the reflex on the opposite side is easily elicited, this should be noted. A possible sequence of testing is shown in the table. Different root levels innervate a number of muscles, and equally the same root level shares in multiple actions, so testing of myotomes is not limited to the actions listed.

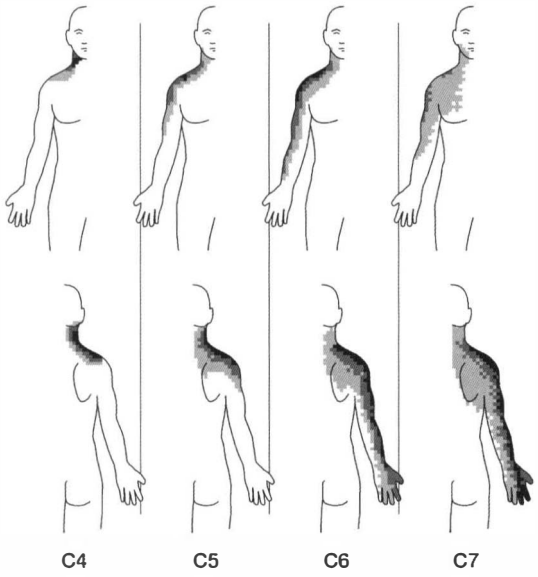
**Table 11.2 Typical signs and symptoms associated with nerve root involvement**

| *Root level* | *Typical area of sensory loss* | *Common motor weakness* | *Reflex* |
|---|---|---|---|
| C4 | Top of shoulder | Shoulder elevation | |
| C5 | Lateral arm | Shoulder abduction | Biceps |
| C6 | Thumb | Elbow flexion | Biceps |
| C7 | Middle finger(s) | Elbow extension | Triceps |
| C8 | Little finger | Thumb extension | |
| T1 | Medial border forearm | Finger abduction/ adduction | |

Source: Kramer 1990; Slipman *et al.* 1998; Butler 2000; Bland 1994

Positive neurological signs and symptoms are usually accompanied by a radicular pain pattern (Figure 11.1).

**Figure 11.1 Cervical dermatomes derived by symptom provocation**




Source: Adapted from Slipman *et al.* 1998

## Examination of movement

It is important that movement testing is done from a standardised start position that allows proper evaluation of movement. Movement whilst in a slouched sitting posture, for instance, is limited and may be uncomfortable, and so does not present a true picture of someone's movement ability. Failure to correct the starting position can cause incorrect conclusions regarding the presenting symptoms. Equally, we want the patient to be in a position that is easy to replicate on the next occasion when we test these movements. Getting the patient to sit in a chair with a back support, 'with your bottom to the back of the chair, and sit upright', will fulfil these criteria as long as these instructions are used on every occasion.

Single movements provide an inadequate mechanical evaluation by themselves and rarely provide enough information to decide on the correct loading strategy. Most importantly, in terms of management we wish to know the effects of repeated movements and any lasting changes that result in the patient's symptoms. Only when movements are repeated do symptom responses become clear, and this testing is discussed below. The initial single movements are important in providing some baseline data about the patient's ability to move. From these we can determine the range and quality of movement and pain response to movement. This information can be compared with range of movement on subsequent occasions to help determine improvement or deterioration in the mechanical presentation, and thus the appropriateness of the management strategies being applied.

If pain is present with the movement, we may also wish to know if the pain is present *during* the movement or at *end-range*. 'Do you have pain as you move, or at the end of movement?' However, this is not easy to analyse either for the therapist or the patient; it is probably more important to note there is pain with a certain movement rather than trying to determine if this is mid- or end-range. If movement is less than expected, we also wish to know if it is limited by pain or by stiffness – the patient may simply be unable to move further, but it is not pain that prevents the movement.

Movements are examined in the following order:

### Protrusion

The patient is instructed to 'extend your chin forward as far as you can, and then return to the neutral position'. Any loss of range of movement is gauged as major, moderate or minor, and any pain with the movement is noted.

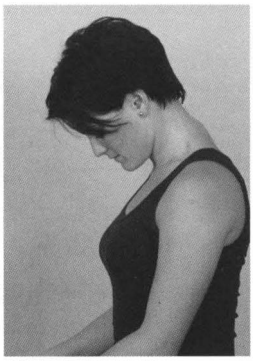
*Photo 4: Protrusion - testing range of movement.*

*4*

### Flexion

The patient is instructed to 'bend your head down and put your chin on your chest, and then return to the neutral position'. Any loss of range of movement is gauged as major, moderate or minor, and any pain with the movement is noted. More specifically, if there is a loss of movement and they are unable to put chin to chest, this can be recorded as distance or the number of clinician's fingers between chin and sternum.

*Photo 5: Flexion - testing range of movement.*

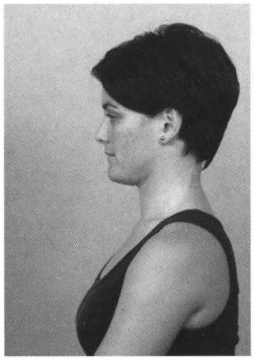


*5*

### Retraction

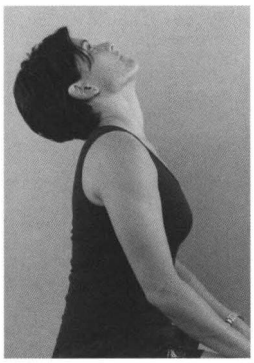
The patient is instructed to 'draw your head backwards as you tuck your chin in, and then return to the neutral position'. Many patients have problems achieving this important movement. There are a number of ways that clinicians can provide input to facilitate this movement if patients are having difficulty achieving it (see Chapter 14). Any loss of range of movement is gauged as major, moderate or minor, and any pain with the movement is noted.

*Photo 6: Retraction - testing range of movement.*



*6*

*Photo 7: Extension - testing range of movement.*



7

### Extension

The patient is instructed to 'bend your head backwards as far as you can to look at the ceiling, and then return to the neutral position'. Any loss of range of movement is gauged as major, moderate or minor, and any pain with the movement is noted.

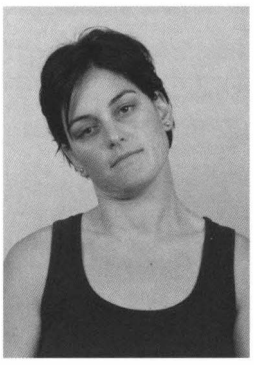
*Photo 8: Rotation - testing range of movement.*

8

### Rotation (right and left)

The patient is instructed to 'look over your right (left) shoulder as far as you can, and then return to the neutral position'. Any loss of range of movement is gauged as major, moderate or minor, and any pain with the movement is noted. Movement to both sides will have to be examined before defining loss of range.

*Photo 9: Lateral flexion - testing range of movement.*



9

### Lateral flexion (right and left)

The patient is instructed to 'take your right (left) ear down towards your right (left) shoulder'. Any loss of range of movement is gauged as major, moderate or minor, and any pain with the movement is noted. Movement to both sides will have to be examined before defining loss of range.

## Repeated movements

The repeated movement part of the physical examination provides the most useful information on symptom response and is the ultimate guide to the management strategy to be applied (McKenzie 1981, 1990). A decrease, abolition or centralisation of pain is a reliable indicator of which movement should be chosen to reduce mechanical deformation. An increase or peripheralisation of pain is just as reliable to indicate which movements should be avoided. This, the cumulative effect of the movement, provides the most important detail concerning the patient's symptomatic response – that is whether they are worse, no worse, better, no better, or the pain has centralised or peripheralised. These responses thus provide the clearest indication for the appropriate management strategy. Sometimes we must also record 'no effect' if at no point during the test procedure there was any change in symptoms.

Only with repeated movement is the paradoxical nature of movement revealed. Whilst a single movement may produce or increase pain, the same movement repeated can bring about an abolition, decrease or centralisation of pain and an increase in range. Repeated movements allow differentiation between the three mechanical syndromes, and also clarify the mechanically determined directional preference of those in the derangement syndrome. Apart from exposing mechanical syndromes, repeated movements are also essential in determining the appropriate timing for restoration of function following trauma in all musculoskeletal problems. *When repeated movements produce less and less pain with each repetition, or produce greater range, these responses indicate the appropriateness of this loading strategy. On the other hand, when more and more pain is experienced with each repetition, that particular exercise is premature or inappropriate. This fundamental response of pain-sensitive musculoskeletal structures to the introduction of loading strategies is a key determinant of the management to be applied.*

Standardised terms should be used to describe the symptomatic response to repeated movements. These are listed in the glossary, and their use is discussed in more detail in Chapter 12. The symptomatic response is recorded three times during the assessment: before movement testing, during the testing, and, most importantly, a few minutes after the test movements. When judging the patient's symptom response, it is essential to know their baseline pain status,

especially the distal extent of any arm pain. 'Sitting there now, are you feeling any of the symptoms you have mentioned? Where is the pain in your neck? If you have pain in your arm, how far down the arm does it extend?'

During the repeated movements the patient may be questioned about symptom behaviour, but keep questions neutral. For instance, 'Is the pain changing?' 'What is happening to the pain in your forearm/arm/shoulder blade?' Avoid leading questions such as, 'Is the pain decreasing?' The response during the movement is not as important as how the patient reports their symptoms after testing, but may be important for monitoring adverse responses in those with distal symptoms.

On completion of ten to fifteen movements the patient is told to relax, and after a minute or two they are questioned again about their symptom status. At this point we wish to know if the area or the severity of symptoms has changed, or if pain that was present before has been abolished, or if pain that was not present before has been produced. Afterwards the patient is 'better', 'no better', 'worse', 'no worse' or 'no effect'. If a patient reports no pain prior to testing, but an improvement in mechanical or symptomatic response occurs during repeated movements, this should also be recorded as 'better' afterwards, and the details noted.

If there is no change during or immediately after the test movements, the joints may not have been stressed adequately and the process may have to be repeated more vigorously. However, it may also be that the pain is not of mechanical origin, because mechanical pain is always affected by movement or position if sufficiently applied. Alternatively, the cervical spine is not causing the problem and other areas should be investigated.

All patients should perform some of the repeated test movements in the sitting position as outlined below, but ***not all test movements will be needed for all patients***. Once a favourable mechanical response is forthcoming or a provisional mechanical diagnosis is made, ***further testing is redundant and unnecessary***. Patients with severe and acute derangements, especially those with a postural deformity, may not be able to tolerate testing in a loaded posture. In such instances, a brief examination in the unloaded position should be conducted.

### Repeated movements in derangement syndrome

In derangement syndrome repeated movements in the direction that produces greater deformation of spinal structures will produce, increase or peripheralise the symptoms, and in addition may frequently cause an obstruction to movement. The performance of movements in the opposite direction will reduce deformation of those structures, cause reduction in the derangement and bring about an abolition, decrease, or centralisation of symptoms. In this syndrome movement is usually impaired, but performance of the appropriate repeated movement may bring about a recovery of all movements, not just the one being repeated. Thus repeated movements are diagnostic of the derangement syndrome as well as confirming the mechanically determined directional preference of the management strategy, to which clues will have been provided in the interview.

Once a repeated movement has been found that decreases, abolishes or centralises symptoms, and/or improves the mechanical presentation, no further testing is necessary and that movement is used in the management strategy.

Very often a definite symptomatic and mechanical response is apparent on the occasion of the first assessment, and the history-taking and physical examination produce a consistent picture. At times the initial response to repeated movements is more equivocal, and after several batches of repeated movement there is no change. In such instances repeated movements should be explored vigorously over the subsequent day(s) and other force progressions considered until elucidation of mechanically determined directional preference is produced.

### Repeated movements in dysfunction syndrome

In articular dysfunction syndrome repeated movements in the direction that puts tension on adaptively shortened structures produces end-range pain on every occasion it is performed. Alternatively, repeated compression of structurally impaired tissue could consistently re-produce the patient's symptoms at end-range. Repeated movements will not make the patient progressively worse; when they return to the neutral position the pain will abate, and neither will pain be peripheralised. On every subsequent occasion the same exercise will generate the same response. There will be no rapid change in range of movement. Thus, repeated movements are diagnostic of dysfunction

syndrome and also reveal the movement that requires repetition to remodel adaptively shortened tissues.

Derangements may also present with end-range pain, but it is usually an end-range pain that changes quickly. Derangement is always managed first, even if a dysfunction is present also. Initially it is not possible to know if there is also an underlying dysfunction. Although this may become apparent after the derangement is reduced, very often this is not the case and the end-range pain was entirely as a result of the derangement.

### Repeated movements in postural syndrome

Patients with posture syndrome will experience no pain on any test movements or their repetition, and they will display no loss of normal range of movement. Only with sustained positioning will these patients experience their pain.

### Selecting repeated movements

Frequently the movements that have the greatest effect on pain are flexion, retraction and extension. For this reason the repeated movement testing initially only uses sagittal plane movements. Except in the case of a lateral deviation, where frontal plane movements are necessary and sagittal plane movements are initially undesirable, sagittal plane movements are always explored first. Frontal plane movements are introduced if sagittal plane movements worsen or peripheralise symptoms. Lateral forces may also be introduced if sagittal plane forces do not improve the symptomatic or mechanical presentation.

Different effects are produced when the test movements are performed in sitting compared with lying. Performing the movements in sitting is functionally easier and much more straightforward for the patient to replicate in their home or workplace. Furthermore, usually movements in sitting are effective in altering the symptoms. Only occasionally, if further symptom response testing is sought, is it necessary to test out the patient's response to the movements in the unloaded posture. Patients with severe and acute derangements, especially those with a postural deformity, may not be able to tolerate testing in a loaded posture. In such instances a brief examination in the unloaded position should be conducted.

Most test movements are done by the patient actively performing the movement, sometimes with the inclusion of added overpressure by

the patient. Only if this force is inadequate to gain sufficient understanding of the mechanical response are clinician's forces used. These are described here also, but are only sometimes necessary.

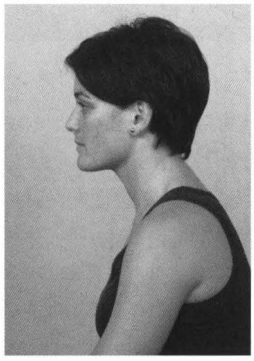
*Once a repeated movement centralises, abolishes or decreases pain, or produces an increase in the range of movement, and thus it is apparent that the mechanically determined directional preference has been determined, further testing is unnecessary.*

All potential repeated movements are described below; however, *it is certainly not intended that all movements should be performed at each initial physical examination.* Most examinations will comprise loaded sagittal plane movements only; if both these and unloaded sagittal plane movements fail to produce a clear symptomatic or mechanical response, two choices are available. If there are clues in the history that sagittal plane is the appropriate direction of movement then overpressure and mobilisation could be included, or the patient could perform repeated movements over the following two or three days. Alternatively, if there are suggestions that frontal plane movements might be appropriate, these could start to be explored at the initial examination. However, beware of abandoning the sagittal plane without full exploration of its potential; it should be remembered that multiple repeat movement tests may actually confuse further rather than clarify. The issue of exploring the lateral component is explored in more detail in Chapter 19.

## Repeated test movements

### Protrusion (sitting)

*Photo 10: Protrusion.*



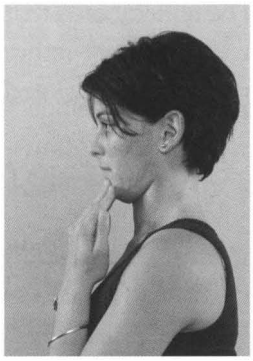
*10*

The patient is reminded to remain sitting upright with their bottom to the back of the chair. Prior to test movements the patient is asked to report the location of any present pain, especially the most distal. The patient is then instructed to extend their chin as far forward as possible, so the neck is outstretched, with the head remaining horizontal and not inclining up or down. Then they return to the neutral sitting position, and the effect this has on their symptoms is recorded. The patient is then instructed to repeat the movement ten to fifteen times, with the maximum movement being achieved in the last few move-

ments. During the repeated movements the patient may be asked, 'Do the movements affect your symptoms? Does it make them better or worse?' Most importantly the pain status, especially the most distal site, is recorded a minute or two *after* the completion of the repeated movements. Note is also made of any changes in range that may have accompanied the movements.

*Photo 11, 12: Retraction (11); retraction with patient overpressure (12).*

*11*



*12*

### Retraction (sitting)

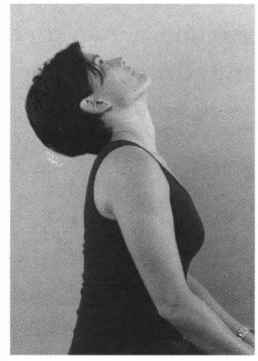
The patient remains sitting upright as far to the back of the chair as possible. Prior to test movements the patient is asked to report the location of any pain currently present, especially the most distal. The patient is then instructed to draw their head as far back as possible, with the head remaining horizontal, facing forward, and not inclining up or down. Then they return to the neutral sitting position, and the effect this has on their symptoms is recorded. The patient is then instructed to repeat the movement ten to fifteen times. The patient is instructed to move as far as they can, and further with each movement, with the maximum movement being achieved. During the repeated movements the patient is asked, 'Do the movements affect your symptoms?' Most importantly the pain status, especially the most distal site, is recorded a minute or two *after* the completion of the repeated movements. Note is also made of any changes in range that may have accompanied the movements. It is quite common that in a movement that was initially limited the range is increased or less painful to perform.

If retraction performed thus has had no clear effect on symptoms, the patient is next instructed in how to apply overpressure. This is achieved by pressing the chin with their fingers at the end of range of the movement. Again, this is repeated ten to fifteen times, and the patient may be asked, 'As a result of performing these movements, is the site of pain changing, or are you having more or less pain than before?' Most importantly, the pain status, especially the most distal site, is recorded a minute or two *after* the completion of the

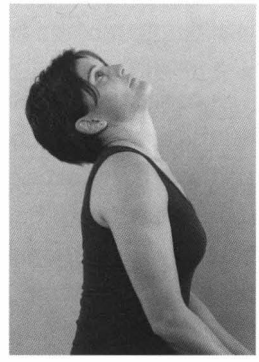
repeated movements, and any change to the mechanical presentation is recorded. If still there is no clear symptomatic response, then clinician overpressure may be used, again as a repeated movement. Again symptom response during and afterwards is monitored.

### Retraction and extension (sitting)

*Photos 13, 14: Retraction/extension (13); retraction/extension with patient overpressure (14).*



*13*



*14*

The patient remains sitting upright with their bottom to the back of the chair. Prior to test movements the patient is asked to report the location of any present pain, especially the most distal. This test movement is a combination of retraction followed by extension, but although they are two movements, they should be performed fluidly as one. The patient is instructed to draw their head as far back as possible, with the head remaining horizontal, facing forward, and not inclining up or down, as they have just done, but now slowly tipping the head backwards as far as is possible or as far as they can tolerate. They then return to the neutral sitting position, and the effect this has on their symptoms is recorded. The patient is then instructed to repeat the movements ten to fifteen times. The patient is instructed to move as far as they can, and further with each movement, with the maximum movement being achieved During the repeated movements the patient may be asked, 'Do the movements affect your symptoms?' Most importantly the pain status, especially the most distal site, is recorded a minute or two *after* the completion of the repeated movements. Note is also made of any changes in range that may have accompanied the movements. It is quite common that in a movement that was initially limited the range is increased or less painful to do.

Should retraction and extension performed thus have no effect on the patient's symptoms, an additional movement can be performed that adds in overpressure at end-range extension. Within the precept of 'minimal force necessary', no additional pressure is necessary if symptom or mechanical presentation has begun to demonstrate

change. For the overpressure, the patient performs the retraction and extension components as described above. When at end-range extension they are instructed to 'Rotate your head from side to side a few times so your nose moves about half an inch either side of the midline. As you do this let your head relax, so that it moves further into extension.' This is repeated five to ten times, and the patient is asked, 'As a result of performing these movements, is the site of pain changing, or are you having more or less pain than before?' Most importantly, the pain status, especially the most distal site, is recorded a minute or two *after* the completion of the repeated movements, and any change to the mechanical presentation is recorded.

**Retraction and extension (lying)**

This position is not commonly used, but may become necessary if the patient cannot tolerate the loaded test positions above (Chapter 14, Procedures 1, 1a, 2, and 2a). This may occur in those with acute derangements with severe symptoms and possibly accompanied by acute deformity. It can also be used if none of the above has any effect on symptoms. In the unloaded position there is less compression acting through the cervical joints, and the movement into extension is accompanied by an element of traction.

The patient lies supine on the treatment table with their head resting on the end. It may be useful to test out their response to retraction and retraction with patient overpressure before continuing, as these movements may be easier to do when unloaded compared with loaded. The patient is then instructed to bend one knee and use the foot to push themselves to the end of the treatment table, at the same time supporting their head with a hand under the occiput. The patient's head and neck should overhang the treatment table to about the level of T3 or T4. First the patient repeats retraction several times, after which they fully extend the head. During this movement they must let go of the head so it is left hanging relaxed, thus attaining maximum range of movement. The effect on the patient's symptoms of performing one movement is noted. As the patient returns to the neutral position, they can use their hand again to support their head. This helps to ensure that they do not pull the head into protrusion as they return to neutral.

The sequence of movements is repeated five times in a rhythmical fashion, during which the effects on symptoms and range are noted. After completion of a cycle of movements, the patient is instructed

to shuffle back down the treatment table and rest their head. The pain status, especially the most distal site, is recorded a minute or two *after* the completion of the repeated movements. If the response is positive, a further set of repetitions may be done. If there is still no clear response in either the symptomatic or mechanical presentation, the patient can apply the additional rotary movement at end-range extension as described in retraction and extension in sitting. As always, changes in symptoms or range are recorded.

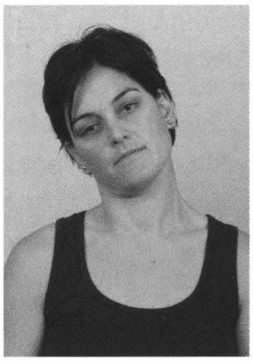
## Exploring frontal plane movements

A large proportion of patients will respond to sagittal plane movements, even in the presence of unilateral or asymmetrical symptoms. Mostly sagittal plane forces achieve symptom and mechanical change more rapidly and effectively than frontal plane movements.

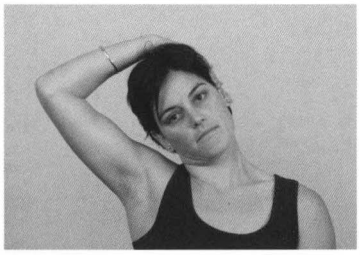
However, in the event that there has been no conclusive symptomatic or mechanical response from the sagittal plane tests outlined above, then it becomes necessary to explore the response to movements in other planes. If after several sets of repeated sagittal plane movements, including overpressure, the clinical presentation is unchanged, the lateral component is explored. Equally, if at any point there is a worsening of symptoms in response to sagittal plane forces in both loaded *and unloaded* positions, the lateral component is also explored. This is most effectively done using lateral flexion or rotation forces.

### Lateral flexion (sitting)

*Photos 15, 16: Lateral flexion (15); lateral flexion with patient overpressure (16).*



*15*



*16*

The patient is sitting upright as far back into the seat of the chair as possible. Prior to test movements, the patient is asked to report the location of any present pain, especially the most distal. The patient is then instructed to retract the head to a neutral position, and then lateral flex *towards the side of pain:* 'Take your right/left ear towards your shoulder'. After a second in that position they are instructed to return to the neutral posture. The effects of one movement on the symptoms are noted. The same movements are then repeated rhythmically ten to fifteen times, returning to the neutral position each time. The patient is instructed to move as far as they can, and

further with each movement, with the maximum movement being achieved. During the repeated movements the patient may be asked, 'Do the movements affect your symptoms?' Most importantly, the pain status, especially the most distal site, is recorded a minute or two *after* the completion of the repeated movements. Note is also made of any changes in range that may have accompanied the movements.

If following repetition there is no conclusive symptomatic or mechanical response, then overpressure is added. The patient is instructed to repeat the same movements of some retraction followed by active lateral flexion. Then they are instructed and shown, if performing left lateral flexion, 'Place your left hand over your head with your fingers reaching to your right ear, and pull your head down to your shoulder'. After a second in that position they are instructed to return to the upright posture. The effects of one movement on the symptoms are noted. The same movements are then repeated rhythmically ten to fifteen times, returning to the neutral position each time. During the repeated movements the patient may be asked, 'Do the movements affect your symptoms?' Most importantly, the pain status, especially the most distal site, is recorded a minute or two *after* the completion of the repeated movements.

If following repetition there is still no conclusive symptomatic or mechanical response, or the patient is unable to provide adequate pressure, then clinician overpressure is added.

#### Rotation (sitting)

The patient is sitting upright with their bottom to the back of the chair. Prior to test movements, the patient is asked to report the location of any present pain, especially the most distal. The patient is then instructed to retract the head, but not fully, and then rotate the head *towards the side of pain:* 'Turn as far as you can as if looking over your right/left shoulder'. After a second in that position they are instructed to return to the neutral posture. The effects of one movement on the symptoms are noted. The same movements are then repeated rhythmically ten to fifteen times, returning to the neutral position each time. The patient is instructed to move as far as they can, and further with each movement, with the maximum movement being achieved. During the repeated movements the patient may be asked, 'Do the movements affect your symptoms?' Most importantly, the pain status, especially the most distal site, is recorded a minute or two *after* the completion of the repeated movements. Note

is also made of any changes in range that may have accompanied the movements.

*Photos 17, 18: Rotation (17); rotation with patient overpressure (18).*

*17*

*18*

If following repetition there is no conclusive symptomatic or mechanical response, then overpressure is added. The patient is instructed to repeat the same movements of some retraction followed by active rotation. Then they are instructed and shown the following, if performing left rotation: 'Place your right hand behind your head with your fingers reaching to your left ear, and your left hand against your chin; apply extra pressure so your head is pushed further into rotation.' After a second in that position they are instructed to return to the neutral posture. The effects of one movement on the symptoms are noted. The same movements are then repeated rhythmically ten to fifteen times, returning to the neutral position each time. During the repeated movements the patient may be asked, 'Do the movements affect your symptoms?' Most importantly, the pain status, especially the most distal site, is recorded a minute or two *after* the completion of the repeated movements.

If following repetition there is still no conclusive symptomatic or mechanical response, or the patient is unable to provide adequate pressure, then clinician overpressure is added.

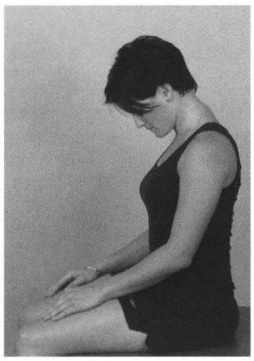
### Flexion (sitting)

Flexion testing is not routinely used in the cervical spine, but may be necessary if previous test movements have failed to produce a conclusive symptomatic or mechanical response.

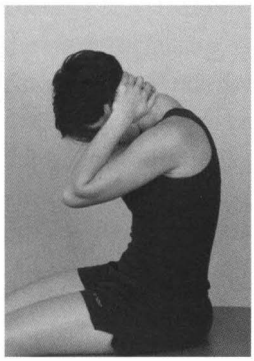
The patient is instructed to sit slouched with the spine flexed. Prior to test movements, the patient is asked to report the location of any present pain, especially the most distal. The patient is then instructed to bend the head forward so the chin touches the sternum. After a second in that position they are instructed to return to the neutral

posture. The effect of one movement on the symptoms is noted. The same movement is then repeated rhythmically ten to fifteen times, returning to the neutral position each time. The patient is instructed to move as far as they can, and further with each movement, with the maximum movement being achieved. During the repeated movements the patient may be asked, 'Do the movements affect your symptoms?' Most importantly, the pain status, especially the most distal site, is recorded a minute or two *after* the completion of the repeated movements. Note is also made of any changes in range that may have accompanied the movements.

*Photos 19, 20: Flexion (19); flexion with patient overpressure (20).*



*19*



*20*

If following repetition there is no conclusive symptomatic or mechanical response, then overpressure is added. The patient is instructed to bend the head forward so their chin touches the sternum, then apply overpressure by interlocking the hands around the back of the head. After a second in that position they are instructed to return to the neutral posture. The effects of one movement on the symptoms are noted. The same movements are then repeated rhythmically ten to fifteen times, returning to the neutral position each time. During the repeated movements the patient may be asked, 'Do the movements affect your symptoms?' Most importantly, the pain status, especially the most distal site, is recorded a minute or two *after* the completion of the repeated movements.

## Static mechanical evaluation

The dynamic or repeated mechanical evaluation outlined above will on most occasions reveal at least one movement that has a significant effect on the symptomatic or mechanical presentation and provide an initial management strategy. In some cases where the identification of mechanically determined directional preference remains elusive, static tests or sustained postures need to be explored, either to provoke obscure symptoms or to decrease persistent symptoms.

A clue for the need to apply static tests is when the patient recounts in their history that sustained postures rather than single or repeated movements provoke symptoms, and that they are better when on the move and worse when still. If repeated movements fail to provoke symptoms, static tests may expose the true nature of the problem. This is especially likely in patients with postural syndrome or patients with intermittent pain from a derangement.

The effect of prolonged relaxed sitting followed by posture correction after the history-taking should be done routinely and is described above. Other static tests are used as related to the history and physical examination. Pre-existing symptoms, especially the most distal, should be noted and then monitored during the sustained loading. Positions can be maintained for up to five minutes, when the patient returns to the neutral position, and again reports symptom intensity and location. Static mechanical evaluation can be conducted in the following postures:

- sitting slouched, head protruded
- sitting upright, head retracted
- retraction and extension in supine lying
- extension in prone lying.

## Testing inconclusive

If testing so far has not produced any conclusive symptomatic or mechanical changes, certain procedures may help to clarify mechanically determined directional preference over the initial and following assessments. If the patient and therapist, where appropriate, have not applied overpressures, these should be applied. If sagittal plane testing has failed to generate any positive responses, frontal plane movements should be tested. Appropriate mobilisation could be applied to help facilitate the process.

If there is still lack of conclusive symptomatic or mechanical change, then the mechanical evaluation should be continued over the next day(s) with the patient performing a specific loading strategy at home. This may be done using sagittal or frontal plane forces depending on any clues gained during the assessment. Otherwise it is more logical to start in the sagittal plane and only introduce frontal movements if this fails to bring about change. Patients should be told about the

expected response and warned about peripheralisation. Often the repetition of repeated movements over several days allows elucidation of the appropriate loading strategy. The presence of a mechanical syndrome should be determined within five treatment sessions; mostly it will be much sooner.

## Other examination procedures

It is generally unnecessary to add further examination procedures than those outlined above. It should be remembered that once a positive symptomatic or mechanical response is gained, further testing is unnecessary at that point. If test movements have so far failed to generate a conclusive symptomatic or mechanical change, there is no guarantee that extra tests will do so – in fact, they are more likely to generate confusion.

Palpation adds very little to a mechanical evaluation and is rarely needed. This is commonly used by chiropractors, osteopaths and manual therapists to purportedly detect manipulative lesions, determine the segmental level, detect hypo- or hypermobility at each segment, or to detect asymmetry. The literature has failed to demonstrate that different clinicians can reliably agree on any of these findings. The relevant literature is summarised elsewhere. Inter-practitioner agreement on the presence of a finding actually constitutes a test of internal validity and is not simply a measurement of reliability (Nansel *et al.* 1989). Poor rates of intertester reliability mean that the existence of such phenomena is unsubstantiated, and therefore these are not valid clinical tests. Furthermore, even if these areas of hypomobility do exist, there is lack of evidence to demonstrate that they are symptom-generators as opposed to normal findings amongst the general population.

Sometimes if testing of the cervical spine has been inconclusive and it is suspected that the pain originates from another site, such as the shoulder or thoracic spine, these sites may require a more detailed examination.

## Mechanical syndromes

### Derangement

This will be the conclusion in the majority of patients; for instance, in a sample of seventy-eight neck pain patients, sixty-two (79%) were

classified as derangement (May 2004a). Once it is determined that a derangement is present, the key management decision concerns the mechanically determined directional preference. The movement that centralised, decreased or abolished the symptoms during the examination is the one chosen for the patient to perform. The movements that the patient reported to aggravate or produce their symptoms are those that need to be *temporarily* avoided. The management strategy should evolve out of the findings of the two parts of the assessment. ***In the treatment of derangement, we must choose the movement that relieves the pain, as this movement decreases the mechanical deformation by reducing the derangement.*** Very often the appropriate movement is painful to perform, but becomes easier with repetition and the patient feels better afterwards.

The majority of patients with derangement require the ***extension principle***. This is applied when extension movements centralise, decrease or abolish symptoms, and the opposite movement, flexion, causes a worsening or peripheralising of symptoms. The extension principle includes a variety of procedures, including retraction with patient and clinician overpressure, retraction and extension, and traction, retraction and extension with rotation.

A smaller group of patients with derangement require the ***flexion principle***. This is applied when flexion movements centralise, decrease or abolish symptoms. The procedures of the flexion principle involve flexion in sitting and lying with patient or clinician overpressure, and can involve a lateral component.

Some patients with derangement require the ***lateral principle***. This is applied when either of the above fails to produce change and lateral flexion or rotation forces cause abolition, centralisation or decrease in symptoms. The procedures of the lateral principle involve lateral flexion and rotation in loaded and unloaded positions, possibly with patient overpressures or clinician mobilisations.

The response of some derangements to *all* mechanical testing is an increase or worsening of symptoms. The working hypothesis in such a case is an irreducible derangement. Before this poor prognosis is conveyed to the patient, a period of mechanical testing and re-evaluation is worthwhile. However, once it is clear that there are no mechanical loading strategies that will decrease, abolish or centralise symptoms, the patient should be advised of the situation. Further

investigation may be necessary; the focus of therapy should change to functional rehabilitation, but certainly a lengthy period of ineffective treatment should not be undertaken.

### Dysfunction

A much smaller number of patients will be placed in the dysfunction category. Again, once it is decided that the patient has a dysfunction, the appropriate direction of movement must be selected. *In dysfunction syndrome the movement chosen is the one that consistently produces the patient's pain, as this movement will gradually remodel the structural impairment.* The movement chosen will reproduce the symptoms on each occasion, but these abate shortly after the movement ceases.

Patients with cervical dysfunction syndrome use a range of treatment principles, multi-directional dysfunctions are not uncommon, and adherent nerve root dysfunctions also occur.

### Postural

Very rarely will patients with pain of purely postural origin present at the clinic. When they do, these patients simply require postural correction and education. However, poor posture is a perpetuating factor in all three syndromes, and most patients have pain to some extent from this cause.

## Inconclusive

Not every patient will display an obvious mechanical response to reductive or provocative loading strategies on the first assessment. Especially if symptoms have been present for some time, a more extended period of mechanical testing may be necessary. As long as baseline measures have been gathered against which to judge change, if the initial response is equivocal it is very often worthwhile to encourage the patients to test out a particular provocative or reductive loading strategy over the next day(s). Multiple sessions of repeated movements performed over several days often provide more definitive information than one or two sessions in the clinic. Within the history and mechanical presentation, there are often clues as to mechanically determined directional preference that become clearer once explored more fully over time.

Even if a non-mechanical syndrome is suspected, it is generally unwise to embark on additional pain provocation testing on the initial visit,

as this often produces false-positive responses. The full diagnostic process should be completed within five clinic sessions, but very often is completed much more quickly than this.

## Conclusions

Having listened to the information provided by the patient about the history of their problem and conducted an appropriate and thorough physical examination, including the relevant repeated movements, certain conclusions can now be drawn.

At this point serious spinal pathology should be excluded from the equation. The patients should have simple mechanical neck pain with or without nerve root symptoms. Mechanically, we wish to know if the patient has a derangement, a dysfunction or a postural problem. The majority of patients will have derangements, a minority dysfunctions, and a few will have postural syndrome. With some patients mechanical testing may be inconclusive, and may need to be continued over a few days to reach a definitive diagnosis or to determine a non-mechanical source of symptoms. If it is determined that a mechanical syndrome is present, then one of the principles of mechanical therapy are selected for the management strategy.

# 12: Evaluation of Clinical Presentations

## Introduction

During the first assessment, data is gathered about the patient's pain and the impact that this is having on their function and normal activity. During the history-taking and physical examination, baseline measures are collected on the symptomatic and mechanical presentations. On all subsequent occasions, clinicians must be evaluating the effect of the management strategies being used against these baseline measures. This evaluation needs to address both the pain, which is frequently the patient's main complaint, and the impaired function. These two elements should be assessed on each occasion and will generally improve or worsen in parallel – in other words, as the pain eases, the function returns to normal. Depending on the effect of the management strategies on the symptomatic and mechanical presentations, these should be continued, abandoned or supplemented with force progressions, as appropriate.

This chapter considers some of the aspects that are relevant to assessment of symptomatic and mechanical presentations, which are the factors that are involved in evaluation and re-evaluation of clinical presentations.

Sections in this chapter are as follows:

- symptomatic presentation
    - site
    - frequency of symptoms
    - severity
    - paraesthesia
    - consumption of analgesics and NSAIDs
    - pain on movement
- assessment of symptomatic response
- use of symptom response to guide loading strategy
- mechanical presentation
- assessment of the mechanical presentation

  - range of movement
  - deformity
  - kyphotic deformity
  - lateral deviation
  - quality of movement
  - functional disability
- use of mechanical response to guide loading strategy
- symptomatic and mechanical presentations to identify mechanical syndromes
- identifying responders
- chronic pain – interpretation of symptomatic responses.

## Symptomatic presentation

Pain is usually the main complaint of patients with musculoskeletal problems, although paraesthesia, numbness or weakness may also be relevant. Pain as an outcome measure is criticised as 'soft' data, which lacks objectivity. However, whilst pain is by its very nature a subjective experience, it can be recorded and assessed in a reliable way, especially when using serial measurements of pain taken from a single individual (Sim and Waterfield 1997). 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. The symptomatic presentation has various dimensions by which changes can be assessed.

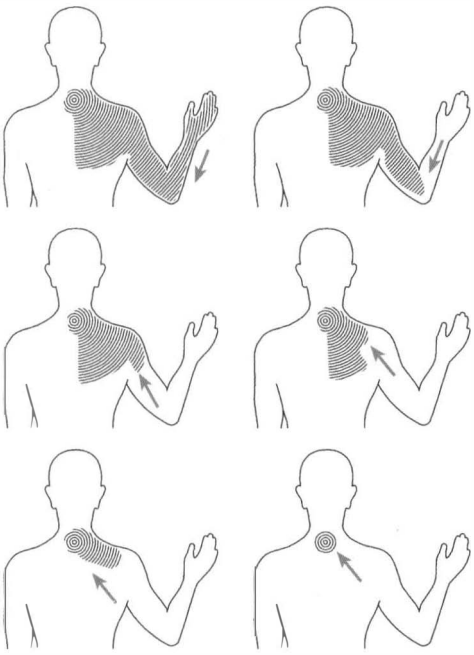
**Table 12.1 Dimensions of symptomatic presentation to monitor progress**

- site of pain
- constant or intermittent
- severity
- paraesthesia
- number of analgesics and non-steroidal anti-inflammatory drugs (NSAIDs)
- pain on movement.

### Site

Pain of spinal origin may centralise or peripheralise (McKenzie 1981, 1990; Donelson *et al.* 1990, 1991); this phenomenon is discussed at length elsewhere. In essence, the further the pain spreads into the limb, the worse the presentation. If the area of symptoms can be reduced or moved further up the arm, this is an improvement. Change in pain site is one of the most important factors used in establishing mechanically determined directional preference, and thus the management strategy to be implemented (Figure 12.1). Just as centralisation offers a good prognosis and is positively sought, its opposite, peripheralisation, should be avoided. It is apparent that not only peripheralisation but also non-centralisation, that is failure to alter the site of the symptoms, is also associated with a poor prognosis (Werneke *et al.* 1999).

**Figure 12.1 Centralisation of distal pain in response to repeated movements**



### Frequency of symptoms

A patient may complain of constant pain. If this subsequently becomes intermittent, an improvement has been made. If an intermittent pain, which is present for most of the day, is reduced to being present only 20% of the day, this also is an improvement. The patient may report

that scapular/neck pain is constant and that arm pain is intermittent. These details should be recorded on the assessment form.

### Severity

Intensity of pain can be assessed in various ways. This can be done formally using a Visual Analogue Scale, with zero representing 'no pain' and ten 'the worst pain imaginable'. They can be asked the same question at a later date for comparison. They can be asked whether their symptoms are severe, moderate or mild. Alternatively, at re-assessment patients can be asked how their present symptoms compare to when they first attended. 'If you had 100 units of pain when we commenced treatment, how many do you have now?' Some patients even volunteer that they are '80% better'.

### Paraesthesia

Patients with cervical radiculopathy may also present with a sensation of tingling, pins and needles, or numbness in the hand. The presence of these symptoms should always be enquired into if the patient presents with pain into the arm or forearm. In such patients a full neurological examination should be conducted, involving appropriate dermatomal, myotomal and reflex testing.

Trauma to the nerve root-dorsal ganglion-spinal nerve complex can cause demyelination or axonal degeneration leading to changes in nerve function. This can be experienced as muscle weakness, sensory deficit or hyperexcitability of the nerve tissue, causing pain (Rydevik *et al.* 1984). Radicular pain is the most common symptom of nerve root interference; sensory deficit and muscle weakness are variable findings. Vague feelings of tingling or sensory impairment can sometimes be reported anywhere in the arm, but marked nerve root interference is usually denoted by a sensory loss in the distal part of the dermatome. Most commonly C6, C7 and C8 are affected, causing loss of sensation in the thumb, middle fingers and little finger respectively. Less commonly, C5 and C4 are involved, affecting the lateral arm and the shoulder respectively (Slipman *et al.* 1998; Butler 2000).

Such symptoms do not respond so directly as pain to repeated movements. There is no 'centralisation' of paraesthesia. Although radicular pain may be centralising or resolving, improvements in sensory deficit usually take place less rapidly, and sometimes not at all. Improvement may occur in one of the ways listed below.

**Table 12.2 Criteria by which paraesthesia may be improving**

- numbness may become more of a 'tingling' feeling
- severity of the numbness may lessen
- constancy of the paraesthesia may lessen
- area of paraesthesia may diminish.

It is important to ensure that there is no worsening of such symptoms with mechanical therapy, which may be judged by the opposite of the above criteria or the onset of sensory deficit.

At times pain will have more or less resolved and the individual can still be left with an area of reduced sensation. If this starts to improve, either lessening in severity, constancy or size, continuing improvements are likely. However, when there is no early easing of these symptoms, recovery is less likely; some individuals are left with a patch of numbness that never improves. They should be reassured that this does occur, but is nothing to worry about.

### Consumption of analgesics and NSAIDs

The number of tablets being taken daily at the beginning of the episode should be recorded and compared with consumption later.

### Pain on movement

Even if movement appears to be full-range, pain may be felt during the movement or at end-range. An improvement is made if the patient reports that over time the difficulty of performing the movement and the degree of discomfort it engenders has lessened, or if there has been an increase in the range of pain-free movement. When other responses are equivocal and a management strategy is unclear, a painful movement is sometimes the only variable that can be assessed to establish mechanically determined directional preference.

Although very often pain responses are a useful determinant of appropriate mechanical therapy, it should also be borne in mind that excessive attention on the pain can heighten the pain response (Arntz *et al.* 1991). By contrast, use of distraction techniques can minimise the pain (Klaber Moffett and Richardson 1995). With some patients the focus should be on function rather than pain; this is especially relevant in patients with chronic pain states.

## Assessment of symptomatic response

Standardised terms (McKenzie 1981; van Wijmen 1994) are used to evaluate the patient's pain responses during mechanical testing (see glossary for summary of terms). The pain status is established before, during and after test procedures. If we wish to compare the effect of movements on the symptoms, *it is vital we know the pain status prior to testing* – it is too late to establish this once the patient has started to do the movements. We are most interested in the effect of the movements a minute or so after testing. The significant response, which helps determine the treatment strategy, is not to a single movement. Response to repeated movements often reveals the paradoxical nature of pain and movement. Whilst a single movement may produce or increase pain, the accumulated and lasting effect of repeating that movement may be to reduce the overall pain. Thus, the key symptomatic response is based on the effects of repeated movements and not on the effects of a single movement.

At baseline the patient is either with or without pain. During the test movements this can be *increased, decreased, abolished, produced* or the movements have *no effect*. Also during the test movements symptoms may show signs of *centralising* or *peripheralising*. The choice of terms to describe responses must wait until the completion of perhaps two or three series of ten movements. The final decision should be made after the patient has had time to assess the effects. It is best to let the patient sit still for a minute after the test movements prior to asking, 'What symptoms do you have now?'

Different terms are used to describe any changes that persist after the completion of one or more series of repeated movements. At this point pain, which was increased or produced by the movements, can either remain *worse* or be *no worse* if it returns to its former state when the movement has ceased, or sometimes it can be *better* afterwards. Pain, which was decreased or abolished during the movements, can either remain *better* or be *no better* when the movement has ceased. If during and after the movements the symptoms remain completely unchanged, they are said to have *no effect*. These terms are listed and defined in the glossary. At first glance they appear very obvious to apply; however, their careful use requires experience, and a thorough understanding of these terms is a pre-requisite of mechanical diagnosis and therapy.

If following test movement the site of pain has changed, symptoms are then reported to be *centralised* or *peripheralised*. If the patient has reported referred pain into the arm, we must know the extent of referral prior to testing. *We should enquire about the most distal point that pain is felt in the arm at that moment*. The movement of pain proximally or distally is a key determinant of mechanically determined directional preference. If after a series of test movements pain that was felt as far as the forearm is now felt only in the arm, then symptoms are in the process of centralising. If pain that was initially felt all across the neck comes to be felt solely in the centre of the neck, again the centralisation process is occurring. However, if pain is made to appear more distally, and further repeated movements increase the distal symptoms, peripheralisation is occurring.

Another favourable symptom response that can occur is production of pain on the first movement, which decreases on repetition. With each repeated movement pain is still felt, but this lessens with each repetition so that by the last movement pain production is minimal or absent. After the test movements the patient returns to being pain-free. Although strictly speaking this 'before and after' response should be recorded as *produced, no worse*, the symptom modification in response to loading strategy is clearly favourable and worth noting. When this response is observed to repeated movements, this should also be recorded afterwards as *better*.

## Use of symptom response to guide loading strategy

Using these standardised terms to define the patient's responses to repeated movements allows us to determine the appropriateness of those particular movements (Table 12.3).

The Traffic Light Guide (on the following page) allows a logical formulation of appropriate mechanical loading strategies based on the patient's symptom responses. If repeated movements abolish the most distal symptoms in the patient's arm or if symptoms give the appearance of moving proximally up the arm, the process of centralisation is occurring and the correct strategy has been selected. Equally, if the patient's symptoms are abolished or reduced after the test movements, the correct movement has been selected and treatment should be continued unaltered. These are both examples of a 'green light' to more of the same procedure. *If the patient is showing*

*improvements with the prescribed management, there is no justification for changing or supplementing it in any way.* In the case of dysfunction, if pain produced by end-range movement ceases afterwards, and this response is consistently produced, this too is a 'green light' for more of the same.

**Table 12.3 Traffic Light Guide to symptom response before, during and after repeated movement testing**

| *Pain status before test* | *Pain response during test* | *Pain response after test* | *Implications (Traffic Light Guide)* |
|---|---|---|---|
| Pain | Increase | Worse | Red |
| | | Not worse | Amber |
| | Decrease | Better | Green |
| | Abolish | Not Better | Amber |
| No Pain | Produce | Worse | Red |
| | | Not worse | Amber/Green (dysfunction) |
| | Produce, better with repetition | No pain | Green |
| Proximal pain | Peripheral pain produced | Worse | Red |
| | | Not Worse | Amber |
| Distal pain | Abolish | Better | Green |
| | Decrease | Not Better | Amber |
| | Increase | Worse | Red |

If peripheral pain is produced by the repeated movements and remains worse afterwards, then the wrong procedure has been applied. If the pain remains worse after test movements then either the direction, speed of movement, or starting position is wrong, and that particular exercise should be modified and, if indicated, stopped. Movements in the opposite direction should be tested or lateral movements need to be fully explored. If movements still aggravate symptoms, non-mechanical problems or an irreducible derangement might be suspected. Movement may have commenced too early during healing and the inflammatory process is being prolonged, the wrong starting position may be being used or the procedure may be too vigorous for the stage of the disorder. Whatever the specific cause, these are indications of a 'red light' to that particular procedure. If everything appears to aggravate the patient's symptoms, they should be spared further testing and reviewed in a week's time.

In the case of the 'amber light', essentially nothing is changed by the test movements. Although they may produce, increase, abolish or decrease symptoms during repeated movements, afterwards the patient reports that they are just the same as they were before the test procedures. In this instance the application of more force is justified to see if the traffic light changes to give a clearer indication of the appropriate direction of therapeutic exercise. Only with an 'amber light' response is it justifiable to use force progressions. More force may be applied by the patient exercising more regularly or with patient overpressure, or by the patient testing the movement over a twenty-four- to forty-eight-hour period. If this does not clarify the response, clinician force becomes necessary.

*Production of pain* and *no worse afterwards* are the expected responses in patients with dysfunction syndrome, and in this instance this response is a 'green light' for that particular exercise. In articular dysfunction if pain is produced on end-range stretch or end-range compression, which ceases on release, that loading strategy should be continued unaltered.

## Mechanical presentation

The mechanical presentation refers to the outward manifestations of the problem that may limit or alter normal movement, posture and function. The mechanical presentation has various dimensions by which it can be assessed; broadly, these address impairment and disability. Impairment refers to an anatomical or physiological abnormality leading to loss of normal bodily ability, such as loss of movement. Disability is the diminished capacity for everyday activities and normal occupation, which is the degree to which the individual is affected by that impairment.

In patients with cervical spine disorders, pain, range of movement, disability and functional limitations have been found to correlate with each other (Hermann and Reese 2001). Although pain, impairment and disability are related to each other, there is not always a close correlation between these different aspects of a clinical presentation. In neck pain patients we cannot measure impairment or disability directly. Instead we get proxy measures of their neck problem by seeing what functional limitations patients report and demonstrate when we examine them. Current functional limitations associated with pain may be the result of an anatomical impairment. It should

also be remembered that these findings measure performance and depend upon the patient's effort and willingness to move. Reduced ability to perform a movement or task may relate to fear of further 'injury' or fear of pain rather than pain itself, and thus result from a patient's beliefs rather than an anatomical impairment.

## Assessment of the mechanical presentation

Neck pain has traditionally and anecdotally been viewed as being less dramatic in its effect on function than back pain. Nonetheless, restrictions in normal function and range of movement are common in neck pain patients, especially if acute. Decreased movement compared to healthy controls and interference with usual activities of living and working are commonly reported in neck pain patients (Jordan *et al.* 1997; Hermann and Reese 2001; Hagen *et al.* 1997b; Chiu and Lo 2002). Changes in these aspects of the clinical presentation can be used to monitor progress.

**Table 12.4 Dimensions of mechanical presentation by which to assess change**

- range of movement
- deformity
- quality of movement
- loss of normal function.

### Range of movement

There is considerable variation in cervical spinal mobility in the general population (Bogduk 2002a). Several tools are now available to make measurement of spinal mobility more 'objective', but reliability has not been adequately tested for most technologies (Chen *et al.* 1999). Clinical utility demands that methods are simple, inexpensive and easy to use, as well as accurate, reliable and sensitive to change. Some sort of trade-off between accuracy and simplicity may be necessary, but should not compromise clinical integrity. Changes in movement patterns that occur over an episode of care are often substantial, especially when patients are seen from the acute stage. Minor alterations that need to be measured in centimetres are generally not relevant. Gross examples of loss of mobility can often be determined by 'eyeballing' and do not need objective tools. At the outset we wish to know from the patient if their present range of movement is abnormal for them.

It is vitally important that the examination process is standardised. Tests should always be done from a consistent position to optimise reliability and sensitivity to change. *Range of movement tests should always be conducted from the same starting position and in consistent ways so that the results can be compared on different occasions.* The patient should be instructed to move their bottom to the back of the chair and sit upright on every occasion that range of movement is being assessed. Increased range of movement is the main improvement for which to look. To determine a baseline measurement, the patient is encouraged to move as far as possible, for instance by saying, 'further, further, further'. The occurrence and severity of pain during movement and the quality of movement are other ways that the mechanical presentation can change.

Loss of movement occurs most dramatically in the derangement syndrome, when, with the onset of pain, all movements can be dramatically reduced. Equally, with derangements there can be rapid improvements in range of movement. In the dysfunction syndrome, the affected movement displays a marked loss of mobility. This will have been present for some time and will only gradually improve. In patients with dysfunction from a whiplash injury or from cervical spondylosis, marked losses can be present in all directions. In the postural syndrome there will be no loss of movement.

#### Deformity

In a small percentage of patients the onset of neck pain is accompanied by a sudden loss of movement that is so severe that they are unable to move out of the abnormal posture. The patient is locked in flexion or protrusion, lateral deviation or wry neck and is unable to self-correct this very visible anatomical misalignment, or if able to correct cannot maintain the correction. This phenomenon only occurs in derangement and must be immediately recognised as it determines treatment.

#### Kyphotic deformity

The patient is locked in cervical protrusion and flexion and is unable to retract or extend.

#### Lateral deviation

The patient is locked in (for instance) right lateral flexion/rotation and is unable to return the head to the neutral position or to achieve left lateral flexion.

Recognition of deformity is straightforward. It will have come on dramatically with the pain, and active attempts to regain a normal upright posture provoke intense pain and prove impossible. The patient generally needs clinician assistance to correct the deformity, and correction must be done in an unloaded position. When patients respond to the appropriate treatment, there are rapid improvements in the deformity and more normal active movement begins to return.

Patients with deformity are instantly recognised. They will have severe symptoms and a marked postural misalignment that is of recent origin. Patients who present with minor deviation from normal alignment, those who have asymmetrical movement losses and patients who are able to correct a pain-relieving posture do not have deformity. In other words, if the presence of deformity needs to be considered and is not obvious, they do not have one.

### Quality of movement

Pain makes people move more cautiously. Not only might there be limitation of normal range of movement, but also the patient may move more slowly and without the normal cadence.

### Functional disability

It is always important to ask which of their normal activities the patient is unable to do because of their neck pain. Work, recreational and domestic responsibilities and activities may be curtailed, and the goal of treatment is always a return to their normal level of function. More formal ways of assessing patient's function should be done using established disability questionnaires. These can be completed within five minutes by the patients themselves and are a good measure of how much they feel their normal lifestyle is affected by the back problem. When used at the beginning and end of an episode of treatment these offer validated, reliable and sensitive research tools that are easily applied in the clinical environment. Examples of questionnaires are given below (Table 12.5). The Neck Disability Index (Vernon and Mior 1991), developed from and similar to the Oswestry Index for back pain, is probably the most straightforward to use and has been tested for validity, reliability and sensitivity to change (Vernon and Mior 1991; Riddle and Stratford 1998).

The topic was reviewed in 2002 (Pietrobon *et al.* 2002) and five standard measures of functional outcome for the cervical spine were listed and reported on. The Neck Disability Index had been revalidated

more times. The Patient-Specific Functional Scale (Westaway *et al.* 1998), in which patients generate a list of problems specific to them rather than mark a common list, is sensitive to functional changes in individual patients, but comparison between individual patients is virtually impossible (Pietrobon *et al.* 2002).

**Table 12.5 Commonly used neck disability questionnaires**

- Neck Disability Index – Vernon and Mior 1991
- Northwick Park Neck Pain Questionnaire – Leak *et al.* 1994
- Neck Pain and Disability Scale – Wheeler *et al.* 1999.

## Use of mechanical response to guide loading strategy

Just as symptom response can be used to guide loading strategy, so also can mechanical responses (Table 12.6). If there is an increase in range with repeated movements or sustained positions, this is interpreted as *better*. If with repeated movements or sustained positions there is a decrease in range, this is interpreted as *worse*. The Traffic Light Guide provides the same therapeutic implications as above – *better* is a green light to more of the same loading strategy, *worse* is a red light requiring that loading strategy be stopped or amended. Often these responses occur with the symptomatic changes outlined above. Sometimes initially only a symptomatic change occurs, or less commonly only a mechanical response occurs. In all three instances the mechanical response has the same therapeutic implications.

**Table 12.6 Mechanical responses to loading strategy**

| *Range of movement change* | *Terminology* | *Implications* |
|---|---|---|
| Increase | Better | Green |
| Decrease | Worse | Red |
| No change | | Amber |

## Symptomatic and mechanical presentations to identify mechanical syndromes

The different mechanical syndromes present with characteristic patterns of symptoms and movement abnormalities. Recognition of these helps in the identification of the different mechanical syndromes. Equally, their absence helps in the discovery of atypical

and non-responders who may require further testing or investigation to elucidate their problem.

The table below indicates the characteristics that may be present in each syndrome. These criteria either ***must*** be present for that mechanical classification to be indicated, shown as +, or are ***variably*** present in that mechanical classification; that is, they ***may*** be present, indicated as (+). If a particular feature is ***never*** found in that syndrome, this is indicated by N. For instance, if there are referred or constant symptoms, the patient cannot have postural syndrome. The table makes clear that the presentation of derangement is much more varied than the other two syndromes. Presentations in dysfunction and postural syndromes are reasonably consistent. In derangement many different signs and symptoms can be present, but not all need be present to classify as derangement.

**Table 12.7 Characteristic symptomatic and mechanical presentations of the mechanical syndromes**

| *Features* | *Postural syndrome* | *Dysfunction syndrome* | *Adherent nerve root* | *Derangement syndrome* |
|---|---|---|---|---|
| ***Symptomatic features*** | | | | |
| Neck/scapular pain | + | + | (+) | (+) |
| Pain to elbow | N | N | (+) | (+) |
| Pain to hand | N | N | + | (+) |
| Pain to hand and neuro | N | N | (+) | (+) |
| Constant | N | N | N | (+) |
| Intermittent | + | + | + | (+) |
| Centralisation/ peripheralisation | N | N | N | (+) |
| Pain during movement | N | N | N | (+) |
| End-range pain with appropriate testing | N | + | + | (+) |
| Sustained loading produces pain | + | (+) | | + |
| Inconsistent pain response to loading | N | N | N | (+) |
| Painful arc, no movement loss | N | N | N | (+) |
| ***Mechanical features*** | | | | |
| Movement loss | N | + | + | + |
| Acute deformity | N | N | N | (+) |

Key: + = must be present, (+) = may be present, N = never present

## Identifying responders

Certain aspects of the history provide clues as to the likely responsiveness to mechanical diagnosis and therapy. Especially suggestive of a good response is a history of intermittent symptoms and variable pain behaviour in response to different postures and activities. Intermittent symptoms indicate that there are times during the day when, as a result of being in certain positions or performing certain activities or for no apparent reason, the patient has no pain. Even in those patients who have had symptoms for years and may be deemed chronic, intermittent symptoms indicate the likelihood of a good prognosis. Neck pain that behaves in this way is demonstrating mechanically responsive pain – certain positions or movements are causing strain on spinal tissues that generates pain, whilst other positions or movements reduce deformation of spinal tissues and relieve the pain. Frequently patients are very aware of postures that aggravate or relieve their symptoms, and educating them to temporarily avoid aggravating factors and make use of reductive factors is straightforward.

Pain variability in response to postures and movements can also be a good predictor of a patient who will respond well to mechanical therapy. They may report that in maintaining certain postures, such as sitting, they experience more or peripheral pain, but this is abolished when they walk about. Variability of pain pattern often indicates a patient who will do well with the management strategies outlined in this book; that is, the symptoms are sometimes on the right side or the left, sometimes only spinal or referred into the arm.

A good indication of patient suitability for this approach to treatment is often obtained on day one during the mechanical assessment. If, during the initial testing procedures, pain centralisation or decrease of pain intensity occurs, this is invariably indicative of a good prognosis. This has been termed a 'green light' in opposition to the plethora of coloured flags that are barriers to recovery. However, it is sometimes necessary to conduct the mechanical evaluation over several days in order to ensure exposure of this response.

## Chronic pain – interpretation of symptomatic responses

With chronic pain, peripheral tissue and central nervous system elements may be sensitised and deconditioned to normal movement; the criteria

of symptom response needs to be different. Under these circumstances normal mechanical stimuli can produce pain, repeated movements may have a 'wind up' effect on pain production, there may be a spread of painful areas, and there may be ectopic nociceptive signals (Dubner 1991; Johnson 1997). These changes make the interpretation of mechanically produced symptom responses difficult and invalidate diagnostic labels applied to particular responses (Zusman 1992, 1994). Psychosocial elements that have been identified as factors in chronic spinal pain and disability are passive coping strategies, fear-avoidance behaviour, lack of self-efficacy and depression (Linton 2000). These characteristics may make patients overly anxious and fearful about pain responses, which they consequently exaggerate. These examples suggest that we should interpret the behaviour of chronic pain to repeated movements somewhat less rigidly. Although most of this work to date has been conducted with low back patients, there is no reason not to assume that the same issues are not important in neck pain patients (Linton 2000). Non-mechanical factors may have become significant factors in perpetuation of pain.

The above effects are unlikely to be present to the same degree, or even in all patients with chronic pain; many such patients respond relatively straightforwardly to mechanical therapy. Most patients with chronic musculoskeletal pain have only mild or moderate symptoms and do not suffer major functional impairment. Only a small proportion of patients with persistent pain are at the severe, disabled end of the spectrum; many respond normally to a mechanical evaluation. If the response is equivocal, test out the patient's response over twenty-four hours or use some other force progression.

However, in the case 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, and 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 may be necessary to concentrate more on trying to improve coping strategies and function rather than focus on pain. Often improvement in general function and the psychological effect of doing something active about their problem can produce a reduction

in pain. Patients with severe levels of dysfunctional behaviour due to persistent pain problems are probably best treated in a multi-disciplinary pain programme or a functional rehabilitation approach rather than on a one-to-one basis.

## Conclusions

This chapter has discussed the means by which patients are assessed on their progress or lack of it. This involves regular interpretation of their pain response and examination of their mobility and function. The way to review and interpret the symptomatic and mechanical presentations in detail has been presented. Using these criteria it can be gauged whether the patient is improving, worsening or unchanging. If they are getting better, nothing should be changed. If they are worse, further assessment is performed and a change in management strategy is necessary. If they are the same, then a progression of forces should be considered and a re-analysis is conducted. Review should ideally be carried out on a daily basis until there is a definite improvement and confirmation of the management strategy.

# 13: Follow-up Evaluations



## Introduction

The review process is an essential part of patient management. We must be able to evaluate the management strategies that have been recommended to see if they are having the desired effect, or if the patient is unchanged. According to the patient's response, the strategy is continued or amended. In the previous chapter the aspects of symptomatic and mechanical responses that help us to evaluate management are presented in detail. It may be helpful to review that chapter before you read this one, which presents the specific way that the review should be conducted.

To be able to conduct the review properly, it is essential to have gathered sufficient detail at the initial assessment. *If baseline details are inadequate, it will at times make it impossible to conduct a thorough review.* Be warned – it is too late to remedy this deficiency in retrospect.

Sections in this chapter are as follows:

- reaching a conclusion
- review process
- implications.

## Reaching a conclusion

The confidence with which a patient can be given a syndrome classification on the first day varies. Sometimes it is very definite that symptoms are centralising and the mechanical presentation improves in one session; at other times the response is less clear. The conclusion made on day one is deemed to be provisional; confirmation of the classification and the appropriateness of the chosen management strategy are made at follow-up. If the response is still equivocal, further testing may be necessary. Sometimes a period of three or four days with several sessions may be necessary to confirm a directional preference or lack of it. Diagnostic classification should be complete within five sessions, but is usually achieved more quickly than this.

Where uncertainty exists concerning the correct management approach, the patient should be reviewed every day if possible until the appropriate treatment principle is confirmed. If the patient is unable to attend, the review should be conducted by telephone. Once symptoms are resolving and the patient is managing successfully alone, review sessions should be scheduled progressively further apart so that they are able to demonstrate independence and gain confidence.

Where uncertainty exists, several strategies may be used to reach a more definite conclusion. These include force progressions (overpressure or mobilisation), force alternatives (for instance, lateral forces or sustained procedures), ensuring end-range is being reached and testing the effects of repeated movements over several days.

If symptoms have been present for some time, it is more likely that the response may be equivocal. It is often helpful at this point to get the patient to test 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 listed in Table 13.1 (from van Wijmen 1994).

**Table 13.1 Different methods of clarifying symptom response**

- test provocative or reductive procedures over two to three days
- use force progressions (overpressure and clinician techniques)
- use force alternatives
  - lateral/rotational forces
  - loaded/unloaded
  - sustained postures
- increase the number of repetitions
- increase the frequency of repetitions
- ensure that movements are to end-range
- stress the joints in one direction and check the effects on pain and movement range in the opposite direction.

## Review process

On the second session and at each subsequent visit a structured, logical and informative review process must be conducted. This is to determine what the patient has been doing regarding previous instructions, the immediate effect of any procedures being done, and if there have been any overall changes. We need to know from

the patient as a result of following instructions if there has been any change: 'With the exercises and postural correction over the last day(s), overall are you better, worse or the same?'

If they are better there is no need to change management in any way, and they should continue with more of the same as long as improvements continue. The patient should be questioned and examined thoroughly to ensure that they are actually 'better' than the previous occasion. If the patient reports an improvement, ask, 'Are you definitely improved, or only possibly improved?' Patients sometimes like to please the clinician, and this question exposes uncertainty. Other questions regarding pain and function clarify the situation. If their response is definitely improved, and supported by symptom location change or symptom abolition and mechanical improvement, the classification is confirmed and the appropriate management strategy has been selected.

If at a later point in the episode the patient stops reporting improvement and says that symptoms are unchanging, then a different management strategy may be necessary. This could involve force progressions or force alternatives.

If they are worse or unchanged, they must be questioned more closely about what they have been doing:

- 'Have you been doing the exercises we discussed?'
- 'How frequently?'
- 'What exercises have you been doing?' Get them to show you; however clear you think you may have been, some patients misinterpret instructions.
- 'Are there any problems that limit your ability to do the exercises?'
- 'What happens to the pain when you do the exercise?'
- 'What happens to the pain when you use posture correction?'
- 'Have you been sitting the way we talked about last time?'
- 'Do you understand the reasons for the exercises and posture correction?'
- Check symptomatic presentation fully:
    - if there is a change, is this definite or doubtful?

- Check symptoms:
  - site for centralisation
  - frequency (constant or intermittent; if intermittent, what proportion of the day)
  - severity
- Check if there has been any change in functional problems.
- Check mechanical presentation:
  - range of movement
  - pain on movement
  - deformity.

It will then be known how regularly they have been doing their exercises and if they are doing them correctly. Their technique may need correcting, but wait until you have heard how it affects their symptoms. They may need encouragement to exercise more regularly, or, less commonly, they might be 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.

The following questions and statements may help the patient to gain more effective management:

- 'If you maintain the correct posture, can you keep yourself free of pain?'
- 'If pain appears, note what you were doing immediately before.'
- 'Particularly note if it came on after sitting or bending.'
- 'If pain does appear, can you get rid of it by doing the exercise(s)?'

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

**Table 13.2 Main elements of review process**

*Mechanical therapy:*

- have they been exercising?
- what exercises?
- posture correction?
- what is the response when they do them?

*Symptomatic presentation:*

- site of pain
- frequency
- severity.

*Mechanical presentation:*

- range of movement
- deformity
- quality of movement
- function.

## Implications

From the review you will also know their symptomatic and mechanical response to performing exercises over a day or two – they will either be better, worse or unchanged. As outlined in Chapter 12, 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. Keen questioning and close analysis of symptomatic and mechanical responses are sometimes necessary to elucidate the true picture.

If better, nothing needs to be changed, only encouragement given and the management strategy maintained unaltered. However, at some point the direction or level of force of mechanical therapy may need to be altered, especially if symptoms stop improving. Do not stick rigidly with one loading strategy because of an initial improvement. If worse, exercises and symptom responses need to be checked, but ensure the patient is actually worse, rather than simply that the exercises 'hurt'. When starting any unfamiliar exercise programme, new pains may be generated; this is not unusual, but may confuse the patient. If they are truly worse, the treatment principle or start position may need to be changed; a derangement may be irreducible, or consideration may need to be given to non-mechanical syndromes. If unchanged, is the patient exercising regularly enough and doing the right exercise? If they have been, force progression may be necessary, or if this has

been attempted already, an alternative treatment principle should be considered. In the case of dysfunction no change would be expected, and they should be encouraged to continue.

In essence, if the patient is better then the provisional diagnosis has been confirmed. The original management strategy was correct, and this should be continued unchanged. If the patient is genuinely worse, a misclassification has occurred: either the patient has been categorised in the wrong syndrome or they have been given the wrong treatment principle. Sometimes the treatment principle may be right, but the start position is wrong. In chronic symptoms there can be a temporary exacerbation of symptoms when activation is started.

If the patient returns and after a thorough assessment of symptomatic and mechanical presentations they are genuinely unchanged, then further analysis is necessary. This takes the form of force progressions until a change occurs. If symptoms start to reduce, abolish or centralise, the directional preference is confirmed; if symptoms start to worsen or peripheralise, an alternative direction must be explored.

## Conclusions

In this chapter, the way the review process is carried out has been presented. This is conducted at each session to determine if the appropriate management strategy is being implemented. Depending on the certainty of response, this will be done with more or less of the detail presented here, but essentially the review involves enquiring about the mechanical therapy component of management and about their symptomatic and mechanical response.

# 14: Procedures of Mechanical Therapy for the Cervical Spine

## Introduction

This chapter contains general descriptions of the procedures that may be needed in mechanical therapy of the cervical spine and indications for their application. The procedures described here include both patient and clinician techniques.

In most situations patient techniques are used first, and these are frequently effective in resolving the problem without the need for more interventions. Provided there is adequate instruction and careful explanation regarding management of the problem, the self-treatment concept can be successfully applied to most neck pain patients. Patients with postural syndrome can only resolve their problem with self-management strategies. Clinician interventions are ineffective without the patient being educated about the role of posture as a cause of their pain. In the dysfunction syndrome only the patient is able to provide the appropriate loading strategies with sufficient regularity to enable a remodelling of the structural impairment. Clinician techniques may aid this process, but on their own are generally inadequate to resolve the tissue abnormality. In the derangement syndrome the majority of patients can successfully manage their own problem, while about 30% cannot recover with exercises alone and need the addition of clinician techniques (McKenzie 1981).

In general, patient techniques are always used first and these are only supplemented by clinician techniques when there is a failure to improve. While the patient is improving with self-management strategies there is absolutely no need to supplement treatment with additional interventions that may encourage patient dependency.

The essential philosophy of this method of management is to give patients, whenever possible, knowledge and understanding of their problem and the tools by which they can treat, manage and control their own pain (McKenzie 1981, 1990). To achieve this it is necessary to depart from the current traditional methods of treatment in which the clinician applies passive modalities or manual procedures to the patient. From that approach the patient attributes his or her recovery, rightly or wrongly, to what was done to them. Consequently

dependency on the clinician develops and the patient returns for assistance whenever problems recur. By avoiding the use of clinician-applied techniques, unless absolutely necessary, and using primarily patient- or self-generated techniques, the patient recognises that they are capable of managing their own problem both now and in the future.

However there are instances where force progressions involving clinician techniques are needed. The role of force progressions and force alternatives in the elucidation of management strategies is discussed. The therapeutic loading strategies that are used involve posture correction, repeated movements and/or sustained postures.

Sections in this chapter are as follows:

- force progression
- force alternatives
- repeated movements or sustained postures
- application of the procedures
- procedures.

## Force progression

This approach to musculoskeletal problems involves a progression of forces, initially starting with patient-generated forces, and only involving clinician-generated forces when needed. This has several advantages (McKenzie 1989, 1990); the patient can regularly apply the procedures throughout the day with far more frequency than would be possible if the patient was treated only in the clinic. If the patient is educated adequately and effectively in self-management, then the responsibility for their condition lies with the individual; the solution to their problem is in their own hands. They become independent of the clinician and are given the opportunity to manage the problem themselves should it recur in the future. Furthermore, should it be necessary to progress forces and include mobilisation or manipulative procedures, the multiple repeated movements that will have preceded these interventions provide a clear indication of the clinically determined directional preference and safety of the proposed loading strategy.

Guidelines about spinal care stress the importance of patient responsibility for management (CSAG 1994, ACHPR 1994). This responsibility can be encouraged if the patient is offered an approach that is based on self-management techniques. In contrast, the primary use of passive therapies, including clinician-generated mobilisation and manipulation, engender patient dependency. Using passive therapies implies that only with the intervention of the clinician can the patient be cured.

It is not always necessary to start with the earliest forces; the patient can enter at the stage that generates a positive effect. This is different for each patient. Clinician-generated forces should never be used before patient-generated forces have been tried. Progression of forces (Table 14.1) is only introduced as needed and is not an inevitable part of management.

Force progression is considered when the previously employed technique brings about no lasting change. For instance, symptoms may increase or decrease during the procedure, but afterwards remain no worse or no better. If a procedure results in the decrease, abolition or centralisation of symptoms it does not need to be progressed or supplemented in any way, provided there is a continued increase of movement to end-range. If a procedure results in the worsening or peripheralisation of symptoms, it should be stopped and force alternatives be considered. Only when symptoms remain unchanged following a procedure should force progressions be considered. Force progression could also include increasing the frequency of exercises and prolonging the period over which exercises are assessed. For instance, a twenty-four hour test period may provide a more definite response than one gained during a short clinic visit. The progression for the application of forces is listed below (McKenzie 1989, 1990).

The progressions are given in the order that most frequently generates a favourable clinical response. However, in determining the appropriateness of loading strategies, some flexibility in the application of force progressions and force alternatives may be required. Application of force progressions and force alternatives should always be conducted with due consideration and attentive interpretation of symptomatic and mechanical responses.

**Table 14.1 Force progression**

- Static patient-generated forces:
  - Positioning in mid-range
  - Positioning at end-range
- Dynamic patient-generated forces:
  - Patient motion in mid-range
  - Patient motion to end-range
  - Patient motion to end-range with patient overpressure
- Clinician-generated forces:
  - Patient motion in mid-range with clinician overpressure
  - Patient motion to end-range with clinician overpressure
  - Clinician mobilisation
  - Clinician manipulation.

## Force alternatives

At times, rather than a force progression, an alternative force is needed. For instance, the response to retraction or extension in sitting may be unclear, or even cause a worsening of symptoms. However, in the same individual, these movements performed in lying may reduce symptoms. If at any point during exploration of sagittal plane movements these are all found to worsen symptoms, including movements performed unloaded, then lateral forces need to be considered. In patients with acute deformity, management always starts in lying and the deformity is initially accommodated using pillows, as too hasty an attempt to recover neutral posture may cause a severe exacerbation of symptoms.

**Table 14.2 Force alternatives**

- starting position, example: loaded or unloaded
- direction of loading strategy, example: sagittal or frontal plane movements
- sagittal force: flexion or extension
- lateral force: lateral flexion or rotation
- lateral direction: towards pain or away from pain
- time factor, example: sustained positioning or repeated movements.

## Repeated movements or sustained postures

Procedures can be used as either repeated movements or as sustained positions. Repeated movements are used most commonly.

The optimum number of movements is about ten repetitions in one 'set'; however, the exact amount can vary depending on the patient's tolerance, response and so on. In certain instances, several sets of exercises may be done in succession. The number of times in a day that the series of exercises should be done varies according to the mechanical syndrome, the severity of the problem and the capabilities of the patient. In most instances a minimum of four or five sets a day is necessary to produce a change; patients should generally be advised to repeat the exercise every couple of hours.

It is essential that movements be repeated in order to gain a true understanding of their mechanical effect. Single movements rarely provide a thorough understanding of the effect of that movement. Sometimes the effect of repeated movements is rapidly apparent, while at other times repeated movements over a period of several days are necessary to produce a clear cut symptomatic and/or mechanical change.

Exercises or mobilisations are generally performed in a rhythmical pattern. The procedure should be followed by a brief moment of relaxation. With each subsequent movement the range or pressure exerted should be increased as long as the symptomatic response is favourable.

Although the eventual goal with all exercises is to perform end-range movements, it must be remembered that this might not initially be tolerated by patients and that movements may need first to be in mid-range. However ultimately, to fully abolish symptoms, end-range movements are required.

On occasions static rather than dynamic procedures should be used. These should be considered, for instance, when symptoms are severe, when there is a poor response to repeated movements, or when a time factor has been indicated in the history. The following procedures are described for dynamic application; however, they can also be used as sustained positions and should then be maintained up to three minutes depending on the symptom response achieved.

The procedures described for clinician overpressures and mobilisation have similar starting positions. However, the distinction between overpressure and mobilisation has to do with the involvement of the patient and the force applied. In clinician overpressure the patient first moves to end-range and then the overpressure from the clinician is

added. With clinician mobilisation, the clinician moves the patient's head to end-range, and then one hand stabilises at end-range and the other hand delivers the mobilisation.

Certain procedures can be adapted depending on the source of the symptoms; for instance, flexion procedures should be preceded by retraction if the upper cervical spine is being targeted. The force alternative of traction can be added to a number of the procedures when this is thought to be appropriate; for example, in lateral flexion supine when the acute deformity of wry neck is present, or retraction in lying with radicular symptoms.

## Application of the procedures

Throughout the application of any of the procedures described below, the symptomatic and mechanical responses to the various loading strategies must be closely monitored. The intensity and location of any pain or other symptoms and the mechanical presentation must be recorded *prior to and then following* the performance of repeated movements or sustained positioning. Abolition, reduction or centralisation of pain is a green flag or green light to proceed. Production, increase or peripheralisation of pain are red flags and the signal to stop.

Close monitoring is essential when a change in direction or progression of loading is introduced for the first time. In such a case only one movement should be carefully applied to determine the potential the progression has to cause exacerbation of symptoms. If the first tentative exploratory movement results in an increase or peripheralisation of symptoms, caution is indicated. The unsuitability of the chosen procedure is suggested and immediate modification is prudent. On the other hand, if no adverse response results, the procedure may be explored further. The precautions implicit in the above requirements ensure the safe practice and delivery of both the diagnostic and therapeutic aspects of this method of practice.

Different positions for delivery of exercises are described to allow for the application of force progressions or force alternatives in loaded or unloaded settings. Some procedures may not be tolerated when loaded, especially in the presence of acute or severe symptoms. The monitoring process described more fully in Chapter 12 must become routine.

The description of the procedures that follow is primarily for the management of patients with the derangement syndrome. However, it is important to note that apart from procedures involving manipulation, the same techniques are applicable for patients with the dysfunction syndrome. There is a difference in application however, in that patients with dysfunction must apply the exercises with more frequency and over a much longer period of time. Therefore in the presence of dysfunction, emphasis must be given to self-applied remodelling techniques.

## Procedures

The procedures are listed according to the treatment principle. They can be performed in a number of different positions (loaded or unloaded) and applied either dynamically or statically. Cervical retraction should be performed prior to the application of all other procedures.

**Table 14.3 Treatment principles**

- extension principle forces
- lateral principle forces
- flexion principle forces.

**Table 14.4 Procedures** (not all in order of force progressions)

Extension principle

*Procedure 1 – Retraction:*

Can be performed in sitting, standing, supine or prone

1a. retraction with patient overpressure

1b. retraction with clinician overpressure

1c. retraction mobilisation

*Procedure 2 – Retraction and extension:*

Can be performed sitting, supine, prone

2a. retraction and extension with rotation

2b. retraction and extension with rotation and clinician traction (supine)

*Procedure 3 – Postural correction.*

Continued next page

Lateral principle

*Procedure 4 – Lateral Flexion:*

Can be performed sitting or supine

4a. lateral flexion with patient overpressure

4b. lateral flexion with clinician overpressure

4c. lateral flexion mobilisation

4d. lateral flexion manipulation

*Procedure 5 – Rotation:*

Can be performed sitting or supine

5a. rotation with patient overpressure

5b. rotation with clinician overpressure

5c. rotation mobilisation

5d. rotation manipulation

Flexion principle

*Procedure 6 – Flexion:*

Can be performed sitting or supine

6a. flexion with patient overpressure

6b. flexion in supine with clinician overpressure

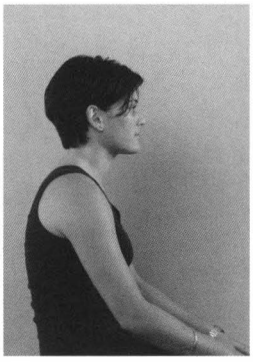
6c. flexion mobilisation in supine

## Extension principle

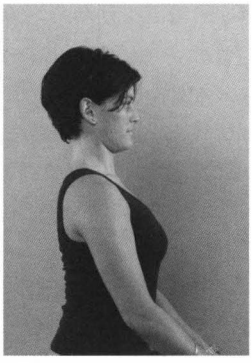
### Procedure 1 – Retraction

Can be performed sitting, standing, supine or prone

*Photos 21, 22: Retraction from erect sitting posture.*



*21*



*22*

#### Retraction in sitting

In this text, retraction means to move the head backwards as far as possible from a protruded position so that it is positioned more directly above the spinal column. Throughout the movement the head must remain horizontal, facing forward, and be inclined neither up nor down.

For instruction, the patient is initially seated on an upright chair with a rather high back, with the sacrum in contact with the back of the chair. The patient should be instructed to sit with the head and shoulders relaxed, thus allowing the adoption of their natural resting posture (Photo 21).

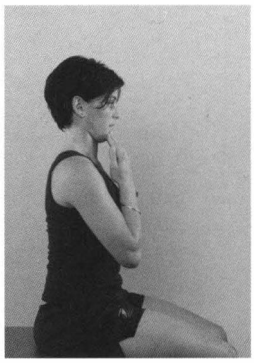
From the relaxed position, the patient is instructed to retract the head as far as possible, keeping the head facing forward and horizontal during the movement (Photo 22). The movement should be made to the maximum end-range of retraction. Once the maximum end position has been reached and held momentarily, the patient may relax back to the start position (Photo 21). During retraction of the head and neck, there should also be an accompanying correction of the shoulder posture.

The same movement should be repeated rhythmically, always returning to the relaxed position after each retraction. With each excursion the patient should be encouraged to move even further than before, so that after five to fifteen movements the maximum possible range of motion has been achieved. In the sitting position most patients can be taught to perform the exercise easily and can become proficient in a matter of five to ten minutes.

### Procedure 1a – Retraction in sitting with patient overpressure

Once the patient is proficient in the practice of the manoeuvre, the first progression can be applied to ensure the patient achieves the maximum end-range of motion. This is achieved by having the patient apply overpressure using the fingertips of one or both hands against the mandible (Photo 23). It is important to avoid flexing the cervical spine when overpressure is applied.

*Photo 23: Patient overpressure applied at the chin.*

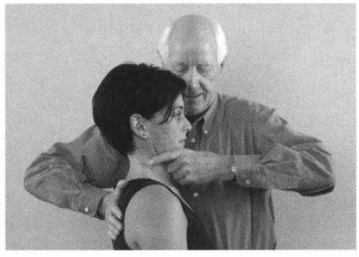


*23*

### Procedure 1b – Retraction in sitting with clinician overpressure

Where patient-generated forces are not achieving resolution of symptoms, the progression of clinician overpressure can be introduced. This can also be useful to assist in teaching the patient the retraction movement. To do this, the clinician places the *heel of the hand* at the level of

*Photo 24: Overpressure is applied through the jaw and across the thoracic spine.*



*24*

the first or second thoracic vertebra. Then the spread of thumb and forefinger of the other hand is applied against the patient's clenched mandible. The patient retracts the head and neck as far as can be tolerated and the clinician applies overpressure with both hands (Photo 24). Ensure that the patient's teeth are approximated so that no adverse pressures affect the tempero-mandibular joint. The movement should be repeated rhythmically five or six times, always returning to the starting position after each retraction.

## Alternative positions for Procedure 1 – Retraction

### Retraction in standing

Retraction in standing should be performed as described for retraction in sitting. It is a useful position as it allows the patient the opportunity to perform retraction regularly throughout the day.

*Photos 25, 26: Retraction may be performed on a pillow if patient is initially unable to tolerate lying without one, as for a patient with kyphotic deformity.*

*25*

*26*

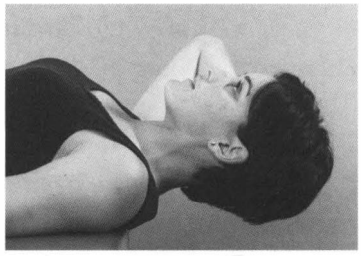
### Retraction in supine with pillow support

The patient should lie supine on the treatment table. In very acute cases and during the initial treatment session, one or two small pillows may be placed under the neck and head to allow for any deformity (Photo 25). The patient should be instructed to retract the head (as described above in sitting) into the pillow, hold for a second or two (Photo 26), and then relax. The movement should be repeated five or six times.

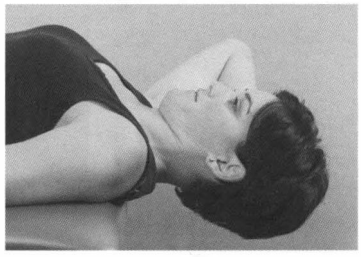
### Retraction in supine without pillow support

The patient lies supine, places one hand behind the occiput, and moves off the end of the treatment table so that the head, neck and shoulders are unsupported down to the level of the third or fourth thoracic vertebra (Photo 27). The patient, while holding the occiput for stability, fully retracts the head for a second or two (Photo 28) and then relaxes to the starting position. The movement should be repeated five or six times. This procedure is a preliminary to the introduction of extension.

*Photos 27, 28: Supine retraction with head over the end of treatment table.*



*27*

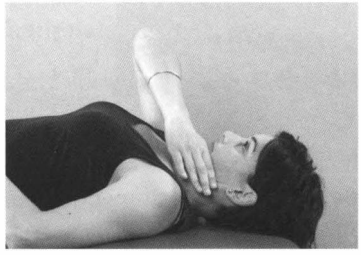


*28*

### Retraction in supine with patient overpressure

To ensure maximum end-range of motion is achieved, the patient can apply overpressure using the fingertips of the other hand against the mandible.

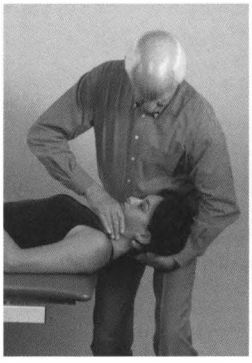
*Photo 29: Overpressure applied through chin with head on the table.*



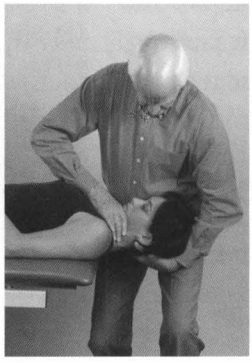
*29*

### Retraction in supine with clinician overpressure

The patient's head is positioned as described above. The clinician stands to one side and supports the occiput with one hand. The thumb and fingers of the other hand are placed over the patient's upper lip or mandible, depending on comfort. The head of the patient is held gently but firmly against the clinician's waist (Photo 30). The patient retracts to end-range, and at the end of the movement overpressure is applied by the clinician (Photo 31). The head is then returned to the neutral position. It is important to ensure that the patient's head is kept in the horizontal plane. The movement should be repeated five or six times.



30

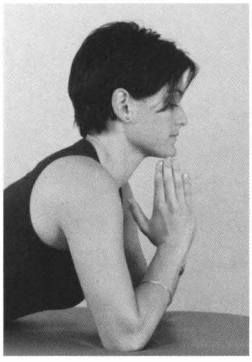


31

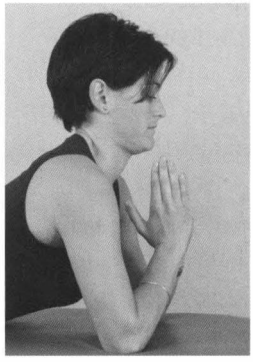
*Photos 30, 31: Patient's head is supported at the occiput (30) and overpressure is applied at end of active range of movement (31).*

### Retraction in prone

The patient lies prone on the treatment table leaning on the elbows so as to raise the upper trunk (Photo 32). The patient retracts the head and neck in the same manner required when the exercise is performed in sitting (Photo 33). After repeating the movement five or six times, the patient rests the chin on their hands.



32



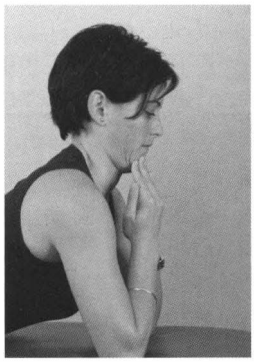
33

*Photos 32, 33: From neutral (32), head is retracted actively (33).*

### Retraction in prone with patient overpressure

The patient can apply overpressure at the end-range of retraction by using the fingertips of both hands against the mandible (Photo 34).

*Photo 34: Retraction prone with patient overpressure.*

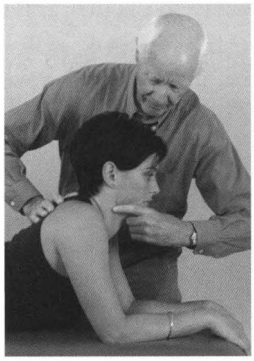


*34*

### Retraction in prone with clinician overpressure

The patient position is as described above. The clinician stands to one side and places the heel of the hand at the level of the first or second thoracic vertebra. The web space of the other hand is placed over the mandible with the clinician's forearms positioned parallel to one another to ensure they are in line with the retraction movement. The patient retracts as far as possible and overpressure is applied by the clinician using both hands (Photo 35). The movement is repeated five or six times; the patient then rests the chin on clasped hands.

*Photo 35: Overpressure is applied through the mandible and the thoracic spine.*



*35*

*Note:* The prone position has the advantage of producing a marked retraction force in the upper thoracic segments, which is often difficult to achieve in supine and in sitting. It may also be more acceptable for older patients, and can be adapted to be performed in sitting with the elbows on a table.

## Application of retraction

Retraction is the essential preliminary procedure for the reduction of posterior derangements in the lower cervical spine. It is also used for the treatment of extension dysfunction in the lower cervical spine.

*Retraction is an essential precursor to other movements required to effectively treat the cervical spine.* Some movements, apparently ineffectual or even aggravating to the patient, can become effective when their application is preceded by repetitive retraction of the head and neck. Limitation of the range of motion in extension and rotation, which may be present while the head remains in a protruded position, can disappear when the movements are carried out with the head in

the retracted position. Further, painful limitation of extension and rotation can become painless when the same movement is performed with the head in the retracted position.

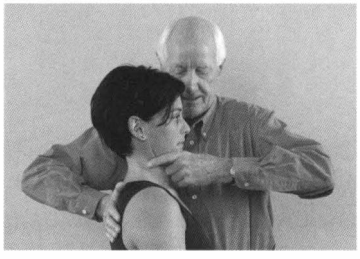
Perhaps the most important reason for performing retraction of the head and neck prior to moving into the extended position is the effect these movements have on referred and radiating symptoms. A well-established test to confirm the origin of radiating symptoms from the cervical spine is to extend and then rotate the neck towards the side of radiating pain or paraesthesia in order to provoke and thus confirm the origin of the problem. The commonly accepted theory is that this test reduces the diameter of the intervertebral foramen and produces or increases peripheral symptoms should the existing nerve roots be compromised. If this test is applied repeatedly, the patient's condition frequently worsens. However, should the head and neck be retracted immediately prior to extending and rotating the neck, a reduction of the referred symptoms frequently follows. This is most likely to occur if the referred symptoms are intermittent.

Retraction is also essential in the management of cervical headaches and for flexion dysfunction in the upper cervical spine. Retraction of the head produces flexion in the upper cervical segments and simultaneously causes extension in the lower segments. It has been demonstrated that more flexion occurs in the upper cervical spine when the head is retracted than occurs when the head and neck are simply flexed.

## Retraction mobilisation

Can be performed in sitting, supine or prone.

*Photo 36: One hand stabilises the head at end-range retraction, and the heel of other hand mobilises through the thoracic spine.*



*36*

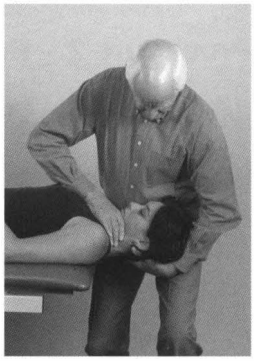
### Procedure 1c – Retraction mobilisation in sitting

Patient and clinician positions are as described for retraction with clinician overpressure (sitting). The clinician's hand on the patient's mandible stabilises the head at end-range of retraction and the clinician applies a postero-anterior force with the heel of the hand on the spinous processes of the upper thoracic

segments (Photo 36). The clinician's forearms should be parallel so that the pressure occurs in the sagittal plane. The mobilisation should be repeated five or six times, and then the patient's head is returned to the neutral position.

## Retraction mobilisation in supine

Patient and clinician starting positions are described in retraction with clinician overpressure (supine). The clinician, by bending the knees, moves the patient's head and neck to the end-range of retraction (Photo 37). The pressure is released and then repeated five or six times before the head is returned to the neutral position. It is important to ensure that the patient's head is kept in the horizontal plane. Some degree of traction may be applied during the procedure.

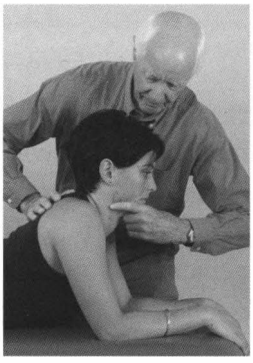


37

*Photo 37: Patient's head is supported at the occiput. End-range retraction is achieved by the clinician bending at the knees; mobilisation is then applied through the mandible.*

## Retraction mobilisation in prone

Patient and clinician starting positions are described in retraction with clinician overpressure (prone). The clinician, with the hand on the mandible, stabilises the head at end-range of retraction. The hand on the spinous processes of the upper thoracic segments applies a postero-anterior force, which achieves an extension movement of the upper thoracic segments (Photo 38). The movement should be repeated five or six times, and then the head returned to the resting position on the clasped hands.



38

*Photo 38: The patient's head is stabilised at end-range retraction and the mobilisation force is applied through the thoracic spine.*

## Application

With all the retraction mobilisation procedures the pressure should be applied in a slow rhythmical way aiming to move further into range with each movement applied. The procedures are used to restore retraction range where patient-generated forces and clinician overpressure have failed to so. They are appropriate to assist in the

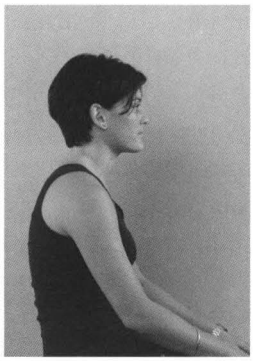
reduction of posterior derangements and may be useful for lower cervical extension dysfunction. As with the application of all procedures, careful monitoring of symptoms is required. Once retraction range has been restored then the use of the mobilisation procedures should cease and patient-generated forces resumed.

## Procedure 2 – Retraction and extension

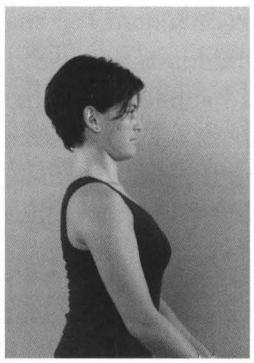
Can be performed in sitting, supine or prone.

### Retraction and extension in sitting

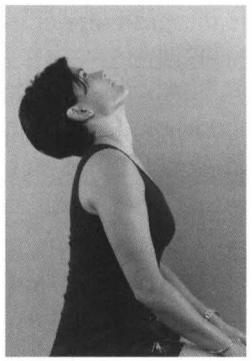
*Photos 39, 40, 41: From neutral (39) the patient first retracts (40) and then extends (41).*



39



40

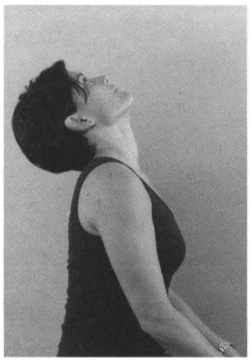


41

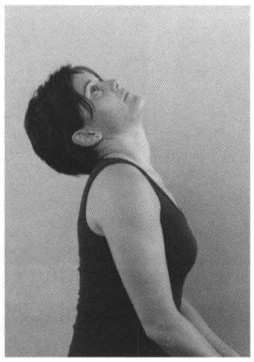
Head and neck retraction and extension are the movements of retraction, followed immediately by movement of the head and neck into the fully extended position. Although there are two movements involved, they should appear to blend smoothly into one continuous motion until finally the neck is fully extended. This procedure can be commenced once the patient is proficient in performing basic retraction and a good range of retraction has been achieved.

The patient is seated as for retraction and retracts the head as far as possible (Photo 40). Once the end-range of retraction has been reached, the patient is instructed to continue the movement by slowly and steadily tilting the head backwards, as far as possible, as if to look skywards (Photo 41). After a second the patient should carefully raise the head to the upright neutral position. The patient may feel more secure by using one hand to provide support behind the occiput and upper cervical spine when first commencing this exercise. The patient repeats the movement of retraction into extension in a rhythmical fashion five or six times.

## Procedure 2a – Retraction and extension with rotation in sitting



42



43

*Photos 42, 43: From full range extension (42) overpressure is applied with slight rotation movements (43).*

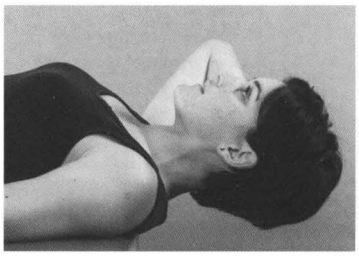
An increase in the range of extension can be achieved with the addition of a rotary component applied while the head and neck are held in the fully extended position. A *minimal rotary* adjustment of the head position is repeated five or six times so that the nose moves only *one centimetre (half an inch) to either side of the mid-line.* During this process the patient is urged to move further and further into extension so as to gain maximum end-range (Photo 43). The patient should then return to the starting position.

## Alternative positions for Procedure 2 – Retraction and extension

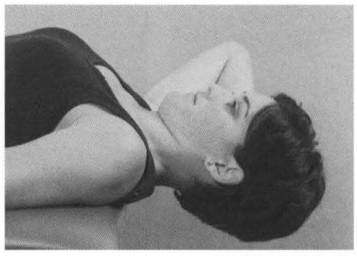
### Retraction and extension with rotation in supine

The patient should be instructed to lie supine over the end of the treatment table so that the head, neck and shoulders are unsupported down to the level of the third or fourth thoracic vertebra. The patient places one hand under the occiput to provide assurance and stability (Photo 44). The patient then fully retracts the head (Photo 45) and is instructed to continue the movement by slowly and steadily tilting the head backwards as far as possible into the end-range of extension (Photo 46). After a second or two the patient, using their supporting hand, should carefully lift the head to the horizontal neutral position. It is important for the patient to avoid actively raising the head by using the neck musculature or bringing head and neck too far forward into flexion during this procedure. The patient may repeat retraction and extension in a continuous rhythm for five or six excursions. The patient can exert full control over the movement by using the hand under the occiput.

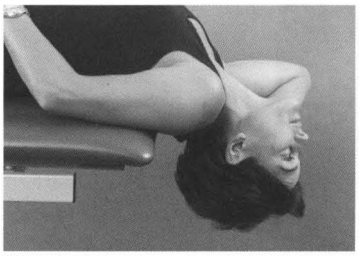
*Photos 44, 45, 46, 47: With head off the end of the treatment table (44), the patient retracts (45) and extends (46); end-range overpressure is applied with a slight rotation movement (47).*



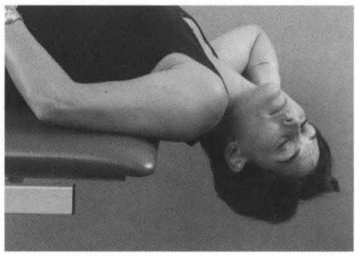
*44*



*45*



*46*



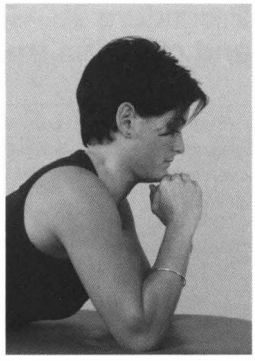
*47*

As with extension in sitting, a small rotary movement can be applied in the extended position to further increase the range of extension. A ***minimal rotary*** adjustment of the head position is repeated five or six times so that the nose moves only ***one centimetre (half an inch) to either side of the mid-line***. During this process the patient is urged to move further and further into extension so as to gain maximum end-range (Photo 47).

*Note:* Some patients may feel unable to tolerate this exercise when performed supine because of dizziness or nausea. This may pass after repetition as the patient becomes accustomed to the exercise. Should this problem persist in the supine position, the prone lying version should be used.

### Retraction and extension in prone

The patient lies prone on the treatment table leaning on the elbows with the chin resting on the hands (Photo 48). Alternatively the patient may interlock the fingers and rest the chin on both thumbs. The patient then retracts and extends the head and neck in the same manner required when the exercise is performed in sitting.



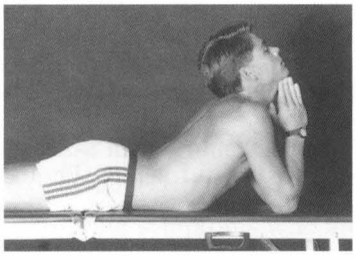
48

*Photo 48: From neutral head is retracted, then extended.*

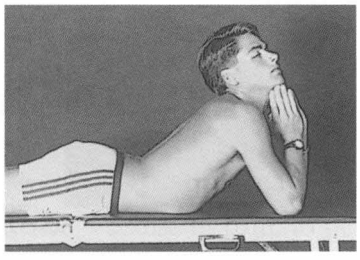
### Retraction and extension with patient overpressure in prone

After repeating the movement five or six times, the patient rests the chin on the outstretched fingertips with the head facing forward and upward in an extended position (Photo 49). To achieve full overpressure, it is important to have the patient as relaxed as possible. To achieve complete relaxation, the patient should allow the upper trunk to sag between relaxed shoulders. As the trunk sags between the shoulders, resistance against the outstretched fingers applies overpressure to the whole of the cervical and the upper thoracic spine. The position can be maintained for two or three seconds.

*Photos 49, 50: Overpressure is achieved by the weight of the upper trunk sagging against the resistance of the fingers (49). Further pressure is achieved by slight rotation movements at end-range extension (50).*



49

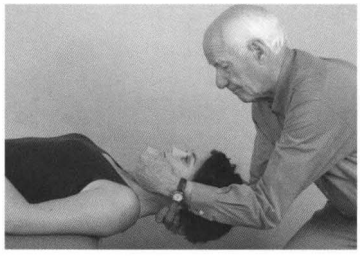


50

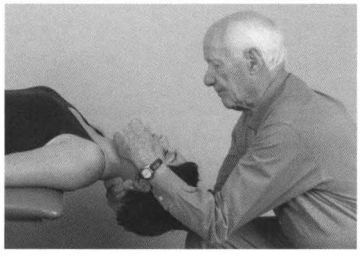
The rotary component described for previous procedures can now be added while maintaining the prone position. The upward pressure from the outstretched fingertips against the underside of the chin should be maintained as the rotation is commenced (Photo 50). Thus overpressure applies a gradual increase in the end-range loading.

## Procedure 2b – Retraction and extension with rotation and clinician traction in supine

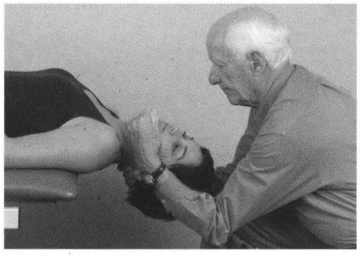
*Photos 51, 52, 53: Longitudinal traction (51) is followed by retraction (52), and then extension (53).*



51



52



53

The movement produced in this procedure is retraction of the head and extension of the cervical spine while under traction applied by the clinician.

The patient lies supine with the head and upper trunk over the end of the treatment table, unsupported down to the level of T3/4. The clinician provides support to the patient's head by placing one hand under the occiput with the thumb to one side and the fingers to the other side of the upper cervical segments. The clinician then places the other hand and fingers under the patient's chin and gently but steadily applies longitudinal traction (Photo 51). While maintaining a firm traction, the clinician fully retracts the patient's head and then extends the cervical spine by drawing the head down to the end of the available range of extension or as far as the patient can tolerate (Photos 52 and 53).

The patient remains completely relaxed throughout the movement. At the end-range of extension the traction forces are slowly but not completely reduced, and the rotary component described in Procedure 2a is applied. While maintaining a little traction the clinician should, in the fully extended position, rotate the head to alternate sides four or five times so that the nose moves only about *one centimetre (half an inch) to either side of the mid-line*. During the performance of this motion, the clinician attempts to obtain maximum end-range of extension.

The manoeuvre must be applied gently and slowly for the first two or three excursions. Throughout, there should be continuous monitoring

of the patient's symptoms. Providing the patient's pain is reducing or centralising or the range is improving, the procedure can usually be repeated five or six times in the first session.

## Application

Retraction and extension are essential components in the process of reduction of posterior derangement. In patients with acute or severe symptoms, persistent efforts to obtain improvement in the range of retraction must be made before extension is applied or appropriate. Retraction and extension are also important prophylactic exercises for patients required to work in prolonged flexed postures. The addition of the rotatory movement when in full extension increases the range of extension, and may be useful in those with more resistant derangements.

Procedure 2b is used for the reduction of posterior derangement in the cervical spine, especially of the very acute or resistant posterior derangement. It is particularly necessary for those patients whose symptoms improve with earlier progressions but who do not remain better as a result of their application. Sometimes it is the only way in which a posterior derangement may be reduced. Cervical extension may be impossible until the clinician applies traction in this way.

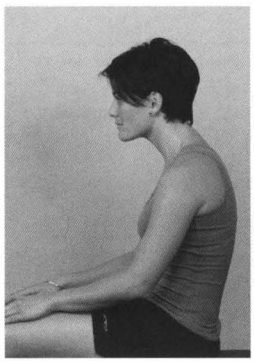
The unloaded lying position allows a better range of extension than can be obtained by performing this movement in either the sitting or standing position. The degree of pain experienced by doing the exercise in the unloaded supine lying position can be significantly less in some patients. This is advantageous when treating patients with acute symptoms who are unable, because of pain, to perform the exercise in the sitting or standing positions. If the patient is able to achieve total relaxation in the extended position, the weight of the head provides overpressure.

The adoption of the alternative prone position enables a greater margin of patient control, and many who are apprehensive about performing this exercise in the supine position are readily able to extend while prone.

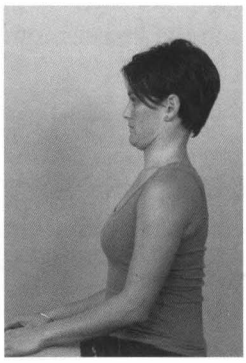
*Some patients are unable to tolerate retraction and extension, especially when performed in supine, because of dizziness or nausea. If this does occur, an alternative position or procedure must be tested.*

## Procedure 3 – Postural correction

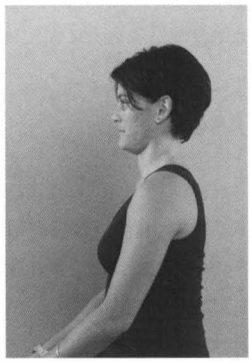
*Photos 54, 55, 56: Extreme of poor posture (54); extreme posture correction (55); followed by slight relaxation (56).*



54



55



56

### Slouch-overcorrect and posture correction

Sitting over the end or side of the treatment table, the patient is instructed to adopt a relaxed slouched posture with the lumbar and thoracic spine flexed and the head and neck protruded (Photo 54). The patient then smoothly moves into the extreme of the erect sitting posture with the lumbar spine in maximum lordosis and the head and chin maximally retracted (Photo 55). Some clinician guidance using gentle hand pressure on the patient's lumbar spine and chin may assist in the learning process. The patient is then instructed to relax back into the slouched position. This cycle should be repeated ten times so that the patient moves from the extreme of the slouched posture to the extreme of the upright extended and retracted posture. After completing ten cycles of the procedure the patient should hold "the extreme of the good position" for a second or two and then release 10% of the strain (Photo 56). *This is the posture the patient must aim for on a daily basis. It is the learning process for maintaining correct posture and is also therapeutic as some patients achieve centralisation of their pain using this procedure alone.*

## Application

Slouch-overcorrect is used to educate patients how to attain correct posture and demonstrates to them the difference between good and bad postures. Patients are often unaware of their body posture, and this procedure, practised regularly, helps them to become conscious of their poor sitting habits. Once the patient is able to attain the correct posture, they are then able to maintain the correction for increasing periods of time. As well as using slouch-overcorrect to retrain postural 'habit', done regularly it is also a useful way of training and

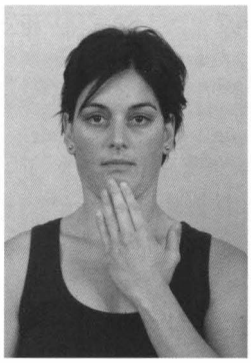
strengthening trunk muscles to support the spine in an upright position. Maintaining correct posture is a strengthening process in itself.

## Lateral principle

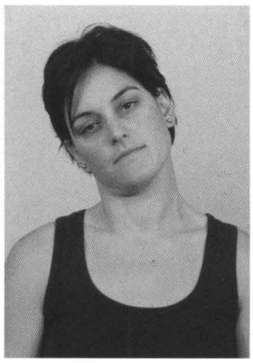
All lateral procedures are preceded by retraction.

## Procedure 4 – Lateral flexion

Can be performed in sitting and supine.



57



58

*Photos 57, 58: Retraction (57) followed by lateral flexion (58).*

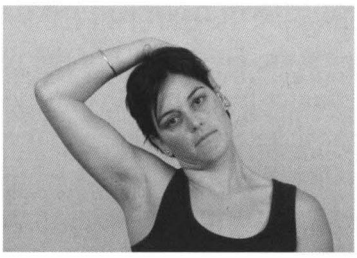
### Lateral flexion in sitting

In derangement, the seated patient first retracts the head (Photo 57) and then laterally flexes *towards the side of pain* (Photo 58). After a second in that position the patient returns to the upright position. The cycle of movement is repeated five to fifteen times so that the full available range is obtained.

## Procedure 4a – Lateral flexion in sitting with patient overpressure

Should the response be inadequate, it may be necessary to apply more pressure. To do this the patient stabilises the upper trunk by holding the seat base with the hand opposite to the side of pain. The patient then retracts, and places the other hand over the top of the head with the

*Photo 59: Overpressure is applied by the hand on the side to which the patient is bending.*

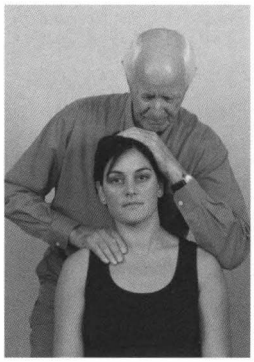


59

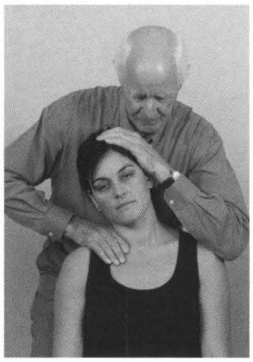
fingers reaching to the ear. With the head still retracted, the patient pulls the head towards the side of pain as far as possible (Photo 59). After a second in this position, and while keeping the hand in place, the patient returns to the upright position. The movement should be repeated about ten times. Care should be taken to avoid any rotation and if possible the movement should appear to be a lateral flexion only.

## Procedure 4b – Lateral flexion in sitting with clinician overpressure

*(Right lateral flexion for right-sided pain is described.)*



60

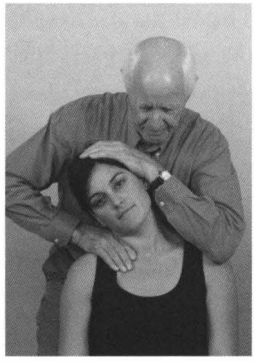


61

*Photos 60, 61: Overpressure is applied by the clinician through both hands, one on the side of the head and the other thumb on the spine.*

Patient position is as described above. The clinician stands behind the patient with the patient's head resting lightly on the clinician's chest. The tip of the clinician's right thumb rests on the right side of the spinous process of the upper thoracic level and the metacarpophalangeal junction of the right index finger rests against the lateral articular pillar of the cervical column at the appropriate level. The clinician's left hand is placed against the left side of the patient's head with the elbow resting on the clavicle and the fingertips on top of the patient's head (Photo 60). The clinician's forearms are positioned parallel to each other. The patient is asked to laterally flex their head to end-range. At the end of the movement, the clinician applies a downward pressure on the side of the patient's head with the left hand and a counter-pressure with the thumb on the spinous process and/or with the metacarpophalangeal junction of the index finger on the articular pillar (Photo 61). This accentuates the lateral flexion movement with pressure being applied with both hands. The position is held for one or two seconds, and then the patient returns to the upright position. The movement is repeated five or six times.

## Procedure 4c – Lateral flexion mobilisation in sitting



62

*Photo 62: The head is stabilised at end-range lateral flexion and mobilisation applied by the hand positioned on the spine.*

Patient and clinician positions are as described above. The clinician, with one hand, laterally flexes the patient's head towards the side of pain to the available end-range. While the head is held in lateral flexion, the clinician applies pressure through the thumb of the other hand on the lateral aspect of the spinous process (Photo 62). An alternative is for the clinician to apply the pressure with the metacarpophalangeal junction of the index finger of the right hand against the lateral articular pillar of the cervical column at the appropriate level. The pressure is applied in a direction towards the opposite shoulder or at the angle that favourably influences the symptoms.

The mobilisation should be repeated in a rhythmical fashion five to six times and then the head is returned to the neutral position. Providing the pain is reducing or centralising, the force applied may be progressively increased so that full end-range motion occurs. The natural coupled movement of rotation that occurs with lateral flexion is of course unavoidable, but obvious rotation of the head and neck must be kept to a minimum.

*Note:* To determine the point at which the motion is to be accentuated, it is necessary to test the effects of the application of pressure at different segmental levels. *The mobilisation and – if found to be necessary, the manipulation – are applied at the level that causes the symptoms to reduce, centralise or abolish. It is not appropriate to choose the level at which the manoeuvre is to be applied by relying on information obtained from palpation or radiography.*

## Procedure 4d – Lateral flexion manipulation in sitting

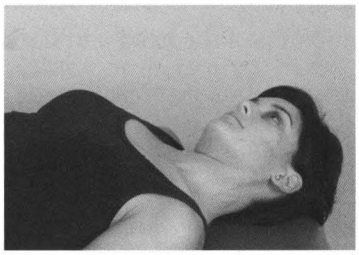
To progress the technique of mobilisation to that of manipulation, the positioning of both patient and clinician can remain the same as for lateral flexion mobilisation. Premanipulative assessment obtained when applying mobilisation techniques will already have determined the available range of motion and confirmed the correct direction of movement.

The clinician laterally flexes the patient's head to end-range (towards the side of pain) and the head is stabilised in this position. With the thumb against the lateral side of the spinous process or the metacarpophalangeal junction of the index finger of the right hand against the lateral articular pillar of the appropriate cervical segment, the clinician then applies a short amplitude, high-velocity movement at the end-range of lateral flexion. The head is then returned to the neutral position and the symptoms evaluated.

This procedure is required for patients whose symptoms are resistant to the previous manoeuvres and is a progression of Procedure 4c. Although the symptoms may be reduced or centralised by the previous procedures, they do not remain reduced and return shortly after the completion of the procedure. The direction of movement has been determined to be appropriate, but previous forces are inadequate to reduce the derangement.

## Alternative positions: Procedure 4 – Lateral flexion

*Photo 63: Lateral flexion in supine lying.*



63

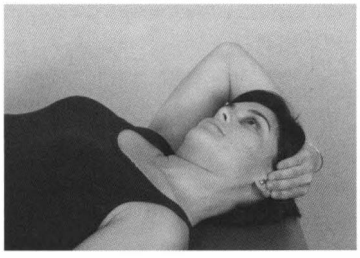
### Lateral flexion in supine

The patient lies supine on the treatment table. The head may be supported on a pillow if necessary. Placing a piece of shiny paper, such as a magazine, under the occiput allows a better lateral flexion movement to be performed. The patient is asked to laterally flex the head and neck towards the side of pain so that the ear approximates the shoulder (Photo 63). It is important for the patient to look straight upwards and avoid rotating the head. The movement is usually performed towards the painful side. The position is maintained for one or two seconds, and then the head is allowed to return to the neutral position. The movement is repeated about ten times.

### Lateral flexion in supine with patient overpressure

Patient position is as described above. With the head retracted, the patient places the hand over the top of the head so that the fingertips cover the ear. The patient pulls the head towards the side of pain as far as possible (Photo 64). The position is held for one or two seconds and the patient returns to the start position. The movement is then repeated about ten times.

*Photo 64: Patient overpressure is applied using the hand.*

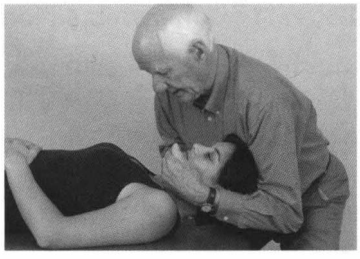


*64*

### Lateral flexion in supine with clinician overpressure

The patient lies supine on the treatment table, which is elevated to a height that enables the clinician to perform the manoeuvre with good control. The patient's head and neck should lie over the end of the treatment table and be supported by the clinician. The patient must remain relaxed throughout the procedure.

*Photo 65: Overpressure is applied by the clinician using both hands.*



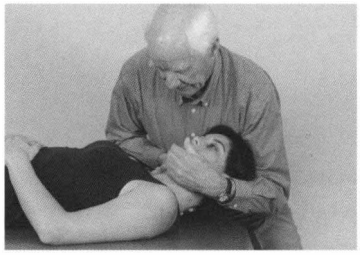
*65*

With one hand on the pain-free side, the clinician holds the patient's mandible and cradles the head between forearm and chest wall. The clinician's other hand is placed so that the metacarpophalangeal junction of the index finger rests firmly against the lateral articular pillar of the cervical column on the painful side. The patient laterally flexes their head towards the side of pain, and the clinician accentuates the movement to the end of range with both hands (Photo 65). The clinician then releases the pressure and the head and neck are returned to the neutral position. The movement is repeated five or six times.

### Lateral flexion mobilisation in supine

The starting positions for the clinician and the patient are as described in the procedure above. With one hand on the pain-free side, the clinician holds the patient's mandible and cradles the head between forearm and chest wall. The clinician's other hand is placed so that the metacarpophalangeal junction of the index finger rests firmly

*Photo 66: The head is stabilised at end-range lateral flexion with one hand while the other hand applies the mobilisation through the articular pillar.*



66

against the lateral articular pillar of the cervical column. The clinician laterally flexes the patient's head *towards the side of pain* (Photo 66). While the head is stabilized at end-range of lateral flexion, the clinician applies a mobilising pressure on the articular pillar. The manoeuvre may be repeated in a rhythmical fashion five to six times. Providing the pain is reducing or centralising, the force applied may be progressively increased so that full end-range motion is obtained. The pressure is then released and the head and neck are returned to the neutral position.

### Lateral flexion manipulation in supine

To progress the technique of mobilisation to that of manipulation, the positioning of both patient and clinician remains the same as for the lateral flexion mobilisation.

The patient's head and neck are moved to the end-range of lateral flexion towards the side of pain. The clinician using the metacarpophalangeal junction of the index finger against the lateral pillar, applies a short amplitude, high-velocity thrust to the end of the range of motion. During this process the hand on the other side stabilises the patient's head and neck. The head and neck are then returned to the neutral position.

## Application

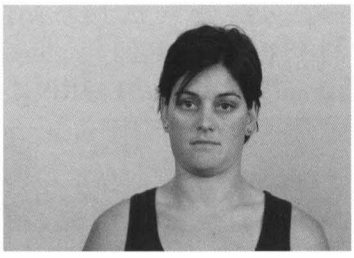
Lateral flexion procedures are used for the reduction of derangement with a relevant lateral component. The conceptual model for the treatment of patients with lateral or posterolateral derangement is that, if pain is felt unilaterally, any displacement present must be towards the side of pain. By laterally flexing towards the painful side, compressive loading in the lateral compartment of the disc moves displaced tissue towards the side of least loading. This is indicated by the movement of pain to the mid-line. Should the motion of lateral flexion be excessive or prolonged, it is not uncommon to hear patients describe that their symptoms have moved to the opposite side. As with all other procedures,

patient-generated forces are used first; force progressions are added only when the symptoms have not responded.

## Procedure 5 – Rotation

Can be performed in sitting or supine.

*Photos 67, 68: From neutral upright posture (67) to rotation (68).*



67

68

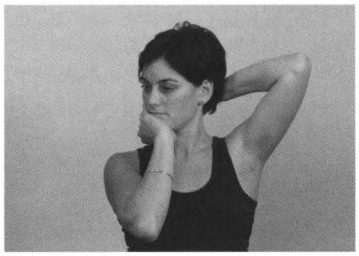
### Rotation in sitting

As with the other cervical procedures, this manoeuvre starts from a position of retraction, which must be retained during the movement of rotation. The patient sits erect in a straight-backed chair. The patient first retracts (Photo 67) and then rotates the head towards the side of pain (Photo 68). After a second in that position the patient returns to the neutral position. The cycle of movement is repeated ten to fifteen times so that the maximum available range is obtained.

## Procedure 5a – Rotation in sitting with patient overpressure

Should the response be inadequate, it may be necessary to add more pressure. The patient retracts the head and places the hand of the non-painful side behind the head with the fingers over the ear on the painful side. The palm of the other hand is placed against the chin on the opposite side (Photo 69). With the head still retracted, the patient turns the head towards the side of pain as far as possible and accentuates the movement by applying overpressure with both hands (Photo 70). After a second in this position, and while keeping the hands in place, the patient should return to the neutral position. The effects on pain are recorded. The movement should be repeated about ten times.

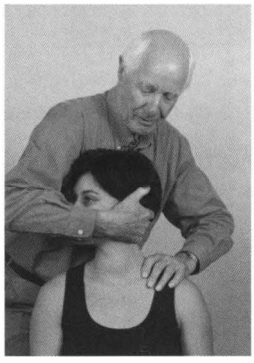
*Photos 69, 70: Overpressure is applied through the mandible and occiput.*



69

70

*Photo 71: Overpressure is applied via the occiput and the spinous process.*



71

## Procedure 5b – Rotation in sitting with clinician overpressure

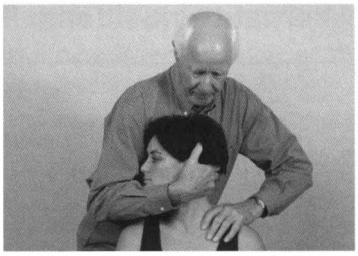
*(Right rotation is described.)*

The patient sits upright in a straight-backed chair with the head in a slightly retracted position. The clinician stands behind the patient with their left hand resting lightly on the patient's left trapezius. The fingers should rest over the clavicle and the tip of the thumb is placed firmly against the left side of the spinous process at the level below that being mobilised. The clinician cradles the patient's head with the right hand with the ulnar border of the right hand being placed along the line of the articular pillar at the symptomatic level. The patient is asked to rotate their head to end of range, at which point the clinician produces a further rotation force using the right arm to rotate the head and the left hand applying a counter-pressure against the spinous process at the segment below. The position is maintained for one or two seconds and then the head is returned to the neutral position. The movement is repeated five or six times.

## Procedure 5c – Rotation mobilisation in sitting

The patient sits upright in a chair with the hands resting on the top of the thighs. The clinician stands behind the patient with one hand resting lightly on the patient's shoulder with the fingers anteriorly and the thumb firmly placed against the spinous process at the desired level on the side opposite to the pain. The clinician cradles the patient's head with the right hand and places the ulnar border of this hand

below the occipital protuberance. The clinician rotates the head to the end-range and stabilises it in this position (Photo 72). With the left thumb against the spinous process, the clinician applies a counter-pressure to accentuate the rotation and then the pressure is released. The manoeuvre may be repeated in a rhythmical fashion five to six times, and then the head and neck are returned to mid-line. The force should be progressively increased to the maximum range, providing the pain is decreasing or centralising.



72

*Photo 72: With the left thumb against the spinous process, the clinician applies a counter-pressure to accentuate rotation, and then the pressure is released.*

Gentle traction can be applied with the arm cradling the head prior to rotating the neck. This procedure is required for those patients whose symptoms are resistant to the previous manoeuvre. To determine the point at which the motion is to be accentuated, it is necessary to test the effects of pressure application at different segmental levels. The mobilisation and – if found to be necessary, the manipulation – are applied at the level that causes the patient's symptoms to decrease, centralise or abolish. *It is not appropriate to choose the level at which the manoeuvre is to be applied by relying on information obtained from palpation or from radiography.*

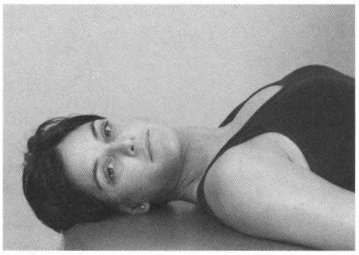
After two or three sessions of mobilisations spread over a period of six or seven days, the patient's symptoms should resolve. If no response is obtained by that time it may be necessary to apply the progression of manipulation, but manipulation should not be applied routinely to all patients.

## Procedure 5d – Rotation manipulation in sitting

With the hands positioned as for rotation mobilisation, the clinician turns the patient's head towards the side of pain so that the cervical spine is at the end-range of rotation. The head is stabilised in this position, and the clinician applies a short amplitude, high-velocity movement *with the thumb against the spinous process* on the other side. The degree of end-range will already have been determined during the premanipulative mobilisation.

## Alternative positions: Procedure 5 – Rotation

*Photo 73: Rotation in supine.*

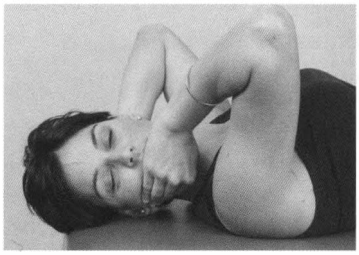


73

### Rotation in supine

Patient lies supine on the treatment table. The head may be supported on a pillow if necessary. Placing a piece of shiny paper under the occiput allows a better rotation movement to be performed. The patient turns the head, generally towards the side of the pain (Photo 73). The position is maintained for one or two seconds, and then the head is returned to the neutral position. The movement is repeated about ten times.

*Photo 74: Rotation with patient overpressure in supine.*



74

### Rotation in supine with patient overpressure

If a progression of force is required, then patient overpressure should be used. The patient turns the head, generally towards the side of the pain. At the end of the rotation range the patient places the heel of their hand along the mandible (Photo 74) and applies an overpressure to accentuate the rotation movement. The position is maintained for one or two seconds and then the head is returned to the neutral position. The movement is repeated about ten times.

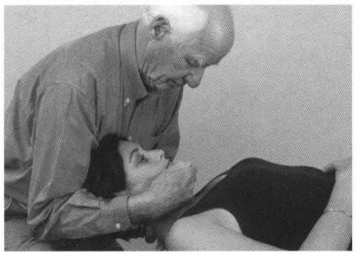
### Rotation in supine with clinician overpressure

*(Right rotation is described.)*

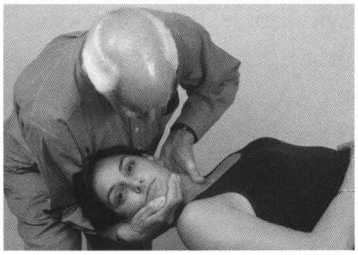
The patient lies supine with their head and neck, supported by the clinician, off the end of the treatment table. The table should be at the height of the lower abdomen or at a position where the clinician can control the manoeuvre. The patient should be relaxed. The clinician stands on the non-painful side of the patient and cradles the patient's head in their right hand with the fingers lightly grasping the patient's mandible (Photo 75). The clinician's left hand is placed so that the radial aspect of the metacarpophalangeal joint of the index finger is placed against the lateral pillar of the cervical spine on the non-painful side at the appropriate level. The patient rotates their head while the clinician cradles the head with the right hand. At the end of the range of rotation the clinician accentuates the rotation movement by applying a rotary pressure to the articular pillar on the non-painful side (left),

and by using the right hand on the mandible. The patient returns the head to the neutral position and the movement is repeated five or six times, progressively increasing the range with each movement.

*Photos 75, 76: Overpressure is applied via the jaw and the articular pillar. With the head stabilised at end-range rotation with one hand/arm the other hand applies the mobilisation force through the articular pillar.*



75



76

### Rotation mobilisation in supine

The patient and clinician positions are as described for rotation with clinician overpressure. With the arm holding the patient's head, the clinician rotates the cervical column to the maximum end-range while the other hand accentuates pressure in rotation at the appropriate level (Photo 76). The pressure is then released and re-applied rhythmically five or six times as a mobilisation. The clinician returns the patient to the neutral position. The motion should initially be carried out *towards the painful side.*

### Rotation manipulation in supine

*(Right rotation is described.)*

The patient and clinician positions are as for rotation mobilisation in supine. Using the right hand, the clinician rotates the patient's head and neck to end-range. With the metacarpophalangeal junction of the index finger of the left hand against the lateral articular pillar of the appropriate segment, the clinician applies a short amplitude, high-velocity movement at the end of the range of motion. The head is returned to the neutral position and the symptoms assessed.

## Application

With all the rotation procedures it is recommended that the patient rotate the head and neck repetitively towards the side of pain in order to decrease, centralise or abolish symptoms. There are two reasons for this recommendation. Clinical experience shows that more patients experience centralisation of their symptoms by rotating towards the pain than occur by rotation away from the pain. However, if no

response is forthcoming, rotation away from the side of pain should be investigated.

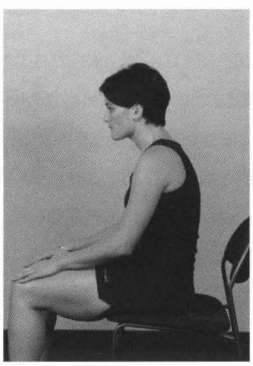
Rotation is used for the treatment of posterolateral derangement and for rotation dysfunction. It is most often required for patients with unilateral symptoms arising from the mid- and upper segments of the cervical spine, that radiate or are referred and that are not reducing or centralising with repetitive sagittal movements. This includes patients with unilateral cervical headache who have not improved with the initial procedures of flexion.

Rotation applied to the mid- and upper cervical segments usually produces change in the patient's symptoms within twenty-four to forty-eight hours of its introduction. If the manoeuvre fails to cause change in the location or intensity of the patient's symptoms within this period, it should be abandoned.

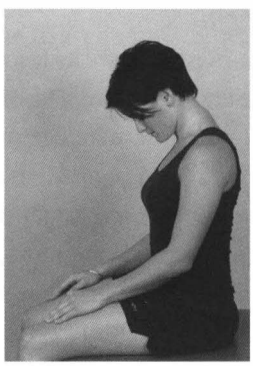
The performance of rotation may be discontinued once the patient's pain has centralised or when improvement ceases. The patient should, however, continue with retraction and extension sitting or if necessary lying in order to obtain complete reduction of the derangement.

## Procedure 6 – Flexion

Can be performed in sitting or supine.



77



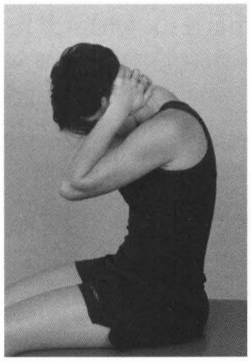
78

*Photos 77, 78: From relaxed sitting to flexion.*

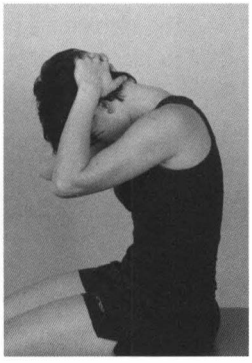
### Flexion in sitting

The patient should be seated and relaxed (Photo 77). The head should be bent forwards so that the chin is as near to the sternum as possible (Photo 78). The patient is asked to return the head to the upright position. The patient should repeat the movement in a rhythmical fashion five to fifteen times.

## Procedure 6a – Flexion in sitting with patient overpressure



79



80

*Photos 79, 80: Flexion with patient overpressure, directed at lower (79) and upper (80) cervical spine.*

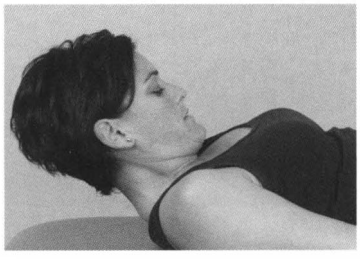
If the response to the exercise is inadequate, the following progression should be applied to ensure that maximum range of motion is achieved. The patient should be instructed to interlock the fingers of both hands behind the upper neck and occiput and repeat the movement as described above. On reaching the end-range position, the patient should apply overpressure with the clasped hands, hold for a second (Photos 79 and 80) and immediately return to the upright position. The patient should repeat the movement in a rhythmical fashion five to fifteen times.

## Alternative positions: Procedure 6 – Flexion

### Flexion in supine

The patient lies supine on the treatment table. The head may be rested on a pillow. The patient lifts the head onto the chest as near to the sternum as possible. The position should be held for one or two seconds and then the head returns to the upright position. The movement should be repeated rhythmically about ten times.

*Photo 81: Flexion in supine - the patient lifts the head to bring the chin towards the sternum.*



81

*Photo 82: Flexion in supine with patient overpressure.*

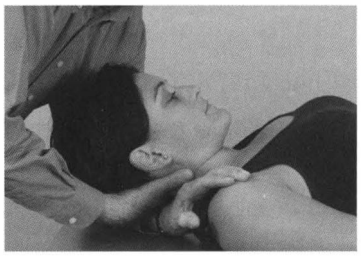
*82*

### Flexion in supine with patient overpressure

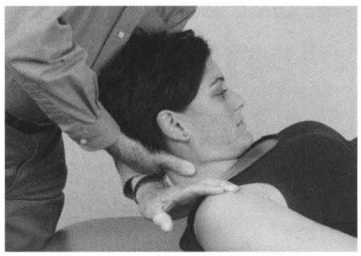
Patient position is as described above. The patient should be instructed to interlock the fingers of both hands behind the upper neck and occiput and repeat the movement as described above. On reaching the end-range position, the patient should apply overpressure with the clasped hands, hold for a second (Photo 82) and immediately return to the lying position. The patient should repeat the movement in a rhythmical fashion five to fifteen times.

## Procedure 6b – Flexion in supine with clinician overpressure

*Photos 83 and 84: Flexion in supine with clinician overpressure.*



*83*

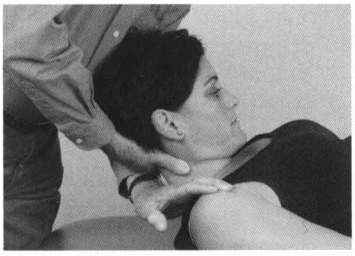


*84*

The patient lies supine with the head at the extreme end of the treatment table. The clinician stands at the end of the table and holds the occiput in the palm of one hand with the finger and thumb cradling the atlas and axis. The clinician's other hand is passed under the wrist or forearm and rests palm down on the patient's shoulder (Photo 83). The patient is asked to flex the chin towards the chest while the clinician raises both forearms, lifting the patient's occiput, and at the same time applying counter-pressure with the hand on the patient's shoulder (Photo 84). The position is held for one or two seconds and then the head is returned to the neutral position. The movement is repeated five or six times.

## Procedure 6c – Flexion mobilisation in supine

*Photo 85: Lower cervical flexion mobilisation.*



85

Patient and clinician positions are as described above. The clinician flexes the patient's head and cervical spine by raising the forearms and the patient's occiput, and at the same time applies counter-pressure with the hand on the patient's shoulder. At the end-range of flexion the shoulders are stabilised and a flexion mobilisation force is applied to the occiput. The force may be applied either sagittally or to either side of the mid-line, depending on the location of pain. The position is held for one to two seconds and then the patient's head and neck are returned to the neutral position. The movement is repeated rhythmically five to six times.

## Application

Flexion can be performed differently depending on whether the upper or lower cervical spine is being targeted. When the focus is on the upper cervical spine for cervical headaches, retraction should be performed before flexion.

Flexion procedures are used for the reduction of anterior derangements, for the recovery of function following a posterior derangement, for the remodelling of a flexion dysfunction and an adherent nerve root. However, flexion procedures are most often required for the treatment of cervical headache.

# 15: Clinical Reasoning



## Introduction

The overall management of patients involves more than examination processes and the treatment techniques selected. A management path is determined by the clinical reasoning process of the examining clinician in addition to the clinical knowledge base and the inherent and learned biases of the clinician. It is also influenced by the clinician's scope of practice, the practice setting, and the bias and preconceived notions of the patient. This chapter examines the role of clinical reasoning in patient management.

Clinical reasoning has been defined as the *"thinking and decision-making associated with clinical practice that enables therapists to take the best-judged action for individual patients. In this sense, clinical reasoning is the means to 'wise' action"* (Jones and Rivett 2004). It is clinical reasoning that matches findings to patterns of clinical presentations, that excludes 'red flags', that considers which examination procedures are necessary and which are not, that determines what to do if responses are atypical or unclear, that addresses patient's concerns, and so on.

On a practical level, clinical reasoning is the process of deciding what problem the patient has, and, from your knowledge base, deciding what can be done about it. First this involves the discovery of the 'character' of that problem – the patient is the best witness to this, and interview skills must be capable of determining a clear 'big picture' of their presentation. Second, a rounded knowledge base is needed to provide practitioners with an understanding of diverse factors: the variety of clinical presentations, serious pathology, the natural history of a condition, pathophysiological changes, management strategies, the evidence base, the effect of an intervention, etc. The third and perhaps most vital element is the ability to reason between the practical reality of the patient's presentation 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. This chapter discusses some of the aspects involved in clinical reasoning as defined in the literature on this topic. However, there are limitations and contradictions about the use of clinical reasoning that are also touched upon. Finally, the chapter presents a clinical example in which a reasoning process based on a mechanical diagnosis and therapy viewpoint is given.

The sections in this chapter are as follows:

- clinical reasoning
- elements that inform the clinical reasoning process
- data-gathering
- knowledge base
- clinical experience
- cognition and meta-cognition
- errors in clinical reasoning
- clinician bias
- mechanical diagnosis and therapy and clinical reasoning
- example of clinical reasoning process.

## Clinical reasoning

Clinical reasoning is the cognitive and decision-making process involved in health care practice that is used in the diagnosis and management of patients' problems (Terry and Higgs 1993; Jones *et al.* 1994; Christensen *et al.* 2002). Two methods for clinical reasoning have been proposed, based either on pattern recognition or on a process of hypothetico-deductive reasoning (Jones 1992; Terry and Higgs 1993).

Hypothetico-deductive reasoning describes a process of hypothesis generation based on information gathered from the patient. The hypothesis is then tested out or further ones generated 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.

An alternative model is based on pattern recognition gained from certain features in a clinical presentation that remind the clinician of previously seen clinical problems. In this model, management strategies are derived from previous experience rather than an experimental 'try it and see' method.

Pattern recognition is only possible with a well-organised knowledge base and plentiful clinical experience; thus, it is generally only available to experienced clinicians. In the face of atypical problems, when pattern recognition is not possible, the expert reverts to hypothesis testing. The novice clinician tends to have to use hypothesis testing more frequently as they slowly develop their own clinical experience (Jones 1992).

Although pattern recognition is a powerful part of expert clinical reasoning, it also probably represents the greatest source of errors in clinical thinking (Jones and Rivett 2004). Three main categories of clinical reasoning errors have been identified: forming a wrong initial concept of the problem; failure to generate plausible hypotheses and test them adequately; inadequate testing and premature acceptance of a hypothesis (Rivett and Jones 2004).

## Elements that inform the clinical reasoning process

Certain factors are said to inform the clinical reasoning process – namely data-gathering skills, aspects of the knowledge base available to the clinician, clinical experience and meta-cognition skills (Terry and Higgs 1993; Jones 1992; Jones *et al.* 1994). It is thus a complex and cyclical process as suggested by the model of clinical reasoning for physiotherapy proposed by Jones (1992). At every stage in this process errors may occur that could affect the reliability or validity of the reasoning process (Jones 1992).

## Data-gathering

Data-gathering is the process of discovery about the patient's problem undertaken during the history-taking and the physical examination. The patient has available the essence of the problem; the skill is in accessing it. Patients know the information that clinicians need to know, but not in the same format and often without the ability to prioritise the key pieces of information, and the unwary clinician may

be flooded with a large amount of irrelevant information by some patients. Data-gathering requires empathy and active listening by the clinician and the knowledge base to ask the appropriate questions when the initial response is unclear. Unless the situation is relaxed, friendly, respectful and non-judgemental, the patient is unlikely to tell his or her whole story.

During the history-taking, considerations and hypotheses are raised and then rejected, or retained for further probing. Does the patient have any features suggestive of serious spinal or nerve root pathology? Does their problem sound mechanical? Is there the suggestion of a mechanically determined directional preference, and what features are there to give an idea about prognosis? Has enough information been gathered on baseline symptomatic and functional levels against which to judge later changes? Does the patient's response to their condition suggest an exaggerated reaction to relatively trivial symptoms? These and other questions should be considered and reflected on during the patient interview. From the history-taking, an overall picture of the patient's condition should have been gained and the main elements of the physical examination should be suggested.

The physical examination is not a routine series of tests performed uniformly on every patient; it should follow on directly from the data-gathering and hypothesis testing of the history-taking. Findings from the physical examination may confirm what is already indicated by the history. It is always important to ensure that sufficient baseline mechanical and symptomatic data is collected against which to make later comparisons. Data collection continues until a decision can be made about management strategies. The decision may be provisional, in which case further data will be gained at the next session and from the patient's response to the proposed management strategy.

Data-gathering does not stop at the end of the first session, but continues on all subsequent occasions to ensure that optimal management is being maintained.

## Knowledge base

Clinical practice requires a wide-ranging breadth of knowledge from different fields. Jones *et al.* (1994) list the following topics as relevant to the knowledge base of physiotherapy: anatomy, physiology, pathophysiology, procedures, patterns of clinical presentation and concepts.

Elsewhere it is stated that hypothesis-making happens in six key areas: the mechanism of symptoms, differential diagnosis, predisposing or contributing factors, precautions or contraindications, management and prognosis (Jones and Butler 1991; Jones 1992; Jones *et al.* 1994). Needless to say, the knowledge base must remain current and therefore be regularly updated.

## Clinical experience

Clinical reasoning also requires clinical experience. It is only having seen hundreds of patient presentations that patterns are recognised and skills of data-gathering are mastered and focussed into generation and confirmation of a hypothesis. This does not happen automatically; the process of assessment must be learned and the potential clinical patterns must be appreciated. It is possible to benefit from others' clinical experience, through case studies and other literature, and through discussion and case reviews with colleagues. It also requires thought – just seeing numerous patients will not necessarily make you a better clinician or allow you to correctly identify meaningful patterns.

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 by itself does not necessarily lead to improved clinical reasoning. It is important to stay open to new ideas and to keep abreast of current literature and evidence, but at the same time to recognise that in physiotherapy certain practices sometimes become widely established with limited credible evidence.

## Cognition and meta-cognition

Cognition refers to the thinking processes involved during data-gathering, the application of a knowledge base, and clinical experience. It is these thinking processes that guide 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). In essence this is thinking about your thought process, being aware of the facets of this discussed above as well as the potential

errors mentioned below. This is no easy process as we tend to think in our 'comfort zone' – *"we are stymied by the fact that we are using our own interpretive filters to become aware of our own interpretive filters!... A self confirming cycle often develops whereby our uncritically accepted assumptions shape clinical actions which then serve to confirm the truth of these assumptions"* (Brookfield 2000).

## 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). Errors may arise from inherent or learned biases.

As a means of determining management strategies, clinical reasoning based on pattern recognition can have drawbacks. Failure to fully explore all options and bias to one's favourite diagnosis can encourage premature dismissal of alternative hypotheses. Pattern recognition, on its own, may be insufficient if it ignores certain complicating factors, such as exaggerated fear-avoidance.

Typical errors of clinical reasoning are making assumptions without further checking; prematurely limiting hypotheses under consideration; failure to gather enough information; attending to those features that accord with a favoured hypothesis while ignoring contradictory information; and gathering redundant information (Jones 1992). Failure to listen carefully to a patient may lead to ignoring a key piece of information and a false trail of hypothesis generation. 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.

Data-gathering skills vary with different presentations. Sometimes close questioning concerning symptomatic responses to different mechanical loads is necessary to determine the correct management strategy. At other times, as in some 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.

## Clinician bias

The clinical reasoning literature generally fails to explore clinician bias. If clinical reasoning was a universal language, all clinicians would process the information in a similar way and reach broadly similar conclusions. As it is, the process is different amongst clinicians; different data is sought and gathered and consequently different end points are reached. Clinicians come to different conclusions, using terms such as instability, hypomobility, 'facet' joint syndrome, osteopathic lesion, fixations, and so on that reflect their training and prejudices rather than an 'objective' truth. Trying to establish patho-anatomical diagnoses is fraught with difficulties regarding intertester reliability and validity. Furthermore, *"it is not satisfactory simply to identify structures involved, as this alone does not provide sufficient information to understand the problem and its effect on the patient, nor is it sufficient to justify the course of management chosen"* (Jones and Rivett 2004).

Conclusions from clinical reasoning thus lie in the eye of the beholder. If the concept described a distinct process, it should lead all clinicians to the same point regardless of profession or training. Clinicians frequently arrive at completely different conclusions using their version of clinical reasoning and there is a lack of universal agreement on what action to take because they have a different knowledge base. Diagnoses are often made based on procedures that lack reliability and validity, with the favoured treatment approaches of the time being commonly applied without clear indications for use. There is a failure to logically link management to a reliable or well-tested examination process.

## Mechanical diagnosis and therapy and clinical reasoning

Clearly elements of the classical description of clinical reasoning are as relevant to the McKenzie Method as any other. Data-gathering, knowledge base, clinical experience and thought processes during the clinical interaction are all central to mechanical diagnosis and therapy. However, there are limitations and problems with the process of clinical reasoning as described.

The list of what knowledge base is needed is considered incomplete, as knowledge from other areas can be required also (McKenzie and

May 2003). Clinicians also need to be informed about epidemiology, the research evidence, and issues about psychology and communication with patients. Any of these factors, and more, may provide useful clinical information on different occasions. The value of some of the more important aspects of the knowledge base has been previously mentioned (McKenzie and May 2003). Whilst earlier descriptions of clinical reasoning had very little to say about patient education, the central role this has in the clinical process has been recognised more recently (Jones and Rivett 2004; Resnik and Jensen 2003). The original clinical reasoning model has been expanded to include a collaborative component with the patient acting as a partner in the clinical reasoning process (Christensen *et al.* 2002). This *therapeutic alliance* has always been a key element of mechanical diagnosis and therapy (McKenzie and May 2000).

In a qualitative study exploring the characteristics of expert clinicians, defined by their better outcomes rather than their years of experience, it was the use of a patient-centred approach to care that distinguished the expert from the average clinician (Resnik and Jensen 2003). In a patient-centred approach a primary aim is empowerment of the patient and increasing self-efficacy, *"accomplished through patient education, avoiding passive modalities, minimizing unnecessary visits, and helping patients to develop self-management strategies"* (Resnik and Jensen 2003). All these elements have always been at the core of mechanical diagnosis and therapy (McKenzie 1981, 1990). It is recognised that management involves both a mechanical intervention (principally patient-centred, with clinician interventions reserved for when patient forces are inadequate), but also an educational component (McKenzie and May 2003). See McKenzie and May (2003, Chapter 18) for a fuller discussion about the importance of a patient-centred management strategy.

In mechanical diagnosis and therapy, pattern recognition is the basis of a classification system of non-specific mechanical syndromes. A syndrome is a characteristic group of symptoms and a distinguishing pattern of responses. The mechanical syndromes described by McKenzie (1981, 1990) allow the novice practitioner easy access to pattern recognition. The value of pattern recognition of a particular syndrome is the automatic link between syndrome and management strategy. Pattern recognition based on centralisation and symptom response has demonstrated reliability (Aina *et al.* 2004). There is a logical

link between the symptomatic and mechanical responses during the physical examination, when a mechanically determined directional preference is established, and the management that follows.

The use of the mechanical diagnosis and therapy assessment form focuses the data-gathering around certain key areas, which should be sufficient in most cases and thus avoids the gathering of redundant information that will not help in decision-making. Repeated movements and progressive loading are used to determine the appropriate management strategy. Force progressions, force alternatives or further procedures or investigations are introduced if needed according to the symptomatic and mechanical responses.

Within mechanical diagnosis and therapy, if there is a failure to establish a syndrome classification initially, there is a system for further investigation and deductive reasoning. History-taking may reveal 'red flags' indicative of serious spinal pathology; force progressions and force alternatives allow exploration of mechanical responses that need further examination; and if there is failure to establish a mechanical syndrome, a non-mechanical cause should be considered.

## Example of clinical reasoning process

In the following illustration some examples of the clinical reasoning process are given in italics. In this clinical example not all possibilities are explored; the main emphasis is on trying to establish a mechanical diagnosis and appropriate management. The data gathered was relevant to this end and another clinician with an alternative perspective could have focussed on other aspects of the case. It should also be noted that the patient initially displayed a number of poor coping responses to her problem, typically labelled 'yellow flags' or 'barriers to recovery'. However, following a thorough assessment process, good listening skills by the clinician, good rapport between clinician and patient, and a convincing management strategy, the patient responded to the intervention and the 'yellow flags' disappeared. This highlights the fact that overly focussing on poor coping strategies, rather than performing a thorough mechanical evaluation, may actually undermine patients' self-efficacy and reduce their ability to cope. By being provided with education and self-management for the problem, in this instance the apparent 'barriers to recovery' rapidly ceased to exist.

## Session one

### The history

A 58-year-old woman is referred to the physiotherapy department. She normally works as a receptionist at a doctors' surgery, but she has been off sick for four weeks with neck and arm pain. The work involves mostly sitting and working at a computer, although she moves around some of the time. She does not normally take any regular exercise and is overweight. Since onset of her symptoms she has been even less active than usual, limiting both her social and domestic activities. On the Neck Disability Index (NDI) she scores thirty-four out of a possible total of fifty, indicating severe self-reported disability (Vernon and Mior 1991); and on a zero-to-ten pain numerical scale she rates her pain as eight.

> *Initial interaction with this woman is rather difficult; she is curt in her replies and gives the impression that the interview is superfluous. She demonstrates several times during the interview very apparent pain behaviours, such as grimacing on movement or clutching and massaging her neck and arm. Her neck problem has led to a major loss of normal work, domestic and social activity, with her functional disability and pain scores being very high. She does not volunteer any keen interest to resume any of these activities. Her work involves a lot of sustained cervical flexion, but as she is generally not very active, so does her non-working life. Initial impressions suggest that her response to her problem is rather exaggerated and disproportional, and that 'yellow flags' may act as barriers to recovery.*

Her present symptoms are right-sided neck, scapular, arm and forearm pain and pins and needles in her thumb and index finger. She is rather uncertain as to when symptoms started, but thinks that about two to three months ago she woke with pain at the base of her neck. Initially it did not worry her much as she thought it would go away, as it had done in the past. This time it did not go away, but over several weeks spread into her shoulder blades and out onto her shoulders. At one point she discussed her problem with one of the doctors, who suggested simple analgesia and some range-of-movement exercises. This seemed to be helping until one morning she woke and the symptoms were mostly on her right side, and then over the following few days spread into her right arm. She remained at work for several weeks more, but the pain in her arm gradually worsened, spread into her forearm and was occasionally accompanied by pins and needles in her fingers. One

of the doctors suggested she have an x-ray, which revealed 'widespread degenerative changes'; she was told these would not get any better. By this time she had pain around
by arm movements – she felt she had developed a shoulder problem as well. She admitted at this point being 'thoroughly fed up with it all'. She was taking NSAIDs and analgesics, neither of which seemed to do much except provide temporary lessening of symptoms. She felt the job was making her worse and asked for a sick note off work and a referral to a 'specialist'. The doctor signed her off work, but instead referred her for physiotherapy. Although initially being off work seemed to be easier, overall in the last few weeks she feels her symptoms are unchanging.

*She is clearly distressed by her apparently unremitting symptoms, in part because she feels she has been mismanaged and wanted to see a 'specialist'. She also has been given some rather unhelpful information concerning the x-rays that were taken, and nothing so far has given her any control over her symptoms. Indeed they have got worse over time and she is now having problems moving her shoulder as well as her neck. These painful sites may well be related rather than separate problems. Although being off work is not making her better, she is reluctant to return to work. However, despite these negative feelings and thoughts, there are pieces of information to suggest a mechanical neck problem resulting from a derangement. The insidious onset, the spread of the pain, the emergence of arm pain and the paraesthesia are all suggestive of derangement, although at this stage more information is required.*

*A lot of effort during this initial session is needed to ensure that she is well informed about the nature of her problem, the role of x-rays to rule out 'serious' disease but provide little else of clinical value, and the importance of movement to recovery. The degenerative changes were present before she had symptoms; they will be present when the symptoms resolve, and they are not necessarily relevant. Information and addressing her specific problems and questions are key to getting her cooperation. It is also essential as early as possible to find strategies with which she can begin to control her symptoms – this is the best way to gain her confidence.*

She reports that the symptoms around her neck and shoulder blade are constant; they are there 'from the moment she wakes up to the moment she goes to sleep', whilst the symptoms in her arm are inter-

mittent. They are in the arm about 75% of the day, but in her forearm only about 25% of the day. The pins and needles are infrequent, perhaps once or twice a day for half an hour or so, and she thinks they are probably less noticeable now than last month. She finds it difficult to identify activities that make her better or worse, as with several activities her response varies depending on the length of time she remains in that position. Sitting with her neck supported eases symptoms at first, but then they get worse; walking around has the same response. She is easiest when she goes to bed, but is woken several times each night by pain, although she usually gets back to sleep relatively easily. She uses two pillows and sleeps mostly on her back; she thinks it is when she turns over that she wakes. There is no position that always makes her better, but she has noticed that the position that most consistently causes her symptoms to worsen is when her neck is bent for a sustained period, as in reading, preparing food or ironing. Shoulder movements and neck movements are painful and both are restricted.

*Certain aspects of the problem have become clearer. The mechanical nature of her symptoms appears to be confirmed by the intermittency of her arm symptoms, and by pain and restriction on neck and shoulder movements. The absence of arm symptoms for at least a portion of the day means that at certain times the pain-generating mechanism is lessened. The physical examination will be used to explore which mechanical factors reduce the pain-generating mechanism. From the history so far no clear relieving factors stand out; however, she has clearly identified sustained flexion as an aggravating mechanical force.*

*It is obviously necessary to take baseline measurements of range of shoulder and neck movements; however, at this point no direct intervention will be aimed at the shoulder. Very often in such instances, where the initial and primary problems are cervical, when this is addressed the apparent 'shoulder' problem goes away. However, this is not always the case, and sometimes it becomes clear that a secondary and genuine shoulder problem has arisen. The true situation will become more evident on later review.*

She reports occasionally feeling nauseous and dizzy when symptoms first got worse and spread down her arm, but not recently. She has had multiple previous episodes of neck pain; she thinks more than ten but cannot be certain of the exact number. In the past these have

always been short-lived, a few days to a week at the most, and also only involved symptoms around her neck. She has never previously sought treatment. For this episode she is still taking the analgesics, two tablets four times a day. A few days ago she ran out of NSAIDs. Since then she does not feel symptoms have changed at all, but wants advice about continuing with them or stopping. She reports that she is not taking medication for any other problem, has had no serious health problem in the past, and feels well except for the neck/arm pain. There is no history of major surgery, accidents or unexplained weight loss.

*History of previous neck pain, as here with multiple self-resolving episodes, is common amongst patients with derangement; this is in accordance with the mechanical-sounding nature of the problem already explored. Her negative responses to various 'red flag' questions further suggest a mechanical neck problem and absence of serious spinal pathology. The NSAIDs do not appear to have helped, and as she has conveniently stopped, at this point it is better to advise no additional tablets until mechanical therapy has been fully explored.*

*As she recognises the thoroughness of the interview and the fact that attempts are made to answer her concerns and questions, she begins to relax and becomes less defensive – this is clearly the first time she has been able to tell her story in full. She is reassured by some of the comments and begins to seem less anxious and demotivated. The mechanical nature of the problem becomes clearer as the history is taken – it sounds like derangement with nerve root involvement.*

Feedback to her includes a brief resume of her case – the present symptoms, aggravating and relieving factors as known, history of the condition and management are all outlined. It is suggested that the next stage is to examine neck and shoulder movements, and she is asked 'if that is all right with you?'

### The physical examination

*This sounds primarily like a neck problem, but there is a need to examine both neck and shoulder to get initial ranges of movement. It has a strong mechanical element, but because of the length of time symptoms have been present she has developed certain anxieties and fears about movement and activity and a degree of depression, as she feels unable to do anything to control her symptoms. She has*

*reported intermittent neurological symptoms, so a baseline neurological examination needs to be done. The only clue for investigating mechanically determined directional preference for movement is a worsening of symptoms with flexion. This suggests that extension movements and postures need to be fully explored. It is better to examine loaded positions first as this is much easier for patients to perform regularly at home or work. It is also important to explore the effect of posture correction – her posture in unsupported sitting is slumped, with her lumbar and thoracic spine in flexion and her head and chin protruded.*

To an enquiry about present symptoms and changes during the interview, she reports that she initially had neck and shoulder pain, but over the last ten minutes this has spread gradually halfway down her arm. On posture correction she reports immediate increase in neck pain, but after about a minute there is a definite easing of arm pain. It is suggested to her that an appropriate sitting posture will help her to control her symptoms. On examination she has a major loss of retraction without any visible posterior glide of her head on her neck; in contrast protrusion is full range and easily obtainable. On attempting flexion she is unable to put her chin on her chest, but is about two centimetres off. On asking her to look up at the ceiling she reveals a major loss; extension occurs mostly in the upper cervical spine and then she compensates by extending her thoracic spine. She is extremely reluctant to extend her lower cervical spine. On examining lateral movements she displays minor and moderate losses of left rotation and left-side flexion, but major losses of both movements to the right. On conducting a neurological examination neither myotomal nor reflex weakness nor an area of sensory loss is found. Finally, in standing, active shoulder movements are examined; flexion, abduction and the hand behind the back position are all painful during movement, but nearly full range. Other movements are no problem; passively she has full range, and resisted tests are inconclusive.

*The early stages of the physical examination appear to confirm a major mechanical component to this patient's problem. Her symptoms displayed peripheralising and centralising responses to changes in posture. Her mechanical presentation displayed selective blockages of movement that are characteristic of posterior derangement. Although reporting intermittent neurological symptoms, no definite signs or symptoms indicating nerve root compromise were*

*elicited. Although reporting pain on shoulder movements, a brief examination does not highlight a specific shoulder problem. The initial focus will be on the neck, her major problem, and return to the shoulder if at a later date this is necessary. Initially she was very reluctant to move, so plenty of encouragement, advice about the importance of movements to help restore joint function, and how to listen to the symptom response to gauge the appropriateness of movements will be needed during the repeated movement section and before she leaves.*

*In terms of exploring repeated neck movements, retraction and extension seem the most promising at this stage. There is the possibility of a lateral component needing lateral forces, but at this stage there are more indicators of the need for sagittal plane forces. There is considerable loss of movement and care must be taken not to rush things too fast, and also to ensure there is sufficient range of retraction before extension is started.*

She reports that the pain in the arm has gone completely, and she relates this to sitting upright during the movement testing. The need to examine the effect of repeating some of the movements to find the most suitable is explained to her, and also that then she will be able to do something regularly at home. Initially she finds retraction difficult to perform, partly as she has so little movement available. After four or five sets of ten to fifteen repetitions, though, the movement is increasing, and she says the more she does the easier it gets. The focus is on her posture and her technique, with encouragement as appropriate. After a number of sets of repetitions she is told to stop and relax, but keep sitting upright. She reports the symptoms still to be right-sided neck, scapular and shoulder pain. On re-examination of her movements, however, there are changes. Retraction now has minor to moderate loss and she is able to extend about halfway with some lower cervical movement now present, but still with considerable pain; both right rotation and side flexion are increased. Upon five to ten repetitions of extension from a neutral head posture the arm symptoms begin to return, but are absent again once she stops. Retraction with patient overpressure is attempted. At first she reports this to be very stiff and painful in the middle of her neck, but again with repetition it gets easier to do, and she gets further back. After three sets of ten to fifteen she reports all movements to be easier, and the symptoms now to be in the neck and scapular area with nothing on the shoulder.

*A positive mechanical response has been generated with posture correction, retraction, and retraction with patient overpressure. This has caused a mechanical response of improved range of movement and symptom response of centralisation, with abolition of arm and shoulder pain. The provisional classification is derangement, the treatment principle is extension, and severity indicators are intermittent arm symptoms and constant neck and scapular pain. There is no need to pursue further tests or look for additional interventions at this point in time. The emphasis now is ensuring she is confident to perform the exercises regularly.*

The positive indication of a mechanical response is explained to her, and that this now provides her with a means of beginning to control her symptoms. She is instructed in posture correction and regular interruption of sitting and neck flexion activities, and told to repeat the retraction exercises at least every two hours, but more regularly if it helps. She is to do ten to fifteen actively and then finish each session with about ten retractions with patient overpressure. She is told that the response to expect is as occurred in the clinic – stiff and painful initially, but gradually getting easier to do, with less and less distal pain. If the opposite happens, which is unlikely, and the pain spreads down the arm, she is told to stop the exercises and wait until the next appointment.

### Session two

She returns to the clinic in two days' time. Very quickly it is apparent she is in a considerably better mood, less anxious and more relaxed about the way she is moving. She reports she has been doing the exercises at least every two hours, often more regularly, and the response was similar to the first day except they have become considerably easier to do. She reports she has been sitting better, regularly getting up and walking around, and even going for walks twice a day, which she now finds help. She has been woken at night by neck symptoms only once the first night and not at all last night. She has had no symptoms in her forearm and only brief symptoms in her arm when she had been sitting and forgot about her posture. She was able to abolish this rapidly with exercises and posture correction. The symptoms are now only sometimes onto the shoulder, and principally in the neck and scapular area. They are still constant there, but on a numerical scale she rates the pain now as three out of ten. This has been the same all today despite regular exercises all morning.

On physical examination retraction has a minor loss; with flexion she is still not able to get her chin to her chest; extension is somewhat improved, but still displays a moderate loss. For rotation and lateral flexion movements to the left are full, and movements to the right are painful with minor to moderate loss of range. Her posture is improved and the quality of her movements is better. Her shoulder movements are checked, and these are now full range and pain-free. Her performance of retraction exercises both actively and with overpressure is accurate.

*At review she reports active participation with her management and demonstrates improved posture and accurate performance of the exercises. She reports positive response to retraction exercises, improvements in site, severity and frequency of symptoms, and is definite that she has improved overall. This is confirmed on examination of range of movement, which shows clearly increased range in all directions. These responses confirm the classification of derangement and the appropriate extension treatment principle. However, she may be reaching a plateau with the present loading strategy. Extension is still limited and it must be seen if it is now appropriate to introduce extension.*

Prior to repeated movements she reports low intensity central to right-sided neck and scapular pain. Repeated active retractions followed by patient overpressure both have the effect of increasing central neck pain, but she reports a return to initial symptoms afterwards. Repeated retraction and extension is performed; again this increases central neck pain, but after repeating two sets of ten to fifteen repetitions it is clear that the right-sided neck pain is getting worse and beginning to spread out to the shoulder.

*She has been making improvements with patient-generated forces up to this point, but continuing with retraction by itself seems no longer to be helping. There is still a substantial loss of extension and it is apparent that this will have to be worked on for further improvement. However, loaded extension again seems to lead to peripheralising of symptoms. Options here are to introduce clinician-generated forces or unloaded forces. Although there might at this point be a lateral component that needs addressing this seems unlikely – she has been responding to sagittal forces, and although there are still losses of right rotation and lateral flexion, the main loss is in extension. Introducing lateral forces before exhausting all sagittal forces is a common clinical error.*

In the loaded position clinician-generated retraction is performed, producing an increase in central neck pain. With repetition this gets no easier, does not seem to get to end-range, and after the second set of repetitions she reports pain spreading out towards the shoulder again.

*At this point she is not responding to loaded force progressions, but before considering lateral forces, unloaded extension forces need to be exhausted. She has been using loaded patient overpressure for nearly two days, so early unloaded forces are not appropriate. The goal at this point is to get her actively performing the next progression of loaded retraction and extension.*

Retraction mobilisation in supine is performed over the end of the plinth. This produces an increase in central neck pain, but over two sets of ten to fifteen repetitions she reports this gets considerably easier. Afterwards the right-sided symptoms remain unchanged. Two further sets of repetitions produce a similar response, but full movement to end-range. After resting for a few minutes in supine she returns to upright sitting, ensuring her head remains in neutral as she does so. Retraction with therapist overpressure in loaded is repeated, again producing an increase in central neck pain, but now the movement feels like it is getting to end-range. She is asked to retract and then extend, but she still finds this very difficult to do because of central neck pain and stiffness. To facilitate she is instructed to put both hands behind her neck with her fingers either side of the spinous processes around the cervico-thoracic junction, pulling the spine forward. This enables her to do the exercise more easily, which increases central neck pain each time, but she demonstrates increasing range. After ten repetitions she reports more central than right-sided neck pain. After a further ten repetitions using her hands to support the movement, she reports only central neck pain. Afterwards she reports it feels considerably easier when she does retraction extension without hands supporting.

*Many different loading strategies needed to be tried to find the appropriate one. There was no clear reason to abandon the sagittal plane as the initial response was good. With unloaded sagittal plane forces of retraction, the patient reports an improving symptomatic response with repetition, and also seems to display a favourable mechanical response. Upon returning to a loaded position, which is a much better position for regular practice, this was no longer*

*causing peripheralisation, but was still difficult in terms of central neck pain. The additional support that the patient could provide allowed her to perform the loaded movement confidently, and so she would be able to do the exercise regularly at home.*

She is much more confident than she was to do the exercises. She reports only central neck pain and an increase in all movements, especially extension, at the end of the session. She is instructed to continue with the same management as before, but now also to add in the retraction extension exercise, with support if needed, and over the next few days to do less of the retraction and more of the retraction extension as long as the response stays good. She is told to expect gradually improving neck pain, and to stop if the symptoms start to peripheralise again. She asks what you think about her going back to work, and you say you think this is a very good idea and next time you can discuss ergonomic advice for her workstation. She suddenly seems very fed-up about being off work and keen to return. You suggest she visit the surgery at the beginning of the next week and make arrangements for returning later that week.

*It is very positive that she volunteered an interest in a quick return to work and demonstrates how once patients have the ability to begin to feel in control of their symptoms how rapidly apparent 'yellow flags' can simply disappear. She is happy to leave the next appointment for four days, another sign of her growing confidence in managing her problem.*

### Session three

She reports first of all that she went to the surgery where she works the day before and has arranged to return to work at the end of the week if you think it is a good idea. She says she is keen to do so, and the surgery is happy to follow any recommendations you might have for her workstation. She reports she has been walking daily and would like to do more exercise in the future – do you think this a good idea, or will it make her neck worse? She reports that over the last four days she has been doing the exercises regularly and doing mostly retraction extension in the last two days. As suggested, the extension movement got gradually easier and easier to do, and in the last twenty-four hours it has only generated increased discomfort at end-range. In the last two days she has had intermittent central and right neck pain only, for less than 50% of the day, of very low severity

that she rates about one on a zero-to-ten scale. At present she has mild central neck pain. Range of movement is checked – she now has full range flexion, chin to sternum, her lateral movements are equal and full, but with pain at end-range or right rotation and lateral flexion; extension has a minor loss with increased central neck pain. Right shoulder movements are full and pain-free and she reports no problems with neck or arm movements in the last few days.

*Everything at this review demonstrates a continuing positive response with nearly full resolution of the problem. All pain is now intermittent, mostly central neck pain and of low severity. Her movements are now virtually all full range, with just a minor loss of extension, and she reports no real problem with activity in the last few days. Not only does she show improvement symptomatically and mechanically, but also she demonstrates a much better frame of mind. She is keen to return to work, she is no longer anxious, depressed and irritable as she was, and she is keen to continue her walking regularly as a start to trying to get fitter. She also seems much more aware of her posture.*

You ask her what she would do about her present symptoms. She performs a few retractions the last with overpressure and then about ten retraction extension exercises. She reports that retraction now has very little effect, and each time she extends she feels it slightly more centrally; afterwards it possibly feels slightly easier, but she is unsure. You demonstrate to her how she can do the same movement with overpressure by doing slight rotations at end-range extension. She does two sets of about ten of these movements, and afterwards reports the abolition of pain and pain-free extension and right lateral movements.

*She is confidently self-managing by this time, and would probably fully reduce the derangement in time with retraction and extension. However, the additional overpressure fully reduces the derangement and fully restores pain-free movement rapidly. As well as continuing to perform the exercises, additional advice should be given to reinforce her self-management skills.*

You recommend that she continue with these exercises for the next week or so, or as the need arises, and also that she stretches all neck movements in all directions once a day. You suggest it is a good idea to do the exercises from time to time to help remember them, and

especially after sustained neck flexion. You discuss with her that the typical history of neck pain is episodic, but that often the movements that helped this time will help next. At the slightest suggestion of recurrence of symptoms or loss of movement she should start the same exercise programme again. You suggest that she needs to regularly interrupt her posture at work, get up and move around; you make a few recommendations regarding seating, lumbar support, screen height, arm rests and so on. You ask her if she would like to come and see you again, maybe when she has been back at work for a few days, and she is happy to leave the review appointment for a week.

### Session four

When she returns for review she has had four days back at work in this and the previous week. She found it tiring to be back the first few days and experienced a bit more aching in her neck, but this settled over the weekend and she has kept on top of the situation this week by exercising regularly and interrupting her posture frequently. She reports mild aching in the morning for an hour or less and infrequent mild aching for brief period during the day in the last three days. This morning there was minimal ache for less than twenty minutes on rising; overall she rates the aching at one, at worst, on a numerical pain scale and on the NDI she now scores two. She feels she is coping well and has every expectation of a full recovery if she continues with the exercises and other aspects of the advice. All movements are full and pain-free. She is happy not to make another appointment, but will phone within the next few weeks if she has any further problems.

> *Although not completely symptom-free, her symptoms are now minimal, brief and occasional only. She demonstrates ability to control and fully abolish the remaining symptoms with exercises, postural correction and interruption of neck flexed postures. She has returned to work, and has in fact increased her normal level of activity with a desire to improve her level of fitness. She is also equipped with knowledge to reduce chances of recurrence, and what to do should one occur. If she is happy to self-manage at this point, further sessions should be avoided unless really needed.*

### Telephone review

The patient was phoned two weeks after the last review. She reported minimal symptoms at the beginning of the first week, but no problems during the last week. She reported work to be going fine; she also mentioned that she had joined a gym and was attending two times a week at the moment, but hoping over time to do more.

## Conclusions

This chapter has considered some of the aspects that contribute towards clinical reasoning. The literature around this topic has been explored, and some of the limitations of the present analysis of the concept have been suggested. Clearly data-gathering and knowledge base are key features, but the idea that sophisticated clinical reasoning equates to performing large numbers of physical examination procedures as well as the emphasis on certain aspects of the process, such as 'yellow flags', has led to an over-complicated and unhelpful assessment process. A case study is presented as an example of how a patient with apparent 'yellow flags' is actually well able to self-manage once provided with appropriate exercises and advice.

# 16: Recurrences and Prophylaxis



## Introduction

As discussed in Chapter 1, recurrences, episodes and persistent symptoms are common experiences in those who have neck pain. At least 40% of those who develop neck pain have future episodes (Lees and Turner 1963; Gore *et al.* 1987; Lawrence 1969; Radhakrishnan *et al.* 1994; Leclerc *et al.* 1999; Kjellman *et al.* 2001; Hill *et al.* 2004; Picavet and Schouten 2003). Persistent neck pain, lasting for two or three months or more, is experienced by about a quarter of the general adult population (Andersson *et al.* 1993; Brattberg *et al.* 1989; Bergman *et al.* 2001; Picavet and Schouten 2003; Makela *et al.* 1991; Hill *et al.* 2004). In fact, single self-limiting, non-recurrent episodes are rare, occurring in only 6% of one sample with neck pain, whilst 39% reported continuous pain and 55% reported episodic symptoms (Picavet and Schouten 2003). Neck pain should probably be viewed from the perspective of the individual's lifetime, from which perspective the importance of self-management appears to be paramount.

Any education or assistance that the patient can be given to try to prevent recurrences, reduce the number or length of episodes or improve their ability to manage the problem should they have a relapse should be an essential part of management. Provision of such education, and encouragement of patients to 'problem-solve' their own difficulties should be part of treatment. Supervision of patients must, in the light of the epidemiology of neck pain, involve the nurturing of self-management strategies. This should be done from the initial assessment, not as an add-on at the end of treatment, and those strategies need to be individualised according to the patient.

Primary prevention refers to risk modification to decrease the susceptibility for an event to occur (Lahad *et al.* 1994). Goals of secondary prevention in musculoskeletal problems could be to prevent or decrease the number of new episodes, shorten the duration of episodes, enhance self-management strategies, decrease the need for seeking health care, or decrease the need for time off work (Linton 1996). Given that no intervention has successfully been shown to reduce the prevalence or incidence of neck pain, primary prevention

appears unrealistic at this point in time. Secondary prevention is perhaps a more realistic goal.

Sections in this chapter are as follows:

- preventative strategies
- future episodes
- evidence.

## Preventative strategies

It is reasonable to advise patients about aspects of the epidemiology of neck pain. Not everyone with neck pain has future episodes, but a considerable proportion have future or persistent episodes. Warnings regarding the natural history of neck pain, which is commonly episodic or persistent, thus represent a responsible aspect of management. Most patients are interested in prognosis and clinicians are responsible for providing this information. Two main aspects should be discussed: what can be done to try to prevent an episode and what can be done should an episode occur.

Physical work factors have been shown to have a relationship with neck pain, although not all studies are consistent in their findings (Ariens *et al.* 1999). Working in static postures, especially involving neck flexion, sitting or driving, are biomechanical loads that have been implicated as risk factors for neck pain in some studies (Grieco *et al.* 1998; Vingard and Nachemson 2000; Makela *et al.* 1991; Andersen *et al.* 2002; Dartigues *et al.* 1988; Kilbom *et al.* 1986; Ignatius *et al.* 1993; Ariens *et al.* 2001b; Jensen *et al.* 1996). Of all the factors predisposing to neck pain, only postural stresses can be influenced and controlled. This potential tool for prophylaxis and management must be developed to the full. To this end, certain issues should be discussed with the patient at several times during the treatment episode; it is important that these issues are not left to the final session.

The following factors should be discussed using appropriate language:

- most neck pain starts without trauma – use this factor to highlight the insidious nature of onset and therefore the probable relationship to ordinary daily and sustained postural stresses
- sustained postural stresses can be controlled if the person is aware of them

- sustained postural stresses are common with continuous activities, such as relaxed sitting
- relaxed sitting involves flexion of the back and trunk, which leads to a protruded head posture
- protruded head posture involves end-range loading of the cervical spine.

Measures that might be used to counteract the effects of sustained loading are as follows:

- maintain lumbar lordosis, with lumbar support if necessary
- maintain upright sitting posture with head over shoulders and chin over chest
- hourly interruption, at least, of sustained sitting to stand up and walk around for a few minutes
- if involved in activities of sustained neck/head flexion:
    - regular interruption of posture by standing/walking around
    - application of retraction/extension/rotation exercises at intervals.

## Future episodes

Despite preventative measures another episode of neck pain may develop; individuals also tend to become less attentive to postural concepts once the pain has receded. Therefore individuals need to be aware that they may have another episode and what they should do about it.

In terms of future potential episodes, the following issues should be discussed:

- postural concepts that were useful last time (see above)
- practicing the exercise(s) that resolved the present problem will help to remember them
- be aware of minor discomfort or problems with movement that may foreshadow the onset of more severe symptoms
- re-instigate the exercise(s) that was/were useful with the last episode of neck pain

- exercises must be performed regularly and over time to end-range
- remember appropriate and inappropriate symptomatic and mechanical responses
- stop exercising if symptoms worsen or peripheralise
- make regular interruptions to sustained working/domestic postures
- consult *Treat Your Own Neck* (McKenzie 1983, 2006) if further information is required
- consult a clinician if symptoms worsen, peripheralise or fail to respond.

## Evidence

Documented evidence for preventative interventions for neck pain is very limited. A systematic review (Linton and van Tulder 2000) on preventative interventions for back and neck pain concluded that:

- there is consistent evidence that back schools are not effective in preventing neck and back pain: level A evidence (strong – consistent findings from multiple randomised controlled trials)
- there is consistent evidence that exercise may be effective in preventing neck and back pain: level A evidence
- there is no good quality evidence on the effectiveness of ergonomics: level D evidence (no evidence in the form of controlled trials)
- there is no good quality evidence on the effectiveness of risk factor modification: level D evidence.

In fact, in the review only one study (Kamwendo and Linton 1991) specifically investigated the prevention of neck pain with the use of a neck school, which did not appear to be effective.

## Conclusions

There has been little documented evidence concerning the efficacy of preventative strategies for neck pain. However, given the high prevalence and recurrence rates, management must address this. Issues of recurrence and preventative strategies should be discussed with patients and what to do should another episode occur. Preventative

and management strategies revolve around postural concepts and appropriate exercise therapy. These issues should be discussed with patients during an episode of care so that they are equipped with sufficient knowledge in this area. Minimally this involves knowledge of the appropriate exercise, recognition of appropriate and inappropriate symptomatic and mechanical responses, and awareness of the importance of everyday sustained loading strategies.

# THE CERVICAL & THORACIC SPINE MECHANICAL DIAGNOSIS & THERAPY

## VOLUME TWO


ROBIN MCKENZIE

CNZM, OBE, FCSP (HON), FNZSP (HON), DIP MDT, DIP MT

STEPHEN MAY

MA, MCSP, DIP MDT, MSC

# THE CERVICAL & THORACIC SPINE MECHANICAL DIAGNOSIS & THERAPY

## BY ROBIN McKENZIE AND STEPHEN MAY

This book is essential reading for any health professional involved in the management of patients with cervical or thoracic pain. Described within are the mechanical measures required for the diagnosis and treatment of these common problems. The precise identification and management of subgroups in the spectrum of mechanical cervical and thoracic disorders has been said to be a priority if we are to improve our methods of management of back and neck problems. This latest book in the series by McKenzie and May provides a system to identify subgroups and consequently provide better strategic solutions.

Robin McKenzie first published his landmark text outlining certain principles and concepts for the diagnosis and management of lumbar spine problems in 1981. Another volume addressing the cervical and thoracic spine was published in 1990. His first publications always stressed the importance of patient self-management and the relevance of this issue has been belatedly recognised by others. Since these publications considerable evidence has demonstrated the importance and relevance of those principles and concepts in the modern management of musculoskeletal problems.

This edition explains the centralisation and peripheralisation phenomena; the use of exercise to induce changes in pain location and intensity; the means of detecting the most effective direction in which to apply therapeutic exercise; differentiation between the pain of displacement, pain of contracture and pain arising from normal tissue; how to differentiate the pain of nerve root adherence from entrapment and sciatica.

This second edition of The Cervical & Thoracic Spine: Mechanical Diagnosis & Therapy parallels the changes in the updated Lumbar Spine text. It has been thoroughly revised and considerably expanded and explores in depth the literature relating to mechanical syndromes and neck and trunk pain in general. There are descriptions of the management of the three mechanical syndromes – derangement, dysfunction, and postural syndrome – as applied to neck, thoracic and headache problems. There is in-depth consideration of the literature relating to a number of issues, such as the epidemiology of neck pain, headaches, serious spinal pathology and whiplash. Operational definitions, descriptions and numerous tables provide clinical signs and symptoms to recognise or suspect mechanical syndromes or other diagnoses.

Robin McKenzie and Stephen May have produced another evidence-based and clinically relevant text for the new century, augmenting the other volumes available that relate to the lumbar spine and extremity problems. It provides a review of relevant general topics as well as the detail of how to evaluate and prescribe appropriate specific exercises and manual techniques. The system described in this book achieves a new benchmark for the non-surgical management of mechanical cervical and thoracic disorders.

# The Cervical & Thoracic Spine Mechanical Diagnosis & Therapy

## Volume Two

Robin McKenzie
CNZM, OBE, FCSP (Hon), FNZSP (Hon), Dip MDT, Dip MT

Stephen May
MA, MCSP, Dip MDT, MSc

Spinal Publications New Zealand Ltd
Raumati Beach, New Zealand

The Cervical & Thoracic Spine: Mechanical Diagnosis & Therapy

First Edition first published in 1990 by Spinal Publications New Zealand Ltd

Second Edition first published in April 2006
Reprinted February 2007, March 2008 by
Spinal Publications New Zealand Ltd
PO Box 2026, Raumati Beach, New Zealand
Email: enquiries@spinalpublications.co.nz



ISBN 10 0-9583647-7-X
ISBN 13 978-0-9583647-7-5

Design by Next Communications
Edited by Autumnwood Editing and Jan McKenzie
Photography by John Cheese
Illustrations by Paul Pugh
Typeset and printed by Astra Print

# Contents



## VOLUME TWO

# List of Figures

# List of Tables

x

# 17: Management of Derangement – Principles

## Introduction

This chapter introduces the principles that are used to manage patients with derangement. This includes the stages of management and the different treatment principles that are necessary to reduce the derangement. Also presented are the treatment pathways used to determine management strategies.

Sections in this chapter are as follows:

- stages of management
    - reduction of derangement
    - maintenance of reduction
    - recovery of function
    - prevention of recurrence.
- management principles
    - extension principle
    - lateral principle
    - flexion principle
    - irreducible derangement.
- treatment pathways in derangement.

## Stages of management

The management of derangement has four sequential stages. Management is a combination of education and mechanical therapy. Although reduction of derangement initially takes precedent, there is likely to be some overlap of the stages. Reduction by itself is of limited value if the patient does not have the ability to maintain improvements. Once the derangement is reduced and improvements maintained, a full restoration of function and confidence to move is vital. At some point throughout the episode of care there should be discussion about recurrence and what to do if this happens.

**Table 17.1 Stages of management of derangement**

- Reduction of derangement
- Maintenance of reduction
- Recovery of function
- Prevention of recurrence.

### Reduction of derangement

Reduction describes the process by which the derangement is progressively lessened. Improving symptomatic and mechanical presentations is a way of monitoring reduction of derangement. This is recorded by centralisation of pain or abolition or decrease in symptoms, and recovery of full range of movements. This may occur on day one or take several sessions.

Following the history and physical examination, a treatment principle will be decided upon to achieve reduction of derangement. The treatment principle chosen is the one that centralises, abolishes or decreases symptoms and increases the range of movement. Treatment principles are categorised as extension, flexion or lateral. Reduction is often attained using end-range patient-generated forces only, although sometimes clinician-generated forces are needed to supplement these.

The reductive process is continuing when peripheral pain is reported to be progressively centralising or decreasing, or if pain located across the shoulders or scapulae is centralising (felt more in the spine), decreasing or ceasing.

When the derangement is fully reduced, pain is abolished and full-range pain-free movement is usually regained. Reduction is complete only when the patient reports none of the original neck or referred pain when undertaking normal daily activities and pain-free movement is restored. In many patients reduction occurs rapidly over days or weeks, but in some patients this process may take several weeks, especially if they do not strictly avoid aggravating factors. Chronic derangements and non-mechanical factors can also elongate the reductive process.

During the process of reduction, the patient may undertake certain activities that impede or reverse the process and cause symptoms to reappear. With cessation of the aggravating positions and performance of the appropriate end-range movements, symptoms should once again start decreasing or centralising.

Procedures that are achieving reduction need not be supplemented in any way, but should be continued until symptoms are abolished, mobility is full and pain-free, and function has returned to normal. If improvements plateau at any point, force progressions, which may include clinician-generated techniques, are added. The minimal force necessary to achieve reduction is used at all times; this ensures that patient involvement and independence is always maximal.

Usually repeated movements are involved in the reductive process, but sometimes, when time is important, sustained procedures are more important, at least initially.

If you are having problems achieving reduction, consider the following:

- Are movements achieving end-range?
- Are force progressions required?
- Do repeated movements need to be done more regularly?
- Are force alternatives required?
- Is reduction being achieved, but not maintenance?

Patients should be made aware of what to expect from the exercises. Movements may initially generate increased neck pain, but reduced shoulder or arm pain. The increased spinal pain can sometimes be quite disconcerting to the patient, so they must be reassured about this. Just as they should be told what to expect, patients must also be informed about indicators to stop the exercises. The main reasons for stopping the programme are a worsening of distal pain or peripheralisation of pain. In such instances patients should be told to stop the exercises and return for review. For the exercises to be held responsible for a worsening of pain, this must occur at the time of performing the exercises, not several hours afterwards. If symptoms appear some hours after doing the movements, when the patient is 'relaxing', their return is due to the posture at the time, not the exercises.

When reduction is complete or nearly complete, the patient may report that the original pain felt on a particular movement is gone, but that they now experience a strain or stiffness. Patients generally recognise this as normal, but occasionally may need to be reassured that this is usual. Thus, when patients report that they experience pain at end-range of extension, it is necessary to clarify the true nature of the problem. They should be asked, 'Is this pain or strain?' Patients

mostly find it easy to make the differentiation. It is usually only necessary to make this distinction at the latter stages of reduction, or when reduction is complete. It should be remembered that this might occur during the initial assessment, so this question should be routinely asked in all circumstances.

In many derangements the reductive movements restore full mobility in all directions. Thus, often in a derangement requiring the extension principle, extension procedures remove the obstruction to extension, but also restore full-range flexion. However, sometimes due to the derangement and avoidance of the aggravating movement, some adaptive changes may have occurred. This only happens when symptoms have persisted for six to eight weeks or longer. It may be noted that flexion has become restricted in a derangement requiring the extension principle.

The derangement is reduced, but pain on end-range movement, which may be limited, could persist because of a dysfunction. This may be recognised only after the derangement has been stabilised for several days. This situation is addressed in recovery of function.

Reduction – key aspects:

- identification of treatment principle that centralises, abolishes or decreases symptoms and restores function
- regular performance of self-management exercise until symptoms are abolished and function fully restored
- regular monitoring of posture to assist reduction
- force progressions only necessary if no initial improvement or improvement ceases
- re-evaluation of treatment principle only necessary if improvement ceases.

### Maintenance of reduction

Maintenance of reduction is about education. The patient must be able to maintain any improvements gained in a treatment session and to reverse any deterioration that happens during normal daily activity. If the patient does not know how to do this, the clinician has failed to do their job thoroughly.

In general, maintenance involves two elements:

- posture – avoidance of aggravating postures
- regular reductive exercise.

For a patient with a derangement being treated with the extension principle, the typical aggravating factor is sustained flexion. Their symptoms may recur when they sit for long periods, typically with a protruded head posture, which usually entails lower cervical flexion and upper cervical extension. This kind of patient needs education about posture correction, posture maintenance, regular repeated extension exercises and intermissions from their seated posture. Other categories of derangement require different information.

Maintenance of reduction is variable. Some reductions are stable in a short period of time and with a limited application of loading strategies, whilst others need a strict application of loading strategies over a more protracted period to bring about and maintain reduction.

The importance of posture in the reductive process and maintenance of reduction is especially important in derangements that require the extension principle. Whilst extension procedures may reduce the derangement relatively easily, successful reduction can often be only temporary if attention is not paid to postural stresses. In particular, sustained sitting with a resulting protruded head posture can prolong pain from a derangement and is generally far more potent a cause of symptom aggravation than bending forward a few times.

For this reason the patient must be taught the importance of postural correction and the link between the lumbar and cervical curves. They should use a lumbar roll for maintenance of the lordosis when sitting, which affects the position of the neck and head. Often they will have identified sitting as an aggravating factor already, so they are receptive to advice on sitting postures. It is often impossible to maintain an upright lordotic posture on a sofa, settee or lounge chair. Patients should be encouraged to use upright chairs, maintain the lordosis with a lumbar roll and regularly interrupt the sitting posture. If symptoms recur, the reductive procedure should be performed. For patients who are at risk of developing pain on sitting or for those who have a history of recurrent or persistent neck pain with prolonged periods of sitting or driving, lumbar rolls should be available for loan or purchase.

Patients may return for review stating that the exercises work in abolishing their symptoms, but the pain returns after a while, and overall they are no better. Further questioning reveals that the return of symptoms occurs when they sit. Another patient may report that generally they are much better and symptom-free, but every time they sit or drive for more than half an hour the symptoms return. Another patient may report that they are now generally free of pain during the day when they are active and moving about, but in the evening, when 'relaxing', symptoms return.

In all such instances force progressions should never be used. In effect the patient is reporting successful reduction of derangement; the problem is in maintaining it. Further discussion must be had about the troublesome nature of sitting and how this relates to neck posture, and the ways to avoid its effects. Instigating clinician procedures at this point will not help the patient deal with the postural stresses of normal activity when they arise. In these circumstances, helpful questions to the patient may be:

- 'If you maintain the correct posture, can you keep yourself free of pain?'
- 'If pain appears, what was it you were doing immediately beforehand?'
- 'Did it come on after sitting or bending?'
- 'If pain does appear, can you get rid of it by doing the exercise(s)?'

Maintenance of reduction – key aspects:

- regular performance of the reductive procedure
- use of postural correction, including lumbar roll if sitting a lot
- avoidance of aggravating factors, especially sustained postures
- regular interruption of sustained postures.

### Recovery of function

Recovery of function is about getting the patient back to where they were before this episode of neck pain. In the clinic we can assess this by looking at range of movement and asking about symptoms, but more importantly we need to know about resumption of usual activities that were curtailed because of this problem. Sometimes patients are fearful about resuming previous activities that they associated with

the onset or aggravation of pain. It is important to reassure and provide the information that they are likely to be better the sooner they resume normal activities.

During maintenance of reduction, patients will have been avoiding certain movements and as a consequence adaptive shortening may have occurred in certain structures that have not been regularly stretched. Even in the absence of adaptive changes the patient may have become overcautious about the aggravating movement, so it is also important to restore their confidence in its use. Recovery of function thus concerns the restoration of restricted movement after the reduction of the derangement and the restoration of the patient's confidence in normal use of the cervical spine. *Failure to recover function after an acute episode can be a potent factor in nurturing fear-avoidance beliefs and thus predisposing patients to chronic symptoms.*

Many patients have no residual loss of movement following derangement. Such patients will have full-range, pain-free movements in all planes. It is unnecessary in these instances to recover function, but obviously this needs to be ensured by assessing all patients' movements prior to discharge. It is important to be aware of the level of function required by individual patients depending on their normal occupational or sporting activities.

Flexion exercises are sometimes required after reduction by extension principle. Because the flexion programme that is being used in the remodelling process could re-aggravate the derangement, which was the problem initially, certain precautions should be observed. This is especially important if the derangement has only recently been reduced. The stability of reduction can be ensured by the following precautions. A time frame is presented as a rough guide for general purposes, but this needs to be used in the context of individual presentations and clinical reasoning. A common fault is delay in introducing flexion for fear of exacerbating the problem; this should not be of concern if the following guidelines are used.

**Table 17.2 Recovery of function – ensuring stability of derangement**

*Determining if derangement is stable:*

- monitor symptomatic response
  - end-range symptoms may be produced, no worse
  - symptoms may become less painful on repetition
  - symptoms should *not* be felt *during* the movement, but at end-range
  - symptoms must *not* be produced and remain worse
  - symptoms must *not* become more painful on repetition
  - symptoms must *not* peripheralise
- monitor mechanical response – range of movement of and response to extension *must* remain unchanged following repeated flexion.

*Introducing flexion:*

- commence with less stressful flexion procedures and progress only if needed, as that procedure is easily tolerated, or bringing no further improvements
  - flexion (day 1-5)
  - flexion with overpressure (day 4-5)
  - flexion mobilisation (day 7-10), but rarely needed
- perform new exercises less frequently, initially only 5/6 repetitions 5/6 times a day
- avoid over-vigorous flexion procedures within first few hours of waking
  - initially perform ten repetitions of flexion from mid-day on, every three hours until going to bed. If the derangement appears stable, the patient may commence the exercise a little earlier in the day and repeat it every two hours.
- following flexion exercises, always perform retraction/extension.

Although the emphasis here has been on flexion, it should be noted that lateral movements are used much more commonly during recovery of function in the cervical spine than the lumbar spine. Again, the same force progressions can be used, with active rotation and/or side flexion being used for the first day or two and then supplemented with patient overpressure.

Recovery of function is complete when all end-range movements are full and pain-free, although a strain may be felt.

Recovery of function – key aspects:

- all movements must be made full-range and pain-free after reduction of derangement

- patients should be made confident to bend and perform other normal activities
- restoration of flexion should proceed in stages, as above
- mobility into extension should remain unchanged after repeated flexion
- recovery of function is rarely required in anterior derangement.

### Prevention of recurrence

Advice concerning neck care in the future is always given to the patient during the treatment episode and prior to discharge. This should include discussion of the following aspects: recurrent nature of neck pain, avoiding prolonged aggravating postures, practice of prophylactic exercises and importance of general fitness.

Discussion and education about prophylactic concepts should commence at the first session and continue on each patient visit. When symptoms decrease or centralise in response to repeated movements, the usefulness of these responses to guide treatment can be explained. The patient can be advised that the procedure that achieves this is their 'first aid' for the future. 'This is the exercise you must do at the first sign of recurrence.'

Numerous questions, opportunities and teaching tools arise during an episode of care, and these must be exploited to the full to maximise future patient understanding and independence. Self-treatment is essential in prophylaxis, and this is impossible without understanding. See Chapter 16 for more detail about prophylaxis.

Prevention of recurrence – key aspects:

- continuance of exercise programme for six weeks to maintain full mobility, flexion and extension
- beware of sustained postures
- lifelong use of lumbar roll
- importance of general fitness
- use of exercises if neck pain re-occurs.

## Management principles

Key to correct management is the identification of the appropriate loading strategy, which will reduce the derangement and improve signs and symptoms. Not all derangements respond to the same loading strategy, and what works in one case may cause the situation to worsen in another. The sub-groups of derangement syndrome depend on the direction of reductive movements. Mechanical evaluation is used to determine this preference for loading strategy in a certain direction. Frequently the opposite movement will worsen the condition. This favour for a particular postural or movement loading is termed 'directional preference' (Donelson et al. 1991).

The fundamental distinction is between derangements that are reduced by movements or postures of extension, flexion or a lateral direction. Within these three treatment principles there is considerable variety of response to the same procedure. For instance, in derangements with a mechanically determined directional preference for extension a few will respond to retraction, whilst most require extension performed in sitting. A few, usually those more severe and acute, need to perform retraction and extension exercises in lying, and a very few need therapist assistance to achieve extension. Similar qualitative decisions are required in derangements that have a mechanically determined directional preference for flexion or lateral movements. Just as derangements themselves are continuums, their reduction should also be seen as a continuum.

Treatment principles are not necessarily stable throughout the reduction of a derangement, although they may be. In one situation it may occur that a patient reduces and then resolves symptoms with a loading strategy entirely in one plane. However, situations may also arise in which initial loading in extension exposes a relevant lateral component with a worsening of peripheral symptoms. The introduction of a lateral force produces rapid centralisation. The patient then requires extension to abolish the remaining central neck pain. In a few minutes the patient has required extension, lateral and extension loading.

In another instance a patient may initially have an obstruction to extension that improves with extension loading. However, after a few days of this loading force, an obstruction to flexion is created and a brief period of flexion loading is required to resolve symptoms.

Such dynamic responses to loading strategies are not universal; many patients will reduce a derangement with loading in a single plane. However, the need for variable loading strategies during the reduction of a derangement occurs frequently enough to need constant awareness of this possibility.

**Table 17.3 Treatment principles**

- extension
- flexion
- lateral
- combination
- irreducible.

### Extension principle

This is the most common mechanically determined directional preference displayed by cervical derangements, with well over 60% responding to these forces (McKenzie 1990). Whether the patient has symmetrical, asymmetrical or unilateral symptoms in the neck, arm or forearm, *it is still necessary in most instances to explore sagittal plane movements first*. Very often extension and flexion are the only movements to be examined, at least initially. There are, however, times when lateral forces must be explored and these situations are outlined first.

In certain instances use of the sagittal plane is avoided or deferred. There is one unusual clinical situation in which sagittal forces are avoided altogether, at least initially, and two situations where use of sagittal plane movements is abandoned, albeit temporarily. In all these situations some element of lateral force is used.

1. *In the case of an acute wry neck or lateral deviation, there should be no testing of the sagittal plane. Lateral forces are applied immediately.*
2. If at any time when exploring sagittal plane movements there is peripheralisation or worsening of unilateral or distal symptoms, these should be abandoned. The response to unloaded extension movements should be assessed before abandoning the sagittal plane altogether. If these also cause peripheralisation or worsening of distal symptoms, lateral forces are applied.
3. In patients with unilateral or asymmetrical symptoms that have not changed following a full exploration of sagittal plane forces,

lateral forces are also applied. Before sagittal forces are abandoned there should have been progression of forces, including clinician mobilisation.

Following the application of lateral forces in any of these clinical situations, sagittal movements may subsequently become necessary.

*Except for the one absolute exclusion criteria of an obvious lateral deviation, repeated movement testing always starts using sagittal plane movements.*

Often the history has already provided clues as to the likely mechanically determined directional preference, and the physical examination simply confirms this. If symptoms are decreased, abolished or centralised by extension forces, or if the mechanical presentation improves, management proceeds with the extension principle.

**Table 17.4 Clues as to need for extension principle (*not all will be present*)**

*History:*

- onset may relate to flexion activity
- activities of flexion produce or worsen symptoms
- activities of extension decrease or abolish symptoms.

*Physical examination:*

- poor posture – slumped sitting and protruded head posture
- posture correction affects symptoms
- loss of multiple movements
- greatest loss of extension
- extension is obstructed and painful
- lateral movements less limited than sagittal movements
- repeated flexion worsens or peripheralises symptoms
- repeated flexion worsens mechanical presentation
- repeated retraction/extension decreases, abolishes or centralises symptoms
- repeated retraction/extension improves extension range
- kyphotic deformity – patient fixed in protrusion/flexion and unable to retract or extend head (rare, severe presentation).

If symptomatic and mechanical responses are not fully elucidated by initial testing in the sagittal plane, then force progressions may be necessary in order to make the situation clearer. This can either be done on day one using overpressure, or else the patient can

perform many sessions of repeated movements over a twenty-four- to forty-eight-hour test period. Many patients respond over this time, and to enable them to resolve their problems using self-mobilising only it is undesirable to use clinician techniques on the first occasion unless absolutely necessary. If at review the situation is still unclear, then force progressions, including mobilisation, should be definitely included at this point. *Unless the sagittal plane is fully explored, the correct reductive movement may not be found.*

In some instances, to clarify uncertainty, symptom provocation can be applied using repeated movements to provoke symptoms. If the patient tests this out over twenty-four hours, they must remain alert to those circumstances that provoke symptoms. Once it is clear that certain loading such as flexion provokes symptoms, this activity is restricted and the opposite principle, in this case extension, is applied. This tactic is not used in patients with acute symptoms.

It is important to note that force alternatives may be necessary, with considerable variation needed in the degree and timing of loading strategies. Some patients require no more than posture correction and advice about maintaining this and about frequent interruption of the aggravating position. Most patients will be treated in sitting and combinations of retraction and extension are used. Some patients gain the most benefit from retraction, but more gain the most relief from extension exercises. *Many patients have to work on regaining retraction before they are able to tolerate full lower cervical extension.* This is a very important point, the ignoring of which frequently leads to the premature abandonment of extension exercises, as they are perceived not to work. In fact, the patient who has insufficient cervical retraction will be unable to extend the lower cervical spine. A few patients, with more severe or acute symptoms, cannot tolerate extension in a loaded position and need to be managed in an unloaded position. In more severe cases, clinician procedures are necessary to allow the patient to regain full extension.

Force progressions and force alternatives may be necessary to establish a mechanically determined directional preference and maintain improvements. Minimal intervention is always best – the simpler the intervention, the easier for the patient to accomplish themselves, and the more likely they are to gain independence of management. Progression does not imply a necessary starting point; patients enter

the spectrum of treatment choices at different levels. Nor does it imply an absolute need to progress, as many respond at minimal levels and no progression of force is required.

**Table 17.5 Extension principle – force progressions and force alternatives**

*Extension principle – force progressions:*

- retraction
- retraction with patient overpressure
- retraction with clinician overpressure
- retraction and extension
- retraction mobilisation.

*Extension principle – force alternatives:*

- posture correction
- retraction (with overpressure) (supine)
- retraction and extension unloaded (supine)
- retraction and extension with patient overpressure (prone)
- retraction with clinician overpressure (supine)
- retraction mobilisation
- retraction and extension with traction and rotation mobilisation (supine).

### Lateral principle

In cervical spine derangements a proportion of patients require some element of lateral principle procedures in their reduction; this is well over 20% of all cervical derangements (McKenzie 1990). These will all be patients with unilateral or asymmetrical symptoms and neck/arm pain. Indications for use of the lateral principle are negative response to sagittal plane movements, and much less commonly acute wry neck. Clues may be unearthed during the history-taking and the need for lateral forces is fully exposed during the physical examination.

Two situations require lateral forces (Table 17.6). The more common is the relevant lateral component without a lateral deviation of the spine. This is when sagittal plane forces are tried, but either cause unilateral or asymmetrical symptoms to worsen or cause no important change in symptoms. The less common situation is the presence of a relevant lateral deviation of the spine or wry neck.

**Table 17.6 Indications for consideration of lateral component**

- Unilateral or asymmetrical symptoms, and 1 or 2:
  1. Indication for temporary abandonment of sagittal plane testing
     - peripheralisation or worsening of peripheral symptoms in response to sagittal movements
     - non-response after full exploration of sagittal plane forces conducted over several days and/or including force progressions.
  2. Indicators to avoid or defer testing of sagittal plane
     - presence of clinically relevant lateral deviation of spine.

*In all instances sagittal movements may later be required.*

In some people the history-taking and early stages of the physical examination do not reveal a clear mechanically determined directional preference. The need for lateral forces becomes apparent only when loading strategies are explored. Positions of or movements into extension cause a worsening or peripheralisation of pain – the need for lateral forces is predicted by the response to loading strategies.

In other patients pure extension forces may not actually cause peripheral symptoms to increase and remain worse, but cause only an increase whilst being performed. Should this response fail to improve, but be repeated each time after multiple repetitions, even with the inclusion of force progressions, again lateral forces should be introduced. Many such patients would, after a brief use of lateral forces, subsequently require pure sagittal extension.

Rarely a very apparent lateral deviation of the neck and head is found – the patient's head is fixed in lateral flexion and possibly flexion and rotation, and they are unable to correct this. In such an example the need for lateral forces is easily predicted, and with the presence of a 'hard' deformity the patient is unable to achieve this alone and clinician assistance is required. In some the obstruction to movement is 'soft' and yields to repeated patient-generated forces. In the presence of a lateral deviation of the spine, pure lateral forces are used.

**Table 17.7 Lateral deviation of cervical spine – definitions**

*Lateral deviation:*

- head and upper cervical spine are visibly and unmistakably shifted to one side
- onset of deviation occurred with onset of neck pain
- patient is unable to correct deviation voluntarily
- if patient is able to correct deviation, they cannot maintain correction
- correction affects intensity of symptoms
- correction causes centralisation or worsening of peripheral symptoms.

*Right and left deviation:*

- a right deviation exists when the vertebra above has laterally flexed and/or rotated to the right in relation to the vertebra below, carrying the head with it
- a left lateral deviation exists when the vertebra above has laterally flexed and/or rotated to the left in relation to the vertebra below, carrying the head with it.

*Contralateral and ipsilateral deviation:*

- contralateral deviation exists when the patient's symptoms are on one side and the head is shifted to the opposite side; for instance, right arm pain with the head laterally shifted/rotated to the left
- ipsilateral deviation exists when the patient's symptoms are on one side and the deviation is to the same side; for instance, right arm pain with the head laterally shifted/rotated to the right.

**Table 17.8 Clues as to need for lateral principle *(not all will be present)***

*History:*

- unilateral or asymmetrical symptoms
- both flexion and extension movements aggravate symptoms
- may report preference for rotation or side flexion
- patient reports sudden, recent onset of postural misalignment (rare).

*Physical examination:*

- lateral movement is asymmetrical, with major loss in one direction
- symptoms centralise or are made better by lateral movements
- symptoms peripheralise or worsen with extension procedures in sitting and lying
- symptoms are overall unchanged after several days' application of extension protocol, including force progressions
    - lateral deviation or wry neck – patient is locked in a position of lateral deformity and is unable to straighten, or if they correct they cannot maintain it (uncommon clinical presentation).

As with the extension principle, procedures associated with the lateral principle can be applied in sitting or lying and have a similar progression of forces from active movements, patient overpressure, therapist overpressure, clinician mobilisation or manipulation. Lateral forces in the cervical spine are principally applied in the plane of side flexion or of rotation. Lateral flexion or rotation is applied depending on the best symptom and mechanical response.

**Table 17.9 Lateral principle – force progressions and force alternatives**

| *Lateral flexion forces (mid-range retraction)* | *Rotation forces (mid-range retraction)* |
|---|---|
| *Loaded – Sitting:* | |
| Lateral flexion | Rotation |
| Lateral flexion patient overpressure | Rotation patient overpressure |
| Lateral flexion clinician overpressure | Rotation clinician overpressure |
| Lateral flexion mobilisation | Rotation mobilisation |
| Lateral flexion manipulation | Rotation manipulation |
| *Unloaded – Supine:* | |
| Lateral flexion | Rotation |
| Lateral flexion patient overpressure | Rotation patient overpressure |
| Lateral flexion clinician overpressure | Rotation clinician overpressure |
| Lateral flexion mobilisation | Rotation mobilisation |
| Lateral flexion manipulation | Rotation manipulation |

### Flexion principle

The flexion principle is rarely required in the treatment of cervical derangement; it is used in less than 5% of all patients (McKenzie 1990). However, flexion forces are commonly required in the management of headache and dysfunction (see appropriate chapters).

In cervical derangements requiring flexion, symptoms will be symmetrical or asymmetrical around the mid- to lower cervical spine, possibly also with anterior or antero-lateral symptoms around the throat. There may also be pain on swallowing. Onset may be related to a motor vehicle accident. Flexion will be obstructed and the patient may not be able to look down at their feet. Repeated flexion produces symptoms. Despite the major loss of flexion, test movements into extension are not obstructed and remain painless.

**Table 17.10 Clues as to need for flexion principle *(not all will be present)***

*History:*

- symmetrical or asymmetrical symptoms
- anterior or antero-lateral symptoms
- pain/problems with swallowing
- recent history of motor vehicle accident
- pain on neck flexion.

*Physical examination:*

- major loss of flexion
- pain-free full range of extension
- repeated flexion increases range.

As with most cervical derangements, management commences with patient procedures and only progresses to clinician procedures if improvements cease, which is unusual. Most derangements respond to pure sagittal flexion; sometimes patients with unilateral or asymmetrical symptoms require additional flexion/lateral procedures.

**Table 17.11 Flexion principle – force progressions and force alternatives**

*Force progressions:*

- flexion
- flexion with patient overpressure
- flexion with therapist overpressure
- flexion mobilisation (supine).

*Force alternatives:*

- flexion with lateral flexion or rotation
- flexion with lateral flexion or rotation with patient overpressure
- unilateral flexion mobilisation.

### Irreducible derangement

In an audit of mechanical diagnosis that included seventy-eight neck pain patients, sixty-two were classified as derangement, of which less than 5% were deemed to be irreducible (May 2004a). These patients generally have symptoms of constant brachialgia accompanied by nerve root signs and symptoms – this group will not respond to mechanical therapy. Again, aspects of the history and physical examination provide clues that a patient may have an irreducible derangement (see Table 17.12).

Although an irreducible derangement may be suspected on day one, the failure to respond should be confirmed over several sessions. Sometimes a patient presents with what appear to be non-responding signs and symptoms, which with re-examination show a more favourable prognosis. *The patient with suspected irreducible derangement should always be examined over at least three sessions to confirm the classification.*

**Table 17.12 Clues to irreducible derangement** ***(not all will be present)***

*History:*

- constant symptoms into hand
- constant paraesthesia or numbness
- arm pain more than neck/scapular pain
- no position relieves pain.

*Physical examination:*

- weakness in relevant myotome
- gross loss of all/most movements
- all loading strategies result in worsening of peripheral symptoms, including unloaded forces
- no movement or position decreases or centralises pain.

## Treatment pathways in derangement

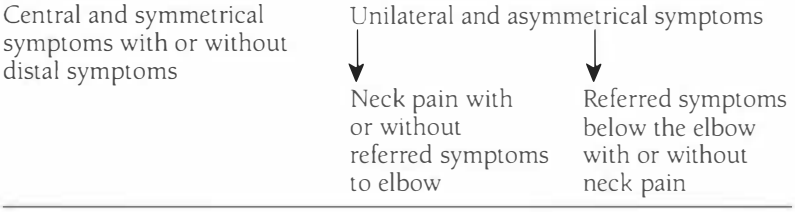
Description of the management of derangement is based on two considerations. The numbering system is no longer used, but the management is based on familiar concepts. The first consideration is the location of pain; the next is the extent of distal symptoms. These issues are decided by the patient's report of present symptoms, which is defined as the pain that is their present problem. This applies to all symptoms being experienced even if not actually present at the time of assessment.

Initially there will be two groups of patients; those with central or symmetrical pain and those with unilateral or asymmetrical symptoms. In those with symmetrical symptoms there may also be referral of symptoms into the shoulders or arms – as long as these referred symptoms are reasonably equally distributed, they should still be considered in the symmetrical group. In those with asymmetrical or unilateral neck pain there may be referral of symptoms; this will be as far as the elbow or below the elbow, and may include paraesthesia.

'Neck' pain may include symptoms principally located around the scapula, shoulder or upper trapezius region. Management is described in terms of these three groups (Figure 17.1). This should include all pain patterns, but if the patient's description of their pain does not clearly fit one of these patterns, consideration should be given to the closest equivalent.

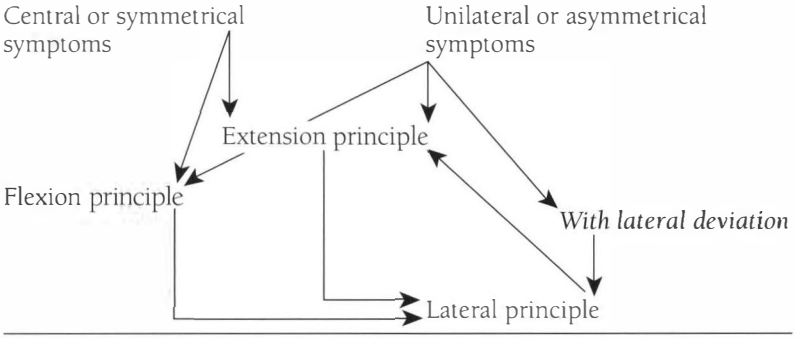
Patients with central symmetrical symptoms are those previously classified as Derangements 1 and 2. Patients with unilateral asymmetrical symptoms as far as the elbow are those previously classified as Derangements 3 and 4. Patients with unilateral asymmetrical symptoms below the elbow are those previously classified as Derangements 5 and 6 (McKenzie 1990). Patients previously classified as Derangement 7 are found in the first two categories.

**Figure 17.1 Derangement – management considerations**




If symptoms are central or symmetrical across the neck, scapulae and shoulder region, management will nearly always be in the sagittal plane, principally involving extension and occasionally flexion. Sometimes patients in this group have distal symptoms; as long as these are relatively equal bilaterally, management is conducted in the sagittal plane. Some patients with more distal and unilateral symptoms need non-sagittal treatment procedures. However, as many of this group respond to sagittal plane forces, this is always explored first. Failure to respond or unfavourable symptom response means lateral loading strategies are explored. Symptom and/or mechanical response always decides management. If a clinically relevant lateral deviation is present, then attention focuses on the lateral direction immediately. Response to loading strategies is dynamic and can change during an assessment or episode of treatment. The clinical reasoning pathways in derangement according to the different treatment principles are shown in Figure 17.2.

**Figure 17.2 Derangement treatment principles and symptoms**




## Conclusions

This chapter has considered the general concepts that need to be understood in order to help patients manage derangement-type problems. The four stages – reduction, maintenance of reduction, recovery of function and prevention of recurrence – are not isolated stages; clinicians need to be aware of all four stages during the management of such patients. They also need to be aware of the different treatment principles (extension, flexion or lateral) that may be involved in the management of such patients. During the history-taking clues will be gained as to whether the patient has a derangement or not; often this is confirmed during the physical examination.

# 18: Management of Derangement – Central and Symmetrical Symptoms

**(Previously Derangements 1, 2 and 7)**

## Introduction

This category encompasses a large proportion of all derangements. Patients report pain centrally or bilaterally in the neck, or across both shoulders, or across both scapulae, or some combination of symmetrical symptoms. Occasionally patients may also report aching bilaterally into both arms. This group comprises those formerly classified as Derangements 1 and 2, and 7. This is a non-specific somatic disorder. Most derangements in this group respond to the extension principle, and a few respond to the flexion principle.

Sections in this chapter are as follows:

- extension principle
    - history and examination
    - management guidelines
    - review
- deformity of kyphosis (previously Derangement 2)
- flexion principle (previously Derangement 7)
    - history and examination
    - management guidelines
    - review.

## Extension principle

### History and examination

A table of clues as to the need for the extension principle is included in the previous chapter. Patients may present in the acute or chronic stage with constant or intermittent symptoms. Most commonly patients have central or symmetrical symptoms around the lower cervical spine. Sometimes these can radiate to the shoulders or scapulae. Symptoms may worsen or be initiated by flexed activities, such as prolonged sitting at a computer, driving or reading. Patients frequently report symptoms improve or ease when on the move and walking about, when the neck tends to be more extended.

Sitting posture is often poor, with reduced lumbar lordosis causing a protruded head posture with concomitant lower cervical flexion. Correcting the posture may centralise or decrease symptoms or sometimes increase them. There is loss of extension, and in more severe cases flexion and lateral movements may be reduced. If lateral movements are affected, there is equal loss to the right and left.

The response to repeated movements will be characteristic. Repeated protrusion or flexion and sustained slumped sitting cause symptoms to increase or spread out from the spine. This response is linked to the number of repetitions or the length of sustaining the flexed posture, and so may not always be immediately apparent. Indeed, in the more acute patient, prolonged exploration of flexion movements should be avoided.

In contrast there will be a positive response to retraction and/or extension, with a decrease or centralisation of symptoms and an increase in range of movement. Often this response may become apparent during several repetitions of the test movements on day one. This response, though, might not be instant, but emerge over several days of repeated movements and posture correction. Often in such cases the beneficial response to the extension principle is confirmed at review twenty-four to forty-eight hours later, when a clear improvement in symptomatic and/or mechanical presentation is noted. Much can be achieved using patient-generated forces in the first few days, and in this way the patient can realise and experience the extent to which self-management can improve their problem. *Therefore it is generally undesirable to use clinician-generated forces during the first session.*

Most patients in this category respond in the loaded position and are able to do the exercises in sitting or standing. This has the advantage of being easy to apply during everyday activities. A minority of patients, with more severe or acute presentations, may need, at least initially, to be treated in an unloaded position. Because this is much less functional, exercising in the sitting position should be started as soon as the response is favourable.

### Management guidelines

Procedures to be used:

- retraction – essential, sometimes all that is required (Procedure 1)

- retraction with patient overpressure – may be needed to regain retraction prior to extension (Procedure 1a)
- retraction and extension – usually essential (Procedure 2)
- posture correction – essential for reduction and maintenance of reduction (Procedure 3).

Regularity:

- ten to fifteen repetitions every two to three hours.

Expected response:

- centralisation, abolition or decrease of symptoms
- possible increase of pain centrally initially
- increase in all ranges of movement that are restricted.

Maintenance of reduction:

- regular performance of retraction/extension exercises to maintain symptomatic and mechanical improvements
- posture correction when sitting
- if symptoms recur when lying:
  - trial of cervical roll
  - trial reduction of pillows
  - maintenance of retraction when rising from lying.
- see Chapter 17, section on *Maintenance of reduction*, for more detail.

#### *Force alternatives*

If the patient is unable to regain retraction or extension in the loaded position, the same movements are attempted unloaded. This requirement is unusual, but may occur in severe or acute derangements or if the patient is apprehensive about performing the exercises in sitting.

- retraction in supine (Procedure 1)
- retraction and extension in supine (Procedure 2)
- retraction and extension in prone (Procedure 2).

### *Force progressions*

Force progressions are used only if improvements plateau or fail to occur. Before undertaking progressions, the patient's exercise technique and postural correction should be checked. Progressions should never be instigated if the patient is able to decrease or abolish symptoms, but these return due to failure to maintain reduction of the derangement. Progressions may also be used to confirm an initial diagnosis if there is some uncertainty; in other words, the procedures are used as part of the assessment process.

- only use one new procedure per session
- wait twenty-four hours before initiating further progressions
- repeat force progressions a maximum of two sessions if no definite improvement occurs
- the patient must continue with home exercise programme, otherwise any benefit from the force progressions will be lost between treatment sessions
- force progressions are stopped once the patient is able to self-manage
- retraction with clinician overpressure (Procedure 1b) – sometimes this is required to enable the patient to regain enough retraction to perform extension adequately
- retraction and extension with rotation and clinician traction supine (Procedure 2b)
- retraction mobilisation (Procedure 1c).

## Review

When the patient returns for review they will be improved, worsened or unchanged. Ensure that the status reported by the patient is their true state. Patients may report their symptoms to be worse when widespread pain has centralised to the middle of the spine; they may report themselves to be unchanged when in fact pain that was constant has become intermittent. Some patients, keen to please and to get better, report an improvement that is difficult to confirm. See Chapter 12 for details of how to analyse clinical presentations and Chapter 13 for the structure of a review process.

### *Patient is better*

If there is improvement in the symptomatic and/or mechanical response at review, management strategy should not be changed.

It is unnecessary to supplement present procedures with any other techniques or interventions if the patient is getting better. Management continues in the same way unless there is a change in status.

Once symptoms are minimal, the frequency of exercising may be reduced if this seems appropriate, and the patient should be told also about performing the exercises at the first signs of recurrence. Maintenance of reduction through the use of posture correction should be reinforced, and the slouch-overcorrect procedure could be introduced to allow the patient to appreciate the different sitting positions.

### *Patient is worse*

There are certain instances that the patient may interpret as being 'worse', which we would not consider as such. When centralisation occurs there can be an accompanying and temporary increase in central pain. When patients with long-standing derangements and an associated obstruction to extension commence the necessary extension principle, procedures there can sometimes be an initial short-lasting increase in symptoms. The patient may be performing the procedures incorrectly or may have misinterpreted instructions and be doing different exercises. New pains may have appeared as a consequence of performing the exercises, which has made the patient reluctant to continue. Do their symptoms improve with the exercises, but get worse later because of insufficient attention to posture? Did they initially get better with the instructions, but then worsened because they increased their activity level too soon? All these instances should not be considered a worsening scenario.

If really worse, pain is usually more widespread. It should be ensured that the patient definitely has symmetrical symptoms. If a condition is truly worsening, the patient should be advised to stop the exercises – patients sometimes improve with this step. If unequivocally worse, their response to flexion principle should be explored. If there is still a worsening response to all procedures, non-mechanical conditions should be considered.

### *Patient is unchanging*

First it should be ensured that the patient is performing the right exercises correctly and with enough regularity, and that they are complying with postural instructions. If this is not the case, further instruction and discussion are necessary to ensure that the patient understands the procedures and their own role in managing the problem. It is best to see the patient daily until certainty of management is established

and the patient is confident about their management strategy. If the patient cannot be reviewed regularly, this can create problems. In such cases attempts to review by telephone are desirable.

If they have been following the treatment principle correctly, but still no improvement has occurred, then force progressions should be implemented. These are done in the following order, with subsequent force progressions applied only if there is failure to improve. If at any point improvement does occur, further progression is unnecessary. Whatever progression is used, the patient must continue to perform the appropriate exercises at home with suitable regularity. Following a force progression, the effects of this procedure should be evaluated at the next review. Force progressions can be repeated on up to two occasions before they should be abandoned if no change ensues. Do not instigate clinician techniques unless it is clear that improvement cannot be achieved by any other means.

- wait twenty-four hours before initiating further progressions
- repeat force progressions a maximum of two sessions if no definite improvement occurs
- the patient must continue with home exercise programme, otherwise any benefit from the force progressions will be lost between treatment sessions
- force progressions are stopped once the patient is able to self-manage
- retraction with clinician overpressure (Procedure 1b)
- retraction and extension with rotation and clinician traction supine (Procedure 2b)
- retraction mobilisation (Procedure 1c).

## Deformity of kyphosis (previously Derangement 2)

This is a rare and acute presentation in which patients usually have central or symmetrical pain. Extension is obstructed and the patient's head is fixed in protrusion and flexion. Symptoms may radiate bilaterally into their arms. Any attempt to extend the neck gives rise to severe twinges of pain, and the patient avoids such movements by maintaining a flexed posture.

It is impossible to carry out a normal physical examination in such a patient as all movements will be extremely limited and repeated movements are too difficult to perform. However, the obvious deformity and inability of this patient to move normally allows easy recognition. If the patient developed the deformity as a result of some trauma, such as a fall or motor vehicle accident, no clinician-generated forces should be used and imaging studies should be undertaken to ensure no serious spinal damage has occurred.

Response to attempts to reduce these derangements is variable and often limited. Sometimes, in the presence of a 'soft' deformity, patient-generated forces are successful in beginning the process of reduction. In the presence of a 'hard' deformity, sometimes clinician-generated forces are necessary to start this process. Often recovery is protracted even when patients do respond, taking several weeks to fully regain extension. Unfortunately some make only minimal improvements.

Because of the nature of the derangement and the difficulty of performing any movements in a loaded position, treatment is always done unloaded.

Procedures to be used:

- the patient should be laid down with head resting on pillows and/or raised treatment table end so that the flexion deformity is accommodated
- retraction in supine in kyphotic deformity
- depending on symptom response, the treatment table end is lowered/pillows removed slowly and gradually, thus letting the head drop back into neutral and then extension
- retraction/extension in supine or prone (Procedure 2)
- if the patient starts to respond they should continue with retraction/extension supine or prone at home (Procedure 2)
- extension with traction and rotation in supine may help to regain extension and might be used from day two for several sessions
- as soon as possible the patient supplements unloaded exercises with retraction and then retraction/extension in sitting.

## Flexion principle (previously Derangement 7)

### History and examination

There may be certain clues found during the history-taking and physical examination that suggest the flexion principle should be used, which are listed in the previous chapter. The patient might report that they have anterior as well as posterior neck pain and that they have pain or problems with swallowing. Such derangements can result from road traffic accidents. On examination there will be marked loss of flexion, but full-range pain-free extension. This presentation is relatively rare.

### Management guidelines

Management is conducted entirely in the sagittal plane using flexion forces.

Procedures to be used:

- flexion – essential (Procedure 6)
- flexion with patient overpressure – essential (Procedure 6a).

Regularity:

- ten to fifteen repetitions every two to three hours.

Expected response:

- centralisation, abolition or decrease of symptoms
- possible increase of pain centrally initially
- increase in flexion range.

### Review

When the patient returns for review they will be improved, worsened or unchanged. Ensure that the status reported by the patient is their true state. See Chapter 12 for details of how to analyse clinical presentations and Chapter 13 for the structure of a review process.

#### *Patient is better*

If the patient reports an improvement in symptoms – centralised, abolished or decreased in intensity of frequency – this should be confirmed by a mechanical change; that is, an increase in flexion range. It is unnecessary to supplement present procedures with any other techniques or interventions if the patient is getting better. Management continues in the same way unless there is a change in status.

*Patient is worse*

If really worse, pain is usually more widespread. It should be ensured that the patient definitely has symmetrical symptoms. If a condition is truly worsening, the patient should be advised to stop the exercises – patients sometimes improve with this step. If unequivocally worse, their further response to extension principle should be explored. If there is still a worsening response to all procedures, non-mechanical conditions should be considered.

*Patient is unchanging*

First it should be ensured that the patient is performing the right exercises correctly and with enough regularity. If this is not the case, further instruction and discussion are necessary to ensure that the patient understands the procedures and their own role in managing the problem. It is best to see the patient daily until certainty of management is established and the patient is confident about their management strategy. If the patient cannot be reviewed regularly, this can create problems. In such cases, attempts to review by telephone are desirable.

If they have been following the treatment principle correctly, but still no improvement has occurred, then a force progression should be implemented. Do not instigate clinician techniques unless it is clear that improvement cannot be achieved by any other means. Use of clinician-generated forces in this derangement is rare.

- the patient must continue with home exercise programme, otherwise any benefit from the force progressions will be lost between treatment sessions
- force progressions are stopped once the patient is able to self-manage
- flexion with clinician overpressure (Procedure 6b)
- flexion mobilisation (Procedure 6c).

## Conclusions

This chapter has described management of patients with central or symmetrical neck pain from a derangement. All such patients require only sagittal plane forces, most need retraction and extension exercises, and most can perform these in sitting. In a very small proportion, symptoms are accompanied by a deformity of kyphosis; extension forces are also required, but must be performed unloaded. Some

patients with central or symmetrical symptoms require flexion forces. Features from the assessment, the management guidelines and the review procedures are also detailed in this chapter.

# 19: Management of Derangement – Unilateral and Asymmetrical Symptoms to Elbow

**(Previously Derangements 3, 4 and 7)**

## Introduction

Patients in this category have unilateral or asymmetrical symptoms in the neck with or without radiating pain into the scapula, shoulder or upper arm. This group mostly encompasses those with non-specific somatic pain in which the pain-generating mechanism causes limited referral of symptoms. This group involves those formerly classified as Derangements 3, 4 and possibly 7 (McKenzie 1981, 1990). In some patients the pain may have started centrally and then moved to a more lateral location, but in others symptoms may have commenced asymmetrically.

The key decision in those with unilateral asymmetrical symptoms is determining the appropriate treatment principle – sagittal or lateral – and in those with a lateral component who require lateral forces, whether rotation or lateral flexion is most appropriate. There may be clues in the history. The first section of the chapter details the physical examination procedures that are used to determine the appropriate loading strategy during the assessment and first review. This details the way to determine if a lateral component is present. The criteria for identifying a lateral component are reviewed. The management of patients with a lateral component, including force progressions and alternatives, is then detailed.

Sections in the chapter are as follows:

- assessment – determining the appropriate strategy
    - response to extension
    - unchanging – further testing
    - review
- identification of a lateral component
- management – lateral component, no lateral deviation
    - review
- management – lateral component, with lateral deviation or wry neck

- flexion principle
    - history and examination
    - management guidelines
    - review.

## Assessment – determining the appropriate strategy

Many patients with unilateral or asymmetrical symptoms respond to extension exercises performed as described for those with symmetrical pain in the previous chapter. At least 65% respond to purely sagittal plane procedures (McKenzie 1990). If there is not an early response to extension, then the lateral component should also be explored. Worsening of symptoms in response to extension leads to an early introduction of lateral forces.

Management using the extension principle is exactly as described in the last chapter. The same starting procedures and force progressions, if needed, are used.

Procedures to be used:

- retraction – essential, sometimes all that is required (Procedure 1)
- retraction with patient overpressure – may be needed to regain retraction (Procedure 1a)
- retraction and extension – usually essential (Procedure 2)
- posture correction – essential for reduction and maintenance of reduction (Procedure 3).

Regularity:

- ten to fifteen repetitions every two to three hours.

Expected response:

- centralisation, abolition or decrease of symptoms
- possible increase of pain centrally initially
- increase in all ranges of movement that are restricted.

Maintenance of reduction:

- regular performance of retraction/extension exercises to maintain symptomatic and mechanical improvements

- posture correction when sitting
- trial of cervical roll if symptoms recur when lying
- maintenance of retraction when rising from lying if necessary
- see Chapter 17, section on *Maintenance of reduction*, for more detail.

### Force alternatives

If the patient is unable to regain retraction or extension in the loaded position, the same movements are attempted unloaded. This requirement is unusual, but may occur in severe or acute derangements, or if the patient is apprehensive about performing the exercises in sitting.

- retraction in supine (Procedure 1)
- retraction and extension in supine (Procedure 2)
- retraction and extension in prone (Procedure 2).

### Response to extension

In response to extension forces, unilateral symptoms may respond in one of three ways, each with different management implications (Table 19.1). They will either be better, worse or unchanged.

**Table 19.1 Response to extension forces in unilateral asymmetrical symptoms and implications**

| *Response to extension forces* | *Implications* |
|---|---|
| Centralisation<br>Abolish pain<br>Decrease pain | Continue with extension forces |
| Increase distal pain | Introduce lateral component. |
| Peripheralisation | See *Management – lateral component, no lateral deviation* for procedures |
| Indeterminate response<br>Increase, not worse | Progress sagittal plane forces *and* explore lateral component and decide on the most appropriate loading strategy. |

#### *Better*

In the first instance, it is apparent that extension forces are appropriate. There is a rapid favourable symptom response, with decrease, abolition or centralisation of pain, and/or a rapid mechanical response with an increase in range of movement. In this situation management would be conducted according to the extension principle, including any

necessary force progressions, as long as improvements continued (Chapter 18). If the response changed, a review would be necessary.

#### *Worse*

Likewise, in the second instance a rapid peripheralisation alerts one to the inappropriateness of pure extension forces and the lateral component is introduced. A relevant lateral component has been determined from symptomatic response. However, before abandoning the sagittal plane, unloaded extension procedures should have been explored, as above.

In patients with asymmetrical or unilateral symptoms where the pain is worse as a result of the initial assessment of the above extension procedures, or has increased laterally or peripheralised, the effect of addressing the lateral component is introduced on day one. See *Management – lateral component, no lateral deviation* for procedures that may be considered.

#### *Unchanging*

When the symptom response is indeterminate, the management strategy is less clear. In these instances it is important to compare symptom response to sagittal movements with the symptom response to lateral movements and determine if there is a clear mechanically determined directional preference for sagittal or lateral forces. Determining the best strategy requires applying a clinical reasoning process, and over-pressures and mobilisation in both planes may be considered on day one to help determine the appropriate loading. If previous testing has not produced a clear symptom response, these procedures may help to clarify a directional preference on day one.

It may be equally valid at times to test the response to repeated movements over twenty-four to forty-eight hours to see if the longer period produces a more clearly favourable response. This is especially appropriate when there are suggestions in the history or physical examination that a positive response is likely.

#### *Unchanging – further testing*

Further testing takes two forms: first, force progressions in extension principle, and second, lateral procedures including force progressions. If at any point extension force progressions cause peripheralisation or worsening of distal pain, exploration of the lateral component must ensue. The loading strategy to use is determined by the most favourable symptomatic and mechanical response.

Force progressions in extension:

- after each force progression, re-assess patient-generated forces as these may now be effective
- if extension forces start to centralise, abolish or decrease symptoms, continue with retraction/extension
- retraction with clinician overpressure (Procedure 1b)
- retraction and extension with rotation and clinician traction supine (Procedure 2b )
- retraction mobilisation (Procedure 1c).

If extension procedures including force progressions have not produced a favourable response, lateral forces should explored. Usually lateral movements are performed to the side of pain, but if no favourable response is generated the other direction can be explored. This is done in the following order:

- lateral force
- lateral force with patient overpressure.

In determining which lateral force to use, bear in mind the patient's comment on aggravating factors, the movement loss shown during the physical examination, and response to repeated movements. Commonly the most affected movement is the one chosen to explore first with repeated movements. Exploration of both movements may be valuable to determine the most effective one:

- rotation or lateral flexion (Procedures 5 and 5a or 4 and 4a).

If symptoms have not centralised, abolished or decreased, clinician-generated forces should be introduced:

- lateral flexion clinician overpressure (Procedure 4b) *and/or*
- rotation clinician overpressure (Procedure 5b)
- lateral flexion mobilisation (Procedure 4c) *and/or*
- rotation mobilisation (Procedure 5c).

The force that generates the most favourable response is chosen for the repeated movement for the patient to perform every two to three hours over the next few days. If there is still a lack of a clear response, a treatment principle is selected for a trial over the next

twenty-four to forty-eight hours. Sometimes there may be clues in the history or physical examination that suggest a certain movement; if not, it is best to first test the response to retraction/extension procedures. The patient should be taught to recognise positive responses, negative responses and fully understand when exercises should be terminated.

In summary, for patients with unilateral asymmetrical symptoms who do not show immediate benefit using extension forces, the lateral component should always be explored. Overpressures and mobilisation in both sagittal and lateral planes can be used to help determine the appropriate loading strategy. In many patients the addition of the lateral component or force progressions clarify the preferred loading. In effect, the clinician-generated procedures are being used as tools of assessment as well as treatment. A thorough knowledge of the chapter on *Evaluation of clinical presentations* (Chapter 12) is necessary in order to interpret the patient's presentation and responses accurately. Sometimes, however, a trial over twenty-four hours is more revealing than repeated movements during the clinical examination.

### Review

When the patient returns for review they will be better, unchanged or worse. Ensure that the status reported by the patient is their true state. Patients may report their symptoms to be worse when widespread pain has centralised to the middle of the spine; they may report themselves to be unchanged when in fact pain that was constant has become intermittent. Some patients, keen to please and to get better, report an improvement that is difficult to confirm.

#### *Patient is better*

If there is improvement in the symptomatic and/or mechanical response at review, management strategy should not be changed. It is unnecessary to supplement present procedures with any other techniques or interventions if the patient is improving. Management continues with the same loading unless there is a change in symptomatic or mechanical responses.

If patient has been using a lateral force, and symptoms have centralised, response to retraction/extension is re-tested.

#### *Patient is unchanging*

If no centralisation or clear improvement has occurred by the review and the patient has been compliant, then force alternatives and progressions should be considered as outlined above. This may involve

sagittal plane procedures and procedures with a lateral component. It will definitely involve overpressure and therapist mobilisation in order to determine the appropriate loading strategy. Some of these should already have been applied on day one. At all times a clinical reasoning process is used, and the clinician is alert to an unfavourable response that means a procedure should be abandoned at that time.

It is important that the sagittal plane is not abandoned prematurely. Sagittal plane procedures should always include the use of sustained positioning, and unless there is a clear worsening of symptoms, overpressure and mobilisation. Worsening means that symptoms are peripheralised or that distal symptoms remain worse; worsening is not indicated by a temporary increase in response to a limited number of repeated movements. Equally, if there is no lasting response to extension forces, a lateral component should be introduced at an early point. See below – *Management – lateral component, no lateral deviation*.

If the patient has been sent away to test response to sagittal plane forces over twenty-four to forty-eight hours, and they have done this, and force progressions have already been used but no change has occurred, then a more thorough exploration of lateral forces ensues. This entails overpressures and mobilisation, but also an extended testing of their response to lateral forces over a twenty-four- to forty-eight-hour period may be beneficial.

### *Patient is worse*

When centralisation occurs there can be an accompanying and temporary increase in central pain. When patients with long-standing derangements and an associated obstruction to extension commence the necessary extension principle procedures, there can sometimes be an initial short-lasting increase in symptoms. The patient may be performing the procedures incorrectly or may have misinterpreted instructions and be doing different exercises. These instances should not be considered a worsening scenario.

In patients with asymmetrical or unilateral symptoms, a worse scenario is indicated when pain has changed from intermittent to constant, increased in intensity, increased laterally or peripheralised. Lateral forces may be required. Pure extension forces should no longer be used, unless there is a change in symptom response.

## Identification of lateral component

The last section dealt with the evaluation process to be conducted on patients with unilateral and asymmetrical symptoms to determine the most appropriate treatment strategy, which may be the extension or lateral treatment principle. This section reviews the clinical presentation that may indicate a relevant lateral component is present. Certain features of history and physical examination provide clues as to the possibility of a relevant lateral component:

- unilateral or asymmetrical symptoms
- activities of both flexion *and* extension aggravate symptoms
- lateral movement is asymmetrical, with major loss in one direction
- presence of lateral deviation of the head and neck
- symptoms centralise or are made better by lateral movements
- symptoms peripheralise, worsen or are unchanged with retraction/extension procedures in lying
- symptoms are overall unchanged after several days' application of extension protocol.

Identification of a relevant lateral component, which requires the lateral principle, occurs in three ways:

1. Peripheralisation or worsening of peripheral symptoms in response to retraction/extension procedures in lying, or a force progression in the extension principle, as long as sufficient time was allowed for extension procedures and force progressions were not introduced too rapidly.
2. Non-response to sagittal plane forces. When there is no benefit from extension procedures, the lateral component should be explored. It is important to assess the response to lateral forces early when this seems appropriate. Equally, it is important on other occasions to make sure that the sagittal plane is not abandoned prematurely and that an extended mechanical evaluation with force progressions is conducted.
3. Presence of lateral deviation or wry neck
    - The 'soft' lateral deviation – the lateral deviation will have accompanied the recent onset of neck pain. The patient presents with a very visible lateral deformity that they are

initially unable to self-correct. On repetition of lateral techniques in lying, some patients can achieve self-correction without clinician assistance.

- The 'hard' lateral shift – the lateral deviation accompanied the recent onset of neck pain. The patient presents with a very visible lateral deformity that they are unable to self-correct. They are unable to bring their head back to the mid-line in lying, or if they can, are unable to maintain this correction. These patients need clinician assistance.

Management of a relevant lateral component is approached differently depending on the presence or absence of a lateral deviation. If there is no lateral deviation of the head, the symptomatic response to sagittal plane forces will have determined the appropriate use of lateral forces. In the first two situations the lateral principle is adopted after a worsening or unchanging symptomatic and mechanical response to sagittal plane evaluation as described above. In the presence of a wry neck, lateral forces are adopted immediately. Management is described below in two sections:

- *lateral component, no lateral deviation – the much more common* situation
- lateral component, with lateral deviation.

## Management – lateral component, no lateral deviation

This section describes the management strategy adopted for unilateral or asymmetrical pain without a clearly discernible lateral deviation of the neck/head *that has not responded to pure sagittal plane procedures*. During the initial and possibly second assessment, extension procedures and progressions will have been explored as described above. These either caused a worsening of symptomatic or mechanical presentations or caused no substantial change. Worsening refers to a change from intermittent to constant pain, peripheralisation or an increase in intensity of distal symptoms. Thus, the sagittal plane has either generated an unfavourable response or, despite progressions, has made no significant change.

Lateral procedures usually involve movements of the neck and head towards the side of pain. Thus, a patient with left-sided neck/shoulder/scapula pain generally uses left lateral flexion or left rotation. This

is the most common direction used, but if no favourable response is generated, the direction away from the painful side should be explored.

Procedures are performed in the order as listed later, with subsequent force progressions applied only if there is failure to improve. If, at any point improvement does occur, further progression is unnecessary. Whatever progression is used, the patient must continue to perform the appropriate exercises at home with suitable regularity. Following a force progression, the effects of this procedure should be evaluated at the next review. Force progressions can be repeated on up to two occasions before they should be abandoned if no change ensues.

Do not instigate clinician techniques unless it is clear that improvement cannot be achieved by any other means. On occasions, however, the application of clinician overpressure during a clinic session helps to determine the appropriate therapeutic loading strategy. After the application of lateral principle procedures, extension procedures frequently become necessary, especially if pain centralises to the mid-line.

The progressions are listed below in the order that most frequently generates a favourable clinical response. However, in determining the appropriateness of loading strategies, some flexibility in the application of procedures may be required. Application of force progressions and force alternatives should always be conducted with attentive interpretation of symptomatic and mechanical responses.

### Force progressions and alternatives – when relevant lateral component is present

In determining which lateral force to use, bear in mind the patient's comment on aggravating factors, the movement loss shown during the physical examination and the response to repeated movements. Often the most relevant items relate to the patient's comment on aggravating factors and perceived movement loss during the physical examination. Commonly the most affected movement is the one chosen to explore first with repeated movements. Exploration of both movements is often valuable to determine the most effective one.

- Rotation *or* lateral flexion (Procedures 5 and 5a or 4 and 4a).

If symptoms still have not centralised, abolished or decreased, clinician-generated forces should be introduced:

- lateral flexion clinician overpressure (Procedure 4b) *and/or*
- rotation clinician overpressure (Procedure 5b)
- lateral flexion mobilisation (Procedure 4c) *and/or*
- rotation mobilisation (Procedure 5c).

Sometimes patients cannot tolerate movements in the loaded position, in which case lateral flexion and rotation are explored in the unloaded position:

- lateral flexion in supine (Procedure 4) *or*
- rotation in supine (Procedure 5)
- lateral flexion in supine with patient overpressure (Procedure 4a) *or*
- rotation in supine with patient overpressure (Procedure 5a)
- lateral flexion in supine with clinician overpressure (Procedure 4b) *or*
- rotation in supine with clinician overpressure (Procedure 5b)
- lateral flexion mobilisation in supine (Procedure 4c) *or*
- rotation mobilisation in supine (Procedure 5c).

The management strategy adopted is that which causes symptoms to be decreased, abolished or centralised with the greatest degree of patient independence. To help determine the appropriateness of different loading strategies, it may be necessary to apply overpressure. In this way the favoured loading can be confirmed.

### Review

When the patient returns for review they will be better, worse or unchanged. Ensure that the status reported by the patient is their true state.

#### *Patient is better*

If there is improvement in the symptomatic and/or mechanical response at review, management strategy should not be changed as long as a favourable response continues. When symptoms centralise, assess ability to return to use of sagittal plane movements.

*Patient is worse*

Ensure the patient is actually worse. If lateral forces have been used, ensure that you have not abandoned sagittal plane movements prematurely. Check technique. If only loaded techniques have been used, unloaded techniques should be tried. Consider non-mechanical syndromes.

*Patient is unchanging*

First it should be ensured that the patient is performing the right exercises correctly and with enough regularity, and that they are abiding by appropriate postural instruction. If this is not the case, further instruction and discussion are necessary to ensure that the patient understands the procedures and their own role in managing the problem. It is best to see the patient daily until certainty of management is established and the patient is confident about their management strategy. If the patient cannot be reviewed regularly, this can create problems. In such cases, attempts to review by telephone are desirable.

If they have been following the treatment principle correctly, but still no improvement has occurred, then appropriate overpressure and mobilisation should be applied as in the order of force progressions indicated above.

## Management – lateral component, with lateral deviation, wry neck or acute torticollis

These patients, few in number, are identified by the obvious lateral deviation of their head and neck. The patient is fixed in flexion, lateral flexion or rotation, or a combination of these. The patient cannot laterally flex, rotate or extend normally. The pain and deformity is usually of very recent onset. The patient may in some cases be able to bring their head back to mid-line, but is unable to maintain this correction. This deformity is termed a *contralateral shift* if away from the painful side or an *ipsilateral shift* if towards the painful side. It is a rare clinical presentation only seen in the young, aged between ten and twenty. The condition is usually short-lived and resolves spontaneously in three or four days. If seen during this period no intervention should be necessary. If, however, it is still a problem after a week, intervention is justified.

**Table 19.2 Criteria for a relevant lateral deviation**

- head and neck are visibly and unmistakably fixed in lateral flexion and flexion, occasionally with some rotation also
- onset of deviation occurred with neck pain
- patient is unable to correct deviation voluntarily
- if patient is able to correct deviation, they cannot maintain correction
- correction affects intensity of symptoms
- correction affects site of symptoms.

*The application of extension in the presence of the lateral deformity can significantly worsen or peripheralise symptoms. The lateral principle is always adopted immediately, and repeated sagittal plane movements, including postural correction, are not explored initially.*

Some patients with this deviation who initially appear to be fixed in lateral flexion can in fact, with repeated movements in supine, begin to self-correct and start to regain the lost movement. If patients respond in this way, they should be encouraged to continue with self-correction of lateral deviation as long as the symptomatic presentation is improving in line with the mechanical presentation. Such 'soft' deformities should start to show some change within two sets of repeated movements; if after this time nothing is altered, clinician assistance should be applied.

Other patients with a 'hard' deformity are completely unable to modify the mechanical and symptomatic presentation in any way. They are truly fixed in the lateral flexed position, and clinician assistance is needed to correct the deformity and regain mobility.

Time is often very important in this condition, so recovery of movement must not be rushed. At all times there is careful monitoring of distal symptoms. Movements are performed to the side of pain.

Procedures to be used:

- lateral flexion in supine (Procedures 4 and 4a)
- lateral flexion in supine with clinician overpressure (Procedure 4b)
- lateral flexion mobilisation – this procedure is done gently and gradually, with careful monitoring of symptom response (Procedure 4c)

- rotation in supine – some patients respond better to this movement than lateral flexion (Procedures 5 and 5a)
- rotation in supine with clinician overpressure (Procedure 5b)
- rotation mobilisation in supine – this procedure is done gently and gradually, with careful monitoring of symptom response (Procedure 5c)
- if one of these procedures produces a positive response, this is continued at home
- if symptoms centralise to mid-line, retraction/extension is tested to determine if sagittal forces have become relevant.

## Flexion principle

### History and examination

A small group of patients with unilateral or asymmetrical symptoms to the elbow require the flexion principle; clues that may suggest patients need this principle of treatment are listed in Chapter 18. There may be certain clues found during the history-taking and physical examination that suggest the flexion principle should be used. The patient might report that they have anterior as well as posterior neck pain, and that they have pain or problems with swallowing. Such derangements can result from road traffic accidents. On examination there is marked loss of flexion, but full-range pain-free extension. This presentation is relatively rare.

### Management guidelines

Procedures to be used:

- flexion (Procedure 6)
- flexion with patient overpressure (Procedure 6a).

Regularity:

- ten to fifteen repetitions every two to three hours.

Expected response:

- centralisation, abolition or decrease of symptoms
- possible increase of pain centrally initially
- increase in flexion range.

Force alternatives:

If there is limited positive response to pure flexion forces, a lateral component is introduced:

- flexion with rotation (Procedure 6)
- flexion with rotation with patient overpressure (Procedure 6a).

**Review**

When the patient returns for review they will be improved, worsened or unchanged. Ensure that the status reported by the patient is their true state.

*Patient is better*

If the patient reports an improvement in symptoms – centralised, abolished or decreased in intensity of frequency – this should be confirmed by a mechanical change; that is, an increase in flexion range. It is unnecessary to supplement present procedures with any other techniques or interventions if the patient is getting better. Management continues in the same way unless there is a change in status.

*Patient is worse*

If really worse, pain is usually more widespread. If a condition is truly worsening, the patient should be advised to stop the exercises – patients sometimes improve with this step. If unequivocally worse, flexion with a lateral component should be tried, and the response to extension principle should be explored. If there is still a worsening response to all procedures, an irreducible derangement or non-mechanical pathology should be considered (Chapter 9).

*Patient is unchanging*

First it should be ensured that the patient is performing the right exercises correctly and with enough regularity. If this is not the case, further instruction and discussion are necessary to ensure that the patient understands the procedures and their own role in managing the problem. It is best to see the patient daily until certainty of management is established and the patient is confident about their management strategy. If the patient cannot be reviewed regularly, this can create problems. In such cases, attempts to review by telephone are desirable.

If they have been following the treatment principle correctly, but still no improvement has occurred, then force alternatives and progression

should be explored. Do not instigate clinician techniques unless it is clear that improvement cannot be achieved by any other means. Use of clinician-generated forces in this derangement is rare.

Force alternatives:

- flexion with rotation (Procedure 6)
- flexion with rotation with patient overpressure (Procedure 6a).

Force progressions:

- the patient must continue with home exercise programme; otherwise any benefit from the force progressions will be lost between treatment sessions
- force progressions are stopped once the patient is able to self-manage
- flexion with clinician overpressure (Procedures 6b)
- flexion mobilisation (Procedure 6c)
- rotation mobilisation (Procedures 5c).

## Conclusions

This chapter has considered the management of patients with unilateral and asymmetrical symptoms, possibly extending to the elbow, that originate from a derangement. The majority of such patients will respond to sagittal extension forces and, unless a lateral deviation is present, an exploration of the extension principle is always performed. If the response is positive, management continues in the sagittal plane. If there is a worsening of distal symptoms, lateral forces are explored. If after an extended exploration of sagittal forces, including force progressions, there is no change in symptoms, lateral forces should also be explored. Lateral forces may either be lateral flexion or rotation, and which is chosen depends upon symptomatic and mechanical response; a clue can be gained from the movement with the greatest loss of movement.

When considering patients with unilateral and asymmetrical symptoms possibly extending to the elbow that originate from a derangement, two other management considerations should be remembered, but are very rarely used. For patients with a lateral deviation, sagittal

forces are initially contraindicated and unloaded lateral forces are performed immediately. A very small number of this patient group respond to flexion forces.

# 20: Management of Derangements – Unilateral or Asymmetrical Symptoms Below the Elbow

**(Previously Derangements 5 and 6)**

## Introduction

This chapter describes the management of patients with symptoms in the forearm that are referred from the neck. These symptoms may be pain and/or paraesthesia and may be accompanied by pain in the arm, shoulder, scapular region or neck. These patients are those previously classified as Derangements 5 and 6, and comprise up to 20% of the neck pain population (McKenzie 1990). Specific cervical pain with clear aetiology is more likely in this group as it includes those with nerve root involvement. This may be suggested by pain patterns, paraesthesia, muscle weakness or reflex loss. Nerve root pathology may be caused by reducible derangements, which respond positively to mechanical therapy. It may be caused by irreducible derangements or degenerative lesions, such as stenosis around the intervertebral foramina. These will not respond directly, although they often settle over time. This group thus includes a number who, by the nature of their pathology, will be unresponsive to mechanical therapy (see section in Chapter 9 about cervical radiculopathy).

Another important distinction to make in those who have apparent nerve root involvement is between symptoms caused by a primary derangement and those caused by a secondary dysfunction that has arisen because of previous derangement or other trauma causing an adherent nerve root. In the first the focus of management is on reduction of derangement, whilst in the latter the focus is on recovery of function. As these two goals are achieved by very different procedures, differential diagnosis is crucial. Such symptoms may also be caused by degenerative changes causing lateral or central stenosis. In the cervical spine, degenerative changes may cause stenosis in the intervertebral foramina that affects the nerve root producing spondylotic radiculopathy, or stenosis in the central spinal canal that affects the spinal cord producing spondylotic myelopathy (Yu *et al.* 1987). Differential diagnosis between these different groups – reducible derangement, irreducible derangement, adherent nerve root or bony stenosis – is therefore the preliminary aim of assessment.

Of these entities, derangement is the most common cause of symptoms below the elbow. Stenosis is unusual, but should be considered

in older patients with protracted histories. Irreducible derangement is likewise unusual, but should be considered when repeated movements only aggravate and no relieving posture or movement is found. This situation is more likely when severe radicular symptoms are present with clear neurological loss, such as constant numbness, muscle weakness or loss of reflex, and if deformity of protrusion and/or wry neck is present. Adherent nerve root is also uncommon, but should be considered in those with a history of arm symptoms for two months or more, which is now intermittent and aggravated by arm movements.

The differential diagnosis in patients with neck symptoms referred below the elbow is considered in this chapter. There then follows a description of management for patients with derangement. For management and further consideration of adherent nerve root and stenosis, see appropriate chapters.

Sections in this chapter are as follows:

- differential diagnosis
- determining the appropriate loading strategy
    - force alternatives
    - review
- management when deformity is present
- non-responders to mechanical diagnosis and therapy.

## Differential diagnosis

Two items of history are important in helping to determine the source: duration of episode and frequency of symptoms. If neck and arm pain are of recent onset, only derangement or stenosis need be included in the differential diagnosis. An adherent nerve root is a secondary product of derangement or some other trauma and takes time to form. Dysfunction of adherent nerve root is unlikely unless two to three months have passed since the beginning of the episode. Onset of the episode is from the time arm symptoms started, not simply neck pain.

If a patient with a derangement presents with intermittent symptoms, even with neurological signs and symptoms, which in such cases are usually mild or intermittent, the prognosis is usually good and reduction likely. If a patient with a derangement presents with constant symptoms, reduction is also possible but rapid recovery is less likely, especially if symptoms are severe and accompanied by constant neurological signs. Once symptoms have been present for two months or more, what were initially constant symptoms may now be intermittent. When symptoms are constant, derangement or stenosis is the cause, but once intermittency develops, adherent nerve root must also be included in the differential diagnosis.

Other items from the history may be helpful in distinguishing the different categories (see Table 20.1). The patient with a bony stenosis is always older, and whilst symptom behaviour in derangement may be variable from one day to the next and during the day, symptoms behave reasonably consistently in the other syndromes. Arm activities may exacerbate symptoms from derangement or from an adherent nerve root.

A limited number of clues are available from the history, and the table is a guideline only; as can be seen, there is considerable overlap between the different groups. Confirmatory features are found during the physical examination. Repeated retraction and extension may start to centralise, abolish or decrease symptoms from a derangement, but have minimal effect on the other conditions. If retraction and extension cause peripheralisation or an increase in distal pain, unloaded extension or lateral movements may cause centralisation in a derangement. If it is not clear from initial repeated movements, testing flexion is usually helpful to distinguish the different syndromes.

In derangement, repeated application of flexion causes distal symptoms to progressively worsen or peripheralise. There may be pain during movement or pain at end-range and extension movement may decrease or become more painful. In the presence of stenosis, repeated flexion may temporarily decrease symptoms, although these will not remain better. In the presence of an irreducible derangement, repeated flexion increases symptoms, and if there is an entrapment – a type of irreducible derangement – the range of movement may progressively increase with repetition. Within five or ten minutes, however, symptoms and movement will have returned to former levels.

**Table 20.1 Clues to the differential diagnosis between derangement, stenosis and adherent nerve root**

| *History* | *Derangement* | *Stenosis* | *Irreducible derangement* | *ANR* |
|---|---|---|---|---|
| Likely age | 20-55 | >55 | 20-55 | 20-55 |
| Stage | Acute to chronic. More likely acute/ sub-acute. | Acute to chronic. More likely chronic. Maybe acute exacerbation. | Acute to chronic. More likely chronic. | Chronic. > 2-3 months |
| Status | Improving/ variable. Spontaneous resolution over time likely. | Unchanging. Spontaneous resolution may occur over time, or acute exacerbation remit. | Unchanging. Spontaneous resolution may occur over time. | Improved since onset, now unchanging. |
| Symptoms | Constant/ intermittent | Constant/ intermittent | Constant | Intermittent |
| Symptom behaviour | Variable: Better/worse | Consistent | Consistent | Consistent |
| Aggravating | Variable: flexion, sitting, may show MDDP. | Extension, lateral flexion towards pain. | Activity temporary aggravation. | Flexion, arm activity/ stretching. |
| Relieving | Variable: possibly upright, on the move, may show MDDP. | Flexion or lateral flexion away from pain. | No activities, staying mid-range. | Avoidance of tension on nerve root. |
| *Physical examination:* | | | | |
| Neurology exam | Possibly | Possibly | More commonly | Possibly – if present, stable over time. |
| Repeated retraction/ extension | Centralise, abolish, decrease or worse, peripheralise. | Increase or produce, no worse. | Increase, no worse. | No effect or neck pain only. |
| Repeated flexion | Worse, peripheralise, PDM/ERP. | Decrease or abolish, no better. | Increase or decrease, no worse/better. | No effect (or produce, no worse). |
| ULTT | Possibly | Unlikely | Possibly | Very positive |

MDDP = mechanically determined directional preference; PDM = pain during movement; ERP = end-range pain; ULTT = upper limb tension test

The distinction between derangement and adherent nerve root (ANR) can also be confirmed using a combination of repeated flexion and the upper limb tension or brachial plexus tension test. Although this position or 'test' has traditionally been taught with the patient supine, it can equally if less accurately and with less control of shoulder depression be examined with the patient standing or sitting. The arm is abducted to 90 degrees, laterally rotated so the palm faces forward, with the elbow extended, and the wrist can then be extended. The course of the nerve can be further tensioned by contralateral cervical side flexion (Butler 2000). A positive tension test tells us very little; this will occur if derangement, irreducible derangement or ANR is present, although it is less likely when stenosis is present. Repeated flexion in the presence of derangement causes symptoms to peripheralise or worsen with or without the tension position applied. In an ANR, only if the adherence is especially severe would flexion by itself produce symptoms, but if the tension position were held repeated flexion would produce arm pain at end-range. The symptoms would not increase, but would recur with every repetition and would quickly disappear once the movements were stopped. Contralateral side flexion would have a similar effect. This is discussed more fully in Chapter 22.

Management of derangement only is considered in this chapter. Management of other conditions, as well as fuller descriptions, are found elsewhere.

## Determining the appropriate loading strategy

A variety of management strategies and responses are not uncommon in this group. For instance, treatment in an unloaded position is more common; responses are sometimes slower, and traction may be required to enable retraction to be performed. At all times careful monitoring of symptom and mechanical response is essential to ensure the appropriate management strategies are being selected. If nerve root signs and symptoms are present, special caution should be exercised when testing movements and postures, as neural tissue is particularly sensitive to inappropriate application of mechanical forces. Any position or movement that produces or worsens distal symptoms, including neurological ones, should be discontinued.

Many patients with unilateral or asymmetrical symptoms with distal referral respond to extension exercises performed as described for those with symmetrical pain. If there is not an early response to extension, then the lateral component should also be explored. Worsening of symptoms in response to loaded extension procedures should lead to early exploration of unloaded extension procedures and possible consideration of lateral forces.

Management using the extension principle is as described in the appropriate chapter. The same starting procedures and force progressions, if needed, are used. However, unloaded strategies are more commonly required.

Procedures to be used:

- retraction (Procedure 1)
- retraction with patient overpressure – may be needed to regain retraction (Procedure 1a)
- retraction and extension – usually essential (Procedure 2)
- posture correction – essential for reduction and maintenance of reduction (Procedure 3).

Regularity:

- ten to fifteen repetitions every two to three hours.

Expected response:

- centralisation, abolition or decrease of symptoms
- possible increase of pain centrally initially
- increase in all ranges of movement that are restricted.

Maintenance of reduction:

- regular performance of retraction/extension exercises to maintain symptomatic and mechanical improvements
- posture correction when sitting
- if symptoms recur when lying
    - trial a cervical roll
    - trial removal of pillows

- maintenance of retraction when rising from lying if necessary
- see Chapter 17, section on *Maintenance of reduction*, for more detail.

### Force alternatives

If the patient is unable to regain retraction or extension in the loaded position, the same movements are attempted unloaded. In this patient group, loaded strategies are more commonly unsuccessful and unloaded strategies need to be explored:

- retraction in supine – may require a degree of traction initially Procedure 1)
- retraction and extension in supine (Procedure 2)
- retraction and extension in prone (Procedure 2).

#### *Response to extension*

In response to extension forces, unilateral symptoms may respond in one of three ways, each with different management implications (Table 20.2). They will either be better, worse or unchanged.

**Table 20.2 Response to extension forces in unilateral or asymmetrical symptoms and implications**

| *Response to extension forces* | *Implications* |
|---|---|
| Centralisation<br>Abolish pain<br>Decrease pain | Continue with extension forces |
| Increase distal pain<br>Peripheralisation | Introduce lateral component.<br>See *Management – lateral component, no lateral deviation* for procedures |
| Indeterminate response<br>Increase, not worse | Progress forces and explore lateral component and then decide on the most appropriate loading strategy. |

#### *Better*

In the first instance, it is apparent that extension forces are appropriate. There is a rapid favourable symptom response, with decrease, abolition or centralisation of pain, and/or a rapid mechanical response with an increase in range of movement. In this situation management would be conducted according to the extension principle, including any necessary force progressions, as long as improvements continued (Chapter 18). If the response changed, a review would be necessary.

### *Worse*

Likewise, in the second instance a rapid peripheralisation alerts one to the inappropriateness of pure extension forces, and the lateral component is introduced. A relevant lateral component has been determined from symptomatic response. However, before abandoning the sagittal plane, unloaded extension procedures should be explored, as above.

In patients with asymmetrical and distal symptoms where the pain has increased distally or peripheralised during the initial assessment with extension procedures, the lateral component is introduced on day one. See *Management – lateral component, no lateral deviation* (Chapter 19) for procedures that may be considered.

### *Unchanging*

When the symptom response is indeterminate, the management strategy is less clear. In these instances it is important to compare symptom response to sagittal movements with the symptom response to lateral movements and determine if there is a clear mechanically determined directional preference for sagittal or lateral forces. The best strategy is determined by applying overpressures and mobilisation in both planes. These may be considered on day one to help determine the appropriate loading. If previous testing has not produced a clear symptom response, these procedures may help to clarify a mechanically determined directional preference on day one.

It may be equally valid at times to test out the response to repeated movements over twenty-four to forty-eight hours to see if the longer period produces a more clearly favourable response. This is especially appropriate when there are suggestions in the history or physical examination that a positive response is likely.

### *Unchanging – further testing*

Further testing takes two forms. First, force progressions in extension procedures; second, lateral procedures including force progressions. If at any point extension force progressions cause peripheralisation or worsening of distal pain, exploration of the lateral component must ensue. The force to finally use is determined by the most favourable symptomatic and mechanical response.

Force progressions in extension:

- after each force progression re-assess patient-generated forces, as these may now be effective

- if extension forces start to centralise, abolish or decrease symptoms, continue with retraction/extension
- retraction with clinician overpressure (Procedure 1b)
- retraction and extension with rotation and clinician traction supine (Procedure 2b)
- retraction mobilisation (Procedure 1c).

If extension procedures including force progressions have not produced a favourable response, lateral forces should be explored. Usually lateral movements are performed to the side of pain, but if no favourable response is generated the other direction can be explored. This is done in the following order:

- lateral flexion (Procedure 4)
- lateral flexion with patient overpressure (Procedure 4a)
- rotation (Procedure 5)
- rotation with patient overpressure (Procedure 5a).

If symptoms have not centralised, abolished or decreased, clinician-generated forces can be introduced:

- lateral flexion with clinician overpressure (Procedure 4b)
- rotation with clinician overpressure (Procedure 5b)
    - lateral flexion mobilisation (Procedure 4c)
    - rotation mobilisation (Procedure 5c).

The force that generates the most favourable response is chosen for the repeated movement for the patient to perform every two to three hours over the next few days. If there is still a lack of a clear response, a treatment principle is selected for a trial over the next twenty-four to forty-eight hours. Sometimes there may be clues in the history or physical examination that suggest a certain movement; if not, it is best to first test the response to retraction/extension procedures. The patient should be taught to recognise positive responses, negative responses and to fully understand when exercises should be terminated.

When determining the most appropriate loading strategy, both symptomatic and mechanical responses should be taken into account.

Sometimes whilst the symptomatic response is unclear, the patient may demonstrate a definite improvement in range of movement that indicates the appropriate loading strategy.

In summary, for patients with asymmetrical and distal symptoms who do not show immediate benefit using extension forces, the lateral component should always be explored. Overpressures and mobilisation in both sagittal and lateral planes can be used to help determine the appropriate loading strategy. In many patients, the addition of the lateral component or force progressions will clarify the preferred loading. In effect, the clinician-generated procedures are being used as tools of assessment as well as treatment. A thorough knowledge of *Evaluation of clinical presentations* (Chapter 12) is necessary in order to interpret the patient's presentation and responses accurately. Sometimes, however, a trial over twenty-four hours is more revealing than repeated movements during the clinical examination.

### Review

When the patient returns for review they will be better, unchanged or worse. Ensure that the status reported by the patient is their true state. Patients may report their symptoms to be worse when widespread pain has centralised to the middle of the spine; they may report themselves to be unchanged when in fact pain that was constant has become intermittent. Some patients, keen to please and to get better, report an improvement that is difficult to confirm.

#### *Patient is better*

If there is improvement in the symptomatic and/or mechanical response at review, management strategy should not be changed. It is unnecessary to supplement present procedures with any other techniques or interventions if the patient is improving. Management continues with the same loading unless there is a change in symptomatic or mechanical responses.

If patient has been using a lateral force and symptoms have centralised, response to retraction/extension is re-tested.

#### *Patient is unchanging*

If no centralisation or clear improvement has occurred by the review and the patient has been compliant, then force alternatives and progressions should be considered as outlined above. This may involve sagittal plane procedures and procedures with a lateral component. It will definitely involve overpressure and therapist mobilisation in

order to determine the appropriate loading strategy. Some of these should already have been applied on day one. At all times the clinician remains alert to an unfavourable response that means a procedure should be abandoned.

It is important that the sagittal plane is not abandoned prematurely. Sagittal plane procedures should always include the use of sustained positioning, and unless there is a clear worsening of symptoms, overpressure and mobilisation. Worsening means that symptoms are peripheralised or that distal symptoms remain worse; worsening is not indicated by a temporary increase in response to a limited number of repeated movements. Equally, if there is no lasting response to extension forces, a lateral component should be introduced at an early point. See *Management – lateral component, no lateral deviation* (Chapter 19).

If the patient has been instructed to assess the response to sagittal plane forces over twenty-four to forty-eight hours, and force progressions have already been used to no avail, a more thorough exploration of lateral forces is required. This entails overpressures and mobilisation and an extended testing of their response to lateral forces over a twenty-four- to forty-eight-hour period.

#### *Patient is worse*

When centralisation occurs there can be an accompanying temporary increase in central pain. When patients with long-standing derangements and an associated obstruction to extension commence the necessary extension principle procedures, there can sometimes be an initial short-lasting increase in symptoms. The patient may be performing the procedures incorrectly or may have misinterpreted instructions and be doing different exercises. These instances should not be considered a worsening scenario.

In patients with asymmetrical or unilateral symptoms, a worse scenario is indicated when pain has changed from intermittent to constant, increased in intensity, increased laterally or peripheralised, or when neurological signs and symptoms have appeared or become more intense. Lateral forces may be required. Pure extension forces should no longer be used unless there is a change in symptom response. If both sagittal and frontal plane movements, both loaded and unloaded, cause any of these worsening symptom responses, further testing should be halted. Rigorous testing in the presence

of a major derangement or neurological symptoms can seriously exacerbate the condition.

## Management when deformity is present

Rarely, patients with constant symptoms of cervical radiculopathy and neurological signs may also present with a deformity of recent onset. This may be principally in one plane or a combination of lateral and sagittal. The patient may be fixed in protrusion and be unable to retract, or be fixed in a combination of lateral flexion and rotation and be unable to bring their head to mid-line. Alternatively, the deformity may consist of some combination of the two.

Such patients should always be treated in the unloaded position. If the principle deformity is in the sagittal plane, then retraction in lying is the first procedure attempted (Procedure 1). Traction may be required to aid in regaining retraction, or if time is important and the position must be sustained. Sometimes retraction and extension with rotation and clinician traction supine (Procedure 2b) is helpful, but should be used with caution. If the patient starts to respond and regain retraction and some extension, and can tolerate these procedures in the loaded position, management is conducted as outlined in earlier chapters for the extension principle.

If the principle deformity is lateral flexion and/or rotation, again, treatment starts in the unloaded position, but lateral movements are addressed first. Both lateral flexion and rotation should be investigated to determine the best symptom and mechanical response (Procedures 4 and 5). As soon as the patient can tolerate the same procedures in the loaded position, this is adopted. If symptoms are centralising, but respond to lateral movements plateaus, the extension principle is explored. Force progressions in either sagittal or lateral plane are as outlined in earlier chapters.

It should be recognised that a high proportion of this group will not benefit from mechanical therapy or any conservative treatment. However, their mechanical response should always be explored in case this is not so. Failure to respond and when all attempts at movement cause an aggravation of peripheral symptoms suggests the need for further investigation. Such patients may improve over the course of time, but are possible surgical candidates for a more rapid improvement in symptoms.

## Non-responders to mechanical diagnosis and therapy

A proportion of patients with pain into the forearm and accompanying neurological signs and symptoms are unresponsive to mechanical diagnosis and therapy, especially if distal and neurological signs and symptoms are constant. During a protracted physical examination over several sessions, exploring loaded and unloaded sagittal and lateral forces, no movement or position will be found that produces a lasting centralisation, abolition or decrease in symptoms. Pathologically, failure to respond may relate to an irreducible derangement or lateral stenosis. Both categories of patients often improve gradually over time; however, there may also be some specific advice that might help bring about a gradual resolution.

Patients with symptoms from stenosis may find that the performance of regular movements away from the stenotic, foraminal closing positions help to relieve the arm pain and allow it to 'settle down'. In effect, advice is given to 'stop irritating' the painful structure in the hope that this allows it to become less sensitive and thus stop the pain-generating mechanism. Typically the patient would be advised to perform regular flexion and contra-lateral lateral flexion.

For patients with irreducible cervical disc herniation, surgery might be a consideration (see Chapter 9). However, there is commonly improvement over time with natural history (Bush *et al.* 1997; Maigne and Deligne 1994; Mochida *et al.* 1998) or if treated conservatively (Rosomoff *et al.* 1992; Saal *et al.* 1996). Patients, some of whom had chronic radiculopathy, improved with an intensive multidisciplinary programme with long-term follow-up (Rosomoff *et al.* 1992). More directly relevant, patients with a confirmed diagnosis of cervical disc herniation, mostly confirmed as disc extrusion, generally responded well to an aggressive programme of physical rehabilitation over three months following pain control measures (Saal *et al.* 1996). In a cohort of twenty-six patients, twenty achieved good or excellent outcomes confirmed by a one-year follow-up (nineteen with disc extrusions), and two came to surgery.

As the symptoms improve over time, the focus of treatment should be on restoring cervical mobility and, if necessary, neural mobility. If the patient is assessed several months after onset, they may have developed a dysfunction with or without an ANR. Careful monitoring of distal symptoms is necessary to avoid any exacerbation of the

radicular symptoms as they commence a remodelling programme. The patient should also be provided with education about postural correction and prophylactic measures including encouragement to resume regular physical activity.

## Conclusions

Patients with neck pain with referred symptoms below the elbow commonly have derangement; the management of this group has been described in this chapter. Initially this involves exploration of sagittal extension forces – if a positive response is forthcoming, management continues with the extension principle. If extension causes distal symptoms to worsen or if there is no response to an extended exploration of extension forces, then lateral forces are explored. If the loading strategy is unclear, exploration of both planes with force progressions is conducted in an attempt to clarify the appropriate management. Some patients in this group will have an irreducible derangement and will not respond to conservative therapy. This is more likely in those patients with constant radicular pain and constant neurological signs and symptoms.

Not every patient in this category will have derangement. In some patients a previous derangement may have resolved and they are left two to three months later with an adherent nerve root. In other patients, usually older, symptoms are due to degenerative changes that have led to stenosis that has impinged on the nerve root. These differential diagnoses are considered elsewhere.

# 21: Cervical Dysfunction Syndrome



## Introduction

In dysfunction syndrome the mobility or function of soft tissues is reduced because of structural impairment. It is a painful disorder caused by loading or stretching tissue that is imperfectly repaired or has become adaptively shortened (McKenzie 1981, 1990). Structural impairment arises from contractures, adhesions, scar tissue or imperfect repair. This is the result of trauma, poor postural habit, degenerative processes or derangement (McKenzie 1981, 1990). In dysfunction syndrome, structurally impaired tissue gives rise to pain with normal mechanical loading.

Dysfunction is the second most common cervical mechanical syndrome, but it is much less frequent than derangement. In an audit of the classification given to 265 patients, seventy-eight had neck pain; of these, six (8%) had pain from dysfunction (May 2004a). Derangement is the most common classification, and it is not possible at the outset to make a diagnosis also of 'underlying dysfunction'. The derangement is always treated first as the main source of symptoms, which may present with end-range pain. Often after the derangement is reduced, no 'dysfunction' remains to be treated.

Sections in this chapter are as follows:

- categories of dysfunction
- pain mechanism
    - trauma
    - derangement
    - degeneration and poor postural habit
- clinical picture
    - onset
    - symptoms
    - intermittent end-range pain
- physical examination
- management of dysfunction syndrome

- instructions to all patients with dysfunction syndrome
- management of extension dysfunction
- management of flexion dysfunction
- management of rotation dysfunction
- management of lateral flexion dysfunction
- management of multiple direction dysfunction.

## Categories of dysfunction

Dysfunction affects peri-articular, contractile or neural structures (McKenzie 1981, 1990; McKenzie and May 2000, 2003). In an articular dysfunction, end-range movement in one or more directions that puts tension or compression on the affected structure provokes the pain. In a contractile dysfunction, pain is experienced during active or resisted movement that loads the affected tissue. Contractile dysfunction occurs predominantly in tendons; muscle tissue, being well vascularised, in general heals uneventfully. The number of lengthy tendons taking heavy loads that exist in the limbs is probably the reason for the common occurrence of contractile dysfunction in peripheral musculoskeletal conditions. Contractile dysfunction is described elsewhere (McKenzie and May 2000). There is also a specific form of dysfunction involving the nerve root or dura complex known as an 'adherent nerve root' (ANR); this is described in the next chapter. In this syndrome, placing tension on the course of the involved nerve reproduces symptoms.

In extremity problems it is relatively straightforward to distinguish articular from contractile dysfunction, whereas in the spine the distinction is not so clear. In the spine the syndrome presents primarily as articular dysfunction, but involvement of contractile tissues cannot be ruled out.

In a spinal dysfunction, when normal movement is attempted the range is restricted and abnormal tissues are prematurely placed on full stretch or compression. End-range articular loading reproduces symptoms. Attempts to move further towards end-range result in pain. Mechanical deformation of free nerve endings within these tissues produces pain at a restricted range of movement. Once a painful restriction is reached, attempts to push further into range increases the pain being experienced as greater mechanical deformation is

being placed on the abnormal tissue. The pain is felt at the end of the existing range and ceases when the end-range stretch is released.

Dysfunction is classified by the direction that is limited and painful, so in flexion dysfunction there is pain and limitation of movement on attempting end-range flexion. In extension dysfunction, there is pain and limitation of movement on attempting end-range extension. In the cervical spine, multi-directional dysfunctions are not uncommon; in this entity, multiple movements are affected. Dysfunction of ANR is dealt with separately (Chapter 22).

## Pain mechanism

Dysfunction syndrome is connected with a history of a past notable event or else arises insidiously as a result of tissue degeneration. The syndrome can follow a traumatic event, most commonly in cervical dysfunction involvement in a road traffic accident or from a previous derangement, or may arise insidiously following poor postural habits or degenerative changes.

### Trauma

Pain in the dysfunction syndrome should be seen in the context of the healing process following soft tissue injury. In ideal circumstances following tissue damage, regardless of the site of injury, healing passes through three stages (Evans 1980; Hardy 1989; Enwemeka 1989; Hunter 1994; Witte and Barbul 1997; Barlow and Willoughby 1992; Carrico *et al.* 1984). Each stage is necessary to restore the damaged structure to optimal function.

Several factors can operate to promote a less than optimal repair if remodelling is not properly implemented. The granulation tissue, which repaired the damage, can later act as glue to prevent movement between tissue interfaces. There may also be increased molecular cross-linkage – these processes may produce adhesion formation and impair collagen gliding (Hunter 1994; Donatelli and Owens-Burkhart 1981). Without the appropriate stresses, the scar tissue remains disorganised and structurally impaired.

Collagen repair contracts from the third week unless appropriate stresses are applied. Contracture of old scar tissue may in fact occur for years after the problem originated (Evans 1980; Hunter 1994). Failure to perform the appropriate tissue stretching leaves the

repair process complete, but the remodelling stage incomplete – the individual may still be bothered by pain and limited function, and the tissue 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. (see Chapter 3, McKenzie and May 2003, for fuller description of healing process).

It is generally not possible or even necessary to identify the specific structure that is at fault in dysfunction syndrome. All connective and muscle tissue heals in the same way by primary formation of granulation tissue and scarring. The only exception is cartilage, which is avascular and lacks the inflammatory response (Barlow and Willoughby 1992). Irrespective of where in the musculoskeletal system damage may have occurred – in ligament, muscle, intervertebral disc, zygapophyseal joint capsule or aponeurosis – healing by fibrous repair will eventually follow. Any of these structures may be a source of dysfunction; precise identification of the structure involved is not necessary to promote the appropriate remodelling strategy.

As mentioned earlier, the most common causative trauma in cervical dysfunction is road traffic accident (RTA) leading to whiplash associated disorders (WAD). Management of this condition is dealt with at length in Chapter 25. These patients may present at an acute or chronic stage. The differential diagnosis of dysfunction only becomes relevant at the chronic stage, when six to eight weeks have elapsed since the accident. If a dysfunction is present repair will have occurred, but the patient has been reluctant to move and has avoided recovery of end-range movements. Often multiple movements have been affected, although this may be asymmetrically, so flexion, extension, rotation and side bending may all be painful and limited.

### Derangement

Not every patient with dysfunction will present with a history of trauma; it may also follow a history of derangement. The patient typically has had an acute episode of neck pain some time in the past that has substantially improved, but not fully resolved. They are left with intermittent pain and a permanent restriction of movement. This is not due to the original derangement, but to tension or compression on the repair itself.

Because stress of the repair is painful, the patient considers the injury to still be present and avoids the end-range movement that produces

pain. Continuing avoidance of the painful end-range movement allows the structural impairment to persist, and without intervention a general deterioration in the range of movement is inevitable. Continuing contracture of the fibrous collagenous scar tissue will further limit mobility, and such inextensible repair causes pain whenever the patient attempts full end-range movement. Movement loss is usually asymmetrical following a derangement.

### Degeneration and poor postural habit

The degenerative process is another common cause of dysfunction in the cervical spine producing limited painful movement. Reduced spinal mobility may also be linked to poor postural habits maintained during earlier decades of life (McKenzie 1981, 1990). This is especially so when the individual is under-exercised, leads a largely sedentary lifestyle and their occupation is predominantly desk-bound or at the wheel of a vehicle.

There is a gradual reduction of cervical spinal mobility that occurs with ageing (Worth 1994; Trott *et al.* 1996; Chen *et al.* 1999). For instance, as people age there is a tendency for their normal head posture to become more protruded and for the distance they are able to retract to decrease significantly (Dalton and Coutts 1994). Those over 50 years of age have about 70% to 90% of the movement that was available at 20, with side bending and sagittal movements most affected (Trott *et al.* 1996; Dalton and Coutts 1994). However, there is considerable variability in the normal range of movement in the older population. It is likely that maintenance of activity and postural habits throughout life have a role in determining ultimate range of movement. *"Neck movement diminishes with age, probably owing in great part to decreasing physical activity and relative immobilization rather than an intrinsic characteristic of advancing age"* (Bland 1994, p. 51).

Movements that are not performed regularly are likely to diminish. Later these may become uncomfortable to do, and are avoided as this is seen as the inevitable accompaniment to old age. Reductions are likely to be associated with considerable soft tissue adaptation, and the movement becomes impossible to perform without producing pain. The individual may assume that this is the anticipated effect of maturity.

Physiologists estimate that up to half of what we currently know as usual ageing is a phenomenon of disuse (O'Brien Cousins 1998). There is abundant evidence that older individuals can positively affect their mobility and physical function by reversing the effects of a sedentary lifestyle and becoming more active (O'Brien Cousins 1998). The effects of stress deprivation on connective tissue are well known and include increased random deposition of collagen, increased collagen cross-links, formation of adhesions and contractures in and between the synovial membrane, capsule and other tissues, and generalised osteoporosis (Akeson *et al.* 1987; Bland 1993; Videman 1987). All these physiological changes have the effect of reducing the available range of movement. Symptoms of stiffness and pain associated with a premature limitation of movement, and related to contracture of the joint capsule, are seen as part of the degenerative process (McCarthy *et al.* 1994; Threlkeld and Currier 1988). When dysfunction results from poor posture and spondylosis, symptoms and movement loss tend to be symmetrical.

## Clinical picture

### Onset

Patients with dysfunction syndrome present with characteristic findings in the history-taking and physical examination. The individual will have a history of trauma or derangement, or in an older individual symptoms may have developed insidiously.

When severe neck pain or significant trauma marked the onset of this episode some time will have elapsed; at least six to eight weeks is probably necessary to allow dysfunction to develop. They may report involvement in a road traffic accident in the past. Since the onset the pain has eased considerably, but is now unchanging. When the onset has been insidious, the patient will be older, poor posture will be obvious, there may be an episodic history of neck pain in the past, and symptoms and functional impairment may well be worsening gradually over time.

### Symptoms

Except in one instance pain from dysfunction is felt locally around the spine with limited radiation possibly to the scapulae or upper trapezius region. Only in the presence of ANR is pain referred into the arm and/or forearm. ANR is a type of dysfunction that can follow the resolution of a derangement with referred symptoms. Besides this

one exception, all dysfunctions present with local neck pain only; this may be symmetrical, unilateral or asymmetrical.

### Intermittent end-range pain

Pain is always intermittent in dysfunction syndrome – this is a key identifying characteristic. It does not persist for long periods, but is always associated with certain limited end-range movements. Sometimes this is apparent to patients; they comment that every time they look over their shoulder, for instance, they produce their pain, but this goes once they return to the neutral posture.

Whether apparent to patients or not, consistency of aggravating factors is another key identifying characteristic. It is always end-range movements that provoke symptoms in articular dysfunction; this is when the adaptively shortened tissue is stressed or compressed. This happens much sooner in a patient with dysfunction than in a normal person, and the greater the loss of function the more often pain is provoked. The same movement(s) consistently provoke their pain, with retraction, extension, flexion and rotation being the most commonly limited and painful movements. End-range pain consistently brought on by the same movement(s) and not amenable to rapid change is another key identifying characteristic of dysfunction.

The patient is usually also aware that they are less flexible than they were. Although painful, patients often also feel that 'it just won't go'. There is a noticeable resistance to movement at the same time as the pain. Patients with dysfunction often experience stiffness and more pain first thing in the morning, which loosens up and gets somewhat easier as the day goes on.

The vital questions in identifying a dysfunction thus relate to consistency of the aggravating factor and relief from symptoms once the aggravating position is released. 'Does looking over your shoulder (for instance) always bring on your symptoms?' 'When you come back to mid-line (for instance), does the pain go away, or does it persist?' If the patient responds that *sometimes* a movement is painful, and *sometimes* not, or that *sometimes* the pain persists for hours at a time, derangement is the likely diagnosis and a dysfunction classification should be discounted. If, however, they respond that *always* when they perform that movement it is painful, but that *always* afterwards the pain goes, a dysfunction is more likely.

## Physical examination

Poor posture may be noted, with protruded head posture most common. When posture and spondylosis are the cause of dysfunction, there may also be an exaggerated cervico-thoracic kyphosis, the so-called 'Dowager's Hump', especially when there is a major loss of extension.

There is always reduced movement in spinal dysfunction. When dysfunction results from some discrete past event such as an accident or derangement, movement loss may be asymmetrical. When dysfunction results from poor postural habit or spondylosis, movement losses are generally symmetrical in all directions and affecting many segments. Upon attempting the movement at premature end-range, pain is produced. In the cervical spine, dysfunction affects sagittal and lateral movements equally, and multi-directional losses of movement are common.

In a dysfunction repeating the painful movement consistently produces symptoms on every occasion at end-range, and there will be no change in range or any other aspect of mechanical presentation. There is no pain during the movement, but only when their full but restricted end-range is achieved. Once the repeated movements cease, pain rapidly abates. Within a few minute of stopping the patient is no worse, and returns to a pain-free state. Repeatedly performing the same movement every time, without fail, reproduce their pain at end-range, which abates when movement ceases. Repeated movements in one direction have no effect on pain or range of the opposite movement. Unlike derangement, there will be no rapid changes in symptomatic or mechanical presentations in dysfunction syndrome. Symptoms and mechanical restriction will persist for many weeks.

A dysfunction classification should be suspected during the history-taking, and the appropriate focussed questions given above should be asked. The physical examination should merely endorse the initial clinical suspicions. The diagnosis should be confirmed at review after twenty-four to forty-eight hours of mechanical evaluation. If at any point there is doubt concerning the classification, hypothesis testing should focus on the diagnosis of derangement, which also frequently presents with end-range pain. This is the most common mechanical syndrome and is susceptible to aggravation if mismanaged. If a derangement is the classification, the derangement is always treated first as the main source of symptoms. They frequently also present

with end-range pain, and it is not possible to know if there is an underlying dysfunction until the derangement is reduced.

**Table 21.1 Articular dysfunction syndrome – criteria *(all will apply)***

*History:*

- gradual onset or six to eight weeks post-trauma
- spinal symptoms only (except adherent nerve root)
- intermittent symptoms
- functional activity/movement that consistently provokes symptoms.

*Physical examination:*

- movement is restricted, and the restricted movement(s) consistently produce concordant pain at end-range, and
- there is no rapid decrease or abolition of symptoms, and
- no lasting production and no peripheralisation of symptoms.

## Management of dysfunction syndrome

The treatment of adhesions, contractures or adaptive shortening as in an articular dysfunction essentially requires the application of movements that encourage the process of remodelling. Only with the application of such loading strategies will normal tissue function be re-established. Ideally such movements commence during the stages of repair and remodelling in the weeks after an injury (Evans 1980; Hardy 1989; Hunter 1994; Barlow and Willoughby 1992). If appropriate and graded tension is applied to injured tissue during the proliferative and remodelling phases of healing, adhesions and contractures will not form and dysfunction is prevented. The longer the time lapse between repair and the initiation of the recovery of full function, the more consolidated the scar tissue. Thus the task of remodelling will be more difficult and the time to recovery will be longer. Once the scar tissue is well consolidated, the *very nature of the abnormal tissue prohibits a rapid recovery of function*.

In such cases the remodelling of collagen by applying a long-term structured exercise programme is necessary. By applying regular stress sufficient to provide tension without damage, collagen undergoes chemical and structural changes that allow elongation and strengthening of the affected tissue. Because tissue turnover is slow, one must recognise that it may be a long process. If the contracture has been present for some time, the remodelling programme has to be followed for several months; Evans (1980) reports that some patients may have

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

The process of recovery in dysfunction is therefore lengthy and should be measured in weeks and months rather than days. During this period, because the nature of healing is slow in this syndrome, patients may become frustrated due to the lack of apparent change. They must be encouraged to persevere as a programme of remodelling is the only solution and warned early on that a relatively lengthy period of rehabilitation awaits them. 'Every day you should try and move a little further than the day before' might be helpful advice. Given the tendency of old scar tissue to contract over time, stretching must be performed frequently if remodelling is to occur. If the intervals between stretching procedures are too long, the length of time when no stretching takes place negates the effect of stretching. In dysfunction syndrome exercises to restore movement and function must be performed repeatedly at two- to three-hour intervals throughout the day – each session should consist of ten to fifteen stretches.

To achieve a remodelling effect, exercise must be firm enough to cause change, but not so excessive as to produce micro-trauma. If no strain pain is felt when the exercise is done, it is a waste of time. *The pain that the patient complains of must be produced each time the stretch is performed.* However, equally important, the pain must quickly subside when the stretching is completed. If pain persists long after the exercises are finished, either over-stretching has occurred, with micro-trauma and further tissue damage, or else the original classification was wrong or has changed and a derangement may be responsible for the exacerbation.

In many instances of cervical dysfunction the condition has developed or been aggravated by postural stresses. Furthermore, pains of postural origin may mask and confuse the analysis of the mechanism of pain production. For this reason it is always necessary to give patients postural instruction to address all components of their problem and ensure improved spinal health in the future.

## Instructions to all patients with dysfunction syndrome

Patients will be attending the clinic with pain. To be told that they must go away and regularly cause the pain that they are complaining about needs a very good explanation to gain their adherence to the programme. As long as patients are given a good justification for performing the exercises, most will follow the advice that is given. Most will understand the idea of scar tissue that needs to be 'stretched' to recover full movement; that stretching the scar hurts, and on releasing the stretch the pain will abate. Reassure patients that when their pain is consistently reproduced they are affecting the necessary tissues – 'if it doesn't hurt it isn't right'. Before giving patients the following guidelines, it is essential that they understand the reason they are performing the exercises.

**Table 21.2 Instructions to patients with dysfunction syndrome**

- exercises must be performed regularly throughout the day, every two to three hours
- if patients are unable to exercise as regularly as recommended, recovery of full function is likely to take longer
- at each session perform ten to fifteen stretches
- if the exercise does not produce their pain, it has not been performed properly
- the exercise must consistently reproduce their pain each time
- the pain should have subsided within ten minutes after the completion of the exercises; mostly it will abate much more quickly
- if pain from the procedures persists constantly afterwards for a long period either overstretching has occurred, in which case repetitions must be reduced, or the original classification was mistaken or has changed – in either case a review is necessary
- if the patient feels they are getting worse they must stop exercising and return for a review appointment
- there will be no rapid changes in range of movement – if they experience a dramatic change in function or range they must return for re-evaluation
- if there is a spread of pain distally or a rapid deterioration in their situation, they must stop exercising and return for a review appointment.

Some caution should be applied if the patient has recently recovered from a derangement or has had regular episodes of neck pain in the past and presents with a flexion dysfunction. In such instances flexion exercises will have to be performed regularly; these, however, can constitute a risk in provoking a derangement. It should be emphasised to the patient that they should never be worse when they have stopped the exercises, they must not provoke constant or

peripheralising pain, and that their ability to retract and extend the neck should be maintained.

Clinician techniques and passive modalities have little or no role in the treatment of dysfunction. Only the patient is able to perform the appropriate exercise with sufficient regularity to ensure remodelling takes place. Manipulation procedures may cause minor trauma and perpetuate the cycle of repair and failure to remodel. Mobilisation may possibly be able to generate the appropriate tissue tension, but without regular exercises the timespan between remodelling sessions is totally inadequate to achieve a lasting change. Stretching two or three times a week or even once a day is insufficient. Heating tissues may make collagen more pliable, but again the effect is short-term; the heating may not be deep enough for the affected tissues, and is in any case unnecessary to achieve improved function. For a discussion about the literature on stretching exercises, see McKenzie and May (2003).

## Management of extension dysfunction

Lower cervical extension dysfunction is relatively common. Once the patient has been given advice about removing postural stresses, they must then be shown the appropriate remodelling exercises. This must be accompanied by a thorough explanation as outlined above. A thorough and convincing explanation to the patient is a prerequisite to gain their committed involvement. Procedures to be used all come under the extension principle.

Procedures to be used:

- retraction – with patient overpressure (Procedures 1 and 1a)
- retraction with extension (Procedure 2).

Regularity:

- ten to fifteen times every two to three hours.

Expected response:

- temporary (ten minutes maximum) localised neck pain
- pain produced with exercise is concordant with patient's complaint
- pain occurs at limited end-range

- pain abates rapidly when exercises stop
- all other movements remain as they were
- improved pain and range within four to six weeks; full recovery may take longer
- it might be found that extension is the more symptomatic movement, in which case the focus should be on that movement.

Maintenance:

- once range of movement and pain are improved, patients should be advised to maintain ten to fifteen repetitions once a day to help prevent recurrence.

Supplementary techniques if needed:

- retraction and extension in supine (Procedure 2)
- retraction and extension in prone (Procedure 2).

Force progressions:

- force progressions are rarely required, but if the patient has very limited retraction, extra force may help improve range
- only use one new procedure per session
- wait twenty-four hours before initiating further progressions
- repeat force progressions a maximum of two sessions if no definite improvement occurs
- the patient must continue with home exercise programme, otherwise any benefit from the force progressions will be lost between treatment sessions
- retraction with clinician overpressure (Procedure 1b)
- retraction mobilisation (Procedure 1c).

## Management of flexion dysfunction

Flexion dysfunction may occur after resolution of a posterior derangement. This is particularly so if the derangement was complicated by referred symptoms, in which case the patient may also present with an adherent nerve root (see Chapter 22). Once the patient has been given advice about removing postural stresses they must then be shown the

appropriate remodelling exercises, which must be accompanied by a thorough and convincing explanation to the patient as outlined above as a prerequisite to gain their committed involvement. Procedures to be used all come under the flexion principle.

As recovery of function following posterior derangement requires flexion forces that may aggravate the initial problem, certain precautions should be exercised. The patient especially should be warned about the development of referred symptoms. However, recovery of flexion should not be avoided as it is equally important for a recovery of full function and optimal spinal health.

**Table 21.3 Recovery of function – ensuring stability of derangement**

*Determining if derangement is stable:*

- monitor symptomatic response
  - end-range symptoms may be produced, no worse
  - symptoms may become less painful on repetition
  - symptoms should *not* be felt *during* the movement, only at end-range
  - symptoms must *not* be produced and remain worse
  - symptoms must *not* become more painful on repetition
  - symptoms must *not* peripheralise
- monitor mechanical response – range of movement of and response to extension *must* remain unchanged following repeated flexion.

*Introducing flexion:*

- commence with less stressful flexion procedures, and progress only if needed, as that procedure is easily tolerated, or bringing no further improvements
  - perform new exercises less frequently
  - avoid over-vigorous flexion procedures within first few hours of waking – during this time period the disc is likely to be under increased pressure as a result of nocturnal re-absorption of fluid
  - initially perform ten repetitions of flexion from mid-day on, every three hours until retirement. If the derangement appears stable, the patient may commence the exercise a little earlier in the day and repeat it every two hours
- following flexion exercises, always perform retraction/extension.

Although it is important to be cautious when introducing flexion procedures in certain circumstances, it is important also not to exaggerate the 'danger' of normal daily movements. We must not be guilty of giving our patients problems and making them over-anxious or fearful of certain movements.

Procedures to be used:

- flexion (Procedure 6)
- flexion with patient overpressure (Procedure 6a)
- retraction and extension – for prophylaxis (Procedure 2).

Regularity:

- initially only five repetitions five times a day
- after a few days if no problems ten to fifteen repetitions every two to three hours
- only introduce overpressure after five or six days, and when symptoms are minimal with flexion only.

Expected response:

- temporary (ten minutes maximum) localised neck pain
- pain produced with exercise is concordant with patient's complaint
- pain occurs at limited end-range
- pain abates rapidly when exercises stop
- all other movements remain as they were
- improved pain and range within four to six weeks; full recovery may take longer.

Progressions:
Force progressions are rarely required, but if improvements slow down or cease the following progression might be considered:

- flexion with clinician overpressure (Procedure 6b)
- flexion mobilisation (Procedure 6c)
- if symptoms are unilateral or asymmetrical sometimes flexion-lateral procedures are necessary
- repeat force progressions a maximum of two sessions if no definite improvement occurs
- the patient must continue with the home exercise programme; otherwise any benefit from the force progressions will be lost between treatment sessions.

## Management of rotation dysfunction

Loss of rotation is seen relatively commonly. Once the patient has been given advice about removing postural stresses, they must then be shown the appropriate remodelling exercises. This must be accompanied by a thorough explanation as outlined above. A thorough and convincing explanation to the patient is a prerequisite to gain their committed involvement. Procedures to be used are all under the lateral principle.

Procedures to be used:

- rotation (Procedure 5)
- rotation with patient overpressure (Procedure 5a).

Regularity:

- ten to fifteen repetitions every two to three hours
- introduce overpressure after five or six days or when symptoms are minimal with rotation only.

Expected response:

- temporary (ten minutes maximum) localised neck pain
- pain produced with exercise is concordant with patient's complaint
- pain occurs at limited end-range
- pain abates rapidly when exercises stop
- all other movements remain as they were
- improved pain and range within four to six weeks; full recovery may take longer.

Progressions:
Force progressions are rarely required, but if improvements slow down or cease, the following progression might be considered:

- rotation with clinician overpressure (Procedure 5b)
- rotation mobilisation (Procedure 5c)
- repeat force progressions a maximum of two sessions if no definite improvement occurs

- the patient must continue with the home exercise programme; otherwise any benefit from the force progressions will be lost between treatment sessions.

## Management of lateral flexion dysfunction

The patient less commonly notes this problem as the movement has less functional use than rotation. It frequently coexists with rotation dysfunction and may require specific exercises. Once the patient has been given advice about removing postural stresses, they must then be shown the appropriate remodelling exercises. This must be accompanied by a thorough explanation as outlined above. A thorough and convincing explanation to the patient is a prerequisite to gain their committed involvement. Procedures to be used are all under the lateral principle.

Procedures to be used:

- lateral flexion (Procedure 4)
- lateral flexion with patient overpressure (Procedure 4a).

Regularity:

- ten to fifteen repetitions every two to three hours
- introduce overpressure after five or six days or when symptoms are minimal with lateral flexion only.

Expected response:

- temporary (ten minutes maximum) localised neck pain
- pain produced with exercise is concordant with patient's complaint
- pain occurs at limited end-range
- pain abates rapidly when exercises stop
- all other movements remain as they were
- improved pain and range within four to six weeks; full recovery may take longer.

Progressions:
Force progressions are rarely required, but if improvements slow down or cease, the following progression might be considered:

- lateral-flexion clinician overpressure (Procedure 4b)
- lateral-flexion mobilisation (Procedures 4c)
- repeat force progressions a maximum of two sessions if no definite improvement occurs
- the patient must continue with the home exercise programme; otherwise any benefit from the force progressions will be lost between treatment sessions.

## Management of multiple direction dysfunction

Multiple direction dysfunction is usually the result of either a road traffic accident or spondylosis and poor posture. If the patient has incurred a whiplash injury and subsequently developed dysfunction, this will have been at least six to eight weeks previously. The original symptoms have eased, but there has been no further recent improvement. Symptoms are intermittent and several movements may be affected, although not necessarily equally. The patient fulfils all the normal criteria to confirm a dysfunction. For a full discussion of management of whiplash associated disorders (WAD) at all stages, see Chapter 25.

When spondylosis or degeneration and poor posture are the cause of dysfunction, onset will have been insidious and the patient is generally over 50, at least. They gradually come to notice symptoms and restricted movement; for instance, crossing the road and looking out for traffic, reversing the car or looking up at the ceiling, or symptoms may have arisen following a particularly vigorous bout of activity. Restriction is usually symmetrical to left and right, with extension and rotations commonly affected. The patient fulfils all the normal criteria to confirm a dysfunction.

Multiple direction dysfunction of whatever cause is treated in much the same way as described above for individual movements, using the same movements and the same force progressions, if these are necessary. However, it is best not to treat all directions from the beginning, but start with one direction only, although it is acceptable to work on right and left rotations simultaneously. It is then possible to gauge response before progressing by adding in other movements. In general it is best to commence remodelling on the movement that is most affected and is the patient's chief limitation. After two or three days the next movement can be added, and so on.

## Conclusions

In spinal dysfunction syndrome pain is due to premature stretch or compression on structurally impaired soft tissue. Thus the patient's pain is consistently reproduced when the appropriate end-range movement is performed. When the loading is released, the pain abates. In order to rectify this situation, a remodelling programme must be instigated. Such a programme must regularly and repeatedly provoke the patient's pain. Recovery of normal range of movement and pain-free function will not occur rapidly, but over a period of weeks or months. In some patients with gross impairment, a remodelling programme may not be effective. Education and an appropriate regular exercise regime are the essential components in the treatment of dysfunction syndrome.

In the cervical spine dysfunctions are seen that affect all planes of movement, sometimes multiple directions are affected, and at times the nerve root/dura complex is involved. The management of the first two has been described in this chapter; recognition and management of an adherent nerve root is described in the next chapter.

# 22: Dysfunction of Adherent Nerve Root (ANR)

## Introduction

Adherent nerve root (ANR) is a specific form of dysfunction that rarely but sometimes occurs as a secondary problem following resolution of derangement with cervical radiculopathy or after surgery. It is the only time in spinal dysfunction when distal pain is experienced. It is also the only time when distal symptoms are repeatedly produced as part of management. Because the history and presentation of ANR are very distinct it is described separately, but in every other way it behaves typically as a dysfunction. Pain is produced at limited end-range, which does not change rapidly, and pain abates once the end-range position is released. In this instance any position that puts tension on the involved nerve provokes symptoms.

This chapter describes the development, clinical presentation and management of ANR. As patients with this syndrome present with arm and/or forearm pain, it should be a consideration in those with distal symptoms.

Sections in this chapter are as follows:

- development of adherent nerve root
- clinical presentation
- history
- physical examination
- upper limb tension test and differentiation of derangement and ANR
- management
- procedures for treating adherent nerve root.

## Development of adherent nerve root

The cause of intermittent persisting cervical radiculopathy may be either derangement or nerve root adherence. Patients with nerve root adherence present with unilateral asymmetrical symptoms with symptoms below the elbow; occasionally pain is felt only in the arm.

Symptoms have improved from onset, but have become unchanging. It should be remembered that nerve root adherence may be developing or fully developed depending on the time since onset when the patient is assessed. The longer symptoms have been present, the longer remodeling will take; a developing nerve root adherence may resolve much more quickly.

The majority of patients with cervical radiculopathy due to disc protrusion or herniation recover without the complication of nerve root adherence or fibrosis resulting from the repair consequent to the herniation. In the case of a resolving derangement, the intensity of the cervical radiculopathy will gradually diminish and in many cases pain becomes intermittent. During this period the range of cervical flexion, contralateral cervical lateral flexion and arm mobility should also increase. If the range of these movements improves as the intensity of the cervical radiculopathy subsides, the patient will not develop nerve root adherence. During recovery from cervical radiculopathy, performance of these movements once a day, as far as pain permits, should help to ensure return to pain-free mobility and full function.

Thus nerve root adherence is an ***uncommon*** complication that may arise following a cervical disc herniation. In an audit of mechanical diagnosis that included seventy-eight neck pain patients, one was classified as an adherent nerve root (May 2004a). Patients who remain cautious of resuming normal activity and movement are more likely candidates for this complication. If, despite overall improvement, the range of cervical flexion and/or lateral flexion and arm movements remain limited and unchanging, patients may well have developed nerve root adherence. Assessment must differentiate between derangement and adherent nerve root. If adherence is the cause of the remaining symptoms, such patients should be provided with a structured exercise programme designed to ***remodel*** any structures that are adherent or contracted. That management is detailed below. Obviously if symptoms are still the result of a derangement, the appropriate ***reductive*** forces must be found; this management is described in Chapter 20.

Brachial Plexus or Upper Limb Tension Tests may be positive whether nerve root adherence or derangement is responsible for the persisting but intermittent symptoms. Information from these tests is unhelpful unless a distinction is made between the syndromes. Is the test positive

because of fibrosis and adhesions about the nerve root, or because of tension or compression acting on the nerve root due to a disc herniation? Failure to identify derangement as the cause of pain has caused much unnecessary prolonging and aggravation of symptoms in patients where treatment has been applied to 'stretch' supposedly shortened or tethered neural structures. Such tension signs disappear after the reduction of derangement.

## Clinical presentation

If radiculopathy persists beyond about twelve weeks and is now felt intermittently rather than constantly, it is possible for the symptoms to arise from two causes:

1. a recurring derangement
2. the development of nerve root adherence or fibrosis.

If after surgery symptoms recur, adherence as a result of surgical scarring may be the cause.

Fibrous repair following disc herniation or protrusion can cause adherence of the nerve root/dura complex to the disc wall, which in turn limits the mobility of the root itself. Any attempt to stretch the tethered structure produces increased tension and causes pain and tightness to be felt in the arm. Thus the patient may not be able to flex or laterally flex their head, and elbow extension with shoulder abduction, especially if combined with wrist extension, is limited. The patient may relate that when they attempt such a movement they feel a tight band in their arm.

The change occurring in the behaviour of symptoms as the primary problem resolves and the secondary problem of nerve root adherence develops is slow and imperceptible. During the transition, the patient is usually unaware of the slow and subtle changes that are occurring to the nature and behaviour of his or her symptoms. This condition is a dysfunction and will behave in the consistent manner of all dysfunctions. It will not produce neurological deficit if the initial episode has not already done so.

**Table 22.1 Adherent nerve root – Clinical presentation *(all will apply)***

*History:*

- history of cervical brachialgia or surgery in the last few months that has improved, but is now unchanging, *and*
- symptoms are intermittent
- symptoms are in the arm and/or forearm
- consistent activities produce symptoms
- consistent movements produce symptoms – typically shoulder abduction, elbow extension, wrist extension with contralateral cervical lateral flexion or cervical flexion
- pain in arm does not persist on ceasing movement.

*Physical examination:*

- shoulder abduction and lateral rotation, with elbow extension, with wrist extension and with contralateral cervical lateral flexion or cervical flexion, is clearly restricted and some elements of it consistently produce concordant pain or tightness at end-range, *and*
- there is no rapid reduction or abolition of symptoms and no lasting production of distal symptoms, *and*
- lateral flexion and/or cervical flexion will improve if arm is flexed
- cervical flexion with arm de-tensioned has no effect
- there will be no rapid changes in mechanical presentation with repeated movement testing.

## History

Where nerve root adherence is the cause of intermittent persisting radiculopathy, the patient describes an improvement from the pain and disability at onset, which occurred between eight and twelve weeks previously, often much longer. However, the status of the condition is now unchanging. The symptoms always behave in the same manner on a daily basis. The same activities always provoke the pain, which may be felt in the forearm only or in the arm and forearm. Some neck pain may be present still or appear in tandem with the arm symptoms. In addition to pain, the patient may describe that they feel 'a tight band' running down the arm when they stretch it. Any activity that exerts tension on the nerve root reproduces the symptoms. Commonly arm activity involving elevation and abduction at the shoulder and extension at the elbow produces the symptoms. The patient may have noticed that flexion or lateral flexion when the arm is abducted and extended is limited and painful. They may have noticed that they failed to regain the normal mobility of their arm since the onset of neck pain. By flexing the arm across their chest, a

de-tensioning position, the patient may avoid the pain, and they may report this to be a 'relieving' position.

Adherent nerve root is a type of dysfunction and will behave consistently, as dysfunctions do. The patient reports that the same activities *always* produce their symptoms, but that the arm pain does not remain once they resume a neutral position. Similarly, the painful movement is *always* restricted.

## Physical examination

A patient with ANR has intermittent symptoms produced in positions that tension the nerve complex; thus they should be pain-free in the neutral starting position for the cervical examination. Most single and repeated cervical movements alone will have little or no effect on arm symptoms in most cervical ANR, although there may be some residual neck pain. Contralateral lateral flexion can be restricted and painful; extension can be full and pain-free. Only if the adherence were especially severe would flexion by itself produce symptoms, but this is rare. Obviously if there is any suggestion of symptoms worsening or peripheralising, derangement should be suspected.

If clues from the history and the early part of the physical examination make the clinician suspect the presence of an ANR, then the Brachial Plexus or Upper Limb Tension Test should be introduced. It can be done actively in the following order; this omits the shoulder depression component, but is easily transferable to home exercises. With the patient standing or sitting upright:

- elbow extension with shoulder abduction at just above 90 degrees, palm facing forwards to include lateral rotation at the shoulder
- plus wrist extension
- plus cervical flexion; and then minus flexion
- plus contralateral cervical lateral flexion.

Compare the ranges of movement with the unaffected side. Patients with cervical ANR find it impossible to perform these movements to full range. If there is little difference, range can be more accurately assessed in supine, but in such circumstances ANR is unlikely. Normally patients are unable to fully extend the elbow with the

shoulder abducted or extend the wrist with the elbow extended, or some similar combination. With the arm in its most extended position, does cervical flexion or lateral flexion become extremely limited and painful? If the arm is put in a less 'tensioned' position, does the range of cervical movement increase? Both these scenarios would suggest ANR. However, a positive tension test tells us very little; this occurs if derangement, irreducible derangement or ANR is present, although it is less likely when stenosis is present. Repeated flexion and/or lateral flexion with and without the Upper Limb Tension Test as outlined above should be done. In the presence of ANR, repeated flexion without the tension position is likely to have little effect; with the tension position it should consistently reproduce the patient's symptoms.

## Upper limb tension test and differentiation of derangement and ANR

Nerve root tension tests for the arm have been termed Brachial Plexus Tension Test (Elvey 1986), Upper Limb Tension Tests (ULTT) (Butler 1991) and Upper Limb Neurodynamic Tests (Butler 2000), and are fully described elsewhere (Butler 1991, 2000). In fact, these are similar tests based on the same movements of the neck and arm that together generate tension in neural tissues as demonstrated by cadaveric studies (Butler 2000). Although these manoeuvres are termed 'nerve tension tests', they also stress local joints, muscles, ligaments and blood vessels, so it is important to exclude local structures as well as spinal. The tests can generate 'positive' responses in asymptomatic individuals, such as an ache, stretch or tingling in the cubital fossa, forearm and/or hand (Kenneally *et al.* 1988). Different tension tests involve multiple movements in the arm and neck and the possible use of sensitising manoeuvres to assist in 'structural differentiation' (Butler 2000). Four tests have been described that supposedly have a bias to the brachial plexus as a whole and median (ULTT1), median (ULTT2a), radial (ULTT2b) and ulnar (ULTT3) nerves. The numbers refer to the most powerful sensitising component of the test (Butler 2000). One refers to shoulder abduction, two to shoulder depression and three to elbow flexion. Butler (2000) recommends that the tests be performed actively before passively, and describes active as well as passive tests. ULTT1, which applies a general stretch to the brachial plexus, is probably the easiest for the patient to perform and the most likely to give a positive response. Although there may be a need to perform tests passively, for instance to take baseline measurements

of range of movement or to include shoulder depression, the active movement is the core of patient management and thus is the focus in this text.

A 'positive' response would be reproduction of the patient's upper limb complaint. If this occurs, components of the test, especially away from the symptomatic area, can be added or taken away to help confirm 'neural' involvement. Unfortunately this still fails to discriminate the source of this involvement, whether derangement or dysfunction. The distinction between derangement and adherent nerve root (ANR) can be confirmed using a combination of repeated flexion/lateral flexion and the brachial plexus tension position (ULTT): shoulder abduction and lateral rotation, elbow extension and wrist extension.

Repeated flexion in the presence of derangement causes symptoms to peripheralise or worsen *with or without* the tension position applied. In an ANR, only if the adherence is especially severe would flexion by itself produce symptoms, but *with* the tension position repeated flexion would consistently produce arm pain at restricted end-range. The symptoms would not increase, but would recur with every repetition and would quickly disappear once the movements were stopped. Contralateral side flexion *with* ULTT would have a similar effect. These tests may be deemed the cervical equivalent of lumbar flexion in standing (*with* ULTT) and flexion in lying (*without* ULTT). In ANR with the ULTT positioned just prior to discomfort, cervical flexion and contralateral lateral flexion will be restricted; once the arm is relaxed across the chest these cervical movements increase in range. This is the same as the restricted flexion in standing in lumbar ANR with the knees fully extended, and the increase in lumbar flexion if the ipsilateral knee is flexed.

Patients with nerve root adherence are usually able to flex their neck without difficulty if the arm is across their chest, a de-tensioning position for the nerves. The nerve root is relaxed and not under tension when this manoeuvre is performed; consequently no pain is experienced even if flexion is repeated many times. Even if several sets of repetitions are done, the response is always the same. The mechanical and symptomatic presentations remain unchanged. The range of and response to extension also remain the same.

If the symptoms were the result of derangement, end-range pain could also be produced. If symptoms were the result of derange-

ment, repetition of flexion would cause the pain to peripheralise or worsen, and remain worse afterwards. In derangement the mechanical presentation may also change, with extension becoming obstructed following repeated flexion.

**Table 22.2 Criteria definition for adherent nerve root (*all will apply*)**

- history of cervical radiculopathy or surgery in the past that has improved, but is now unchanging, *and*
- symptoms are intermittent, *and*
- symptoms in the arm and/or forearm, may include 'tightness', *and*
- flexion/lateral flexion with shoulder abduction and lateral rotation, elbow extension with/without wrist extension is clearly restricted and consistently produces concordant pain or tightness at end-range, *and*
- there is no rapid reduction or abolition of symptoms and no lasting production of distal symptoms.

## Management

Nerve root adherence is a sequel to the repair process itself and is essentially a dysfunction. Management should provide a regular remodelling programme that will eventually alter the adhesions that limit mobility. To remodel contracted or scarred fibrous structures, it is necessary to stress the affected tissue without disrupting it and causing further damage. At the same time it is necessary to ensure that stress is applied with enough regularity and force as to cause remodelling.

*During the process of remodelling, some arm pain and tightness or discomfort must be felt, but any discomfort so produced should subside within ten minutes.*

*It should be noted that nerve root adherence is the only condition in which the deliberate provocation of distal symptoms can be permitted during the application of the treatment itself.*

Remodelling may use cervical movements, arm movements or a combination. Cervical movements may involve flexion and/or lateral flexion. The appropriate movement is the one that reproduces the patient's arm symptoms. Often both cervical and arm movements may be used, and stronger remodelling forces will use a combination. As neural tissue can be extremely sensitive to mechanical pressure,

it is wiser to initially commence the remodeling programme gently, with limited numbers of progressions and less vigorous procedures. If using cervical flexion in the remodelling process, which could cause recurrence of the derangement, certain precautions should be observed. The stability of reduction can be ensured in the following ways using the progression of forces as outlined below:

- test stability of neck flexion first
- use less forceful flexion movements initially
- always follow flexion procedures with a few extensions
- perform new exercises less frequently, initially only five to six repetitions five to six times a day
- monitoring symptomatic response
    - symptoms may be produced, no worse
    - symptoms may become less painful on repetition
    - symptoms must *not* be produced, and remain worse
    - symptoms must *not* remain more painful after repetition
    - symptoms must *not* remain peripheralised after repetition
- monitoring mechanical response – range of movement of and response to extension – should remain unchanged following repeated flexion
- if range of movement reduces or pain remains worse, suspect derangement
- avoid over-vigorous flexion procedures within first few hours of waking
    - initially perform ten repetitions of flexion from mid-day on, every three hours until retirement. If reduction of the derangement appears stable, the patient may commence the exercise a little earlier in the day and repeat it every two hours.

## Procedures for treating adherent nerve root

- commence with less stressful flexion procedures and progress as that procedure is easily tolerated or has no effect
    - cervical flexion (Procedure 6)

  - cervical flexion with patient overpressure (Procedure 6a)
  - cervical lateral flexion away from the pain (Procedure 4)
  - cervical lateral flexion with patient overpressure (Procedure 4a)
- add in non-cervical movements
  - elbow extension in shoulder abduction with lateral rotation
  - elbow extension in shoulder abduction/lateral rotation with wrist extension
  - elbow extension in shoulder abduction/lateral rotation with neck flexion/lateral flexion
  - elbow extension in shoulder abduction/lateral rotation with wrist extension with neck flexion/lateral flexion.

Force progressions are rarely required in the treatment of ANR; if necessary this will be flexion mobilisation (Procedure 6c) without and then with ULTT.

Recovery of function in ANR will take several weeks at least. The longer it has been present, usually the longer rehabilitation lasts. The patient will notice improvement as arm activities become easier and the range of movement of the ULTT position increases. This test with/without cervical flexion/lateral flexion can be used to monitor improvements.

## Conclusions

This chapter has described the clinical presentation, means of classification and management strategies for patients with cervical adherent nerve root. It must be emphasised that patients presenting with this condition are not that common.

# 23: Postural Syndrome



## Introduction

Postural syndrome is a painful disorder caused by prolonged end-range static loading of normal soft tissues continued until the point when mechanical stress triggers discomfort (McKenzie 1981, 1990; McKenzie and May 2000, 2003). Pain from the postural syndrome is caused by mechanical deformation of soft tissues or vascular insufficiency arising from prolonged positional or postural stresses affecting the articular structures or the contractile muscles, their tendons or the periosteal insertions.

In spinal postural syndrome pain arises from mechanical deformation of articular structures, whilst in extremity postural problems pain is as likely to come from vascular deprivation. Pain continues as long as the posture is maintained, but abates as soon as the posture is released. No pathology is present; as a consequence there is nothing to 'treat', and medicines or manipulation received for this syndrome are pointless and ineffective.

Many experience pain from this syndrome. It is especially common in schoolchildren and students who spend many hours sitting bent over books or computer screens. Most people learn that a simple change in posture abolishes symptoms, and it does not bother them when they are active and busy at other times. Consequently individuals rarely seek treatment and patients with only postural syndrome are rarely seen in clinical practice. It is the least common of the three mechanical syndromes encountered by health professionals, making up only a few percent of all neck pain patients who seek treatment. In a multi-site audit of mechanical diagnoses involving 265 patients, one neck pain patient was classified with postural syndrome out of seventy-eight with neck pain (May 2004a). However, pain of postural origin frequently exacerbates and perpetuates symptoms in all mechanical problems and must be addressed.

Sections in this chapter are as follows:

- pain mechanism
- effect of posture on symptoms in normal population

- clinical picture
- physical examination
    - posture syndrome versus minor derangement
- postures involved
- management of postural syndrome
- postural syndrome – aggravating factor sitting
    - correction of sitting posture
    - attaining correct sitting posture
    - maintaining correct sitting posture
- postural syndrome – aggravating factor lying
    - modification of lying posture
- postural syndrome – aggravating factor standing
    - management of postural syndrome
- consequences of postural neglect
- management of postural syndrome.

## Pain mechanism

Actual tissue damage is not always necessary to provoke pain in innervated structures (Bogduk 1993). Mechanical pain may result if prolonged mechanical forces are applied that *stress or deform* free nerve endings contained within the tissue. This disappears when the application of that force is terminated, and this occurs by a mere change of position. Such transient pain has been termed 'physiological' pain, and is related to the intensity of the noxious stimulus in a measurable way (Woolf 1991; Woolf *et al.* 1998). It warns the body of potential damage and, as long as the mechanical stress is transient and interrupted before actual tissue damage occurs, no long-term sequelae will ensue. Such mechanical stress fails to cause protracted symptomatology, as it is insufficient to cause tissue damage. If actual tissue damage occurs, the inflammatory response is unleashed with protracted pain (Levine and Taiwo 1994).

An example is the pain incurred during prolonged slouched sitting that disappears on 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 appears. If the finger is bent suddenly by applying a brief, rapid end-range force, the pain receptor system is activated immediately, but briefly. Thus pain can appear eventually after moderate prolonged loading or can appear immediately as a result of sudden short-lived overstretching. In both cases the pain ceases on release of stretching. In neither case, however, is damage to tissue necessary to cause the experience of pain.

Pain of postural origin in the cervical spine is usually produced by mechanical stress at end-range. It is most commonly a sustained end-range loading that eventually causes sufficient tissue deformation to provoke pain.

The greater the mechanical loading and deformation become, the greater is the intensity of pain. If the painful position is prolonged, the pain becomes more diffuse, widespread and difficult to define and may expand distally (Harms-Ringdahl 1986). If such loading on return to a neutral position has caused no structural damage, the pain becomes less diffuse and more localised to its point of origin, until it quickly subsides.

## Effect of posture on symptoms in normal population

Harms-Ringdahl (1986) explored the effects of sustained slumped postures in volunteers without current or past neck symptoms. They maintained a posture of lower cervical and thoracic flexion and extreme upper cervical extension that is seen in a typical protruded head posture. All ten volunteers began to perceive pain within two to fifteen minutes, which increased with time, eventually forcing them to discontinue the posture after sixteen to fifty-seven minutes. Once they discontinued the position, the symptoms abated. Most experienced similar pains the next day, but when these occurred is not reported. Pain was generally localised around the neck and upper scapulae, but radiated into the arms in a few individuals. This study demonstrated how individuals without pre-existing spinal symptoms can have transient pain created by sustained mechanical loading.

During this sustained protruded head posture, the muscular activity in the trapezius, splenius, thoracic erector spinae and rhomboids was monitored and found to be generally very low. Consequently, it seems reasonable to conclude that sometimes pain due to extreme

spinal positions is provoked by mechanical load on articular and peri-articular structures rather than sustained muscular activity (Harms-Ringdahl 1986).

Gooch *et al.* (1991) studied ***in vivo*** creep of the cervical spine in sustained flexion in a mixed group of patients and controls. Over the ten-minute period, creep occurred in those who were able to sustain the position with the effect of increasing the angle of cervical flexion. A third of the forty-seven individuals were unable to sustain the original position due to discomfort, but were able to remain in a less flexed posture. Twelve of the original experimental group withdrew before completing the full ten minutes due to pain; most of these were in the 'neck pain' group, but one was in the control group. The study demonstrates that a sustained posture of cervical flexion can lead to an increase or production of neck pain.

Abdulwahab and Sabbahi (2000) also looked at the effect of sustained neck flexion for twenty minutes in patients with cervical radiculopathy and in controls. This had the effect of significantly increasing the radicular pain in the patient group, but also produced mild pain in the control group, who were without prior neck symptoms. These studies demonstrate the role of sustained cervical flexion in producing pain of postural syndrome, as well as the more obvious exacerbation of pre-existing cervical syndromes.

These three studies use posture as an independent variable and measure pain as the dependent outcome, thus informing us directly about the relationship between posture and pain.

Other studies that have examined the relationship between cervical posture and neck pain have used cross-sectional study designs that have examined postures in symptomatic and non-symptomatic groups to determine if they differ. In this type of study design, a direct and causal link between posture and pain cannot be determined, although association or lack of it may be. Some studies have failed to find an association between the angle of the cervicothoracic kyphosis and the presence or absence of neck pain (Refshauge *et al.*1995), and between individuals' normal sitting head posture and a history of pain in the previous month (Grimmer 1996).

However, in other studies people with cervicogenic headaches have demonstrated a more protruded head posture than non-headache

populations (Watson 1994). Individuals with increased cervicothoracic kyphosis have an increased incidence of interscapular pain, and those with a more protruded head posture have an increased incidence of neck, scapular and head pains (Griegel-Morris *et al.* 1992). In a group reporting more areas of pain this was associated with a more protruded head posture and less range of extension compared with less symptomatic individuals (Haughie *et al.* 1995).

## Clinical picture

Patients with solely postural pain are usually under 30 years old, often much younger – schoolchildren may be brought to the clinic by concerned parents. Individuals are generally sedentary due to their occupation, studies or lifestyle. They usually have spinal pain only, without referral, but may describe pain in the thoracic and lumbar regions as well. Frequently they have been getting symptoms for months, which have been getting gradually worse – pain is now coming on more quickly and occurring more often. It is this gradual deterioration that is more likely to cause them to seek help rather than any dramatic onset of severe neck pain. With the passage of time the more often structures are stressed to the point of being painful, the more sensitive they become to mechanical stimuli and symptoms are more easily provoked. Less mechanical stimulus is needed to produce the symptoms and they come on after a decreasing period of time.

Pain in the postural syndrome is always intermittent, and may sometimes not be present for days at a time. Pain is only brought on by prolonged static end-range postures – problems may arise during sitting, standing or lying. If they are in these positions for brief periods or are generally active, they are pain-free. For instance, they may lead a more active life style at the weekend and have no symptoms at this time. When constantly moving and changing position, they avoid the stresses arising from end-range static postures. If sitting is the provoking posture pain will not come on immediately, but only after a prolonged period of static positioning. Once the individual changes their position, or gets up and walks around, symptoms disappear. They move fully and freely, and are perfectly all right until they resume the same position for a period of time. The link between a particular posture, time and the onset of symptoms should be reasonably obvious in their history. Usually the patient has failed to make this connection. Pain from the postural syndrome is never

induced by movement, is never extensively referred and is never constant. There is no loss of movement, no sign of joint abnormality and no mechanical presentation. There is nothing to see other than the poor posture itself. There is no pathology; symptoms arise from mechanical loading.

## Physical examination

The examination will be unproductive, except relating to one aspect of the presentation. There is no deformity, no loss of movement and no response to repeated movements. Para-clinical tests are negative. These 'negative' results in fact provide confirmatory data for the diagnosis of postural syndrome.

The only relevant 'positive' result relates to posture. The patient's sitting, and often standing, posture is poor. If enough time has elapsed during the history-taking and the patient is positioned in their provocative slouched sitting posture, their symptoms are produced. At this point if they are encouraged to move from that position by correcting their posture, the symptoms are abolished and the relevance of the poor posture to their pain will have been demonstrated to the patient. Equally, once the patient stands, the symptoms abate.

Sometimes it may take longer, with a sustained posture for up to half an hour, before the pain is felt. If this is the case it may be necessary to ensure that the patient sits for this length of time in order to provoke the symptoms. Once the pain is present it is simple to educate the patient in the relevance of posture to their problem. Posture correction or standing abolishes the symptoms. They thus learn the importance of their position and how their sitting posture can either provoke or relieve their symptoms. It is vital that the patient appreciates the intimate link between posture and pain in this syndrome; only then will they be able to deal with it. If it is not possible to reproduce the patient's neck symptoms during the initial examination, they must be instructed in postural correction and advised to assess the relationship between posture and pain next time the pain is felt.

**Table 23.1 Postural syndrome – criteria *(all will apply)***

*History:*

- local pain
- intermittent pain
- pain associated with time spent in a particular posture
- pain does not persist
- painless movement and activity.

*Physical examination:*

- poor posture
- full range of movement
- no deformity
- no problems with curve reversal
- repeated movements do not reproduce pain
- pain only produced by sustained loading in relevant position, which is then relieved on moving from that posture.

### Posture syndrome versus minor derangement

Those who are unfamiliar with the system of mechanical diagnosis and therapy have confused the posture and derangement syndromes in patients with lumbar spine problems (Riddle and Rothstein 1993). The same errors of classification may occur in the cervical spine. In fact, the symptomatic and mechanical presentation of these two syndromes are very different, although both are affected by posture. In the case of a patient with derangement there can be referred pain, pain on movement, deformity or blockage to movement, persistent pain after the posture is corrected or the aggravating position released, and in general a much more severe presentation.

Sitting is the most common cause of neck pain in the posture syndrome. It is also an extremely common cause of aggravation of pain in derangement, but pain behaviour in the two syndromes is distinctly different. If an individual is having neck pain that is due to postural syndrome that is caused by sitting, there will be a clear association between the posture, when sustained for a sufficient period, and their pain – as frequently occurs in derangement. Upon rising and moving the pain rapidly ceases, only re-occurring when they resume the sitting position for a sustained period, and when tested all movements are full and pain-free. Pain in derangement has a vastly more significant effect in terms of pain and function. Another key difference between the two is prevalence in those seeking health

care. Postural syndrome is rarely seen in the clinic, whilst derangement syndrome is very commonly seen.

Patients not infrequently describe being woken by pain. If they relate that a simple change of position quickly relieves the problem, this is from postural syndrome. Derangement syndrome may also be triggered by an awkward sleeping position, but in derangement the symptoms persist after a change of position.

Clinicians with limited experience of the method have mistaken posture syndrome for derangement, and vice versa. There is more potential to mis-classify a derangement syndrome as a postural syndrome as more of them are seen in the clinic. Furthermore, this classification error would more likely produce inappropriate and ineffective management.

## Postures involved

Sitting is the most frequent cause of pain of postural origin, and many patients name this as the only provoking factor. They may complain that pain is produced after spending a certain time, for instance quarter of an hour or so, in any sort of chair or when driving. Others may complain that the pain only comes on after working at the computer for a while.

During prolonged sitting when the relaxed posture is assumed, spinal muscular activity decreases (Harms-Ringdahl 1986; O'Sullivan *et al.* 2002; Dolan *et al.* 1988; Andersson *et al.* 1975). Typically a slumped posture ensues, with reduced lumbar lordosis, exaggerated cervicothoracic kyphosis and protrusion of the head and upper cervical spine. If this position is maintained, end-range loading can occur with flexion of the lower cervical
cervical
and enough mechanical deformation may ensue to produce pain. It is important to understand the link between the position of the lumbar and cervical spine. With lumbar lordosis maintained, the head and neck can be positioned in an upright posture; with the lumbar spine in flexion, there is concomitant lower cervical flexion and upper cervical extension.

Sometimes individuals are woken by neck pain, but this quickly abates when they change their sleeping position. Occasionally individuals complain of pain after prolonged standing, usually when working in prolonged neck flexion. The same criteria as above must apply in all circumstances. Other postures are less likely to produce pain of postural origin as they generally allow people a much greater opportunity to alter their position.

## Management of postural syndrome

Once patients are made aware of the link between their posture and their pain, most find it relatively easy to self-manage this syndrome. However, it is vital that this correlation between their position and symptoms is made apparent to them. If the patient is finding this difficult to accept, positioning them in a sustained posture so that symptoms are provoked is usually sufficient to convince them of the cause. Once the link is well established, they need advice on correcting posture and interruption of the aggravating factor. If they avoid end-range stresses to soft tissues for two to three weeks, the problem will resolve.

Repeated exposure to the aggravating posture over time leads to a gradual deterioration with increased frequency of symptoms. Conversely, correcting the posture enables the patient to remain pain-free for longer and longer time periods. If the causative posture is avoided, the sensitivity of tissue nociceptors to mechanical stimuli diminishes over time. Thus, after two weeks of posture correction, resumption of the aggravating position is tolerated for longer before pain is triggered. Obviously this should not be encouraged, as it will set them once more on a deteriorating pathway.

As well as advice to avoid the causative posture, patients must be shown how to maintain correct positions. Patients should be warned that the adoption of new postures might cause the temporary development of 'new' pains, which will subside within a week. Management is thus founded on a thorough understanding of the effects of posture, and a combination of avoidance and performance – avoid the aggravating factor and perform the corrective procedures.

**Table 23.2 Management of postural syndrome**

- education on link between posture and pain
- education on link between lumbar and cervical postures
- education on posture correction
  - attain correct posture
  - maintain correct posture
- education on avoidance of aggravating posture
- posture correction
- slouch-overcorrect.

## Postural syndrome – aggravating factor sitting

Sitting is the most common cause of pain in the posture syndrome. The individual with posture syndrome is exposed to long hours of sitting due to occupation, study, unemployment or hobby. The postural habit when sitting for a period of time, and the effect this may have on symptoms, is best observed if the patient is seated without a back support as on the examination couch, rather than in a chair. Posture in sitting is often slumped, with no attempt to maintain an upright position actively. The cervicothoracic spine is flexed and the head and upper cervical spine protruded. The patient is resting on articular and peri-articular structures; with sufficient and repeated loading, eventually pain is triggered. The best time to observe their relaxed sitting posture is when they are not aware you are doing so, for instance while the history is being taken. During this period the effect of sustained relaxed sitting on the patient's posture may be noted. At the end of this part of the mechanical evaluation, question the patient about the presence of pain. This may have come on during the interview, and if present, posture correction rapidly abolishes symptoms. *If a direct link between posture and pain can be clearly demonstrated to patients, their compliance to the management programme is rapidly achieved.* For this reason the patient should have the history taken whilst sitting unsupported, as described.

Allow the patient to relax unsupported to expose the true nature of their sitting posture. Correction or further investigation of slouched sitting posture and its effect on symptoms is the hallmark finding in the posture syndrome. The rest of the physical examination will be normal, with full range of movement and no pain on repeated movements.

### Correction of sitting posture

In a review of the optimal sitting posture it was concluded, regarding the cervical spine, that minimising forward head posture and cervical flexion is associated with higher comfort ratings (Harrison *et al.* 1999). It should be explained to patients that when we sit, especially when preoccupied, a relaxed posture is adopted. The spine takes up the shape of the chair, or if sitting unsupported, eventually the slouched posture is adopted. Unless a conscious effort is made, or a well-designed chair with appropriate support is used, it is a universal phenomenon that within a short period of sitting individuals will adopt a relaxed, slouched posture. This flexed posture places ligaments, capsules and other peri-articular and articular structures under tension. If this posture is maintained, as creep occurs, greater tensile stress is placed upon these soft tissues. Eventually, if maintained without respite, enough mechanical tension can be generated to trigger nociceptor activity.

That simple mechanical tension eventually becomes painful is easily demonstrated to the patient using the analogy of the 'bent finger', especially if the patient's own finger is used for educational purposes. By holding their finger in end-range extension, first discomfort and then a dull ache is produced, but as soon as it is released the aching abates and within a minute is completely gone, no damage having occurred.

The patient needs to be convinced that the same process is at work in the spine. If pain is of postural origin there is never any lasting repercussion – when the pain is provoked by sitting slouched, it is abolished by correcting the posture. Hopefully during the first treatment session the patient's symptoms will be produced after prolonged sitting – the period of history-taking allows fifteen to twenty minutes for this to occur. Upon completing the interview, the patient should be questioned about any symptoms that are now present. If they do indeed report the onset of symptoms during this period, then the effect of posture correction must be explored. If symptoms are due to posture syndrome, posture correction will abolish them. When pain is shown to be so clearly related to position, the patient quickly accepts the logic of correcting the sitting posture. If the pain does not abate when the posture is corrected, but increases, changes location, or stays the same, another mechanical syndrome, derangement in all probability, is the cause of symptoms. Pain from derangement

syndrome may also centralise or be reduced or abolished by posture correction.

If it is not possible to produce the patient's symptoms during the interview, then they must be instructed to test out the effect of posture correction on the next occasion that symptoms develop. 'The next time pain occurs, can you abolish it by correcting your posture?'

Posture correction involves:

1. attaining correct sitting posture
2. maintaining correct sitting posture.

### Attaining correct sitting posture

To encourage the patient to attain and maintain the corrective posture, they must be convinced of its value. Most patients, when they are fully aware of the relationship between posture and the production of pain, quickly accept the need to alter their postural habits. They need to understand the correct sitting posture as well as recognise a poor posture, and they need to be able to control their trunk during posture correction and maintenance. A 'good posture' is defined here as a position in which the lumbar spine is positioned in a moderate degree of lordosis, and the head and shoulders are evenly aligned over the pelvis with the chin over the chest – moderately retracted rather than protruded. The patient should be shown how to recognise end-range positions and encouraged to use mid-range postures.

To understand and attain the correct sitting posture, the 'slouch-over-correct' procedure is introduced (Procedure 3). This procedure allows patients to feel the difference between a poor slouched posture and a fully overcorrected posture. It is neither good nor desirable for patients to maintain this overcorrected position. The best sitting posture is gained by releasing the last 10% of the overcorrected sitting position. The lumbar lordosis and position of the head should be similar when sitting to that which is present when standing.

If the slouch-overcorrect procedure is practised three times daily, ten to fifteen times at each session, the patient will in a matter of a few weeks have re-educated their postural habit. They will no longer perceive the slouched posture as 'normal'; they will come to find that the corrected posture is now 'normal' for them. As well as practising slouch-overcorrect in order to retrain their postural 'habit' and to

train their muscles to hold their trunk upright, the procedure should be done regularly whenever pain arises. Painful postures should be frequently and rapidly interrupted. The increased sensitivity to mechanical stimuli
exposure to those mechanical loads will gradually abate. Each time the slouched posture is resumed and pain re-triggered, then the threshold at which mechanical stimuli cause pain will remain low. If the painful position is avoided altogether sensitivity returns to normal, and short periods of slouched sitting no longer hurt.

### Maintaining correct sitting posture

When sitting for prolonged periods it is essential that a lumbar lordosis be maintained at all times. The patient must be made aware of the link between the position of the lumbar spine and that of the cervical spine, and how by maintaining the lumbar lordosis the cervical spine can be positioned in a neutral posture (Procedure 3). The lumbar lordosis can be maintained in two ways:

1. actively, by muscular control when sitting on a seat and not using a backrest;
2. passively, with the use of a lumbar roll or support when sitting in a seat with a backrest. The lumbar roll keeps the lumbar spine in moderate lordosis while driving, sitting at work or relaxing. Without it the lordosis is lost if the person leans back in the chair or concentrates on something other than the maintenance of the lordosis. With the lordosis maintained, a neutral head posture can also be maintained.

Active control of the lumbar lordosis is demanding, and someone used to the slouched posture will have problems maintaining this position actively for long. However, it is good for the patient to practice this posture actively several times a day, holding it as long as they can for up to a few minutes. The performance of this regular active posture correction will improve their overall postural control. It has the additional benefit of strengthening the muscles responsible for maintaining the upright position.

Patients frequently complain about the effort to maintain the correct sitting posture, especially if they are actively maintaining the position. Many describe a strain pain or say that the new position is uncomfortable. These new postural stresses are to be expected, and if patients do not complain of 'new pains' it is likely that they have not been adequately

practising or maintaining the correct posture. Adjustment to a new posture results in short-lived transitional aching, usually of a different quality and location than the original pain of which they complained. These are commonly felt further up the back in the thoracic region, and should not last longer than five or six days.

A lumbar roll has a significant effect on the maintenance of the lordosis, with increasing support producing increasing lordosis (Andersson *et al.* 1979). As long as the support is level with the lumbar spine, the exact level is less important as it does not much influence the ultimate angle (Andersson *et al.* 1979). It is most appropriate to place the support level with the lower lumbar spine, which is the area of greatest stress. The individual must push their pelvis to the back of the chair; otherwise the support is wasted. A cushion is not suitable as it simply moves the whole spine away from the chair without influencing the degree of lordosis of the lumbar spine.

A good sitting posture is often difficult to obtain on sofas or settees because the shape of the chair causes posterior rotation of the pelvis, which in turn reduces the lordosis (Keegan 1953). Good posture is easier to attain and maintain on a straight-backed chair. If any benefit is to be gained from a lumbar roll in a lounge chair, then cushions should be used first to prevent the support being absorbed by the upholstery. Once the posture of the lumbar spine is improved, the upright posture of the cervical spine and head should follow automatically. The patient may need some reference for where the head should be, for instance 'the ears should be over the shoulders', or 'if you drop a line from your chin this should be over your chest, rather than the space in front of your body'.

Particular work or recreational factors may need to be discussed with the patient. For instance, use of a computer screen, computer games or bi-focal lenses. In each instance a correct overall spinal posture is important, as well as maintaining the neck in mid-range position, avoiding sustained end-range loading and regular interruptions from the task.

In postural retraining the problem lies in loss of awareness of the correct posture, not in an inability to assume it. Lumbar rolls, expensive office furniture and ergonomically designed work stations will all have no effect on postural habits unless the individual is aware of the correct posture. Likewise, strengthening of the muscles of the spine will

have no effect on posture if the individual is not 'bodily' aware of the correct sitting position. No strengthening exercise can teach the patient the correct posture. Once the correct posture is attained, stronger muscles may help the patient maintain it. Regular use of the slouch-overcorrect procedure is a helpful way for the patient to learn how to attain the right posture. The patient will also be motivated to improve his posture as a result of improving symptoms. Actively maintaining the correct posture is the best way to strengthen the postural muscles and to enforce a new bodily posture. By regularly attaining and maintaining a better posture this will become easier to do, and after three or four weeks a new postural habit can become normal.

## Postural syndrome – aggravating factor lying

Individuals frequently awake with neck pain, presumably arising from end-range or awkward positions of the head and neck assumed and maintained during sleep. Derangements can arise in this way and symptoms will persist for a few days or more and be accompanied by restricted painful movements. Pain from cervical postural syndrome fades away once the individual gets up, and there are no effects on movements. However, this may occur regularly if the person sleeps in an awkward position that sustains the neck in an end-range posture or uses unsuitable pillows.

If resting through the night is causing pain, two factors need to be investigated:

1. The lying posture itself. This is different for each person and must be dealt with individually. Sleeping postures are habitual and can be difficult to influence. A typical position is end-range rotation that occurs in those who sleep prone.
2. The surface on which the person is lying. The pillow is usually more important than the mattress in cervical postural pain. A soft and malleable pillow is most appropriate, which provides support for the head in a horizontal position, but avoids pushing the head into flexion or lateral flexion.

### Modification of the lying posture

Patients can be encouraged to alter their sleeping posture if this is indicated, but it may be difficult to achieve. An extreme strategy to avoid prone lying is to tape an object to the abdomen, which disturbs the individual enough to change their posture.

If the surface seems to be inappropriate, a cervical roll can be placed inside the pillow. This fills the gap between the shoulders and the head and provides direct support for this area. This should only be used with one pillow. The cervical roll usually works quickly or not at all, and should be tried for a few nights.

## Postural syndrome – aggravating factor standing

Patients rarely report cervical postural pain that occurs in standing, presumably as this position allows greater postural variety and they escape sustained end-range postures. Occasionally someone who works standing in a position of sustained neck flexion or protruded head posture may present.

For management, the same principles apply. If sustaining the causative posture can reproduce symptoms, the effects of resuming a more neutral posture are quickly established. If symptoms cannot be replicated in the clinic, but lack of other examination findings suggest pain from postural syndrome, then the patient is advised on appropriate strategies. They need to interrupt the sustained flexed/protruded posture, and if pain occurs observe the effect of correcting that posture.

## Management of postural syndrome

- education on link between posture and pain
- education on link between lumbar and cervical postures
- education on posture correction
    - attain posture
    - maintain posture
- education on avoidance of aggravating posture
- posture correction.

*Pain of postural origin arises from postural neglect; through postural correction they can stop their pain and also prevent its onset.* As long as the link between pain and posture has been clearly demonstrated to patients, and they have been adequately educated, most are well able to treat themselves.

In avoiding end-range postures in the cervical spine, the interconnectedness of lumbar and cervical postures must be made apparent to patients. The avoidance of sustained protruded head or neck flexion postures necessitates maintenance of the lumbar lordosis. Without correcting the lumbar posture, correction of the cervical posture will not occur.

When management by education is completed successfully, it should be explained to the patient that, although the present pain has been relieved, recurrence of similar symptoms is possible if postural care is neglected for extended periods. The consequences of postural neglect should be discussed.

## Consequences of postural neglect

The effects of postural habits have long-term implications on the human shape (McKenzie 1981, 1990). The commonly observed posture of protruded head, rounded shoulders and flattened spine may become habitual. As age advances, permanent postural 'set' may occur – head protruded, shoulders rounded, dowager's hump, loss of lumbar lordosis and the erect posture replaced by a slight stoop. This is likely to be accompanied by considerable soft tissue adaptations. Positions that are frequently adopted, such as flexion, will be maintained, whilst movements that are rarely performed, such as extension, become steadily more difficult to achieve. Long-term postural neglect can lead to adaptive tissue shortening, causing dysfunction syndrome.

As men and women age, their natural head position tends to progress to a more forward position; their ability to retract the head declines, whilst protrusion range is maintained, and there is an overall decline in antero-posterior mobility (Dalton and Coutts 1994). Between young adulthood and older age there is a reduction in all planes of cervical movements of 20 – 45% (Worth 1994), and a reduction in all planes of lumbar movements of about 30% (Twomey and Taylor 1994). In a meta-analysis of normative cervical motion, multiple studies demonstrated a decrease in cervical range with age (Chen *et al.* 1999). Although a large part of this may be the natural effects of ageing, there is also an element of variability in the degree to which people become restricted in range of movement and in resting postures. The mean range of movement decreases decade by decade,

but the standard deviation gets proportionally bigger as people age (Youdas *et al.* 1992). This indicates considerable individual variability amongst an overall decrease in range.

This means that protruded head positions and stooped postures are not simply an inevitable consequence of ageing. Movement that is lost because of tissue adaptation could have been retained had affected soft tissues been regularly stretched. If end-range movement is neglected, eventually that movement is lost forever. Postural 'sets' that arise from long-term postural neglect and tissue adaptation result from postural habit as much as the consequence of age. Loss of function can be prevented if end-range movements are regularly performed and posture corrected throughout life.

Thus, initially, poor postural habits produce pain of postural origin without loss of function. Prolonged postural neglect leads to adaptive shortening. If flexion is regularly performed but extension rarely, the anterior structures of the joints shorten and the posterior structures lengthen. Extension becomes more and more difficult to perform. Adaptive shortening implies loss of function and movement. Whenever shortened structures are placed on stretch, they will induce discomfort or pain. Furthermore, the decreased movement must inevitably lead to impairment of nutrition in the intervertebral disc, contributing to disc degeneration. *The shortening of soft tissue, caused by poor postural habit and inadequate exercise, can be prevented by regular postural correction and adequate performance of the relevant exercises* (McKenzie 1981, 1990). Furthermore, habitual poor posture predisposes to derangement.

## Conclusions

Only the patient can rectify pain of postural origin. No externally given treatment can alter the aggravating factor, which is their postural habit. To dispense treatment for a condition that can only be resolved through patient education is negligent health care. The essence of management for this condition is education and postural correction.

# 24: Headache



## Introduction

Headache is a commonly reported symptom with a variety of causes, both serious and benign. Some headaches arise secondary to a neck problem with the primary complaint in the neck – such patients should be classified with one of the mechanical syndromes and managed in the same way as described elsewhere in the book. Some patients attend primarily with the complaint of headache, although there may be some secondary neckache – this chapter addresses this group. Headache means pain anywhere in the area of the occipital, parietal, temporal or frontal regions, and may also include symptoms around the eyes. Some of these patients have headaches that arise from upper cervical joints, and some will respond to mechanical therapy. There is a range of other causes of headache and some of these and the classification of headache are considered.

The task of a mechanical assessment, as in other areas of the spine, is first to determine those who are inappropriate for treatment and, with accompanying 'red flag' features, should be referred for further investigation. This differential diagnosis is performed principally on features from the history. Second, headache patients need to perform a mechanical evaluation as described below – some will demonstrate a beneficial or recognisable symptomatic response, and this group will benefit from mechanical therapy. Lastly, there will be a group who demonstrate no consistent mechanical response, whose symptoms are from some non-mechanical source.

Sections in this chapter are as follows:

- epidemiology of headache
- causes of headache
- differential diagnosis
- cervicogenic headache
- neuroanatomy of cervicogenic headache and experimental evidence
- mechanical diagnosis and therapy and headaches

- classification
  - derangement
  - dysfunction syndrome
  - postural syndrome
- history
- physical examination
- mechanical assessment
  - retraction – sitting (Procedure 1)
  - other tests
- management of mechanical cervical headache
  - correction of the lying posture
  - modification of the lying posture.

## Epidemiology of headache

Headaches are extremely common in the general population and a very common reason for seeking health care. The literature on the prevalence of headache in forty-four studies was summarised in 1999 (Scher *et al.* 1999). At age 40 there was an estimated prevalence in males of 25% in Europe and just over 60% in North America, and in females of 70 – 80% in both these areas (Scher *et al.* 1999). It is unclear why there is such a marked difference between European and North American males. Lifetime prevalence may be higher, with recent population studies giving figures of 83 – 93% of respondents reporting headache ever (Boardman *et al.* 2003; Ho and Ong 2003). These studies make clear that headache is extremely common. About 2 – 4% of the general population report chronic daily headache, which may have persisted for years (Hagen *et al.* 2000; Lanteri-Minet *et al.* 2003).

Complaints of headache have constituted 1.5 – 7% of patients visiting primary care physicians in North America (Becker *et al.* 1987; Hasse *et al.* 2002) and 4% in a UK general practice (Phizacklea and Wilkins 1978). A range of diagnoses was given: most commonly tension headaches, vascular, migraine, sinusitis and upper respiratory tract infection (Becker *et al.* 1987; Phizacklea and Wilkins 1978).

## Causes of headache

There are multiple listed causes of headaches (see Table 24.1). The most comprehensive attempt to classify headaches was made by the International Headache Society (IHS 1988), updated by a second edition in 2004. This listed thirteen separate groups, such as headaches associated with vascular disorders, substances or their withdrawal, non-cephalic infection, metabolic disorder, cranial neuralgias and so on (IHS 1988). More unusual causes are associated with serious pathology and systemic conditions and are obviously not appropriate for management by physical therapy. The more common causes are migraine, tension-type headaches and cervicogenic headache, which, although not always listed in differential diagnosis, have come to be accepted by the IHS. Other headache types include chronic paroxysmal hemicrania, cluster headaches and hemicrania continua. Some are simply Latinate descriptions of symptom features; for instance, the latter describes a continuous unilateral headache.

**Table 24.1 Differential diagnosis of headache**

| *Type of lesion* | *Diagnosis* |
|---|---|
| *Intracranial* | Brain tumour<br>Brain abscess<br>Subdural haematoma |
| *Meningeal irritation* | Acute meningitis<br>Chronic meningitis<br>Syphilis<br>Tuberculosis<br>Cryptococcosis<br>Sarcoid<br>Cancer |
| *Cranial* | Metastic neoplasms<br>Paget's disease |
| *Vascular disturbances* | Migraine<br>Temporal arteritis (associated with polymyalgia rheumatica)<br>Sub-arachnoid haemorrhage<br>Hypertension<br>Carotid/vertebral artery dissection |
| *Toxic states* | Infections/alcoholism/lead/arsenic |
| *Extracranial* | Lesions of eye<br>Lesions of middle ear<br>Lesions of nasal sinuses<br>Lesions of oral cavity |
| *Psychogenic* | Conversion hysteria/anxiety states<br>Muscle tension |

Continued next page

| *Type of lesion* | *Diagnosis* |
|---|---|
| *Other* | Cervicogenic<br>Cluster<br>Post-traumatic<br>Exertional<br>Post-coital. |

Source: Berkow *et al.* 1992; Mainardi *et al.* 2002; D'Andrea *et al.* 2002

It is hoped that any patients with headaches associated with serious pathology have been recognised and do not seek treatment from a musculoskeletal specialist. However, as this may not be the case and as musculoskeletal specialists can be front-line clinicians, an awareness of 'red flags' as relevant to headaches is very important. These are suspected 'red flags' and their diagnostic acumen has not been formally tested. Severe headache has been the initial and salient symptom in a number of case studies in which the patient was finally diagnosed with acute myocardial infarction, carotid artery dissection, intracranial tumour, lung carcinoma or hypertension (Famularo *et al.* 2002; Mainardi *et al.* 2002; Pfund *et al.* 1999; Abraham *et al.* 2003; Spierings 2002; Vazquez-Barquero *et al.* 1994).

**Table 24.2 Possible 'red flag' indicators of serious pathology in headaches**

- progressive worsening of headache
- recent severe onset/'thunderclap' headache
- onset of headache after exertion
- onset of headaches > 50 years old
- history of major trauma
- nausea/vomiting
- temporal/occipital headache, with visual changes
- preceding sore throat/respiratory infection
- history of cancer
- problems with speech/swallowing
- visual changes – diplopia, ptosis, blurring
- associated symptoms – progressive weakness, convulsions, blackouts, mental changes, systemically unwell.

Source: Berkow *et al.* 1992; Pfund *et al.* 1999; Oh *et al.* 2001; Makofsky 1994

Prevalence rates for the different types of headaches vary, but problems with classification mean that the true prevalence rates may only be estimates. In eighteen studies on the prevalence of migraine, using the International Headache Society's (IHS) diagnostic criteria, the

estimated prevalence at age 40 in the general population was 6 – 7% in males and 15 – 22% in females in the Americas and Europe (Scher *et al.* 1999). Population studies since then have estimated similar levels: 6 – 9% in men and 11 – 17% in women (Hagen *et al.* 2000; Dahlof and Linde 2001; Lampl *et al.* 2003; Ho and Ong 2003). Prevalence increases up to age 40 and then declines.

In a population sample of 826 individuals, using limited IHS criteria, 2.5% of the general population and 18% of the frequent headache population were deemed to have cervicogenic headache (Nilsson 1995). Several studies have suggested the same range of cervicogenic headaches, around 15 – 20% of all headaches (Haldeman and Dagenais 2001). The mean age is 43, 79% are female, and mean duration of symptoms is 6.8 years (Haldeman and Dagenais 2001).

Tension headache is considered by some the most common type of headache with a one-month prevalence in the general population for mild and episodic symptoms of between 20% and 50% (Rasmussen *et al.* 1991; Rasmussen 2001; Ho and Ong 2003). The prevalence of chronic, daily tension-type headaches in the general population is about 2 – 3% (Bahra and Goadsby 2000; Rasmussen 2001, Ho and Ong 2003). Prevalence is higher in women. As with other types of headache, prevalence declines with age, and a family history is common. Late onset headaches are generally unusual and may indicate serious pathology.

Cluster headache is rare, with estimated prevalence between 0.07% and 0.4%, and unlike other headaches is more common in men (Dodick *et al.* 2000). Hemicrania continua is also considered to be uncommon (Bigal *et al.* 2002a). Exertional headaches are thought to be rare, but one study found a prevalence rate of 12% (Sjaastad and Bakketeig 2002).

Several studies have investigated the proportion of different types of headaches in several hundred consecutive patients seeking treatment (Gallai *et al.* 2002; Mongini *et al.* 2003; Cassidy *et al.* 2003; Fishbain *et al.* 2001). Migraine without aura is usually the most common (57 – 70%), with fewer diagnosed with migraine with aura (6 – 55%), episodic or chronic tension-type headache (14 – 34%), cluster headache (3 – 5%) and cervicogenic headache (34%). However, these prevalence figures should probably be taken as estimates, and most of these groups did not include cervicogenic headaches. Due

to problems with classification, considerable variability in diagnosis has been found at different sites (Beghi *et al.* 2003).

**Table 24.3 Diagnostic criteria for migraine without aura and episodic tension headache**

| *Migraine without aura* | *Episodic tension headache* |
|---|---|
| 1. At least 5 headaches fulfilling points 2-4 | 1. At least 10 headaches fulfilling points 2-4 |
| 2. Headaches last 4-72 hours | 2. Headaches last 30 minutes to 7 days |
| 3. Headache has at least two of following:<br>a. unilateral<br>b. pulsating<br>c. moderate to severe<br>d. aggravated by routine activity | 3. Headache has at least two of following:<br>a. pressure/tightening<br>b. mild to moderate<br>c. bilateral<br>d. no aggravation by routine activity |
| 4. Headache accompanied by at least one:<br>a. nausea/vomiting<br>b. photophobia and phonophobia | 4. With headache both of the following:<br>a no nausea/vomiting<br>b. no photophobia and phonophobia or only one, not the other |
| 5. Other headaches excluded. | 5. Other headaches excluded. |

Source: International Headache Society (IHS 1988)

## Differential diagnosis

The prevalence figures suggest that the differentiation between the different headache types is straightforward, uncontroversial and simple to make, but this is not the case. One problem is the use of different diagnostic criteria. For instance, Haldemann and Dagenais (2001) list five different criteria for cervicogenic headache, which have certain consistent features, but each includes distinctive characteristics. The most extensive classification criteria produced by IHS has been criticised on several counts (O'Driscoll 1999). It has had limited publication in a specialist journal, and therefore is not easily available; it is lengthy and very detailed for normal clinical practice. The groupings within the classification are based on structures and pathophysiological processes, whereas this is a particularly complex area that is relatively poorly understood in chronic benign headaches. The classification criteria are inconsistent, with some based on structure and others based on systemic disorders and still others on external factors, which might give rise to overlap (O'Driscoll 1999).

Furthermore, due to the lack of empirical findings a large part of the work was based on expert opinion and consensus, and thus has been subject to a number of challenges (Gobel 2001). The authors, however, do acknowledge its limitations and state that it was primarily intended for research rather than clinical purposes (IHS 1988).

Other issues have highlighted the potential for confusion over classification (Chou and Lenrow 2002). There are several overlapping features between cervicogenic headaches, migraine and tension-type headaches, and it is recognised that reliable differential diagnosis in clinical practice is still a problem (Leone *et al.* 1998). Incorrect application of the IHS criteria has been reported, with clinicians failing to gather full data, failing to make a specific diagnosis, or giving very different proportions of headache types when they do (Gallai *et al.* 2002; Blumenthal *et al.* 2003; Beghi *et al.* 2003). There are several reports of overlap, with more than one set of diagnostic criteria being met by the same patient, and migraine, tension-type, cervicogenic or other headache type being reported in the same patient (Bono *et al.* 1998, 2000; Antony 2000; Pfaffenrath and Kaube 1990; Sjaastad and Bovim 1991; Bigal *et al.* 2002b; Sanin *et al.* 1994; Fishbain *et al.* 2001). Even apparently distinct features for cervicogenic headache, such as onset with neck position, movement or trauma and other neck-associated symptoms are frequently found in other types of headaches (Fishbain *et al.* 2001). This suggests these are not discrete categories that are distinct from each other. Although significant differences in diagnostic features have been reported between cervicogenic headache, migraine and tension-type headache, few features are reported solely in one type of headache (Vincent and Luna 1999). Using a constellation of features is diagnostically more accurate than relying on single features.

Critics have come up with a range of reasons why the IHS classification system may be unstable. This may be because of overlapping symptoms, fluctuating patterns of symptomatology, the obscuring effect of self-medication, the possibility that different headache types are not in fact distinct disorders but share a common pathophysiological basis, and the limited validity and reliability of the classification criteria (Beghi *et al.* 2003).

## Cervicogenic headache

The first description of cervicogenic headache was in 1983 (Sjaastad *et al.* 1983), and the IHS classification was amended in 1988 to include headaches related to neck problems. Since then several groups have published diagnostic criteria or amended earlier ones (Sjaastad *et al.* 1990, 1998; Merskey and Bogduk 1994; Meloche *et al.* 1993; Jull 2002). Although these contain certain common features, they also contain many inconsistencies. Differences include the location of pain, whether it is unilateral or bilateral, and whether it can change sides or not. Some include a positive response to nerve blocks, one included radiographic criteria, some stipulated neck trauma, one focussed on abnormalities in local muscles and one included additional symptoms. Most agree that pain starts around the occipital area and can be aggravated by neck movement. Some state aggravation by posture as well, most note a decrease in cervical range of movement and most include neck tenderness to palpation or reproduction of headache on palpation. It does not appear that any specific tests or clinical finding has been determined to be pathognomonic of cervicogenic headache (Haldeman and Dagenais 2001). Features that most clearly distinguished cervicogenic headache from other headache types were: unilateral, side-locked headache with neck pain and headache associated with neck movements or postures (Vincent and Luna 1999; Bono *et al.* 1998).

There was initially reluctance by some headache specialists to accept the concept of cervicogenic headaches (Bogduk 2001). As with other headache classifications, there are problems with recognition. The validity of the diagnostic criteria to delineate a unique entity has been challenged, as there is considerable overlap with migraine and tension-type headache (Leone *et al.* 1998; Antonaci *et al.* 2001). The overall reliability of making a diagnosis of cervicogenic headache from history and physical examination is moderate, kappa 0.51 (van Suijlekom *et al.* 1999). Items from the history had kappa values between 0.08 and 0.76. In the physical examination pain provocation movements were more reliable (kappa 0.53 – 0.50) than range of movement tests (kappa 0.32 – 0.41), with provocation of headaches by manual pressure on the zygapophyseal joints the least reliable (kappa 0.16 – 0.23). Overall, agreement on the existence of cervicogenic headache amounted to 76%, similar to migraine (77%), with tension-type headaches being the least reliably detected (45%) (van Suijlekom *et al.* 1999).

In a reliability study to detect painful upper cervical joint dysfunction by manual examination in forty subjects with and without headaches, overall agreement was generally excellent, with kappa values mostly 1.00 (Jull *et al.* 1997). However, of twenty volunteers without symptoms, three were judged to have upper cervical joint dysfunction. It is claimed that manual examination can differentiate different headache types, but the claim is based on unpublished data, and a caveat is made that tenderness over cervical joints is present in all headache types and in those with no headache (Jull and Niere 2004). In another reliability study in which the examiners used both active and passive movements, the most reliable were tests for pain and range of protraction, retraction and retraction with overpressure (Hanten *et al.* 2002). Of the remaining eleven accessory movements, seven achieved kappa values less than 0.5.

## Neuroanatomy of cervicogenic headache and experimental evidence

Ironically, despite remaining controversies regarding diagnosis, the neuroanatomical mechanism for cervicogenic headaches is one of the best understood (Bogduk 2001). Cervicogenic headache appears to be a form of referred pain from the upper three cervical segments (Bogduk 1994; Pollman *et al.* 1997). The mechanism for this is the 'trigeminocervical' nucleus in the upper part of the spinal cord (Bogduk 1994). Within this area, terminals from the trigeminal nerve and the upper three cervical nerves overlap and ramify in the same section of spinal cord. The trigeminal is the fifth cranial nerve, and its branches provide the cutaneous nerve supply for most of the head and face (Williams *et al.* 1980). This convergence of afferents from two separate regions of the body into neurons in the central nervous system provides the anatomical substrate for referred pain. Furthermore, cervical rami 2 and 3 provide the cutaneous innervation to the areas of the occiput, and the area of the head posterior and inferior to the ear. Thus, problems in the upper cervical spine can potentially be perceived in any area of the head or face.

In asymptomatic volunteers it has been demonstrated that stressing the atlanto-occipital, the lateral atlanto-axial or C2 – 3 zygapophyseal joints evokes pain in the occipital or suboccipital regions (Dreyfuss *et al.* 1994b; Dwyer *et al.* 1990). Patients with headache have had their symptoms abolished by nerve block at O – C1 (atlanto-occipital),

C1 – C2 (lateral atlanto-axial) and C2 – C3 zygapophyseal joints (Busch and Wilson 1989; Aprill *et al.* 2002; Bogduk and Marsland 1986, 1988; Lord *et al.* 1994). Collectively these studies attest to the clinical reality of headaches stemming from upper cervical joints. However, their recognition is not easy, except with the use of anaesthetic joint blocks, as to date no clinical features of cervicogenic headache, including the distribution of symptoms, have been validated (Bogduk 2001). Headache as main complaint and tenderness over the C2 – C3 zygapophyseal joint had a positive likelihood ratio of 2:1 for the joint to be the source of symptoms (Lord *et al.* 1994), but only provides for a 60% confidence of the right diagnosis. Pain in the occipital or suboccipital region, tenderness on palpation of the lateral atlanto-axial joint, and restricted rotation at that level had a positive predictive value of only 60% at detecting headaches stemming from C1 – C2 (Aprill *et al.* 2002).

## Mechanical diagnosis and therapy and headaches

Patients who attend musculoskeletal specialists with a primary complaint of headaches may be suitable for mechanical diagnosis and therapy. Patients who have a secondary complaint of headache, but a primary complaint of neck pain, are managed as explained in other parts of the book. Amongst those with primary headache it must be remembered that the symptom can indicate serious pathology, although rare, and such patients must always be screened for the existence of other 'red flag' features (Table 24.2). Furthermore, headache may be due to migraine or other conditions that may not be amenable to mechanical therapy. Because of problems with the validity and reliability of diagnostic classifications, the proportion of headache patients that belong in each category is as yet unclear. It may in the future be revealed that far more patients with headache are in fact amenable to mechanical therapy than traditionally thought. Hopefully also in the future clinical features of those who do and do not respond to mechanical therapy may be recognised more clearly.

In the absence of pain patterns or other clinical features from the history indicating upper cervical joint problems, the symptomatic and mechanical response to repeated movements and sustained postures is the best way to recognise responders from non-responders to mechanical therapy. With the exclusion of 'red flag' features, it is reasonable to provide a trial of mechanical therapy as described below to determine

responsiveness. Failure to alter symptoms or mechanics by four to five sessions or after two weeks, including force progressions and force alternatives, should lead to the abandonment of mechanical therapy. It is likely in such cases that the headache is due to a non-mechanical condition or is a non-responder.

Atypical, inconsistent and sometimes even contradictory responses can be obtained during the mechanical evaluation of patients with mechanical cervicogenic headache. Experience has shown that whereas in other regions of the spine these types of responses might indicate non-responders to mechanical therapy or even serious spinal pathology contraindicating mechanical therapy, in cervicogenic headache such responses are not unusual. As long as the 'red flag' features are monitored, mechanical evaluation should be continued in such instances to determine if a mechanical response is forthcoming.

Because of the association between upper cervical procedures and serious, sometimes fatal outcomes in a few patients, some attempt must be made to establish the relative safety of end-range cervical procedures. The method of doing this is imperfect and the issues around this topic are discussed in Chapter 8.

Aims of the history and physical examination are thus:

- exclude 'red flag' pathology
- determine if headache is cervical in origin
- determine if headache is mechanical in origin
- determine mechanical diagnosis
- proceed with appropriate mechanical therapy.

Later stages of mechanical therapy may include:

- force alternatives/progressions if necessary
- end-range mobilisations/manipulations; if considered, first perform end-range sustained testing (described in Chapter 8).

## Classification

Mechanical headaches are classified as derangement, dysfunction or postural syndrome. It should be recognised that mechanical syndromes may behave atypically when symptoms are primarily

headache, which may be due to the unique anatomy of the upper cervical region. For instance, with derangement headache symptoms can be abolished, but not always easily reproduced. For this reason the original description involved a separate headache syndrome (McKenzie 1990). However, despite atypicality, certain core features can be similar to the symptomatic and mechanical responses found in derangement – rapid abolition of symptoms or increase in range of movement in response to repeated movements or sustained postures.

McKenzie (1990) considered that the application of mechanical diagnosis and therapy in the population with a primary complaint of headache was unsuccessful with a larger proportion of this group than in any other region. This may relate to the higher proportion of non-mechanical causes of headache symptoms compared to other musculoskeletal complaints.

### Derangement

Headache derangement can present in a typical fashion; for instance, with loss of range of movement, abolition of symptoms in response to repeated movements, and radiating symptoms to the temporal area localising to the occiput. However, there are several ways in which cervicogenic headaches due to derangement may be atypical in presentation or response and are dissimilar to the way derangements present in other parts of the spine. This may be due to the unique anatomy of the upper cervical segments. They are atypical in the following ways:

- limitation of range of movement is not necessarily present or is too small to be perceived
- after being made better, symptoms cannot necessarily be made worse or reproduced
- symptoms are more responsive to sustained postures than repeated movements
- symptoms are affected by end-range position, but not always affected by movement
- during the early stages of treatment the headache can increase for a few hours and then subside; in this situation, persist with treatment for four to five days before abandoning the attempt.

The most common reductive forces are upper cervical flexion, which may be retraction or a combination of retraction and flexion, and rotation less commonly.

To generate a positive response, sometimes force progressions are needed. For instance, in some patients with restricted movement, repeated retraction causes symptoms to increase, but retraction with overpressure causes symptom relief and a sudden increase in range of movement. In others constant headache may cease with retraction overpressure, but return as soon as overpressure is released. However, flexion with overpressure gradually resolves symptoms in some such patients. In those patients who respond to posture correction, symptoms are quickly reproduced on resumption of the faulty posture. Only by maintaining correct posture over several weeks does resolution of symptoms occur (McKenzie 1990). It is possible that the explanation for this is the gradual desensitisation of sensitised articular or peri-articular mechanoreceptors.

### Dysfunction syndrome

Dysfunction syndrome when present as a cause of cervicogenic headache is usually typical of articular dysfunction elsewhere, but such patients do not commonly present for treatment. Headache is produced with end-range movement, or sometimes end-range with overpressure, produced consistently, and abates when end-range position is released. Patients should report consistency of aggravating factors. The most common remodelling forces are, again, upper cervical flexion and rotation.

### Postural syndrome

Cervical headaches commonly arise from static loading in end-range positions that cause mechanical deformation of peri-articular structures. *Complete relief of headache on performing postural correction* confirms the presence of postural syndrome. The great majority describe that prolonged sitting, especially driving and office work, is the most troublesome posture. In this position with relaxed sitting a protruded head posture is adopted, which is often the causative loading in cervical headache due to postural syndrome. The patient often fails to attribute the headache to poor posture, instead blaming stress, workload or fatigue. If headache is due to postural syndrome, correction of the protruded head posture should immediately abolish the headache.

## History

Taking a history from patients with headache is much the same as patients with other symptoms. The site of symptoms is recorded, the initial onset, history this episode, aggravating and relieving factors, frequency, history of headache, and history and effect of medication. It is important to have a clear understanding of the frequency history, how often and for how long the headaches last, as this may help in evaluating clinical response. As in other parts of the spine, the aggravating and relieving factors may provide clues as to the mechanical nature of the problem, or lack of it. It is important to be alert to 'red flag' features in the history (Table 24.2), and be aware of severity, age at onset and cause of onset, as well as any other accompanying symptoms.

Special questions should relate to presence of:

- dizziness
- nausea
- tinnitus
- visual disturbance
- difficulties with speech or swallowing
- drop attacks/loss of consciousness
- vomiting
- other associated symptoms.

Whereas the first three symptoms may accompany cervicogenic problems, the others are more likely to indicate other, potentially more serious problems. Dizziness may be due to cervicogenic, vestibular or vertebral artery insufficiency or a number of other problems (see Chapter 8 for discussion).

In cervicogenic headaches, pain typically starts in the occipital region and can radiate to the forehead, temples, orbital region, vertex or ear. It can be accompanied by dizziness, nausea and even photophobia (Haldeman and Dagenais 2001; Pollman *et al.* 1997). Less commonly headache is reported in the orbital and frontal regions. Occipital headaches are frequently symmetrical, whilst headaches in other regions are frequently unilateral and may occur on alternating sides.

Various clues in the history may alert the clinician to the potential for mechanical therapy, or lack of it. As in non-specific neck pain in general, headache arising from mechanical disturbance of the cervical spine tends to be intermittent and episodic in nature. If symptoms are intermittent and a consistent causative factor can be identified, management should be reasonably straightforward. Headache is nearly always affected by positioning, but not always by movement. Cervical headaches commonly arise from static loading in end-range positions; many describe that prolonged sitting is the most troublesome posture. With relaxed sitting a protruded head posture is adopted, which is often the causative loading in cervical headache.

**Table 24.4 Possible clues to mechanical nature of cervicogenic headache**

- intermittent symptoms
- symptoms associated with consistent activity
- symptoms produced with sustained activity in one posture.

Patients that report constant symptoms or symptoms unrelated to mechanical loading, such as prolonged sitting or sleeping, may prove less responsive to mechanical therapy, but a trial of mechanical therapy is still justified.

## Physical examination

Physical examination for patients with headache is much the same as patients with other problems. A baseline record is made of posture, range of movement and pain response during movement. All cervical movements are evaluated; usually the most relevant are retraction, extension, flexion and rotation. Although restricted range of movement can be one of the distinguishing characteristics between cervicogenic and other types of headache (Zwart 1997), this is not always present or is too small to see and absence of apparent limitation is a frequent finding (McKenzie 1990). Significant differences in range have been found in rotation and flexion/extension between cervicogenic headache and patients with migraine and tension-type headache and controls (Zwart 1997).

The pain status at rest, the effect of posture correction and then the effect of repeated movements are noted. In addition to active repeated movements, overpressures and sustained positions are often required to explore symptomatic and mechanical responses fully. Thus, test

procedures include end-range positions maintained for up to two to three minutes at a time. Confirmation of the mechanical nature of the problem is made by inducing a change in pain location or severity in response to repeated movements or sustained positions (McKenzie 1990). Symptoms may localise to the occiput or occasionally change sides. For those with limited movement, the mechanical response may also confirm the mechanical nature of the problem.

Relative to end-range positions for upper cervical segments, it should be remembered that sagittal end-range is obtained by using retraction and protrusion in addition to flexion and extension, rather than flexion and extension only (Chapter 4).

If when patients attend the clinic they are free of headache, it may be impossible to prove a mechanical cause-and-effect relationship between symptoms and cervical loading. In such instances the patient should be advised to re-attend when the headache next occurs.

## Mechanical assessment

The mechanical assessment is used to try to confirm the cervical and mechanical basis for the headache. Mechanical cervicogenic headaches occur most commonly from static end-range loading (McKenzie 1990). Mechanical response is confirmed when repeated movements or sustained positions cause characteristic symptoms and/or mechanical responses for the three syndromes as outlined above.

### Retraction (sitting) – (Procedure 1)

The patient should be sitting with their bottom to the back of the chair and upright. Prior to test movements, the patient is asked to report the location and intensity of the present headache. The patient is then instructed to draw their head as far back as possible, with the head remaining horizontal, facing forward and not inclining up or down. The movement must be made to maximum end-range retraction. Then the patient should add overpressure with fingers on their chin, still ensuring that the head remains facing forward. With each additional overpressure the head should move further into end-range, and each time the overpressure should be maintained for several seconds. After five or six excursions, the end-range position should be maintained for up to three minutes depending on the patient's tolerance.

If this fails to induce symptom response, then clinician overpressure to retraction is added. Again, this should be maintained for up to three minutes. This should be done during the initial assessment if patient overpressure has failed to produce symptom change.

### Other tests

If the headache remains completely unaffected by retraction with clinician overpressure, then alternative forces should be explored. This should be done in a similar way using end-range active movements, patient overpressure to end-range, and clinician overpressure to end-range until a symptom response is generated. Again, all these procedures should be sustained for up to three minutes. The order of force alternatives is as follows:

- flexion (Procedure 6)
- retraction and flexion modified to maximise force on upper cervical area
- rotation (Procedure 5)
- combination flexion/rotation
- combination extension/rotation.

Once a procedure is found to affect the patient's symptoms, no further testing is necessary. If none of these procedures affect the headache, it may be that it is not mechanical cervical in origin. However, prior to the complete abandonment of mechanical evaluation, it is worth exploring the patient's response over twenty-four to forty-eight hours. If there was any hint in the history or physical examination for any particular directional preference, this should be explored; otherwise upper cervical flexion positions are best utilised as being the most common movement to generate a positive response.

## Management of mechanical cervical headache

If it has been established that the headache is cervical in origin and mechanical in nature by the previous test movements, management usually consists of postural advice and an exercise component. The exercise involves the movement that has been found to abolish or decrease symptoms for derangement and reproduce symptoms for dysfunction, whilst for postural syndrome, posture correction is the key component. The sequence below describes the normal force alternatives and force progressions that may be added to affect symptoms:

Procedures to be used:

- posture correction – essential procedure, occasionally all that is necessary whenever patient is seated (Procedure 3)
- retraction with patient overpressure, repeated/sustained – essential (Procedure 1a).

Regularity:

- every two hours at least, or when indicated by symptoms.

If after two to three days headache severity or frequency is improving, management continues with retraction. However, the length of time needed to evaluate the effect of repeated movements depends partly on the pattern of headache frequency that the patient reports in the history. If there is limited improvement or failure to maintain improvement, ensure that the patient has followed the postural advice and performed the exercises in the correct manner. Force progressions and force alternatives are considered in the following order with subsequent procedures performed only when there is failure to improve. If at any point improvement does occur, further progressions are unnecessary. Whatever progressions are used, the patient must continue with the appropriate home exercise plan regularly. Following one force progression or force alternative, the effect of this should be evaluated at the next review. Force progressions can be repeated on up to two occasions before they should be abandoned if no change occurs. Do not instigate clinician techniques unless it is clear that improvement cannot be achieved by the use of patient techniques only.

Force progressions:

- flexion with patient overpressure, may need to be modified to maximise force to upper cervical spine (Procedure 6a)
- flexion with clinician overpressure (Procedure 6b)
- flexion mobilisation (Procedure 6c)
- rotation with patient overpressure in sitting (Procedure 5a)
- rotation with clinician overpressure (Procedure 5c)
- rotation mobilisation (Procedure 5c)
- extension – unusually some patients respond to extension rather than flexion (Procedure 2).

Very few patients with cervicogenic mechanical headache fail to respond to the procedures that have been described. Certain patients, however, who appear to have a mechanical headache but gain no lasting benefit from these procedures, may benefit from appropriate upper cervical techniques of mobilisation or manipulation. Only a clinician with the appropriate manipulation skills should perform this.

### Correction of the lying posture

Some patients describe that their headache is consistently present on waking each morning, but was not there the night before. Such a consistent pattern is usually the result of a mechanical headache. If resting through the night is causing pain, two factors need to be investigated:

1. The lying posture itself. This is different for each person and must be dealt with individually. Sleeping postures are habitual and can be difficult to influence. A typical position is end-range rotation that occurs in those who sleep prone.
2. The surface on which the person is lying. The pillow is usually more important than the mattress in cervical postural pain. A soft and malleable pillow is most appropriate, which provides support for the head in a horizontal position, but avoids pushing the head into flexion or lateral flexion.

### Modification of the lying posture

Patients can be encouraged to alter their sleeping posture if this is indicated, but it may be difficult to achieve. An extreme strategy to avoid prone lying is to tape an object to the chest or abdomen, which disturbs the individual enough to change their posture.

If the surface seems to be inappropriate, a cervical roll can be placed inside the pillow. This fills the gap between the shoulders and the head and provides direct support for this area. This should only be used with one pillow. The cervical roll usually works quickly or not at all, and should be tried for a few nights.

## Conclusions

This chapter has dealt with patients who report their primary symptom to be headache, not those whose primary complaint is neck pain with some associated headache. Headache is a symptom with numerous causes. It can be a symptom of serious cranial pathology, and

the first task is to ensure that no 'red flag' features are present. It is then necessary to determine if the headache is cervical in origin and mechanical in nature. The series of end-range test positions to use in the physical examination and management of mechanical cervicogenic headache have been described. A higher proportion of patients fail to respond to mechanical therapy with headache than other symptoms. The reason for this may be a non-mechanical and non-cervical cause for these symptoms.

# 25: Cervical Trauma or Whiplash Associated Disorders

## Introduction

*"One thing is certain about whiplash – it is something that happens in a motor vehicle accident. Every other aspect of whiplash has been controversial, or remains so in some quarters"* (Barnsley *et al.* 2002). Whiplash associated disorders (WAD) is the term applied to a collection of signs and symptoms that are said to result from a road traffic accident (RTA) or some other trauma. The predominant symptoms are neck pain and headache, and the sign is restricted neck movement. A mechanical evaluation should still be performed with such patients, although sometimes this should be done with caution. Patients with neck pain from a traumatic onset may not display typical mechanical responses as soft tissue injury may have occurred. It may be necessary to allow time for the healing and repair process necessary for such conditions. However, despite the onset, many such patients still present with a mechanical syndrome. The time since onset is an important consideration in determining classification and management. This chapter considers certain aspects of the condition.

The chapter has the following sections:

- what is whiplash?
- is whiplash real?
- signs and symptoms
- classification of WAD
- natural history
- prognostic factors
- management of WAD – literature
- management – mechanical diagnosis and therapy
    - investigations
    - acute/sub-acute stage
    - chronic stage.

## What is whiplash?

Whiplash is a familiar term to patients and clinicians. It generally denotes neck symptomatology that has commenced during or shortly after a motor accident. Strictly, the causal event for a true 'whiplash' injury is said to be a rear-end collision at a modest speed into a stationary vehicle in which the victim is facing forwards (Bogduk 1986). In practice, anyone involved in a car accident that develops symptoms is likely to be considered to have 'whiplash'. Neck pain that develops after a diving incident or some other high-velocity impact to the neck is also often included in the term. A major review (Spitzer *et al.* 1995) of the topic gave the following definition:

*"Whiplash is an acceleration-deceleration mechanism of energy transfer to the neck. It may result from rear-end or side-impact motor vehicle collisions, but can also occur during diving or other mishaps. The impact may result in bony or soft-tissue injuries (whiplash injury), which in turn may lead to a variety of clinical manifestations (Whiplash Associated Disorders)"*, Spitzer *et al.* 1995.

The kinematics of whiplash are discussed by Bogduk (1986). As the car and seat are propelled forward, the body and shoulders are moved likewise; the head resists forward displacement and is thrown into extension. Once the inertia of the head is overcome, the leverage provided by the neck allows the head to be propelled into acceleration, and it is catapulted into flexion. Head acceleration can be as high as 12G in the extension phase and 16G in the flexion stage.

The possible pathological events during this occurrence are numerous and include damage to a range of soft tissues, the intervertebral disc, zygapophyseal joints, the odontoid process, the temporomandibular joint and other structures, even including the brain (Bogduk 1986). The pathophysiology of whiplash is discussed in detail elsewhere (Barnsley *et al.* 1994a; Bogduk 1986; Barnsley *et al.* 2002). The most likely structures to be injured are the zygapophyseal joints, the intervertebral discs and the upper cervical ligaments (Barnsley *et al.* 2002). In a systematic review of autopsy studies of the cervical spine of road traffic fatalities, pathoanatomical lesions were found in the cervical discs, endplates and zygapophyseal joints (Uhrenholt *et al.* 2002). These were generally of a subtle nature that would not be detected by imaging studies, especially radiography.

Following whiplash injury, disc herniations (Pettersson *et al.* 1997; Jonsson *et al.* 1994; Davis *et al.* 1991), ligament damage and fractured end-plate (Davis *et al.* 1991), cervical muscle dysfunction (Nederhand *et al.* 2000), faulty vestibular functioning (Chester 1991), and vertebral artery occlusion (Giacobetti *et al.* 1997) have all been reported. Damage to the intervertebral disc and zygapophyseal joint in the cervical and lumbar spines has been reported in autopsy studies from road accident victims (Taylor and Twomey 1993; Twomey *et al.* 1989). However, despite these accounts of specific pathology, other investigators using sophisticated diagnostic imaging have been unable to detect soft-tissue injuries in acute patients who nonetheless developed symptoms (Barton *et al.* 1993, Ronnen *et al.* 1996).

In over three hundred patients with chronic neck pain following various neck traumas, using provocation and abolition of pain with intra-articular injections as the criteria, 53% had a symptomatic disc and 26% a symptomatic zygapophyseal joint (Aprill and Bogduk 1992). In a group of 56 patients, symptom response in both structures was observed in 41%, to individual structures in about 20% each, with neither joint implicated in 17% (Bogduk and Aprill 1993).

One research group has demonstrated that one of the most common sources of chronic neck pain or headache following whiplash are the cervical zygapophyseal joints. Several studies of consecutive patients with chronic symptoms referred to tertiary care have been undertaken. Very strict criteria were used, with double intra-articular injections to make the diagnosis. Repeat injections are necessary because there is roughly a 40% placebo response to a single injection. Between 27% and 54% of these groups were identified with pain of zygapophyseal joint origin (Barnsley *et al.* 1995; Lord *et al.* 1994, 1996a). Symptomatic segmental levels were predominantly C2 – C3 and C5 – C6.

## Is whiplash real?

There is controversy within the medical literature between those who believe in the validity of WAD and those who claim it to be a myth. It has been argued that 'whiplash syndrome', as opposed to brief and insignificant neck pain, is no more than a cultural construct within a biopsychosocial model of pain (Ferrari and Russell 1997, 1999). It is suggested that WAD exists only in cultures in which pain following RTA is expected, bred in an environment in which anxiety and fears are nurtured by health professionals, lawyers and patients alike. These

authors *"believe that the 'whiplash syndrome' is an example of illness actually induced by society, in general, and by physicians in particular"* (Ferrari and Russell 1997).

The following are mentioned in support of their viewpoint:

- studies that demonstrate no injury with sophisticated imaging studies, as mentioned above
- the failure to cause injury in animals except with exceptional force
- the lack of neck pain in those who crash cars for fun, or let themselves be involved in 'crashes' for the sake of experiment
- the variability of symptoms in different cultures, which appears to reflect legal and medico-legal determinants rather than the rate of accidents.

They attempt to show that reporting 'injuries' in part reflects compensation systems, and that the prevalence of chronic symptoms varies in different countries. Where individuals do not witness WAD-type illness behaviour, the argument is that they don't get whiplash. In this regard the study from Lithuania (Schrader *et al.* 1996) is much quoted: *"Where there is little knowledge or expectation of the potential of a whiplash injury to lead to chronic symptoms, and where involvement of insurance companies, litigation and even the therapeutic community is rare....* [there is] *no increased prevalence of chronic neck pain after a rear-end collision, when compared with the background risk of chronic neck pain in uninjured control subjects"* (Ferrari and Russell 1999). The study from Lithuania (Schrader *et al.* 1996) found symptoms in 35% in a retrospective investigation of police records of those involved in rear-end collisions one to three years previously; this compared to 33% in a non-injured control population. This study has been criticised (Freeman *et al.* 1999) as the numbers were insufficient to study the natural history of the condition. In a similar study in which individuals who had neck pain after an accident ten years earlier were compared to a control group, reports of neck pain were 55% and 29% respectively and were significantly different between the groups (Bunketorp *et al.* 2005).

Despite there being a *"virtual lack of proof of a causal relationship to the injury mechanism after which the syndrome has been named"* (Stovner 1996), the condition has excellent face validity as it is a

condition recognised by clinicians and patients, and a multitude of descriptive studies give it good descriptive validity.

Freeman *et al.* (1999) produced a review that challenged those authors who refute the existence of the whiplash syndrome. As a result of their literature review, Freeman *et al.* (1999) determined that there is *no* epidemiological or scientific basis for the following ideas:

- whiplash injuries do not lead to chronic pain
- chronic pain from whiplash is psychogenic
- the risk of chronic neck pain among acutely injured whiplash victims is the same as the prevalence of chronic neck pain in the general population.

Furthermore, previous neck injury has been found to be a significant risk factor for subsequent neck pain (Croft *et al.* 2001), and in a population study in which 814 individuals reported chronic and persistent neck pain, 30% had a history of neck trauma (Guez *et al.* 2003).

## Signs and symptoms

Neck pain is the most commonly reported symptom following an RTA; other symptoms have been described (Table 25.1).

**Table 25.1 Presenting signs and symptoms from whiplash**

*Common symptoms (> 70%, multiple studies)*

- neck pain
- neck stiffness/limited range of movement
- headache

*Moderately common symptoms (20 – 70%, multiple studies)*

- shoulder pain
- numbness/paraesthesia in arm
- low back pain

*Uncommon symptoms (<20%)*

- arm pain
- scapulae pain
- visual symptoms
- auditory symptoms
- muscular tenderness
- dizziness
- nausea

Continued next page

*Occasional reports*

- dysphagia
- weakness
- Horner's syndrome
- tinnitus
- concussion
- insomnia
- psychological disturbance, such as
  - anxiety
  - depression
  - irritability
- short-term memory disturbance
- cognitive impairment.

Source: Bogduk 1986; Hohl 1974; Pearce 1989; Hildingsson and Toolanen 1990; Norris and Watts 1983; Watkinson *et al.* 1991; Maimaris *et al.* 1988; Barnsley *et al.* 1994a; Brison *et al.* 2000; Radanov and Dvorak 1996; Radanov *et al.* 1992, 1996; Mayou *et al.* 1993; Wallis *et al.* 1996

## Classification of WAD

As the identification of specific pathology without the use of intrusive injection technology is highly problematical, classification is usually based on symptomatology. Most commonly this is related to duration of symptoms, which are described as either acute or chronic. The Quebec Task Force (QTF) classification attempts to portray some level of the severity of symptoms resulting from a whiplash injury (Spitzer *et al.* 1995).

**Table 25.2 QTF classification of WAD**

| *Grade* | *Clinical presentation* |
|---|---|
| 0 | No neck pain<br>No mechanical signs |
| 1 | Neck pain, stiffness or tenderness only<br>No mechanical signs |
| 2 | Neck pain ***and***<br>Mechanical signs |
| 3 | Neck pain<br>Mechanical signs ***and***<br>Neurological signs |
| 4 | Neck pain ***and***<br>Fracture or dislocation.* |

Mechanical signs = reduced movement, tenderness.
Neurological signs = sensory, motor or reflex deficit.
* This group is obviously not for normal conservative management.

Source: Spitzer *et al.* 1995

This classification scheme has been found to have prognostic value, in that higher grades have been associated with poorer outcomes at six, twelve, eighteen and twenty-four months (Hartling *et al.* 2001). However, this classification scheme only gives an indication of severity and is unhelpful in prescribing management. As far as mechanical diagnosis and therapy is concerned, classification is determined by the mechanical evaluation. In the acute stage non-mechanical conditions may be common, no mechanically determined directional preference is detected and the patient is treated as for any trauma during the inflammatory stage; however, this frequently changes in the subsequent weeks. In the chronic stage multi-directional dysfunction, derangement or a chronic pain state may be present.

## Natural history

The epidemiological literature on WAD principally concerns those individuals who develop symptoms; however, various reports would suggest that 50% or less of those involved in car accidents actually develop symptoms (Galasko *et al.* 1993; Thomas 1990; Freeman *et al.* 1999). Most studies that consider the natural history of the disorder are based on patients who have attended Accident and Emergency departments. This is likely not to include those who had what they considered to be trivial and brief symptoms, and may only relate to those with more severe symptoms, greater anxiety about those symptoms or those with a desire to seek compensation. The studies on the natural history of WAD may therefore present a biased sample.

Bearing these limitations in mind, a multitude of studies consistently demonstrate high prevalence rates of neck pain one to two years after a whiplash injury, with about 50% or more of consecutive inception cohorts still reporting symptoms (Deans *et al.* 1987; Hildingsson and Toolanen 1990; Norris and Watts 1983; Gargan and Bannister 1990; Watkinson *et al.* 1991; Squires *et al.* 1996; Gargan *et al.* 1997; Maimaris *et al.* 1988; Brison *et al.* 2000; Mayou and Bryant 1996). Three reviews (Freeman *et al.* 1998; Spitzer *et al.* 1995; Barnsley *et al.* 1994a) have considered the natural history of WAD from the methodologically stronger studies. They concluded that an average of 33% at nearly three years (Freeman *et al.* 1998), 27% to 66% at six months or more (Spitzer *et al.* 1995) and 14% to 42% (Barnsley *et al.* 1994a) of individuals develop chronic symptoms. Approximately 10% will have constant severe pain indefinitely (Barnsley *et al.* 1994a).

Clearly there is considerable individual variation in the natural history of WAD; it does not follow a uniform or a predictable course. For many it would seem that the incident is very trivial; no or minimal symptoms ensue and no or minimal health care is sought. For those who seek health care, the outcome appears to be dichotomous. Many improve in the first few weeks and become asymptomatic within a few months. However, a substantial minority of those who seek health care have persistent symptoms – further improvements occur in this group, but recovery appears to be less and less likely the longer symptoms persist. Those with long-term symptoms may comprise at least a third of all those who seek help. *"Viewed simplistically, the outcome for an individual patient is dichotomous; either the neck pain will resolve in the first few months or it will persist indefinitely"* (Barnsley *et al.* 2002).

## Prognostic factors

Various studies, generally of poor quality, have tried to identify factors that are associated with persistent symptoms, and a wide range of potential prognostic factors have been considered (Hohl 1974; Deans *et al.* 1987; Hildingsson and Toolanen 1990; Norris and Watts 1983; Watkinson *et al.* 1991; Maimaris *et al.* 1988; Gargan and Bannister 1990; Hartling *et al.* 2001; Stovner 1996; Allen *et al.* 1985; Olney and Marsden 1986; Mayou and Bryant 1996; Pennie and Agambar 1991; Gargan *et al.* 1997; Radanov *et al.* 1991, 1994; Harder *et al.* 1998). Many studies are limited by retrospective design, selection and follow-up bias, small numbers, incomplete follow-up and lack of standardised outcome measure (Boon and Smith 2002).

The strongest and most consistent factor associated with a poor prognosis is severe initial symptomatology. This is especially so if associated with headache, arm pain, neurological signs or symptoms, or restricted range of movement. Several studies suggest that female gender and older age denote poorer prognosis. The type of collision and whether symptoms develop immediately or later may be factors in prognosis. The role of psychological factors, litigation and radiological findings are sometimes hypothesised to have a role in outcome, but the literature would suggest otherwise.

Baseline psychological variables do not appear to predict future pain (Radanov *et al.* 1991, 1994). One study that looked at psychological variables as defined by the General Health Questionnaire found that

within a week scores were normal in 82% of the group. However, in the group that developed persistent and intrusive symptoms, at three months scores had become abnormally high in 81% of the patients (Gargan *et al.* 1997). Outcome at two years was predicted both by these raised scores and restricted neck movement. These findings suggest that the disorder has both physical and psychological components, but the psychological response develops after the physical damage and these responses are established within three months of injury. Psychological features exhibited by these chronic pain patients would appear to be the consequences of somatic symptoms and not their cause (Wallis *et al.* 1996; Radanov *et al.* 1996). Sterling *et al.* (2003a, 2003b) found that psychological distress and fear-avoidance beliefs decreased in those whose symptoms became mild or resolved, but remained high in those whose symptoms were reported as moderate or severe at six months.

Litigation is common after whiplash injuries and it has been hypothesised that this has a prognostic influence on outcome. However, several studies discount the prognostic value of whether or not compensation is sought and whether a settlement has been reached (Pennie and Agambar 1991; Mayou and Bryant 1996; Paramar and Raymakers 1993). In fact, multiple studies have failed to demonstrate an association between symptom resolution and compensation (Boon and Smith 2002). Many patients improve prior to settlement and many remain symptomatic after settlement of litigation.

The prognostic significance of radiographic findings is unclear (Boon and Smith 2002). Study findings are contradictory: some studies report a link between cervical spondylosis and persistent symptoms, but these often fail to account for age or severity of initial symptoms.

A systematic review of prognostic factors with emphasis on high-quality papers found strong evidence for high initial pain intensity being an adverse prognostic factor (Scholten-Peeters *et al.* 2003). They found strong evidence that older age, female gender, psychological response and compensation were *not* associated with adverse prognosis.

## Management of WAD – literature

As in other areas of musculoskeletal medicine, physiotherapy management is something of a lottery with a wide range of techniques and modalities used (Maxwell 1996), for some of which the evidence is

lacking. Most of the research in this area refers to acute WAD; the optimum management for chronic WAD has not been determined.

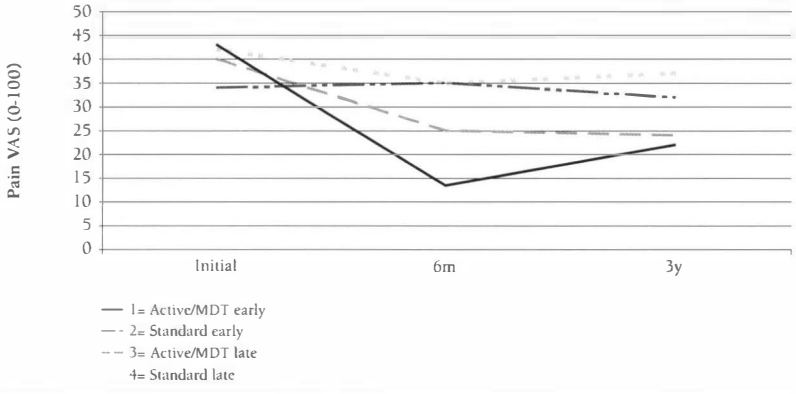
Several systematic reviews of conservative treatments for acute whiplash have recently been conducted. The general conclusions of all are similar – early activity is best, and the use of collars or rest leads to poorer outcomes. Peeters *et al.* (2001) concluded that active treatments show a beneficial long-term effect, and that 'rest makes rusty'. Bogduk (2000) found a home exercise programme better than rest, and stated that traction, electromagnetic therapy, collars, TENS, ultrasound, spray and stretch and laser should not be used. Magee *et al.* (2000) found studies indicated a weak-to-moderate positive effect for exercise, educational advice on posture and manual therapy. The QTF supported the use of exercises, advice on posture and mobilisation to help promote activation (Spitzer *et al.* 1995). Guidelines for physiotherapy management of whiplash derived from the available evidence recommended active interventions such as education, exercise therapy and training of functions and activities (Scholten-Peeters *et al.* 2002).

Thus, management of acute whiplash should consist primarily of advice about normal activity, no or minimal sick leave, no use of collar and a regular and progressive exercise programme starting with rotation and retraction. An active treatment approach is supported by numerous studies (Mealy *et al.* 1986; McKinney *et al.* 1989; McKinney 1989; Borchgrevink *et al.* 1998; Soderlund *et al.* 2000; Rosenfeld *et al.* 2000, 2003). Use of collars and rest produced worse outcomes than active treatment (Mealy *et al.* 1986; McKinney *et al.* 1989; McKinney 1989; Borchgrevink *et al.* 1998). Mobilisation is better than collar and rest (Mealy *et al.* 1986), but in the long term leads to worse outcomes than exercise and advice (McKinney 1989).

Rosenfeld *et al.* (2000, 2003) compared an active to a standard intervention, with both instigated either within the first four days (groups 1 and 2) or more than two weeks after the injury (groups 3 and 4). The active intervention consisted of two phases: initially information, postural control and cervical rotation exercises every hour, and a second phase if symptoms had not resolved using mechanical diagnosis and therapy (groups 1 and 3). The standard intervention involved advice initially to rest, and then to perform exercises a few weeks after trauma two or three times a day (groups 2 and 4). Sick leave was significantly less and changes in pain intensity significantly

more in the active treatment group both short- and long-term (Figure 25.1). At three years only the early active intervention group had a total cervical range of movement similar to a matched asymptomatic control group. It is clear from this and other studies that movement should start as early as possible and be performed regularly, whereas a more cautionary approach leads to considerably worse long-term outcomes.

**Figure 25.1 Outcomes of whiplash: MDT versus standard intervention**




Source: Rosenfeld *et al.* 2003

Lengthy treatment episodes should be avoided with this group of patients. Of the forty-three patients in the two active treatment arms of the trial, twenty-five had three or less sessions and the mean number of treatment sessions was just under four (Rosenfeld *et al.* 2000). Twenty-seven of the forty-three (63%) were still symptomatic at twenty days and received a mechanical evaluation and specific exercise therapy.

## Management – mechanical diagnosis and therapy

### Investigations

Radiography or imaging studies are not routinely needed for patients with WAD. Radiological studies are generally unremarkable or show pre-existing degenerative changes that are not relevant to the present symptoms (Teasell and Shapiro 2002). It should also be noted that several studies demonstrate the insensitivity of plain radiographs to detect significant bony injury (Barnsley *et al.* 2002). Serious injuries do occur during motor vehicle accidents, but these are relatively rare and should be detected at the time of the accident. The pathology of

particular concern are fractures or ligament instabilities that make the patient at risk of a spinal cord lesion. In a database of 22,858 patients involved in accidents and admitted to hospital for more than seventy-two hours, admitted to a high dependency unit, or died in hospital as a result of trauma, just over 2% were diagnosed with spinal injuries (Robertson *et al.* 2002). Less than 0.4% of the total database had spinal cord lesions. However, in a group of three hundred patients with fracture-dislocations, gross disability in a third resulted from error or lack of suspicion on the part of the examining physician (Bohlman 1979). Any indication that the patient has an upper motor neurone lesion or major instability in the neck requires urgent specialist referral and immobilisation of the neck prior to the patient leaving the clinic. See section on 'red flags' (Chapter 8) for more detail.

### Acute/sub-acute stage

If the patient is seen within the first few weeks following trauma exercises should start ***straightaway***, although they will have to be progressed gradually and with due care to the symptom response. A normal mechanical evaluation is performed if possible and if the patient displays any mechanically determined directional preference indicating a derangement, they are treated in the normal way. Failure to display a mechanical response may indicate that pain is due to trauma and the subsequent inflammatory response. See McKenzie and May (2000) for a résumé of the healing process. During the healing or repair stage the patient is encouraged to regain all lost movement by gradually expanding their range of movement and the movements they are performing. Regular and frequent movements performed throughout the day are essential for optimal tissue repair as the collagen heals along the lines of stress. Movement encourages transport of fluid and metabolites, facilitates the removal of exudates and promotes healing (Twomey and Taylor 1993).

Some patients are reluctant to move as movement produces or increases pain. Patients must be reassured about the normality of this during the healing process, and also that the best long-term outcome is ensured if they start to move as early and regularly as possible. It is the clinician's duty to provide the appropriate education and to placate patients' anxieties and fear of movement. The appropriate symptom response should be discussed: that movements should be far enough to provoke some discomfort, which should settle quickly after, and the range of movements should be gradually increased.

Patients must also be made aware of the posture of their head and neck. Relaxed sitting posture usually involves a protruded head posture, which should be avoided during the repair process. It will be more comfortable and better long-term if a neutral head position can be maintained – head over shoulders. This should be discussed with the patient, including the importance of maintaining a lumbar lordosis to achieve it.

Management thus consists of the following:

- education of patient concerning role of movement during healing process
- posture correction (Procedure 3)
- exercises, every one to two waking hours, moving as far as possible, progressively increasing the range of movement
- all movement directions should be worked at eventually
- a suggested order of recovery is as follows, but the order is less important than the regular movement:
    - rotation (Procedure 5)
    - lateral flexion (Procedure 4)
    - extension (Procedure 2)
    - flexion (Procedure 6).

The order that movements are regained and the rate that end-range is achieved depends initially on patient's symptomatic and mechanical responses, and a process of clinical reasoning is used as the concepts outlined above are applied. Patient overpressures may need to be used toward the end of the rehabilitation process to ensure full movement is regained.

It is emphasised again that patients must be evaluated to determine if a mechanically determined directional preference exists; this would indicate a derangement and the need for specific directional exercises rather than general exercises. Sometimes this only becomes apparent during subsequent evaluation, so it is always important to have an awareness of the possibility of derangement with this patient group. If seen in the very acute stage, pain may prevent an adequate assessment; this should be conducted as soon as it is feasible.

### Chronic stage

The management of chronic whiplash can be more problematical; the evidence regarding conservative management of this group is virtually non-existent. Some in this group will belong to the small percentage with constant intractable pain, for whom prognosis is poor. However, as in acute patients a mechanical assessment should be conducted. An unreduced derangement may be present requiring a specific mechanically determined directional preference of movement. Multiple direction dysfunctions also occur in this group as a result of reluctance to move in the earlier stages of recovery. If a mechanical syndrome is not detected, failure to respond is likely to be high; poorer prognosis is more probable the longer symptoms have been present and if previous therapy has been unsuccessful. For those deemed to be in a chronic pain state, a cognitive-behavioural approach has been suggested (Shorland 1998).

## Conclusions

Although the existence of whiplash as a clinical entity is still debated in the medical community, both patients and clinicians largely accept it. WAD is the occurrence of neck pain and headache arising as a direct result of a road traffic accident – other symptoms occur with less frequency. Such neck pain does not always result from car accidents; available studies suggest that about 50% or less of those involved in accidents go on to develop significant neck symptoms. Of those who do develop neck pain, the natural history is extremely varied and unpredictable. Whilst the majority appear to resolve their symptoms within the first few months, the rate of resolution then slows considerably and a significant minority of patients will be left with persistent symptoms. Numerous factors have been found to be suggestive of a poor outcome, but for many of these the evidence is contradictory. The most consistent factors across multiple studies associated with poor outcome involve severity and spread of symptoms.

Management of acute whiplash is dependent upon exercise, advice and patient-centred care. The available evidence makes clear the importance of a return to normal activity and movement as quickly as possible. Regular repeated movements are essential, starting with rotation and retraction, to recover full range of movement. Posture correction and interruption of static postures are also important. A graded return to full activity should be supplemented by a full

mechanical assessment to determine the presence of derangement and the need for a particular mechanically determined directional preference. In the absence of this a more general approach is required. For chronic whiplash patients the evidence is much less convincing, and the possibility of persistent and unrelenting symptoms must be considered. Again, an exercise, patient-centred approach is essential. Multiple direction dysfunction is not an unusual finding in those with chronic symptoms.

# 26: Thoracic Spine – Epidemiology, Pain, Anatomy, Biomechanics

## Introduction

Literature about many aspects of the thoracic spine is generally very limited. There is speculation about certain aspects, but little documentation. This chapter aims to be an introduction to the following management chapters by presenting some of the limited documented evidence that is available concerning the thoracic spine. It deals with the following aspects in the following sections:

- thoracic epidemiology
- thoracic pain
- thoracic anatomy
- thoracic biomechanics
- abnormal morphology.

## Thoracic epidemiology

Understanding of thoracic pain epidemiology is handicapped by two major drawbacks: the lack of good quality literature and the problem of definition of thoracic pain. The literature investigating the epidemiology of thoracic spine pain is very limited. In one of the few relevant population-based studies of 35- to 45-year-olds, 66% reported spine pain in the previous year, 15% reported pain in the upper back compared to 56% in the low back and 44% in the neck (Linton *et al.* 1998). This equates to a population prevalence of about 3% for upper back pain. Most other reports of thoracic pain prevalence are from clinical studies rather than population studies. Surveys of osteopath clinics have reported thoracic pain in 3 – 14% of all patients (Welch *et al.* 1995; Burton 1981; Hinkley and Drysdale 1995), chiropractors have reported thoracic/chest pain in 7% of over a thousand patients (Pedersen 1994), and surveys of nearly three thousand musculoskeletal patients seen by physiotherapists reported 77 (2.6%) thoracic pain patients in total (May 2003, 2004a, 2004b). This would mean between 5% and 17% (mean 10%) of all spinal problems are thoracic in origin. Only 2% of all intervertebral disc disease is said to occur in the thoracic spine (Kramer 1990).

Estimating the true prevalence of thoracic pain is confounded further by the definition of thoracic pain according to site. Pain in the upper thoracic region around the scapulae and upper chest may originate from the cervical as much as the thoracic spine. Has 'thoracic pain' included scapular pain that is referred from the neck? Would it include pain possibly referred from the thorax to the shoulder, xiphisternum, and buttock and anterior thigh (Singer and Edmondston 2000)? The occurrence of thoracic region pain, initially regarded as originating from cardiac, pancreatic, renal or some other visceral disease but after long delays attributed to the thoracic spine, is not uncommon (Bechgaard 1981; Whitcomb *et al.* 1995; Grieve 1994). The example of the misdiagnosis involved in chest pain is considered in Chapter 2 – initially interpreted as cardiac, it is frequently found to be musculoskeletal in origin.

Despite this lack of clarity and although the figures provide only estimates of prevalence, it seems fair to conclude that this region is troubled far less than the cervical and lumbar areas by pain. It can only be speculated why this is so – perhaps it is the stability provided by the rib cage, the perpetual rhythmic motion provided by respiration, or the greater stability of the intervertebral discs. Even less is known about other aspects of thoracic pain epidemiology, such as recurrence rates.

## Thoracic pain

As mentioned above, there is ample room for confusion between symptoms that emanate from the thoracic and cervical spines. Several studies have indicated that pain around the scapular and shoulder region commonly arise from cervical discogenic or zygapophyseal joint disorders (Cloward 1959; Smith 1959; Whitecloud and Seago 1987; Grubb and Kelly 2000; Dwyer *et al.* 1990; Aprill *et al.*1990). However, stimulation of thoracic structures has also caused pain in the chest and scapular region (Bogduk 2002c). Any combination of neck and scapular or shoulder pain is probably referred pain from cervical structures.

Innervation of thoracic structures is assumed to be similar to other areas of the spine, and thus the same structures can potentially be sources of pain: vertebrae, dura mater, intervertebral discs, longitudinal ligaments, muscles, zygapophyseal and rib joints (Bogduk 2002c; Valencia 1994). Some of these structures have been formally

investigated with pain provocation or pain relief studies, proving they can be genuine sources of pain. Injections of hypertonic saline into thoracic muscles and ligaments have produced local and referred pain in volunteers (Kellgren 1939; Feinstein *et al.* 1954). Local pain has been evoked by distension of zygapophyseal joint capsules (Dreyfuss *et al.* 1994a) and by discography (Wood *et al.* 1999) in normal asymptomatic volunteers. Single and double zygapophyseal joint injections have been used to provide patients with pain relief (Dreyfuss *et al.* 1994c; Wilson 1987; Manchikanti *et al.* 2002c) and discography has reproduced patients' thoracic pain (Wood *et al.* 1999; Schellhas *et al.* 1994).

Morphologically internally deranged discs with annular tears, intrinsic degeneration and Schorl's nodes were much more likely to be painful than normal-appearing control levels with discography (Schellhas *et al.* 1994). These injections often reproduced concordant pain posteriorly, laterally or anteriorly in the chest wall. Although sometimes the location of pain matched the anatomic location of annular tears around the disc circumference, the pain provoked was highly variable and not predictable. Disc herniations have been reported to produce 'band-like' chest pain commonly, and interscapular, epigastric and lower extremity pains less commonly (Brown *et al.* 1992).

Patterns of somatic referred pain from these structures vary between studies, and there is considerable overlap between different motion segments (Bogduk 2002c). Although stimulation at higher segmental levels tends to produce pain higher in the thorax, this was not always the case. Pain tended to refer in bands around the lateral and anterior chest wall. Upper thoracic segments produced pain around the scapular and upper chest wall, middle thoracic segments below the scapular and around the ribs, and lower thoracic segments into the lumbar area, pelvis and below the ribs. Given the overlap and vagueness of pain patterns, identification of the segmental level by the site of pain is unlikely.

Despite summaries of thoracic radicular pain presenting with severe, band-like symptoms around the chest wall (Grieve 1994; Mellion and Ladeira 2001), no scientific study investigating the differentiation between somatic and radicular thoracic pain could be located.

As mentioned in the previous section, differentiation between thoracic pain of visceral or somatic origin is far from clear. Generally, although

not always, patients with visceral disease have accompanying non-musculoskeletal symptoms (Sparkes 2004). For instance, complaints of abdominal discomforts, bowel or bladder alterations, poor general health, symptoms affected by food, nausea or vomiting might highlight a visceral cause of thoracic pain. The questions in Table 26.1 have been found to be helpful in identifying a musculoskeletal cause of abdominal symptoms.

**Table 26.1 Indicators of musculoskeletal cause of abdominal pain**

*'Yes' response by patient:*

- Does coughing, sneezing or deep breathing exacerbate symptoms?
- Do activities such as bending, twisting, sitting, lifting or turning over in bed exacerbate symptoms?
- Was onset associated with a fall, trauma or lifting incident?

*'No' response by patient:*

- Does eating certain foods make the pain worse?
- Did your bowel habit change with onset of symptoms?
- Any weight change associated with onset of symptoms?

Source: King 1998 in Sparkes 2004.

## Thoracic anatomy

For detailed descriptions of thoracic clinical anatomy, see other texts (for example, Singer and Goh 2000; Valencia 1994; Singer 1994; Mercer 2004a; Singer *et al.* 2004; Edmondston 2004); all that will be presented in this section is a brief résumé of key features. The shape and size of the twelve thoracic vertebrae and intervening intervertebral discs vary considerably from T1 – T12. Upper thoracic vertebrae are similar to cervical and lower thoracic similar to lumbar (White and Panjabi 1978a). Most notably the size of the vertebral body increases substantially with more caudal segments, reflecting the increase in body load. With the posterior components of the vertebrae there are variations in shape, orientation and structure. Furthermore, the thoracic vertebrae have additional articulations at the costovertebral and costotrasverse joints with the ribs. The spine with the ribs and sternum has an important protective role for the heart, lungs and major blood vessels. The thoracic kyphosis is a primary curve, its shape determined by the vertebral bodies and discs, which are thicker posteriorly; this is most marked in the mid-thoracic region (Singer and Goh 2000).

The spinal canal of the thoracic spine is relatively narrow, with the narrowest section generally found in the mid-thoracic region (Kramer 1990). The intervertebral foramina are relatively large and lie at the level of the vertebral bodies, rather than at the level of the intervertebral discs as in the cervical and lumbar spine. This means that nerve root impingement is rarer here than in the other regions (Kramer 1990).

Very little is known about the detailed morphology of thoracic intervertebral discs (Mercer 2004a). Preliminary work suggests a structure similar to cervical discs down to T9 – T10 (Mercer 2004a); with a thicker anterior than posterior annulus fibrosus, a posterolateral deficiency in the annulus fibrosus and the presence of uncovertebral clefts. Clefts and fissures are extremely common. Below T10 the intervertebral disc begins to look more like a typical lumbar disc.

## Thoracic biomechanics

Serious study of movement and biomechanics of the thoracic spine is very limited, consisting mostly of cadaver studies or unvalidated research techniques (Mercer 2004b). The ribcage and the complex interaction between spine and ribcage present significant methodological problems in the study of thoracic biomechanics and movements (Edmondston 2004). Most reports of range of movement appear to be based on one early cadaver study, and whilst coupled movements seem to occur, report of coupling is inconsistent and contradictory (Mercer 2004b). Biomechanical models of the thoracic spine have been suggested (Lee 2002; Edmondston 2004), but at present there is insufficient evidence to support any particular pattern of coupling in the thoracic spine (Mercer 2004b). It is suggested that sagittal plane movements are a combination of sagittal rotation and translation, and lateral movements combine rotation and lateral flexion (Stokes 2000; Lee 2002).

The protective role of the thoracic spine for the chief organs means that stability and protection are gained at the expense of mobility. The presence of the ribs, the additional vertebral-rib joints, the configuration of the zygapophyseal joints and the spinous processes, and the fact that the thoracic intervertebral discs are thinner than in other regions are all factors that limit mobility. Movement of the trunk involves movements at multiple vertebral joints posteriorly, the ribs and the joints of the sternum anteriorly.

Just as the anatomy of the upper and lower thoracic spine differs, so do the biomechanics. It is suggested that the extent of coupling of movements is partly dependent on the slopes of the zygapophyseal facets (Valencia 1994). The obliquity varies from 60 degrees, nearly 90 degrees and nearly 0 degrees in the upper, middle and lower thoracic spine respectively. It is suggested that coupling is similar to cervical segments in the upper thoracic (rotation coupled with lateral flexion), but becomes weaker or changes in middle and lower segments (White and Panjabi 1978a). Range of movement is different also between the upper and lower thoracic spine.

Measuring the range of movement at the thoracic spine is technically difficult (Valencia 1994). Unlike the cervical and lumbar spines, where a plethora of range of movement analyses has been performed, the movement of the thoracic spine has been rarely investigated. Many reports of thoracic movement (White and Panjabi 1978a; Grieve 1991; Valencia 1994; Mercer 2004b) seem to be based on a single early cadaver study (White 1969), often taking the data from a slightly later review in which the authors estimate ranges based on previous work and their analysis (White and Panjabi 1978b). There is some inconsistency of reporting (Grieve 1991; Valencia 1994; Mercer 2004b), and even a chapter on thoracic biomechanics does not mention range of movement (Lee 2002).

There are, however, a few studies, using different techniques, *in vivo* and *in vitro*, that have been done, but which give different measurements (Edmondston 2004). These studies would suggest less than 5 degrees of sagittal movement at most segmental levels, with greater movement in the lowest thoracic segments. Between 4 and 8 degrees of lateral flexion have been reported at most levels, with possibly a greater range in the lowest segments. Rotation appears to be greatest in the upper thoracic spine, possibly 8 to 12 degrees at each segment, less in the mid-thoracic spine, and even less, perhaps less than 3 degrees, in the low thoracic region.

Clearly a certain amount of caution should be used in understanding thoracic movement from such a database. It could be suggested that on the whole sagittal plane mobility is very restricted in the thoracic spine compared to both the cervical and lumbar spine, except in the lowest two or three segments when range begins to equate with lumbar flexion and extension. Lateral flexion appears to be reasonably equally distributed across all segments of the spine, although

individually the range at each section is small. Rotation is varied, the cervical and mid- and upper thoracic spine being quite mobile and the lowest three or four segments of the thoracic and the lumbar spine being much more restricted (White and Panjabi 1978a).

## Abnormal morphology

The thoracic spine is prone to the usual range of degenerative and maturation changes that affect other areas of the spine, plus some that principally affect this region. With age the thoracic kyphosis tends to increase, a process that is accentuated by reduced physical activity, postural habit and female gender (Singer 2004). A number of specific pathologies, some asymptomatic, can exacerbate this; these include ankylosing spondylitis, Scheuermann's disease, diffuse idiopathic skeletal hyperostosis, Paget's disease and osteoporosis (Sparkes 2004). An exaggerated thoracic kyphosis can have repercussions on the cervical spine and respiratory function (Singer 2004).

Intradiscal pressures are high in the thoracic spine in standing and when holding a weight, although these are not so affected by flexion as disc pressures in the lumbar spine (Polga *et al.* 2004). High disc pressure leads to intervertebral disc herniations into the vertebral bodies, known as Schmorl's nodes, which are most common in the thoracic spine (Hilton *et al.* 1976; Hilton and Ball 1984); their clinical significance is unknown. Extensive degenerative changes occur in the disc and zygapophyseal joints, including the development of osteophytes especially in the lower thoracic area (Grieve 1994; Singer 2004; Nathan 1962), but these changes are largely asymptomatic.

A number of degenerative conditions especially affect the thoracic spine; these include Schmorl's nodes and Scheuermann's disease, mentioned in Chapter 27. Osteoporotic fractures are probably most common in the mid- and lower thoracic spine (Stokes 2000; Bennell and Larsen 2004). Diffuse idiopathic skeletal hyperstosis (DISH) is a condition that results in the ossification of spinal ligaments, particularly in the thoracic spine (Singer 2000, 2004). It is rare below 40 years of age and may also involve enthesophytes or bony spurs in ligaments at other joints. The condition is frequently asymptomatic and is an incidental finding on x-ray. If it is symptomatic there may be thoracic back pain and morning stiffness, which may be accompanied by loss of movement. Radiographic features of the disease have

been found in 25% of males and 15% of females over the age of 50 (Weinfeld *et al.* 1997). The limitation of thoracic extension may affect treatment of cervical or lumbar problems. Individuals with DISH may on mechanical evaluation appear to have a mechanical dysfunction – with the long period of time this may have been present, resolution is unlikely.

Paget's disease is a disorder that leads to extra bone activity principally affecting the spine, skull, pelvis and femurs in middle-aged and older patients (Altman *et al.* 1987; Collins 1956). The lumbar and thoracic spines are most commonly affected. There may be dull pain, deformity and increased thoracic kyphosis, but it is generally asymptomatic. Reports of Tietze's syndrome are limited and generally old; incidence is likely to be rare (Gregory *et al.* 2002). The disease is said to produce anterior chest wall pain, most commonly at the second and third costochondral junctions, and usually in young people (Gregory *et al.* 2002). Swelling may be present; the aetiology is thought to be inflammatory, and natural history is self-limiting.

As in other regions of the spine, disc herniations are common in the asymptomatic population, with prevalence rates possibly as high as 37% (Wood *et al.* 1995, 1997). These morphological abnormalities can be present and not be associated with pain, and equally exist over time without change in size and remain asymptomatic (Wood *et al.* 1997). Equally, as in other regions of the spine, when disc herniations are the cause of symptoms many respond to conservative care, even of a non-specific nature (Brown *et al.* 1992).

Structural deformity of scoliosis in the frontal plane occurs more frequently in the thoracic spine than elsewhere. The reasons for this are not fully known, and equally the aetiology is not fully understood although a genetic component is known to exist (Stokes 2000). It usually occurs as a thoracic or thoracolumbar curve convex to the right and a lumbar curve convex to the left (Saada *et al.* 2000). Such spinal curve anomalies are mostly idiopathic (70%) and related to a growth disturbance during the growth period; less common is congenital scoliosis, which occurs through a defect in the vertebral column. Idiopathic scoliosis develops from childhood to adolescence and is said to affect 2 – 3% of 10-to-16-year-olds (Singer 2000), with female dominance in the most severely affected. Four main curve patterns have been described: thoracic, lumbar, thoracolumbar and double major curves – each is said to have its own characteristics and

end-point. *It is important to remember that minor or even moderate scoliosis maybe an incidental finding that is unconnected to symptoms* (Dieck *et al.* 1985).

Conservative treatment of scoliosis consists mostly of bracing, exercises and electrical stimulation of trunk muscles, but efficacy is uncertain and results may be more dependent on lack of progression of deformity than the treatment itself (Findlay and Eisenstein 2000; Stokes 2000). A detailed consideration of this topic is beyond the scope of this book.

## Conclusions

This chapter has aimed to be an introduction to the next few chapters that are about the management of thoracic spine problems. The literature on the epidemiology, pain, anatomy and biomechanics of the region has been briefly explored. The limited evidence base and generally inadequate quality of what is available should lead to caution about drawing firm conclusions on many topics related to the thoracic spine.

# 27: Classification of Thoracic Spine Problems

## Introduction

The classification of thoracic pain will be similar to that of cervical pain syndromes (see Chapter 5). Patients with cases of serious spinal pathology must first be excluded (see Chapter 8). Remaining patients will be examined as described in Chapter 28. Most will demonstrate a mechanical response and be classified as derangement, dysfunction or postural syndrome. A few, within three to five sessions of full exploration of symptomatic and mechanical responses, will prove unclassifiable and might be placed in one of the 'Other' categories considered below. If at any time concern is generated by an unusual presentation or atypical responses to mechanical evaluation, re-assessment or referral for further investigations should be considered. This chapter outlines the classification process in the following sections:

- serious thoracic spinal pathology
- mechanical syndromes
- other categories
- Scheuermann's disease.

## Serious thoracic spinal pathology

Thoracic spine pain is often found in lists of 'red flags' indicating serious spinal pathology (CSAG 1994; Waddell 2004). Not all pain originating in the thoracic spine is serious, and much of it is normal mechanical pain. However, as there is a much lower prevalence rate of thoracic pain compared to lumbar and cervical, proportionately there is a higher incidence of serious pathology in this region. A range of serious pathologies can occur in the thoracic spine, some more commonly in this region than at the cervical or lumbar regions. In a survey that included thirty-three patients deemed to have thoracic pain and who underwent a mechanical evaluation, two (6%) were thought to have serious spinal pathology compared to less than 2% and 0% in the lumbar and cervical regions (May 2004b). It is essential that Chapter 8 is read in full to help in the identification of serious pathologies, and Table 8.1 is consulted as a checklist of possible 'red

flags' indicating serious pathologies. *As these conditions have been described, their descriptions are not repeated in this chapter.*

However, the following points should be noted: tumours occur more commonly in the thoracic spine than other spinal regions (Table 8.1). Although thoracic disc herniations are uncommon, a higher proportion of them appear to lead to spinal cord involvement than in other regions (Table 8.2). The thoracolumbar junction is reported as the most common site for non-osteoporosis-related spinal column fractures (Huler 1997); the earliest osteoporotic fractures are typically seen in the thoracic spine (Kanis and Pitt 1992). It has been suggested that as mechanical T1 lesions are so rare, clinicians should always be aware of non-mechanical and serious pathology (Mellion and Ladeira 2001). Although extremely rare, spinal infection occurs nearly as commonly in the thoracic as in the lumbar spine (Table 8.1). *If any serious spinal pathology is suspected or there is concern about an atypical response to the mechanical evaluation, such patients must be referred for further investigation.*

It should also be noted that visceral conditions may refer pain to the chest wall and be mistaken for musculoskeletal disease, although the reverse is probably more common. Visceral conditions will not respond to mechanical evaluation and there may be clues in the history, such as pain on exertion with a heart condition.

## Mechanical syndromes

Derangement, dysfunction and postural syndromes are all found in the thoracic spine. Their presentations are more fully described in Chapter 6, and are only briefly outlined here. Derangement syndrome is most common and is characterised by a varied clinical presentation and typical responses to loading strategies. Pain may be central or symmetrical or radiate laterally around the chest wall, or even present with patches of pain on the anterior chest wall. Worsening or peripheralisation of symptoms away from the spine may occur in response to certain postures and movements. A decrease, abolition or centralisation of symptoms and the restoration of normal movement occurs in response to therapeutic loading strategies. Most commonly therapeutic loading strategies use extension or lateral principles of treatment, the latter using rotation forces, and management is described in Chapter 30.

Dysfunction presents in a similar way to other areas of the spine. Pain is always intermittent and produced by consistent end-range loading, with always some limitation to the range of movement. There is commonly some preceding history, such as a previous derangement, trauma, Scheuermann's disease or long-term poor postural habits, and there is never any rapid change to symptoms. Typically therapeutic management consists of extension or lateral principles of treatment (rotation forces) and is described in Chapter 31.

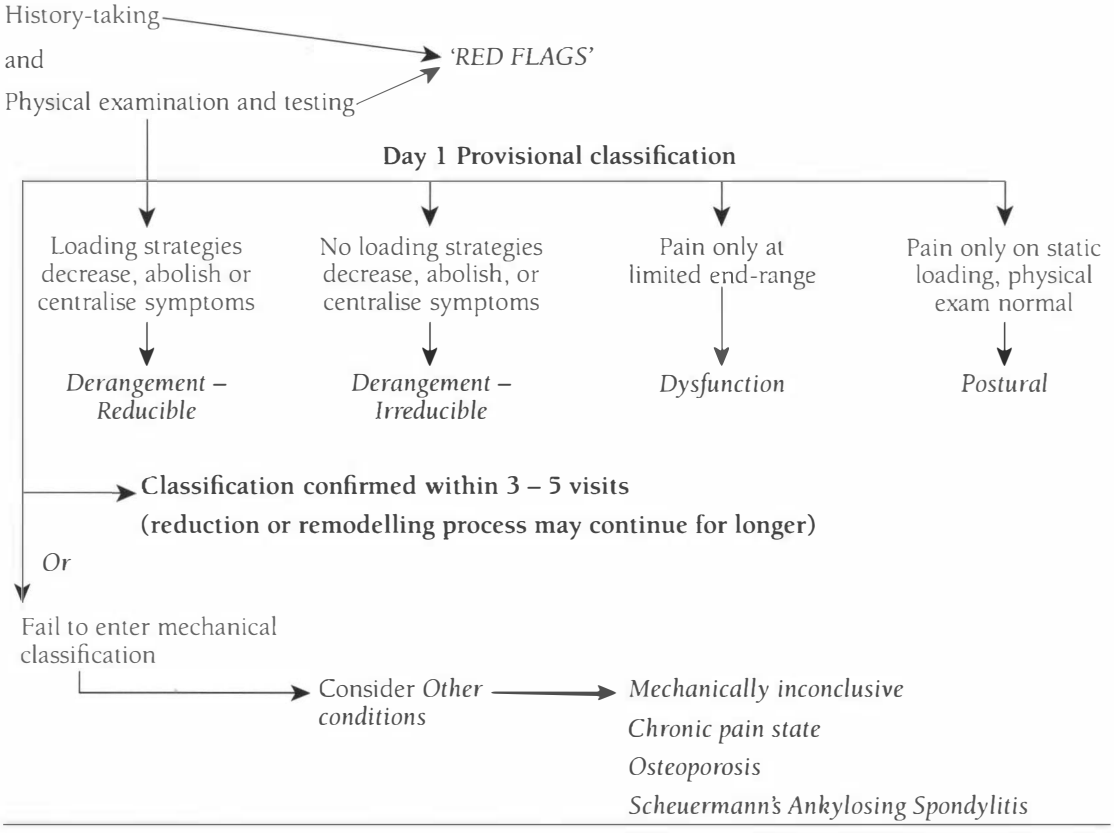
Patients with postural syndrome of the thoracic spine rarely present for treatment, but its clinical presentation is the same as other areas of the spine (Chapter 6). Patients may report concurrent lumbar, thoracic and cervical pain of postural origin. Pain from postural syndrome is only produced by sustained end-range loading, usually sitting. As soon as the poor sitting posture is corrected or changed, the pain goes and physical examination is entirely normal. Management is described in Chapter 31.

## Other categories

As in other spinal regions, once serious pathologies have been excluded, an extended mechanical evaluation within three to five sessions will demonstrate one of the mechanical syndromes in the majority of patients. Failure to demonstrate a mechanical response might lead to the consideration of other possible conditions. *For these 'Other' categories to be included, however, it is essential that force progressions and force alternatives are fully but safely explored to exclude a mechanical response.* In some instances, such as possible osteoporosis, force progressions are contraindicated.

'Other' categories include osteoporosis, ankylosing spondylitis, Scheuermann's disease, mechanically inconclusive, or chronic pain state. Osteoporosis and ankylosing spondylitis are reviewed in Chapter 8 and elsewhere (McKenzie and May 2003, Chapter 12). These are conditions whose management may involve physiotherapy, but they require special consideration and appropriate diagnosis. The description of the latter two is as in Chapter 9.

**Figure 27.1 Classification algorithm for thoracic spine**




## Scheuermann's disease

This is a poorly defined clinical entity with an exaggerated thoracic kyphosis (Saada *et al.* 2000). Scheuermann's disease is a cause of structural kyphosis of the thoracic or thoracolumbar spine that is confirmed radiographically by the presence of anterior wedging of at least three adjacent vertebrae of five degrees or more, osteophytes and Schmorl's nodes (Lowe 1990; Singer 2000, 2004). Its aetiology is unknown, but a genetic component is suggested (Graat *et al.* 2002). Estimates of its prevalence vary between 0.4% and 10% or more (Singer 2000; Saada *et al.* 2000; Moquin *et al.* 2003).

Onset usually occurs around puberty. There is poor posture and pain with certain activities (Lowe 1990; Moquin *et al.* 2003); however, pain is not always present (Graat *et al.* 2002). The natural history is unclear; it can involve progressive structural kyphosis during the growth period. The back pain tends to disappear once the individual reaches skeletal maturity. Once established, on examination there

is usually an angular kyphosis accompanied by a compensatory hyper-lordosis of the lumbar spine. The kyphosis is fixed and remains when the patient extends; in some patients this is accompanied by a scoliosis. Thus Scheuermann's disease may be a relevant consideration in adolescents with poor posture and exaggerated thoracic kyphosis. In adults Scheuermann's disease may be the cause of structural kyphosis, but be asymptomatic. The role that postural correction and exercise might have in this disease has not been properly evaluated. Surgery has been proposed to prevent progression of the deformity (Lemire *et al.* 1996; Moquin *et al.* 2003).

## Conclusions

This chapter has described the classification system used in the thoracic spine. The first duty of the clinician is to detect patients who have serious spinal pathology. A series of 'red flags' might indicate the presence of serious pathologies, and any such patients should be referred for further investigation – these are detailed in Chapter 8. It should be remembered also that thoracic pain may result from visceral disorders, although ascribing visceral disease to thoracic musculoskeletal conditions seems more commonly documented. All other patients are given a mechanical evaluation, and most patients will be classified as derangement, plus fewer with dysfunction, and fewer still with postural syndrome. Suspicion of osteoporosis should be maintained if the patient is elderly and female or ankylosing spondylitis in young men when the response is atypical. If following a detailed and prolonged mechanical evaluation over three to five sessions it is not possible to classify the patient in one of the mechanical syndromes, one of the 'Other' categories might be considered. These categories include mechanically inconclusive, chronic pain state, osteoporosis, ankylosing spondylitis and Scheuermann's disease.

# 28: Thoracic Spine Assessment



## Introduction

Most components of the interview with patients with thoracic spine problems are the same as that conducted when assessing cervical spine patients; therefore the history-taking is not presented in detail. A few points distinct to the thoracic spine are addressed in this chapter, but the detail of the history-taking is the same as that covered in Chapter 10 (cervical history). The physical examination involves the use of movements that are more specific to the thoracic spine. It is also important with symptoms that are located around the scapulae to distinguish between pain that is most influenced by cervical or by thoracic movements. The physical examination, including differential diagnosis, is considered in more depth.

Sections in this chapter are as follows:

- history
    - differentiating cervical and thoracic symptoms
- physical examination
    - flexion
    - extension
    - rotation
    - repeated movements
    - erect sitting flexion
    - erect sitting extension
    - erect sitting rotation
    - extension in lying
    - prone
    - supine
- static mechanical evaluation
    - role of palpation
- conclusions following the examination
    - further testing.

## History

As mentioned in the introduction, most items of history-taking are the same as those used for neck pain patients, and much of the detail in the chapter on cervical history is relevant here. However, some items that are relevant specifically to the thoracic spine need further comment.

In the thoracic spine the patient's age may alert one to the possibility of Scheuermann's disease, which affects adolescents, whilst osteoporosis is a consideration in older patients, especially older women. These and other specific conditions that are relevant to the thoracic spine are considered in the chapters on serious spinal pathology (Chapter 8) and other diagnostic considerations (Chapter 9).

If clinicians wish to use an established functional disability questionnaire, as we are not aware of one developed specifically for the thoracic spine it is probably best to use one developed for low back pain rather than neck pain, as generally the functional questions are more relevant.

In non-specific thoracic spine problems symptoms are generally felt around the trunk. The narrowness of the thoracic spinal canal makes a spinal lesion such as a disc herniation at risk of causing an upper motor neuron lesion (Kramer 1990). This may produce lower limb signs and symptoms with minimal thoracic involvement. It is important to be aware of such serious spinal pathologies in the thoracic spine as in other regions; these are considered in more depth in Chapter 8.

Two other important considerations regarding symptoms are their distribution and site. Regarding distribution, a distinction should be made between symptoms that are central or symmetrical and those that are unilateral or asymmetrical. As in other spinal regions, these different types of distribution ***may*** require different management strategies.

Centralisation and peripheralisation do occur in the thoracic spine, but symptoms most commonly spread out from the spine laterally, with the pain referred in bands around the trunk. Thoracic pain syndromes can refer pain to the front of the chest, and also they may present as isolated patches of pain over the trunk – the symmetrical or asymmetrical distribution of symptoms should still be considered.

Thoracic presentation in isolated patches on the trunk is probably the reason why spinal problems have been mistaken for visceral disease in the past. Thus, when monitoring symptom response in thoracic spine problems, the most distal symptoms are usually those that are felt most anteriorly or laterally and centralisation is noted when symptoms move toward the spine.

In the thoracic spine onset may either be for no apparent reason or related to trauma, such as whiplash or sudden twisting movements, or sustained positions. Aggravating and relieving factors that may affect symptoms are similar to the lumbar spine: bending, sitting, standing and lying; rotating or twisting the trunk. Sleep is commonly disturbed, and activities that involve thoracic activity, such as laughing, coughing or deep breathing, are frequently painful.

Pain in the thoracolumbar region may be either from the upper lumbar or lower thoracic spines; however, both respond to the same repeated movements. Pain in the cervicothoracic region is more likely to originate from the cervical spine than the thoracic, but again the same repeated movements would produce similar responses wherever the origin of symptoms. In effect, these 'border' regions should be considered to be part of the functional lumbar and cervical spine respectively, and the examination proceeds as described for those regions with some minor adaptations to increase forces in the thoracic region. If symptoms and movement are being improved with, for instance,extension of the cervical and upper thoracic spines, it is not particularly important to pinpoint the segmental level that is causing the disturbance. However, sometimes it may be important to differentiate the origin of cervical and thoracic symptoms.

### Differentiating cervical and thoracic symptoms

Since the classic study by Cloward (1959) in which he stimulated cervical discs at surgery and reproduced interscapular pain, several studies have confirmed that pain around the scapulae is commonly caused by cervical discogenic disorders (Roth 1976; Connor and Darden 1993; Parfenchuck and Janssen 1994; Schellhas *et al.* 1996); it can also result from stimulation of cervical zygapophyseal joints (Dwyer *et al.* 1990; Aprill *et al.* 1990). Thoracic disc disease, at least of a serious nature, is reported as being uncommon at the upper three or four segments (Arce and Dohrmann 1985; Singounas *et al.* 1992). Pain provocation studies involving thoracic zygapophyseal joints suggest limited patterns of referred pain two or three segments inferior

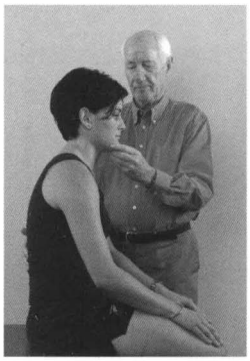
to the joint injected (Dreyfuss *et al.* 1994a). Given this background, pain above a line drawn at the inferior border of the scapulae should be judged to be of cervical origin until proven otherwise (McKenzie 1990). Any associated shoulder and arm pain and any sensory abnormalities in the arm are much more likely to be associated with neck problems.

Other clues to differentiate symptoms of cervical or thoracic origin can be found in the aggravating factors. If neck or arm movements are implicated the former is likely to be the origin; if coughing, deep breathing, laughing or trunk rotation is implicated the latter is likely to be the origin.

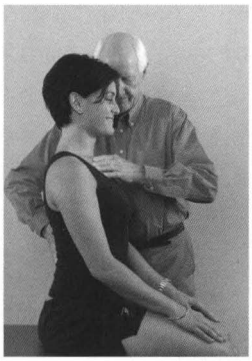
If symptom response to initial movements is uncertain and further clarification is necessary, the cervical spine should be examined whilst the thoracic spine is immobilised. This can be done with the patient adopting a slumped sitting posture with the head protruding and the thoracic spine and lumbar spines flexed. It may be necessary for the clinician to restrain or stabilise the thoracic spine in some cases. The location and intensity of pre-test pain should be established once the patient is in this position, and then cervical test movements are performed. If symptom response correlates with cervical movement – for instance worse with flexion, better with extension – then this is the likely source. If cervical movements do not particularly affect symptoms, the thoracic spine is the more likely source. If symptoms gradually worsen whichever way the cervical spine is moving, the thoracic spine, being placed in sustained flexion, may be the source.

## Physical examination

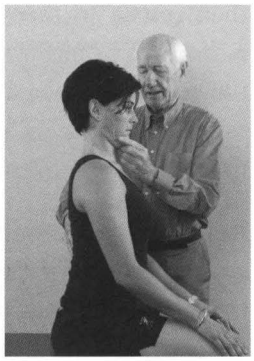
The principles of assessment in the thoracic spine are just the same as those applied to the cervical and lumbar spines; that is, single movements are performed to examine range, then repeated movements are performed and the symptoms and mechanical responses noted. Movements that centralise, abolish or decrease symptoms are indicated; movements that peripheralise or increase symptoms are temporarily avoided. As in other regions of the spine, clues to directional preference may be gained during the history-taking. Movements examined are flexion, extension and rotation in erect sitting. Extension can also be examined in prone or supine; pre-test symptoms are always noted prior to repeated movements.



86



87



88

*Photos 86, 87, 88: From slumped position (86), gentle pressure on the spine and sternum restores the lordosis (87). Gentle pressure at the chin and thoracic spine corrects the head posture (88). Symptom response is monitored before and after.*

The posture should be examined. The normal thoracic spine is kyphotic, but an increase should be noted. A protruded forward head posture is often associated with increased thoracic kyphosis, especially around the cervicothoracic junction area. Scoliosis may be present but not relevant to the symptoms (Dieck *et al.* 1985). The relevance or lack of relevance of any postures is best tested by changing the posture and noting symptom response. Thus, if the patient is sitting with increased thoracic kyphosis and protruded head, symptoms are noted, posture correction is performed and any symptom change is recorded (Procedure 2).

Movements are examined in the following order:

### Flexion

The patient is instructed to 'bend their trunk forward, bringing their head and shoulders towards their knees and then return to the starting position'. Any loss of range of movement is gauged as major, moderate or minor and any pain with the movement is noted.

### Extension

Sitting upright on the treatment table the patient is instructed to 'stretch the head, neck and trunk backwards as far as possible and then return to the starting position'. Any loss of range of movement is gauged as major, moderate or minor and any pain with the movement is noted.

### Rotation

The patient sits upright on the treatment table with hands clasped across the sternum and the elbows and hands at chest height. The patient is instructed to 'turn to the right (left), keeping the hands clasped together, pointing the elbow as far behind as possible, and

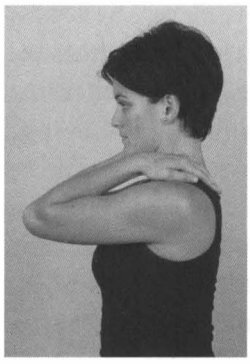
then return to the starting position'. Ensure that true rotation is performed by ensuring the hands remain on the sternum, rather than the patient simply sliding their arms around the trunk. Any loss of range of movement is gauged as major, moderate or minor and any pain with the movement is noted.

### Repeated movements

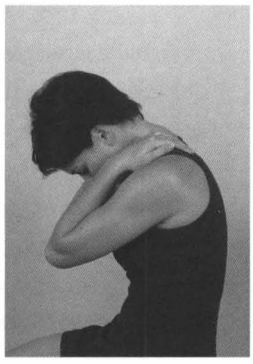
The repeated movement part of the physical examination provides the most useful information on symptom response and is the ultimate guide in identifying the management strategy to be applied (McKenzie 1981, 1990). A decrease, abolition or centralisation of pain is a reliable indicator of which movement should be chosen to reduce mechanical deformation. An increase or peripheralisation of pain is just as reliable to indicate which movements should be avoided. This, the cumulative effect of the movement, provides the most important detail concerning the patient's symptomatic response – that is, whether they are worse, no worse, better, no better or the pain has centralised or peripheralised. These responses provide the clearest indication for the appropriate management strategy. The role of repeated movements is discussed more fully in Chapter 11, and the terminology to record symptom responses is described in Chapter 12.

### Erect sitting flexion

The intensity and location of existing symptoms are noted, in particular the location of the most distal symptoms. The patient sits upright on the treatment table with hands over the shoulders to apply overpressure. The patient is instructed to slump so that the spine, from the neck to the sacrum, is in a fully flexed position. On reaching maximal flexion the patient returns to upright erect sitting. The effects of performing the movement once are recorded. The test movement should be repeated ten to fifteen times, or enough times to influence the symptoms, with overpressure being applied if the initial active movements have no effect. Symptom response is noted during the repeated movements, and most importantly a minute or so after a cycle of repeated movements.



89

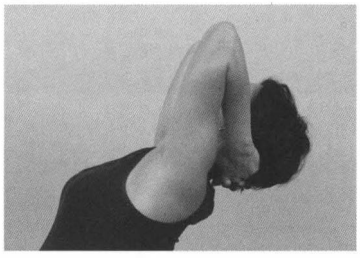


90

*Photos 89, 90: Flexion – overpressure can be applied through the upper thoracic transverse processes.*

### Erect sitting extension

*Photo 91: Extension.*



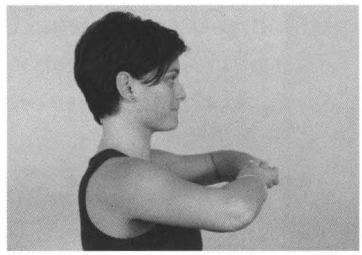
91

The intensity and location of existing symptoms are noted, in particular the location of the most distal symptoms. The patient sits upright on the treatment table with hands clasped behind the neck. The patient is instructed to arch backwards to extend the trunk as far as possible and point the elbows towards the ceiling. On reaching maximal extension, the patient returns to upright erect sitting. The effects of performing the movement once are recorded. The test movement should be repeated ten to fifteen times, or enough times to influence the symptoms. Symptom response is noted during the repeated movements, and most importantly a minute or so after a cycle of repeated movements. Sometimes overpressure applied by the clinician is necessary to generate the symptom response.

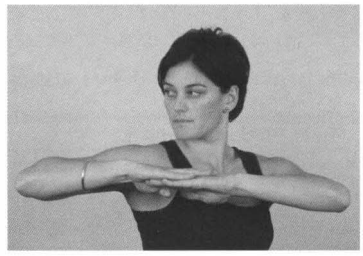
### Erect sitting rotation

The intensity and location of existing symptoms are noted, in particular the location of the most distal symptoms. The patient sits upright on the treatment table with hands clasped across the sternum and the elbows and hands at chest height. The patient is instructed to turn to the right (left), keeping the hands clasped over the sternum, and point the elbow as far behind them as possible. Ensure that true rotation is performed by ensuring the hands remain on the sternum, rather than the patient simply sliding their arms around the trunk. On reaching maximal rotation the patient returns to upright erect sitting. The effects of performing the movement once are recorded.

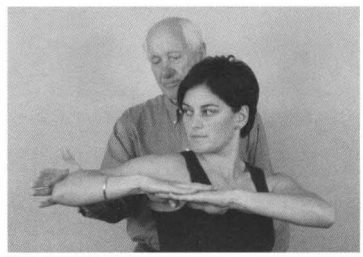
*Photos 92, 93, 94: Rotation overpressure is applied by increasing the speed of the manoeuvre, for instance by getting the patient to strike the clinician's hand with the elbow.*



92



93



94

The test movement should be repeated ten to fifteen times, or enough times to influence the symptoms. As repetitions are performed the patient is instructed to move further into rotation; this is best done by rotating swiftly and vigorously as if striking an object behind with the elbow. Symptom response is noted during the repeated movements, and most importantly a minute or so after a cycle of repeated movements.

### Extension in lying

Further testing can be performed in an unloaded posture if information from the previous test movements has been insufficient. Extension should be performed in prone as for the lumbar spine and in supine as for the cervical spine. The former affects predominantly lower thoracic segments, whilst the latter affects upper thoracic segments.

### Prone

The intensity and location of existing symptoms are noted, in particular the location of the most distal symptoms. The patient lies prone on the treatment table with hands under the shoulders as for the traditional extension in lying exercise. The patient is instructed to straighten the arms and lift their upper body whilst the lower half, pelvis down, remains on the table. Upon fully extending the elbows, the patient returns to prone lying. The effects of performing the movement once are recorded. The test movement should be repeated ten to fifteen times, or enough times to influence the symptoms. With repetition the patient must fully extend the elbows and let the trunk sag from scapulae region to pelvis, and push the chest forward. These additions should ensure maximum thoracic extension is achieved. Symptom response is noted during the repeated movements, and most importantly a minute or so after a cycle of repeated movements.

### Supine

The intensity and location of existing symptoms are noted, in particular the location of the most distal symptoms. The patient lies supine on the treatment table with the head and neck unsupported to the level of the fourth thoracic vertebra. They lower the head and neck until the cervicothoracic region is fully extended. Upon gaining maximum extension, the patient returns to a neutral position using their hands for support. The effects of performing the movement once are recorded. The test movement should be repeated ten to fifteen times, or enough times to influence the symptoms, with the repetitions ensuring that maximum range is gained. Symptom response is noted during the repeated movements, and most importantly a minute or so after a cycle of repeated movements.

## Static mechanical evaluation

On occasions repeated movements and overpressures are not the appropriate mechanical forces to provoke or influence symptoms, especially in postural syndrome. In such patients sustained postural loading is necessary to provoke symptoms. Most commonly this is due to sustained thoracic flexion, as in the slumped sitting posture; symptoms may also be present in the cervical and lumbar regions. In such cases postural correction and the resumption of an erect sitting posture will abolish symptoms. This is best tested by having the patient maintain a relaxed, unsupported sitting posture during the history-taking. Usually such patients adopt a slumped posture, and it will be sustained for fifteen to twenty minutes whilst the patient is being interviewed. At the end of the interview, the procedure of posture correction is performed and symptoms present before and after correction are recorded (Procedure 2).

For testing sustained positions the same procedure is adopted for different postures. Intensity and location of pain is noted prior to the adoption of the position; the posture is sustained for at least three minutes. Sometimes a longer period will be required; symptom response is noted during the sustained posture ***and*** on return to erect sitting posture. It is important to note that responses to sustained postures are different for different mechanical syndromes. In derangement syndrome flexion may sometimes give temporary relief despite the directional preference being for extension, but more typically there is an increase or peripheralisation of symptoms. In patients

with thoracic extension dysfunction, sustained flexion will have no effect, with symptoms only being reported on end-range extension. Sustained loading in flexion, given the appropriate time period, will generate symptoms in patients with postural syndrome.

Sustained postures are as described in the chapter on procedures, and may include the following. The sleeping position may have to be examined if this is one of the aggravating factors.

- sitting sustained flexion
- extension in lying prone
- extension in lying supine
- rotation in sitting.

### Role of palpation

The ability of clinicians to agree on findings obtained from palpation of motion abnormalities or segmental levels has not been substantiated in the lumbar spine (McKenzie and May 2003) nor in the cervical spine (see Table 9.1). Inter-practitioner agreement on the presence of a finding actually constitutes a test of internal validity and is not simply a measurement of reliability only (Nansel *et al.* 1989). If inter-rater reliability is poor the clinical phenomenon may not exist, and certainly not in any consistent way that gives it clinical value. Little similar work has been done that specifically pertains to the thoracic spine, but extrapolation from the other areas and the little work that has been done suggests no better levels of inter-rater reliability for motion palpation (Christensen *et al.* 2002; Horneij *et al.* 2002). Consequently, palpation of thoracic motion segments or costotransverse joints should not be routinely used as part of the mechanical evaluation.

## Conclusions following the examination

It should be possible at the end of the examination to reach a provisional diagnosis. If derangement is present symptoms will have centralised, abolished or decreased in response to repeated movements. Alternatively, repeated movements may produce a clear positive mechanical response, such as improvement in range of movement. A treatment principle of extension or lateral (rotation forces) will have been selected to reduce the derangement, and posture correction

will also have been advocated. If dysfunction is present an end-range movement will have consistently produced symptoms, which will have swiftly faded once the repeated movements have ceased. A treatment principle of extension, lateral or flexion will have been selected to reproduce symptoms and remodel the dysfunction. If postural syndrome is present, pain will have come on whilst the patient was in slumped sitting and will have been abolished by posture correction; there will be no other findings on physical examination, all movements being full and pain-free.

### Further testing

If a provisional diagnosis cannot be made because there is an absence of any of the above mechanical or symptom responses, further testing may be necessary. This may take the form of repeated movements over the following few days, and/or force progressions during the first or the second sessions to determine if either of these options produces a clearer response. Force progressions to be used:

- extension with patient overpressure – using edge of chair (Procedure 1a)
- extension with clinician overpressure (Procedure 1b)
- extension mobilisation (Procedure 1c)
- extension manipulation (Procedure 1d).

If symptoms are asymmetrical or unilateral and either worsen in response to extension forces or show no change after force progressions, lateral forces should be used:

- rotation (Procedure 3)
- rotation with patient overpressure (Procedure 3a)
- rotation with clinician overpressure (Procedure 3b)
- rotation mobilisation in extension (Procedure 3c).

If no clear beneficial mechanical or symptom response is generated after thorough testing over three to five sessions, non-mechanical pathologies should be suspected (see Figure 27.1).

## Conclusions

This chapter has outlined the assessment process for thoracic spine problems. This is very similar to the history-taking and physical

examination conducted in other regions. A few different questions are asked during the history, and some different movements are explored during the physical examination as outlined above. As in other regions, the aim of assessment is to exclude serious spinal pathology and then to conduct a mechanical evaluation to determine the mechanical syndrome and the appropriate treatment principle.

# 29: Procedures of Mechanical Therapy for the Thoracic Spine

## Introduction

This chapter contains general descriptions of the procedures that may be utilised in mechanical therapy of the thoracic spine and indications for their application. The procedures described here include both patient and clinician techniques.

Use of any of these procedures should only be considered with a full understanding of mechanical diagnosis and therapy, and it is important that there is familiarity with the first sections of Chapter 14, which introduce the use of cervical procedures. In particular it is vital that the user has a thorough understanding of the following principles:

- force progression
- force alternatives
- repeated movements or sustained postures
- assessment of symptomatic and mechanical responses
- treatment principles.

*A thorough understanding of the appropriate way to interpret the symptomatic and mechanical response is essential in order to safely and effectively conduct a mechanical evaluation and manage the patient.* A careful monitoring of the patient's response to different procedures is vital; this issue is considered in depth in Chapter 12, but is not reiterated with each procedure.

## The procedures

The procedures are listed (Table 29.1) according to the treatment principles:

- extension principle forces
- lateral principle forces
- flexion principle forces.

They can be performed in a number of different positions (loaded or unloaded) and applied either dynamically or statically.

**Table 29.1 Procedures** (not all in order of force progression)

*Extension principle*

*Procedure 1 – Extension:*

Can be performed sitting (mid-thoracic), supine (upper thoracic), prone (mid- and lower thoracic).

1a. extension with patient overpressure

1b. extension with clinician overpressure

1c. extension mobilisation

1d. extension manipulation.

*Procedure 2 – Posture Correction.*

*Lateral principle*

*Procedure 3 – Rotation:*

3a. rotation with patient overpressure

3b. rotation with clinician overpressure

3c. rotation mobilisation in sitting or in prone extension

3d. rotation manipulation in prone extension.

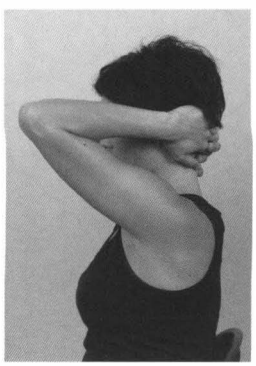
*Flexion principle*

*Procedure 4 – Flexion:*

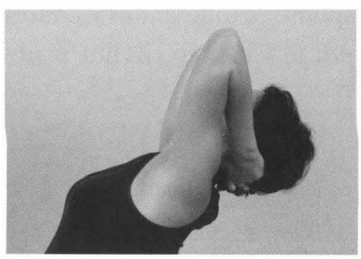
4a. flexion with patient overpressure.

## Extension principle

*Photos 95 and 96: Extension for mid-thoracic spine.*



95



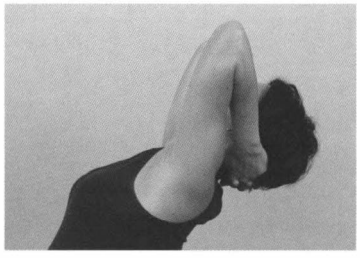
96

### Procedure 1 – Extension (in sitting)

The patient sits on the treatment table with hands clasped behind the neck for support. The patient then extends the thoracic spine lifting the elbows upwards as far as possible. The extended position is maintained briefly before the patient returns to the starting position. The movement is repeated about ten times, with each repetition increasing the range to maximum.

### Procedure 1a – Extension (in sitting) with patient overpressure

*Photo 97: Overpressure for mid-thoracic spine using a chair.*

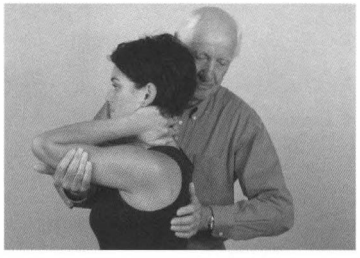


97

The patient sits in an upright chair with the hands clasped behind the neck for support. The patient then extends the thoracic spine by lifting the elbows upwards as far as possible using the top of the chair as a fulcrum. The position is maintained briefly before the patient returns to the starting position. The movement is repeated up to ten times, with each repetition increasing the range to maximum.

### Procedure 1b – Extension (in sitting) with clinician overpressure

*Photos 98 and 99: Overpressure is applied through the arms and thoracic spine.*



98

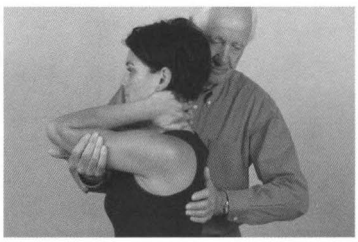
99

The patient sits on the treatment table with hands clasped behind the neck for support. The clinician stands to the side of the patient with one arm under the patient's elbows and the heel of the other hand against the spinous process at the appropriate level. The patient then actively extends the upper thoracic spine as far as possible. By lifting the patient's elbows with one hand and applying pressure to the appropriate level with the heel of the other hand, extension overpressure is applied briefly and the patient returns to the starting position. The procedure is repeated five or six times.

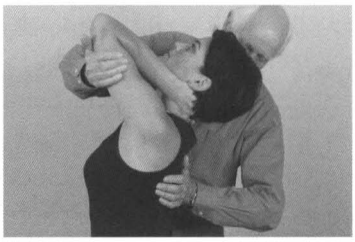
### Procedure 1c – Extension mobilisation (in sitting)

The patient and clinician positions are as described above. The manoeuvre is essentially the same as that described for clinician over-pressures except that the patient remains relaxed throughout. The clinician lifts the patient's elbows and applies resistance overpressure with the heel of the hand to the spinous process at the appropriate

*Photos 100 and 101: One arm stabilises the patient at end-range thoracic extension while the other hand applies extension mobilisation to the thoracic spine.*



*100*

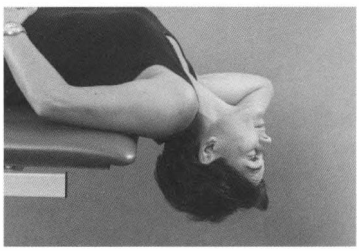


*101*

level. The patient's thoracic spine is held briefly at end-range and then returns to the neutral position. The procedure is repeated five or six times.

## Alternative positions for extension

*Photo 102: Once off the end of the treatment table, the patient retracts and then extends the head for upper thoracic extension.*



*102*

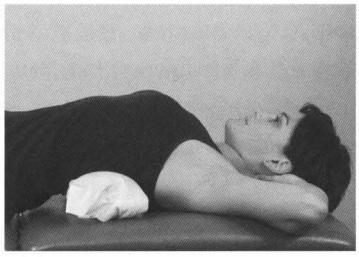
### Upper thoracic: Retraction and extension in supine

This is the same as the equivalent cervical procedure described in Chapter 14. The patient lies supine on the treatment with their head and neck over the end, unsupported down to the level of T3/4. During this process the patient should provide support for the head with one hand under the occiput. In this position the patient fully retracts the head and neck and lowers the head towards the floor into a fully extended position. If tolerable, the supporting hand may be removed and the head, neck and upper thoracic spine allowed to hang relaxed. After two or three seconds the patient should return the head to the starting position by lifting the head with the supporting hand and at the same time tucking in the chin. Care should be taken to avoid actively raising the head by using the neck musculature. The retraction and extension movement should be repeated about ten times slowly and rhythmically.

### Mid-thoracic: Sustained extension in supine

The patient lies supine on the treatment table and a tightly rolled towel is positioned under the thoracic spine. In this position the patient is encouraged to increase the depth of their exhalation to achieve a greater range of thoracic extension. To achieve further extension in the thoracic spine, the patient can place their hands above their head.

*Photo 103: Sustained supine lying over rolled towel for mid-thoracic spine.*

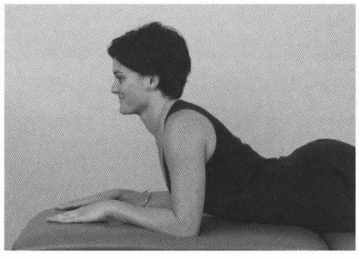


*103*

### Mid- and lower thoracic: Sustained prone lying in extension

The patient lies prone on the treatment table, leaning on their elbows. The patient relaxes in this position, allowing the spine to sag, which applies a passive overpressure to the thoracic spine. The position is sustained for a maximum of three minutes and then the patient returns to the prone lying position.

*Photo 104: Sustained prone lying in extension.*



*104*

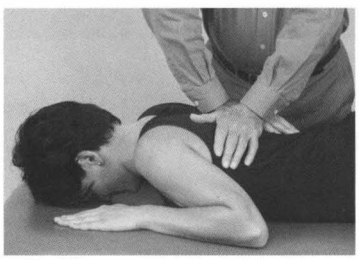
### Procedure 1a – Extension (in lying) with patient overpressure

The patient lies prone on the treatment table with the hands palm down alongside the shoulders as for extension in lying for the lumbar spine. The top half of the body is pressed upwards by straightening the arms while the lower half of the body is allowed to sag with gravity. The top half of the body is then lowered to the starting position. The exercise is then repeated ten to fifteen times. After a few repetitions it is important that the arms are fully straightened and the patient sags the trunk from the mid-scapula to the pelvis to obtain as much extension as possible. The hand position can be varied if necessary if symptomatic response is improved by this adjustment.

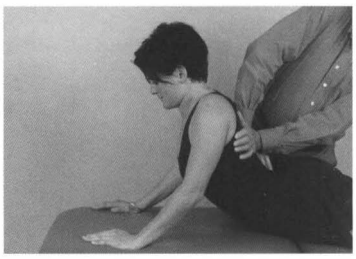
### Procedure 1b – Extension (in lying) with clinician overpressure

Standing to one side, the clinician crosses the arms so as to place the hypothenar eminences at the appropriate level on the transverse processes on either side. One hand is parallel to the spinous process and the other perpendicular to it so that the hands are at 90 degrees to each other.

*Photos 105 and 106: Overpressure is applied as the patient extends.*



*105*



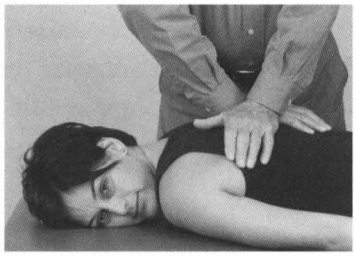
*106*

Once the hands are positioned, the elbows are fixed just short of full extension. The clinician's body weight is used to apply a firm symmetrical and constant pressure to the selected segment. This pressure is maintained as the patient performs extension in lying, but should not impede the patient's ability to extend. As the patient extends it will be necessary to change the angle of the overpressure, from directly downward to about 45 degrees, so that the force remains parallel to the motion segment. This change in angle may require the clinician to adjust the distribution of body weight from one leg to the other. The level of force can be changed depending on the response of the symptoms, and the pressure can be increased if the response indicates. The procedure is repeated five or six times.

### Procedure 1c – Extension mobilisation

An adjustable-height treatment table is preferred to perform this procedure most effectively. It should be at a level that allows the clinician to be positioned directly over the patient so as to deliver the mobilising force perpendicular to the spine. The patient lies prone with their arms by their side and near the edge of the table.

*Photo 107: Mobilisation is applied bilaterally through transverse processes.*



*107*

The clinician stands beside patient and places the hands and arms as described for extension in lying with clinician overpressure. Once the hands are positioned and a firm pressure applied to the selected segment, the elbows can be fixed just short of full extension. By alternately leaning forwards to increase pressure and backwards to reduce pressure, the clinician delivers a perpendicular rhythmical alternating cycle of force that is repeated up to ten times.

The hands must remain in contact throughout this procedure. Each pressure is a little stronger than the previous one, depending on the patient's tolerance and the resulting pain. The intent is to move further into range and attain end-range with the last few pressures if possible.

If a treatment table with an adjustable end is being used, the procedure can be performed in varying degrees of extension. The starting position would always be neutral, but if a progression is required the mobilisation could be performed with the patient placed in a position of extension.

**Procedure 1d – Extension manipulation**

Extension manipulation is used if a favourable response was previously generated with extension mobilisation, but symptoms subsequently returned.

The patient and clinician start positions for this procedure are described in the previous procedure. The clinician stands to one side of the patient with arm and hand positions the same as for extension mobilisation above. Once the hands are positioned, a firm pressure is applied to the selected segment to near end-range. The elbows should be fixed just short of full extension. Leaning forward to increase pressure, the clinician delivers a perpendicular short amplitude high-velocity thrust to the selected segment by causing a sudden extension of the elbows. The hands are then removed completely.

The segmental level at which the manipulation is performed is decided by symptom response during the application of extension mobilisation, which always precedes this procedure. The appropriate level is the one at which symptoms were only temporarily decreased, centralised or abolished. Only one manipulative thrust should be performed at any one treatment session.

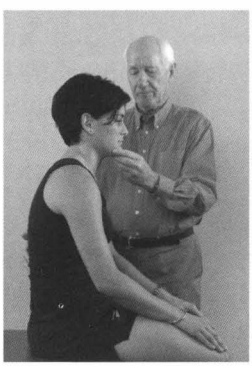
### Application

Extension procedures are commonly used for the treatment of thoracic derangement. They are also used for extension dysfunction. Extension in sitting is used for mid-thoracic symptoms, extension in supine (as for the cervical spine) for the upper thoracic spine and extension in prone (as for the lumbar spine) for the mid- and lower thoracic spine.

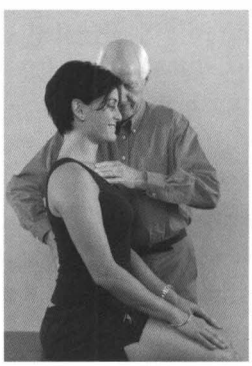
In the case of dysfunction, symptoms are produced with the procedure but abate once the procedure is finished. Where the procedures are applied for derangement, the desirable response is for the symptoms to be decreased, centralised or abolished afterwards.

### Procedure 2 – Posture correction

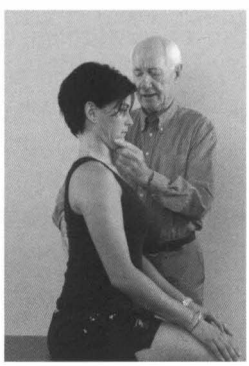
*Photos 108, 109, 110: From slouched position (108), gentle pressure at spine and sternum restores the lordosis (109). Gentle pressure at chin and thoracic spine corrects the head posture (110). Symptom response is monitored before and after.*



*108*



*109*



*110*

#### *Slouch-overcorrect and posture correction*

Sitting over the end or side of the treatment table, the patient is instructed to adopt a relaxed slouched posture with the lumbar and thoracic spine flexed and the head and neck protruded (Photo 108). The patient smoothly moves into the extreme of the erect sitting posture with the lumbar spine in maximum lordosis and the head and chin maximally retracted (Photo 109). Some clinician guidance using gentle hand pressure on the patient's lumbar spine and mandible may assist in the learning process (Photo 110). The patient is then instructed to relax back into the slouched position. This cycle should

be repeated ten times so that the patient moves from the extreme of the slouched posture to the extreme of the upright extended and retracted posture. *After completing ten cycles of the procedure, the patient should hold the extreme of the good position for a second or two and then release 10% of the strain. This is the posture the patient must aim for on a daily basis. It is the learning process for maintaining correct posture and is also therapeutic as some patients achieve centralisation of their pain using this procedure alone.*

### Application

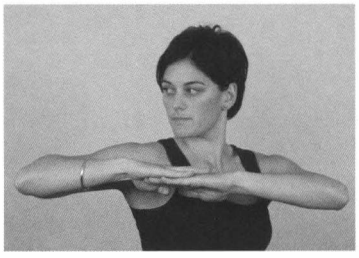
Slouch-overcorrect is used to teach patients how to attain correct posture. The ability to attain and maintain good thoracic posture is essential for the maintenance of thoracic derangements and in the management of thoracic extension dysfunction.

## Lateral principle

### Procedure 3 – Rotation (in sitting)

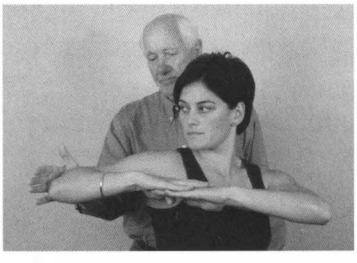
The patient sits upright on the treatment table with fingers interlocked, arms horizontal and elbows at approximately chest height. The patient turns to the side of pain, swings around as far as possible and then returns to the neutral start position. The movement is repeated about ten times, each time trying to increase the range of movement.

*Photo 111: Rotation in sitting.*



*111*

*Photo 112: Rotation overpressure is applied by increasing the speed of the manoeuvre, for instance by getting the patient to strike the clinician's hand (112).*

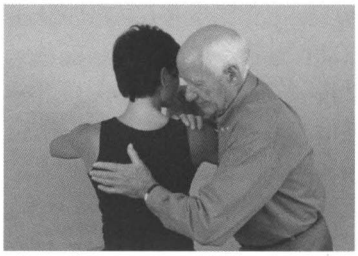


*112*

### Procedure 3a – Rotation (in sitting) with patient overpressure

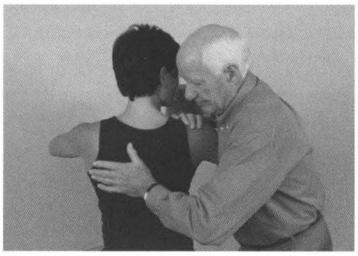
The patient performs rotation as described above. Then, to achieve the overpressure, the speed and force of the procedure are increased. This is best done by the patient swinging around rhythmically and vigorously, as if trying to strike an out-of-reach object with the elbow. It is important that the fingers remain interlocked and the arms remain horizontal across the upper chest. The procedure is repeated about ten times.

*Photo 113: Clinician overpressure is applied anteriorly and posteriorly at both shoulders.*



*113*

*Photo 114: One hand stabilises at end-range rotation, the other applies the mobilisation.*



*114*

### Procedure 3b – Rotation (in sitting) with clinician overpressure

The patient first performs rotation and rotation with patient overpressure as above. The clinician stands at the patient's side. When the patient is rotating to end-range, the clinician applies overpressure at the shoulders with one hand anteriorly and one hand posteriorly on opposite shoulders. The procedure is repeated five or six times.

### Procedure 3c – Rotation mobilisation (in sitting)

(Described for right rotation mobilisation.)

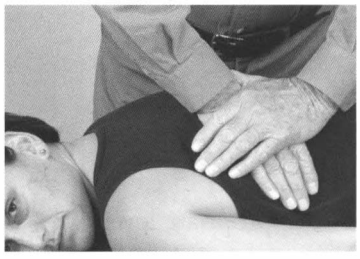
The patient first performs rotation and rotation with patient overpressure as above. The clinician stands at the patient's side. When the patient is at end-range rotation, the clinician stabilises the right shoulder with their right hand and applies the mobilisation pressure on the contralateral rib angle five or six times. The patient then returns to the neutral position.

### Procedure 3c – Rotation mobilisation (in prone extension)

To perform this procedure most effectively, an adjustable-height treatment table is recommended. This should be at a level that allows the clinician to be positioned directly over the patient to enable delivery of the mobilising force perpendicular to the spine. The relaxed patient lies prone with their arms by the side and head turned to one side near the edge of the treatment table.

The clinician stands to one side of the patient and places the hands and arms as described for extension in lying with clinician overpressure. The clinician crosses hands and places the heel of the hypothenar eminences on the transverse processes of the same lumbar segment. One hand is parallel to the spinous process and the other is perpendicular to it, so that they are 90 degrees to each other.

*Photo 115: Unilateral mobilisation may be applied with both hands through transverse process.*



*115*

A gentle pressure is applied and released first to one side and then to the other. By repeating the pressure on alternate sides, a rhythmical rocking effect is obtained. As pressure is applied on one side a simultaneous reduction in pressure must occur on the other; thus, some extension force is always present, as well as an alternating lateral force. Pressure is achieved by applying the weight of the body through the arms, and the pressure is gradually increased with the intent of moving further into range. The technique is repeated five or six times on one segment and/or repeated on adjacent segments as necessary.

If pressure on one side is producing a favourable response, then mobilisation is restricted to that side only. The clinician stands on the opposite side to that which is to be mobilised; one hand is placed on top of the other to perform the mobilisation on one transverse process. About five or six mobilisations are performed, each one with more force, with the force directed anteriorly and slightly medially.

**Procedure 3d – Rotation manipulation (in prone extension)**

Rotation manipulation in prone extension is used if a favourable response has previously been generated with unilateral rotation mobilisation in extension, but symptoms have subsequently returned.

The patient and clinician start positions for this procedure are the same as those described in the previous procedure. The clinician stands on the side opposite that which is to be mobilised. Place one hand on top of the other to perform the mobilisation on one side; force is directed anteriorly and slightly medially. *The manipulation is indicated only if all previous procedures have given temporary relief.*

The clinician leans over the patient with the arms perpendicular to the spine and applies unilateral pressure slowly until the spine feels taut. Then a high-velocity thrust of very short amplitude is applied and immediately released.

The segmental level at which the manipulation is performed is decided by symptom response during the application of rotation mobilisation in extension, which always precedes this procedure. The appropriate level is the one at which symptoms are decreased, centralised or abolished but return when the mobilisation pressure is released. Only one manipulative thrust should be performed at any one treatment session.

#### Application

Rotation procedures are key in the management of derangements requiring lateral forces. They are used for unilateral symptoms from derangement not responding or worsening in response to sagittal plane forces. Most commonly patients respond when rotation is towards the side of pain; however, if this fails to generate a positive response or causes worsening, the opposite rotation should be explored using the same procedures. The procedure is also used for rotation dysfunction.

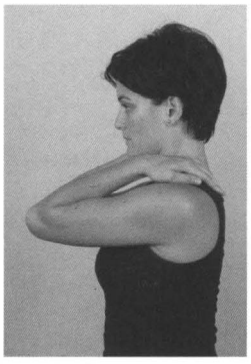
## Flexion principle

#### Procedure 4 – Flexion (in sitting)

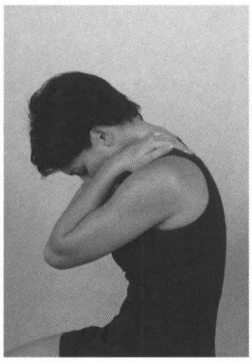
The patient sits upright on the end or side of the treatment table and slouches into a fully flexed position. The position is maintained for a few seconds and then the patient returns to the neutral upright posture. The movement is performed about ten times.

#### Procedure 4a – Flexion (in sitting) with patient overpressure

The patient sits in the same position as for flexion and interlocks the fingers of both hands behind the lower cervical and upper thoracic vertebrae. The patient repeats the flexion exercise while simultaneously applying overpressure at the end of the range of movement. The overpressure is maintained for a few seconds and the patient returns to the upright position. The movement is repeated up to ten times.



116



117

*Photos 116 and 117: Flexion is performed; overpressure is applied through the upper thoracic transverse processes.*

### Application

Flexion procedures are rarely used therapeutically in the thoracic spine. The most common use of flexion procedures in the thoracic spine is provocative testing of posterior derangements or non-mechanical problems. Flexion procedures may help to establish the force direction that worsens the patient, therefore helping to establish what improves their condition. Worsening with all test movements highlights unsuitability for mechanical therapy and the need for further investigation.

Flexion procedures are also appropriate for the management of flexion dysfunction in the thoracic spine. Flexion with clinician overpressure and flexion mobilisation are rarely required; however, where patient-generated forces have not achieved a positive response, these force progressions are appropriate.

# 30: Thoracic Derangement – Management

## Introduction

As in other parts of the spine, derangement is the most common mechanical syndrome in the thoracic or dorsal spine. Because of the limited pathway of thoracic nerves, referral of pain is generally limited to the trunk itself. Symptoms tend to present either centrally and symmetrically or unilaterally. Referral may extend around the rib cage, and occasionally patients present with patches of pain anteriorly and little or nothing posteriorly. Whether symptoms are distributed symmetrically or asymmetrically, management generally starts with the use of sagittal plane forces. Lateral forces are only introduced if asymmetrical or unilateral symptoms fail to show improvement with the use of sagittal plane procedures.

Sections in this chapter:

- management of central and symmetrical symptoms
- extension principle
    - force progressions
- patient review
    - extension principle
- management of asymmetrical and unilateral symptoms
- extension principle
    - response to extension
- lateral treatment principle
    - force progressions.

## Management of central and symmetrical symptoms

A loss of thoracic extension can sometimes be difficult to distinguish. Rather than there being a loss of range of movement, the patient may have full range of movement, but pain at end-range. In more severe cases, both flexion and extension can be clearly limited and painful. Rather misleadingly, some patients with thoracic derangement find relief of symptoms in the flexed posture, whilst extension increases

pain, usually centrally. This may give the mistaken impression that the patient's mechanically determined directional preference is for flexion. However, whether sustained or repeated, the patient is no *better* after flexion, and such a patient actually requires extension to reduce a posterior derangement. Symptoms may be localised around the spine or radiating over the rib-cage; occasionally there may be additional symptoms or only around the sternum. The extension principle is always used; the type of extension exercise depends on the level of symptoms.

## Extension principle

Procedures to be used for lower thoracic problems:

- prone lying in extension sustained for three to four minutes – particularly important if symptoms are severe or constant (Procedure 1)
- extension in lying, prone as used in the lumbar spine – essential (Procedure 1)
- posture correction – essential for reduction and maintenance of reduction (Procedure 2).

Procedures to be used for upper thoracic problems:

- extension in sitting – essential (Procedure 1)
- extension in lying, supine as used in the cervical spine – if unloaded position is preferred (Procedure 1)
- posture correction – essential for reduction and maintenance of reduction (Procedure 2).

Force alternatives (some patients respond better to exercises in the loaded position):

- extension in standing
- extension with patient overpressure (Procedure 1a).

Regularity:

- ten to fifteen repetitions every two to three hours.

Expected response:

- centralisation, abolition or decrease of symptoms
- possible increase of pain centrally initially
- increase in all ranges of movement that are restricted.

Maintenance of reduction:

- regular performance of extension exercises to maintain symptomatic and mechanical improvements
- posture correction when sitting
- temporary avoidance of sustained flexion activities.

### Force progressions

Force progressions are used only if improvements plateau or fail to occur. Before undertaking progressions, the patient's exercise technique and postural correction should be checked. Sometimes patients are able to abolish or reduce symptoms with repeated movements, but they return because of poor postural control and poor maintenance of reduction. In such instances force progressions are not needed; rather, there needs to be greater emphasis on posture. Progressions may also be used to confirm an initial diagnosis if there is some uncertainty; in other words, the procedures are used as part of the assessment process.

- only use one new procedure per session
- wait twenty-four hours before initiating further progressions
- repeat force progressions a maximum of two sessions if no definite improvement occurs
- the patient must continue with home exercise programme, otherwise any benefit from the force progressions are lost between treatment sessions
- force progressions are stopped once the patient is able to self-manage:
    - extension with clinician overpressure (Procedure 1b)
    - extension mobilisation (Procedure 1c)
    - extension manipulation (Procedure 1d).

## Patient review

### Extension principle

When the patient returns for review they will be improved, worsened or unchanged. Ensure that the status reported by the patient is their true state. Patients may report their symptoms to be worse when widespread pain has centralised to the middle of the spine; they may report themselves to be unchanged when in fact pain that was constant has become intermittent. Some patients, keen to please and to get better, report an improvement that is difficult to confirm. See Chapter 12 for details of how to analyse clinical presentations and Chapter 13 for the structure of a review process.

#### *Patient is better*

If there is improvement in the symptomatic and/or mechanical response at review, management strategy should not be changed. It is unnecessary to supplement present procedures with any other techniques or interventions if the patient is getting better. Management continues in the same way unless there is a change in status.

Once symptoms are minimal the frequency of exercising may be reduced if this seems appropriate and the patient should be told also about performing the exercises at the first signs of recurrence. Maintenance of reduction through the use of posture correction should be reinforced. Ensure that function is fully recovered and that flexion is full and pain-free; however, such complications are unusual in thoracic derangements.

Discuss prevention of recurrence with the patient this is based upon:

- continue with extension in lying twice a day for several weeks after recovery
- use of extension exercises after activities of sustained flexion
- use of correct sitting posture
- full resumption of normal activities
- resume or commence regular exercise programme at suitable level.

#### *Patient is worse*

There are certain instances that the patient may interpret as being 'worse', which we would not consider as such. When centralisation occurs there can be an accompanying and temporary increase in

central pain. When patients with long-standing derangements and an associated obstruction to extension commence the necessary extension principle procedures, there can sometimes be an initial short-lasting increase in symptoms. The patient may be performing the procedures incorrectly, or may have misinterpreted instructions and be doing different exercises. New pains may have appeared as a consequence of performing the exercises, which has made the patient reluctant to continue. Do their symptoms improve with the exercises, but get worse later because of insufficient attention to posture? Did they initially get better with the instructions, but then worsened because they increased their activity level too soon? All these instances should not be considered a worsening scenario.

If really worse, pain is usually more widespread. It should be ensured that the patient definitely has symmetrical symptoms. If a condition is truly worsening, the patient should be advised to stop the exercises – patients sometimes improve with this step. If unequivocally worse, non-mechanical conditions should be considered.

### *Patient is unchanging*

First it should be ensured that the patient is performing the right exercises correctly and with enough regularity, and that they are abiding by postural correction. If this is not the case, further instruction and discussion are necessary to ensure that the patient understands the procedures and their own role in managing the problem. It is best to see the patient daily until certainty of management is established, and the patient is confident about their management strategy. If the patient cannot be reviewed regularly, this can create problems. In such cases, review by telephone is desirable.

If they have been following the treatment principle correctly, but still no improvement has occurred, then force progressions should be implemented. These are done in the following order, with subsequent force progressions applied only if there is failure to improve. If at any point improvement does occur, further progression is unnecessary. Whatever progression is used, the patient must continue to perform the appropriate exercises at home with suitable regularity. Following a force progression, the effects of this procedure should be evaluated at the next review. Force progressions can be repeated on up to two occasions before they should be abandoned if no change ensues. Do not instigate clinician techniques unless it is clear that improvement cannot be achieved by any other means.

- extension with clinician overpressure (Procedure 1b)
- extension mobilisation (Procedure 1c)
- extension manipulation (Procedure 1d).

## Management of asymmetrical and unilateral symptoms

Many such patients respond to the extension principle of treatment as set out in the previous section, although a few require the lateral principle of treatment. Patients with asymmetrical symptoms may well present with end-range painful extension or loss of extension, but impairment of rotation is also common. With rotation it is generally easier to determine when there is a loss of movement, although again, in minor cases the presentation may only involve pain at end-range. Patients with painful limitation of rotation are more likely to be those who require the lateral principle of treatment.

## Extension principle

Procedures to be used for lower thoracic problems:

- prone lying in extension sustained for three to four minutes – particularly important if symptoms are severe or constant (Procedure 1)
- extension in lying, prone as used in the lumbar spine – essential (Procedure 1)
- posture correction – essential for reduction and maintenance of reduction (Procedure 2).

Procedures to be used for upper thoracic problems:

- extension in sitting – essential (Procedure 1)
- extension in lying, supine as used in the cervical spine – if unloaded position is preferred (Procedure 1)
- posture correction – essential for reduction and maintenance of reduction (Procedure 2).

Force alternatives (some patients respond better to exercises in the loaded position):

- extension in standing

- extension with patient overpressure (Procedure 1a).

Regularity:

- ten to fifteen repetitions every two to three hours.

Expected response:

- centralisation, abolition or decrease of symptoms
- possible increase of pain centrally initially
- increase in all ranges of movement that are restricted.

### Response to extension

In response to extension forces, unilateral symptoms may respond in one of three ways, each with different management implications (Table 30.1). They will either be better, worse or unchanged.

**Table 30.1 Response to extension forces in unilateral asymmetrical and implications**

| *Response to extension forces* | *Implications* |
|---|---|
| Centralisation<br>Abolish pain<br>Decrease pain | Continue with extension forces |
| Increase distal pain<br>Peripheralisation | Introduce lateral forces |
| Indeterminate response<br>Increase, not worse. | Progress forces *and* explore lateral component and then decide on the most appropriate loading strategy. |

#### *Better*

In the first instance it is very apparent that extension forces are appropriate. There is a rapid favourable symptom response, with decrease, abolition or centralisation of pain, and/or a rapid mechanical response with an increase in range of movement. In this situation management would be conducted according to the extension principle, including any necessary force progressions, as long as improvements continued. If the response changed, a review would be necessary.

Maintenance of reduction:

- regular performance of extension exercises to maintain symptomatic and mechanical improvements
- posture correction when sitting

- review of lying posture (if appropriate)
- temporary avoidance of sustained flexion activities (for maintenance of reduction).

### *Worse*

Likewise, in the second instance, a rapid peripheralisation or lasting increase in pain severity is an alert to the inappropriateness of pure extension forces and that the lateral component may be required. In the thoracic area peripheralisation refers to the spread of pain away from the vertebrae. A relevant lateral component has been determined from symptomatic response.

In patients with asymmetrical or unilateral symptoms where the pain is worse as a result of the initial assessment of the above extension procedures, or has increased laterally or peripheralised, the lateral principle of treatment is introduced on day one.

### *Unchanging*

When the symptom response is indeterminate, the management strategy is less clear. In these instances it is important to compare symptom response to sagittal movements with the symptom response to lateral movements and determine if there is a clear mechanically determined directional preference for sagittal or lateral forces. Determining the best strategy requires applying a clinical reasoning process, and overpressures and mobilisation in both planes may be considered on day one to help determine the appropriate loading. If previous testing has not produced a clear symptom response, then these procedures may help to clarify a mechanically determined directional preference on day one.

Further testing on day one takes two forms. First, force progressions in extension procedures; second, lateral procedures including force progressions. If at any point extension force progressions cause peripheralisation or worsening of distal pain, lateral principle procedures are used. The principle adopted is determined by the most favourable symptomatic and mechanical response.

Force progressions in extension:

- extension with clinician overpressure (Procedure 1b)
- extension mobilisation (Procedure 1c).

If extension procedures including force progressions have not produced a favourable response, lateral forces should be explored. Usually lateral movements are performed to the side of pain, but if no favourable response is generated the other direction can be explored. This is done in the following order:

- rotation in sitting (Procedure 3)
- rotation with patient overpressure (Procedure 3a)
- rotation with clinician overpressure (Procedure 3b).

If initial lateral principle procedures appear to have no effect, therapist-generated force can be added to help clarify:

- rotation mobilisation (Procedure 3c)
- rotation mobilisation in extension (Procedure 3c).

The force that generates the most favourable response is chosen for the repeated movement for the patient to perform every two to three hours over the next few days. If there is still a lack of a clear response, a treatment principle is selected for a trial over the next twenty-four to forty-eight hours. Sometimes there may be clues in the history or physical examination that suggest a certain movement; if not, it is best to first test the response to extension procedures. The patient should be instructed about what constitutes positive and negative responses and when they should stop exercising.

It may be equally valid at times to test out the response to repeated movements over twenty-four to forty-eight hours to see if the longer period produces a more clearly favourable response. This is especially appropriate when there are suggestions in the history or physical examination that a positive response is likely. If when the patient returns the response is still unclear, force progressions and force alternatives are explored as detailed above.

In summary, for patients with unilateral asymmetrical symptoms who do not show immediate benefit using extension forces, the lateral component should always be explored. Overpressures and mobilisation in both sagittal and lateral planes can be used to help determine the appropriate loading strategy. In many patients the addition of the lateral component or force progressions clarifies the preferred loading. In effect, the clinician-generated procedures are being used as tools of assessment as well as treatment. A thorough

knowledge of the chapter on *Evaluation of clinical presentations* (Chapter 12) is necessary in order to interpret the patient's presentation and responses accurately. Sometimes, however, a trial over twenty-four hours is more revealing than repeated movements during the clinical examination.

## Lateral treatment principle

Procedures to be used:

- rotation (Procedure 3)
- rotation with patient overpressure (Procedure 3a).

Regularity:

- ten to fifteen repetitions every two to three hours.

Expected response:

- centralisation, abolition or decrease of symptoms
- possible increase of pain centrally initially
- increase in all ranges of movement that are restricted.

Maintenance of reduction:

- regular performance of rotation exercises to maintain symptomatic and mechanical improvements
- posture correction when sitting (if appropriate)
- check sleeping posture (if appropriate)
- temporary avoidance of sustained flexion activities (if appropriate).

### Force progressions

Force progressions are used only if improvements plateau or fail to occur. Before undertaking progressions, the patient's exercise technique and postural correction should be checked. Progressions should never be instigated if the patient is able to decrease or abolish symptoms, but allows symptoms to return due to failure to maintain reduction of the derangement. Progressions may also be used to confirm an initial diagnosis if there is some uncertainty; in other words, the procedures are used as part of the assessment process.

- repeat force progressions a maximum of two sessions if no definite improvement occurs
- the patient must continue with home exercise programme and posture correction, or any benefit from the force progressions will be lost between treatment sessions
- force progressions are stopped once the patient is able to self-manage:
    - rotation with clinician overpressure (Procedure 3b)
    - rotation mobilisation (Procedure 3c)
    - rotation mobilisation in extension (Procedure 3c)
    - change to sagittal plane movements if symptom response dictates.

## Conclusions

This chapter has described the management of thoracic derangement syndrome and the procedures used to do this. If the patient presents with central or symmetrical symptoms, procedures from the extension principle
symptoms patients may also respond to extension procedures, but the lateral principle, using rotation procedures, will also be used in some patients. The symptomatic and mechanical responses (Chapter 12) determine the appropriate mechanical forces to be used.

# 31: Thoracic Dysfunction and Postural Syndrome – Management

## Introduction

These mechanical syndromes are uncommon in the thoracic spine, but as in the other spinal regions dysfunction is more common than postural syndrome. Management of both is described in the cervical section and is briefly outlined here.

Sections in this chapter are as follows:

- dysfunction syndrome
- postural syndrome.

## Dysfunction syndrome

In dysfunction syndrome, the mobility or function of soft tissues is reduced because of structural impairment. It is a painful disorder caused by loading or stretching tissue that is imperfectly repaired or has become adaptively shortened (McKenzie 1981, 1990). Structurally impaired tissue gives rise to pain with normal mechanical loading. In the thoracic spine dysfunction may develop because of trauma, derangement, long-term poor postural habits or pathologies that affect the region, such as Scheuermann's disease or osteoporosis (see Chapter 27). Fuller description of the pain mechanism and clinical picture is provided in Chapter 21. The thoracic dysfunctions most likely to be seen are extension and rotation – these will be described. If other directions of dysfunction are seen they would be treated in the same manner, but using the appropriate treatment principle.

**Table 31.1 Articular dysfunction syndrome – criteria *(all will apply)***

*History:*

- spinal symptoms only
- intermittent symptoms.

*Physical examination:*

- movement is restricted, and the restricted movement(s) consistently produce concordant pain at end-range, *and*
- there is no rapid decrease or abolition of symptoms, *and*
- no lasting production and no peripheralisation of symptoms.

Usually extension and/or rotation are limited and painful; if the loss of movement is small this maybe difficult to detect. The chapter on cervical dysfunction contains more detail about the mechanical syndrome and also more about the educational component of management of this syndrome (see Table 31.2).

**Table 31.2 Instructions to patients with dysfunction syndrome**

- exercises must be performed regularly throughout the day, every two to three hours
- if patients are unable to exercise as regularly as recommended, recovery of full function is likely to take longer
- at each session perform ten to fifteen stretches
- if the exercise does not produce their pain, it has not been performed properly
- the exercise must consistently reproduce their pain each time
- the pain should have subsided within ten minutes after the completion of the exercises; mostly it abates much quicker
- if pain from the procedures persists constantly afterwards for a long period, either overstretching has occurred, in which case repetitions must be reduced, or the original classification was mistaken or has changed – in either case a review is necessary
- if the patient feels they are getting worse, they must stop exercising and return for a review appointment
- there will be no rapid changes in range of movement – if they experience a dramatic change in function or range, they must return for re-evaluation
- if there is a spread of pain distally or a rapid deterioration in their situation, they must stop exercising and return for a review appointment
- posture correction should complement the exercise programme.

### Extension dysfunction

A thorough and convincing explanation to the patient is a prerequisite to gain their committed involvement. Procedures to be used all come under the extension principle and can include any of the following. The procedure used is the one that most effectively generates a symptom response and that the patient can fit easily into their daily routine.

- extension in standing
- extension in sitting (Procedure 1)
- extension in sitting with patient overpressure (Procedure 1a)
- extension in lying – prone (as used in the lumbar spine) – for lower thoracic problems (Procedure 1)

- extension in lying – supine (as used in the cervical spine) – for upper thoracic problems (Procedure 1)
- posture correction (Procedure 2)
- avoidance of sustained flexion activities (especially important in patients with osteoporosis and other degenerative changes).

Regularity:

- ten to fifteen times every two to three hours.

Expected response:

- temporary (ten minutes maximum) localised thoracic pain
- pain produced with exercise is concordant with patient's complaint
- pain occurs at limited end-range
- pain abates rapidly when exercises stop
- all other movements remain as they were
- improved pain and range within four to six weeks; full recovery may take longer.

Maintenance:

- once range of movement and pain are improved, patients should be advised to prevent recurrence to maintain ten to fifteen repetitions once or twice per day.

Force progression:

- force progressions are rarely required
- repeat force progressions a maximum of two sessions if no definite improvement occurs
- the patient must continue with home exercise programme, or any benefit from the force progressions will be lost between treatment sessions
- extension with clinician overpressure (Procedure 1b)
- extension mobilisation (Procedure 1c).

### Rotation dysfunction

Procedures to be used:

- rotation (Procedure 3)
- rotation with patient overpressure (Procedure 3a).

Regularity:

- ten to fifteen times every two to three hours.

Expected response:

- temporary (ten minutes maximum) localised thoracic pain
- pain produced with exercise is concordant with patient's complaint
- pain occurs at limited end-range
- pain abates rapidly when exercises stop
- all other movements remain as they were
- improved pain and range within four to six weeks; full recovery may take longer.

Maintenance:

- once range of movement and pain are improved, patients should be advised that to prevent recurrence, maintain ten to fifteen repetitions once or twice per day.

Force progression:

- force progressions are rarely required
- repeat force progressions a maximum of two sessions if no definite improvement occurs
- the patient must continue with home exercise programme, or any benefit from the force progressions will be lost between treatment sessions
- rotation with clinician overpressure (Procedure 3b)
- rotation mobilisation (Procedure 3c)
- rotation mobilisation in extension (Procedure 3c).

## Postural syndrome

Postural syndrome is a painful disorder caused by prolonged static loading of normal soft tissues continued until the point when mechanical stress triggers discomfort (McKenzie 1981, 1990; McKenzie and May 2000, 2003). Pain from the postural syndrome is caused by mechanical deformation of soft tissues or vascular insufficiency arising from prolonged positional or postural stresses affecting the articular structures or the contractile muscles, their tendons or the periosteal insertions. In thoracic postural syndrome, pain most likely arises form mechanical deformation of articular structures. Pain continues as long as the posture is maintained, but abates as soon as the posture is released.

Many experience pain from this syndrome. It is especially common in schoolchildren and students who spend many hours sitting bent over books or computer screens. It is not uncommon for patients to have symptoms in more than one region of the spine. Most people learn that a simple change in posture abolishes symptoms, and it does not bother them when they are active and busy at other times. Consequently, individuals rarely seek treatment and patients with postural syndrome are rarely seen in clinical practice. It is the least common of the three mechanical syndromes encountered by health professionals. Sleeping postural problems sometimes occur, especially if there has been a new sleeping surface or the individual is sleeping on an unfamiliar surface as when on holiday.

Pain of postural origin frequently exacerbates and perpetuates symptoms in all mechanical problems and usually needs to be addressed. Treatment of thoracic postural syndrome is just as described in the cervical section. Fuller description of the pain mechanism, clinical picture and management are provided in that chapter.

**Table 31.3 Postural syndrome – criteria *(all will apply)***

*History:*

- local pain
- intermittent pain
- pain associated with time spent in a particular posture
- pain does not persist
- painless movement and activity.

Continued next page

*Physical examination:*

- poor posture
- full range of movement
- no deformity
- no problems with curve reversal
- repeated movements do not reproduce pain
- pain only produced by sustained loading in relevant position, which is then relieved on moving from that posture.

Postures most commonly involved are sitting and standing. A key component of management is education, ensuring that the patient is aware of the link between their habitual posture and the pain, and also aware of the link between lumbar and thoracic posture. In sitting, the offending posture is usually associated with a flexed lumbar spine, increased thoracic kyphosis and protruding head posture. In standing, postural pains are usually associated with an exaggerated lumbar lordosis, increased thoracic kyphosis and protruding head posture.

**Table 31.4 Management of postural syndrome**

- education on link between posture and pain
- education on link between lumbar and thoracic postures
- education on avoidance of aggravating posture.

*Sitting:*

- education on posture correction
  - attain posture
  - maintain posture
- posture correction (Procedure 2)
- slouch-overcorrect.

*Standing:*

- attain mid-range lumbar lordosis, posterior pelvic tilt, abdominal muscles, raising chest, reduce thoracic kyphosis, retract head and neck.

*Lying:*

- check sleeping surface – rather than a firm mattress, patient may need to create a more concave surface to allow for thoracic kyphosis
- check sleeping posture.

## Conclusions

This chapter has considered the identification and procedures to be used in the management of dysfunction and postural problems in the thoracic spine. Both syndromes would present with intermittent symptoms and consistent and characteristic responses to mechanical testing. Their clinical appearance is uncommon, however, if encountered, management is as described above.

# Appendix



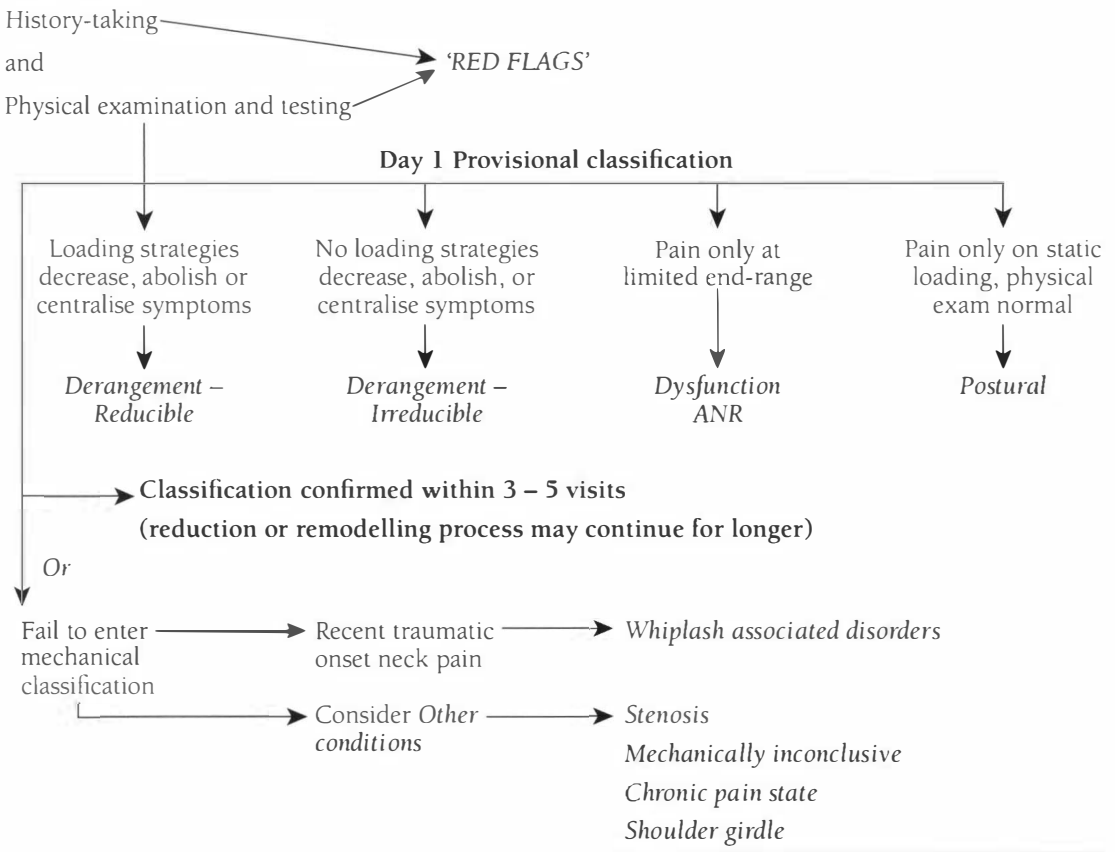
## Classification and operational definitions

| Category | Definition | Criteria* |
|---|---|---|
| ***Mechanical syndrome*** | | ***Symptom response*** |
| *Reducible derangement* | Internal disc displacement with competent annulus | Centralisation<br>Abolition<br>Decrease |
| *Irreducible derangement* | Disc displacement with incompetent or ruptured annular wall | Peripheralisation<br>Increase in peripheral pain<br>No centralisation, reduction or abolition |
| *Articular dysfunction* | Soft tissue structural impairment affecting peri-articular structure | Intermittent pain when loading restricted end-range |
| *Contractile dysfunction* | Soft tissue structural impairment affecting contractile structure | Intermittent pain when loading musculotendinous unit |
| *Adherent nerve root* | Adhesions producing functional impairment of nerve root or dura | Intermittent pain when neural tissue placed under tension |
| *Postural syndrome* | Prolonged mechanical deformation of normal soft tissues | Pain only with prolonged loading<br>Physical examination normal |
| ***'Other'*** | ***Exclusion of above*** | ***Lack of above responses, plus the following*** |
| *Spinal stenosis* | Extensive degenerative changes that cause narrowing of spinal or intervertebral canal | Signs/symptoms of upper or lower motor neurone lesion<br>Increase on extension, decrease on flexion |
| *Mechanically inconclusive* | Unknown intervertebral joint pathology | Inconsistent response to loading strategies<br>No obstruction to movement |
| *Chronic pain* | Pain-generating mechanism influenced by psychosocial factors or neurophysiological changes peripherally or centrally | Persistent widespread pain<br>Aggravation with all activity<br>Exaggerated pain behaviour<br>Inappropriate beliefs and attitudes about pain |
| *Thoracic outlet syndrome* | Compression of neurovascular bundle at the shoulder girdle causing diffuse arm pain and neurological symptoms | Symptoms with raised arm activity<br>At least two pain provocation tests positive |

| Category | Definition | Criteria* |
|---|---|---|
| ***Serious spinal pathology suspected*** | | ***Symptom response*** |
| *Cord lesions* | Compression of spinal cord by bony or soft tissue due to degenerative, traumatic or pathological changes | Hyper-reflexes<br>Babinski positive<br>Bilateral/quadrilateral paraesthesia<br>Bilateral/quadrilateral weakness |
| *Cancer* | Growth of malignant tumour in or near vertebrae | Age > 55<br>History of cancer<br>Unexplained weight loss<br>Constant, progressive pain unrelated to loading strategy, not relieved by rest |
| *Fracture* | Bony damage to vertebrae caused by trauma or weakness due to metabolic bone disease | Significant trauma<br>Trivial trauma in individual with osteopenia |
| *Spinal infection* | Infection affecting vertebrae or disc | Systemically unwell<br>Febrile episode<br>Constant severe neck pain unrelated to loading strategy |
| *Ankylosing spondylitis* | One of the systemic inflammatory arthropathies affecting spinal and other structures | Lumbar, thoracic and cervical exacerbations and remissions<br>Marked morning stiffness<br>Persisting limitation all movements<br>No directional preference, but better with exercise, not relieved by rest<br>Systemic involvement<br>Raised ESR, + HLA B27 |

* The operational definitions provided below present the criteria in more detail. These give the symptom responses and time scale by which classification should be recognised.

**Classification algorithm for cervical spine**




Refer page 450 for classification algorithm for thoracic spine.

## Operational definitions

The operational definitions describe the symptom and mechanical behaviours and the time scale needed to document each category.

## Reducible Derangement

- Centralisation: in response to therapeutic loading strategies, pain is progressively abolished in a distal to proximal direction, *and*
- each progressive abolition is retained over time until all symptoms are abolished, *and*
- if back pain only is present, this moves from a widespread to a more central location and then is abolished, *or*
- pain is decreased and then abolished during the application of therapeutic loading strategies.
- The change in pain location, or decrease or abolition of pain remain better, *and*

- should be accompanied or preceded by improvements in the mechanical presentation (range of movement and/or deformity).

*Time scale*

- A derangement responder can be identified on day one, *or*
- a derangement responder will be suspected on day one and a provisional diagnosis made. This will be confirmed by a lasting change in symptoms after evaluating the response to a full mechanical evaluation within five visits.
- Decrease, abolition or centralisation of symptoms is occurring but the episode may not have completely resolved within five visits.
- Aggravating factors may precipitate deterioration in symptoms and a longer recovery process.

### Irreducible Derangement

- Peripheralisation of symptoms: increase or worsening of distal symptoms in response to therapeutic loading strategies, *and/or*
- no decrease, abolition or centralisation of pain.

*Time scale*

- An irreducible derangement patient will be suspected on day one and a provisional diagnosis made. This will be confirmed after evaluating the response to a full mechanical evaluation within five visits.

### Dysfunction

- Spinal pain only, *and*
- intermittent pain, *and*
- at least one movement is restricted, and the restricted movement consistently produces concordant pain at end-range, *and*
- there is no rapid reduction or abolition of symptoms, *and*
- no lasting production and no peripheralisation of symptoms.

### ANR

- History of cervical radiculopathy or surgery in the last few months that has improved, but is now unchanging, *and*
- symptoms are intermittent, *and*
- symptoms in the arm and/or forearm, including 'tightness', *and*
- upper limb tension test is clearly restricted and consistently produces concordant pain or tightness at end-range, *and*
- there is no rapid reduction or abolition of symptoms and no lasting production of distal symptoms.

*Time scale*

- A dysfunction/ANR category patient will be suspected on day one and a provisional diagnosis made. This is confirmed after evaluating the response to a mechanical evaluation within five visits.
- If the patient fails to fit all criteria another category must be considered.
- Rapid change will not occur in this syndrome, *and*
- symptoms gradually reduce over many weeks, as range of movement gradually improves.

## Postural

- Spinal pain only, *and*
- concordant pain only with static loading, *and*
- abolition of pain with postural correction, *and*
- no pain with repeated movements, *and*
- no loss of range of movement, *and*
- no pain during movement.

*Time scale*

- A posture category patient will be suspected on day one and a provisional diagnosis made. This will be confirmed after evaluating the response to a mechanical evaluation within two/three visits.
- If the patient fails to fit all criteria, another category must be considered.

'Other' categories are only considered on failure to enter a mechanical diagnosis within five treatment sessions. To be designated into 'Other' category patients will fulfil:

- 'Other' criteria, *and*
- criteria for specific 'Other' category as listed below.

## 'Other'

- No centralisation, peripheralisation or abolition of symptoms, *or*
- does not fit derangement, dysfunction or posture criteria.
- No lasting change in pain location or pain intensity in response to therapeutic loading strategies, *and*
- fulfils relevant criteria in suspected 'Other' pathology listed below.

## Indicators for possible 'red flags'

### Possible cancer

- age (>55)
- history of cancer
- unexplained weight loss
- constant, progressive pain not affected by loading strategies; worse at rest
- multiple, systemic symptoms.

### Other possible serious spinal pathology

One of the following:

- systemically unwell, *or*
- widespread neurology, *or*
- history of significant trauma enough to cause fracture, *or*
- dislocation (x-rays will not always detect fractures), *or*
- history of trivial trauma and severe pain in potential osteoporotic individual, *or*
- or sudden and persistent extremes of pain causing patient to 'freeze'.

### Possible inflammatory disorders

- gradual onset, *and*
- marked morning stiffness, *and*
- persisting limitation of movements in all directions
- peripheral joint involvement
- iritis, psoriasis, colitis, uretheral discharge
- family history.

### Stenosis

- age (>50)
- possible nerve root signs and symptoms
- extensive degenerative changes on x-ray
- extension provokes symptoms.

### Mechanically inconclusive

- symptoms affected by spinal movements
- no loading strategy consistently decreases, abolishes or centralises symptoms, nor increases or peripheralises symptoms
- inconsistent response to loading strategies.

### Chronic pain state

- persistent widespread symptoms
- all activity increases symptoms
- exaggerated pain behaviour
- mistaken beliefs and attitudes about pain and movement.

### Thoracic outlet syndrome

- diffuse neck/shoulder/arm symptoms of pain/paraesthesia
- provoked with raised arm activities
- positive concordant pain response to at least two TOS provocation tests.

# References




Abdi S, Datta S, Lucas LF (2005). Role of epidural steroids in the management of chronic spinal pain: a systematic review of effectiveness and complications. Pain Physician 8.127-143.

Abdulwahab SS, Sabbahi M (2000). Neck retractions, cervical root decompression, and radicular pain. JOSPT 30.4-12.

Abraham PJ, Capobianco DJ, Cheshire WP (2003). Facial pain as the presenting symptom of lung carcinoma with normal chest radiograph. Headache 43.499-504.

AHCPR (1994). *Agency for Health Care Policy and Research – Acute Low Back Problems in Adults.* In: Eds Bigos S, Bowyer O, Braen *et al*. Department of Health and Human Services, US.

Aina A, May S, Clare H (2004). The centralization phenomenon of spinal symptoms – a systematic review. Manual Therapy 9.134-143.

Aker PD, Gross AR, Goldsmith CH, Peloso P (1996). Conservative management of mechanical neck pain: systematic overview and meta-analysis. BMJ 313.1291-1296.

Akeson WH, Amiel D, Abel MF, Garfin SR, Woo SLY (1987). Effects of immobilisation on joints. Clin Orth & Rel Res 219.28-37.

Aldrich F (1990). Posterolateral microdiscectomy for cervical monoradiculopathy caused by posterolateral soft cervical disc sequestration. J Neurosurg 72.370-377.

Allen KL (1952). Neuropathies caused by bony spurs in the cervical spine with special reference to surgical treatment. J Neurol Neurosurg Psychiat 15.20-36.

Allen MJ, Barnes MR, Bodiwala GG (1985). The effect of seat belt legislation on injuries sustained by car occupants. Injury 16.471-476.

Altman DG (1991). *Practical Statistics for Medical Research*. Chapman & Hall, London.

Altman R, Brown M, Gargano G (1987). Low back pain in Paget's disease of bone. Clin Orth Rel Res 217.152-162.

Andersen JH, Kaergaard A, Frost P *et al*. (2002). Physical, psychosocial, and individual risk factors for neck/shoulder pain with pressure tenderness in the muscles among workers performing monotonous, repetitive work. Spine 27.660-667.

Andersson GBJ, Ortengren R, Nachemson AL, Elfstrom G, Broman H (1975). The sitting posture: an electromyographic and discometric study. Orth Clinics Nth Am 6.105-120.

Andersson GBJ, Murphy RW, Ortengren R, Nachemson AL (1979). The influence of backrest inclination and lumbar support on lumbar lordosis. Spine 4.52-58.

Andersson GBJ, Bostrom MPG, Eyre DR *et al*. (1997). Consensus summary on the diagnosis and treatment of osteoporosis. Spine 22.63S-65S.

Andersson HI, Ejlertsson G, Leden I, Rosenberg C (1993). Chronic pain in a geographically defined general population: studies of differences in age, gender, social class, and pain localization. Clin J Pain 9.174-182.

Antonaci F, Ghirmai S, Bono G, Sandrini G, Nappi G (2001). Cervicogenic headache: evaluation of the original diagnostic criteria. Cephalalgia 21.573-583.

Antony M (2000). Cervicogenic headache: prevalence and response to local steroid therapy. Clin Exp Rheumatol 18.2S.S59-S64.

APA (1988). Australian Physiotherapy Association. Protocol for pre-manipulative testing of the cervical spine. Aus J Physio 34.97-100.

Aprill C, Dwyer A, Bogduk N (1990). Cervical zygapophyseal joint pain patterns II: A clinical evaluation. Spine 15.458-461.

Aprill C, Bogduk N (1992). The prevalence of cervical zygapophyseal joint pain. A first approximation. Spine 17.744-747.

Aprill C, Axinn MJ, Bogduk N (2002). Occipital headaches stemming from the lateral atlanto-axial (C1-2) joint. Cephalalgia 22.15-22.

Arce CA, Dohrmann GJ (1985). Herniated thoracic disks. Neurologic Clinics 3.383-392.

Arem AJ, Madden JW (1976). Effects of stress on healing wounds. 1. Intermittent noncyclical tension. J Surg Res 20.93-102.

Ariens GAM, Borghouts JAJ, Koes B W (1999). Neck pain. In: Eds Crombie IK, Croft PR, Linton SJ, LeResche L, von Korff M. *Epidemiology of Pain*. IASP Press, Seattle.

Ariens GAM, Bongers PM, Hoogendoorn WE, Houtman ILD, van der Wal G, van Mechelen W (2001a). High quantitative job demands and low coworker support as risk factors for neck pain. Spine 26.1896-1903.

Ariens GAM, Bongers PM, Douwes M, Miedema MC, Hoogendoorn WE, van der Wal G, Bouter LM, van Mechelen W (2001b). Are neck flexion, neck rotation, and sitting at work risk factors for neck pain? Results of a prospective cohort study. Occup Environ Med 58.200-207.

Arntz A, Dreessen L, Merckelbach H (1991). Attention, not anxiety, influences pain. Behav Res Ther 29.41-50.

Arseni C, Nash F (1960). Thoracic intervertebral disc protrusion. J Neurosurg 17.418-430.

Ashby EC (1977). Abdominal pain of spinal origin. Ann Roy Coll Surg England 59.242-246.

Aspinall W (1989). Clinical testing for cervical mechanical disorders which produce ischemic vertigo. JOSPT 11.176-182.

Aspinall W (1990). Clinical testing for the craniovertebral hypermobility syndrome. J Orth Sports Phys Ther 12.47-54.

Assendelft WJJ, Bouter LM, Knipschild PG (1996). Complications of spinal manipulations: a comprehensive review of the literature. J Family Pract 42.475-480.

Atkinson R, Ghelman B, Tsaris P, Warren RF, Jacobs B, Lavyne M (1982). Sarcoidosis presenting as cervical radiculopathy. A case report and literature review. Spine 7.412-416.

Auld AW, Buerman A (1966). Metastatic spinal epidural tumors. An analysis of 50 cases. Archives of Neurology 15.100-108.

Awerbuch M (1995). Different concepts of chronic musculoskeletal pain. Ann Rheum Dis 54.331-332.

Bahra A, Goadsby P (2000). Chronic tension-type headache. In: Barton S, Adams C, Amore LGCA *et al. Clinical Evidence, Issue 4*. BMJ Publishing Group, London.

Balci AE, Balci TA, Cakir O, Eren S, Eren MN (2003). Surgical treatment of thoracic outlet syndrome: effect and results of surgery. Ann Thorac Surg 75.1091-1096.

Banerjee R, Palumbo MA, Fadale PD (2004). Catastrophic cervical spine injuries in the collision sport athlete, part 1. Epidemiology, functional anatomy, and diagnosis. Am J Sports Med 32.1077-1087.

Barker S, Kesson M, Ashmore J, Turner G, Conway J, Stevens D (2000). Guidance for pre-manipulative testing of the cervical spine. Manual Therapy 5.37-40.

Barlow Y, Willoughby J (1992). Pathophysiology of soft tissue repair. Br Med Bull 48.698-711.

Barnekow-Bergkvist M, Hedberg GE, Janlert U, Jansson E (1998). Determinants of self-reported neck-shoulder and low back symptoms in a general population. Spine 23.235-243.

Barnett GH, Hardy RW, Little JR, Bay JW, Sypert GW (1987). Thoracic spinal canal stenosis. J Neurosurg 66.338-344.

Barnsley L, Lord S, Bogduk N (1993a). Comparative local anaesthetic blocks in the diagnosis of cervical zygapophyseal joint pain. Pain 55.99-106.

Barnsley L, Lord S, Wallis B, Bogduk N (1993b). False-positive rates of cervical zygapophyseal joint blocks. Clin J Pain 9.124-130.

Barnsley L, Lord S, Bogduk N (1994a). Whiplash injury. Pain 58.283-307.

Barnsley L, Lord SM, Wallis BJ, Bogduk N (1994b). Lack of effect of intraarticular corticosteroids for chronic pain in the cervical zygapophyseal joints. New Eng J Med 330.10447-1050.

Barnsley L, Lord SM, Wallis BJ, Bogduk N (1995). The prevalence of chronic cervical zygapophyseal joint pain after whiplash. Spine 20.20-26.

Barnsley L, Lord SM, Bogduk N (2002). The pathophysiology of whiplash. In: Eds Malanga GA, Nadler SF. *Whiplash*. Hanley & Belfus, Inc, Philadelphia.

Barton D, Allen M, Finlay D, Belton I (1993). Evaluation of whiplash injuries by technetium 99m isotope scanning. Arch Emergency Med 10.197-202.

Bassols A, Bosch F, Campillo M, Canellas M, Banos JE (1999). An epidemiological comparison of pain complaints in the general population of Catalonia (Spain). Pain 83.9-16.

Bechgaard P (1981). Segmental thoracic pain patients admitted to a medical department and a coronary unit. Acta Medica Scand. Supplementum V.644.87-89.

Becker LA, Iverson DC, Reed FM, Calonge N, Miller RS, Freeman WL (1987). A study of headache in North American primary care. J Roy Coll Gen Pract 37.400-403.

Bednarik J, Kadanka Z, Vohanka S, Stejskal L, Vlach O, Schroder R (1999). The value of somatosensory- and motor-evoked potentials in predicting and monitoring the effect of therapy in spondylotic cervical myelopathy. Spine 24.1593-1598.

Bednarik J, Kadanka Z, Dusek L *et al.* (2004). Presymptomatic spondylotic cervical cord compression. Spine 29.2260-2269.

Beghi E, Monticelli ML, Amoruso L *et al.* (2003). Prevalence, characteristics, and patterns of health care use for chronic headache in two areas of Italy. Results of a questionnaire interview in general practice. Cephalalgia 23.175-182.

Bennell K, Khan K, McKay H (2000). The role of physiotherapy in the prevention and treatment of osteoporosis. Manual Therapy 5.198-213.

Bennell K, Larsen J (2004). Osteoporosis. In: Eds Boyling JD, Jull GA. *Grieve's Modern Manual Therapy* (3rd ed.). Churchill Livingstone, Edinburgh

Bergman S, Herrstom P, Hogstrom K, Petersson IF, Svensson B, Jacobsson LTH (2001). Chronic musculoskeletal pain, prevalence rates and sociodemographic associations in a Swedish population. J Rheumatol 28.1369-1377.

Berkow R, Fletcher AJ, Bondy PK *et al.* (1992). *The Merck Manual of Diagnosis and Therapy* (16th ed.). Merck & Co., New Jersey.

Bernat JL, Greenberg ER, Barrett J (1983). Suspected epidural compression of the spinal cord and cauda equina by metastatic carcinoma. Clinical diagnosis and survival. Cancer 51.1953-1957.

Bertalanffy H, Eggert HR (1988). Clinical long-term results of anterior discectomy without fusion for treatment of cervical radiculopathy and myelopathy. A follow-up of 164 cases. Acta Neurochir 90.127-135.

Bertilson BC, Grunnesjo M, Strender LE (2003). Reliability of clinical tests in the assessment of patients with neck/shoulder problems – impact of history. Spine 28.2222-2231.

Best RA (1999). Non-cardiac chest pain: a useful physical sign? Heart 81.450.

Bidese T, Buckley S, Cameron I *et al.* (2001). *Guidelines for the Management of Whiplash Associated Disorders.* Motor Accidents Authority, Sydney.

Bigal ME, Tepper SJ, Rapoport AM, Sheftell FD (2002a). Hemicrania continua: comparison between different classification systems. Cephalalgia 22.242-245.

Bigal ME, Sheftell FD, Rapoport AM, Lipton RB, Tepper SJ (2002b). Chronic daily headache in a tertiary care population: correlation between the International Headache Society diagnostic criteria and proposed revisions of criteria for chronic daily headache. Cephalalgia 22.432-438.

Bisschop P (2003). The Cervical Spine – Non-mechanical disorders. In: Ombregt L, Bisschop P, ter Veer HJ (2003). *A System of Orthopaedic Medicine* (2nd ed.). Churchill Livingstone, Edinburgh.

Bland JH (1993). Mechanisms of adaptation in the joint. In: Eds Crosbie J, McConnell J. *Key Issues in Musculoskeletal Physiotherapy*, 88-113. Butterworth Heinemann, Oxford.

Bland JH (1994). *Disorders of the Cervical Spine. Diagnosis and Management* (2nd ed.). W.B. Saunders Company, Philadelphia.

Bland JH (1998) Anatomy and pathology of the cervical spine. In: Giles LGF, Singer KP. *Clinical Anatomy and Management of Cervical Spine Pain*. Butterworth Heinemann, Oxford.

Blumenthal HJ, Weisz MA, Kelly KM, Mayer RL, Blonsky J (2003). Treatment of primary headache in the emergency department. Headache 43.1026-1031.

Blunt SB, Galton C (1997). Cervical carotid or vertebral dissection: an undiagnosed cause of stroke in the young. BMJ 314.243.

Boardman HF, Thomas E, Croft PR, Millson DS (2003). Epidemiology of headache in an English district. Cephalalgia 23.129-137.

Boden SD, Clark CR (1998). Rheumatoid arthritis of the cervical spine. In: Eds The Cervical Spine Research Society Editorial Committee. *The Cervical Spine*. Lippincott-Raven, Philadelphia.

Bogduk N (1982). The clinical anatomy of the cervical dorsal rami. Spine 7.319330.

Bogduk N (1986). The anatomy and pathophysiology of whiplash. ClinBiomech 1.92-101.

Bogduk N, Marsland A (1986). On the concept of third occipital headache. J Neurol Neurosurg Psych 49.775-780.

Bogduk N, Marsland A (1988). The cervical zygapophyseal joints as a source of neck pain. Spine 13.610-617.

Bogduk N, Windsor M, Inglis A (1988). The innervation of the cervical intervertebral disc. Spine 13.2-8.

Bogduk N (1993). The anatomy and physiology of nociception. In: Eds Crosbie J, McConnell J. *Key Issues in Musculoskeletal Physiotherapy*. Butterworth Heineman, Oxford.

Bogduk N, Aprill C (1993). On the nature of neck pain, discography and cervical zygapophyseal joint blocks. Pain 54.213-217.

Bogduk N (1994). Cervical causes of headache and dizziness. In: Eds Boyling JD, Palastanga N. *Grieve's Modern Manual Therapy* (2nd ed.). Churchill Livingstone, Edinburgh.

Bogduk N (1997). *Clinical Anatomy of the Lumbar Spine and Sacrum* (3rd ed). Churchill Livingstone, New York.

Bogduk N (2000). Whiplash: "Why pay for what does not work?" J Musculoskeletal Med 8.29-53.

Bogduk N (2001). Mechanism and pain patterns of the upper cervical spine. In: Ed. Vernon H. *The Cranio-Cervical Syndrome. Mechanism, Assessment and Treatment*. Butterworth Heinemann, Oxford.

Bogduk N (2002a). Biomechanics of the cervical spine. In: Ed. Grant R. *Physical Therapy of the Cervical and Thoracic Spine* (3rd ed.). Churchill Livingstone, New York.

Bogduk N (2002b). Innervation and pain patterns of the cervical spine. In: Ed. Grant R. *Physical Therapy of the Cervical and Thoracic Spine* (3rd ed.). Churchill Livingstone, New York.

Bogduk N (2002c). Innervation and pain patterns of the thoracic spine. In: Ed. Grant R. *Physical Therapy of the Cervical and Thoracic Spine* (3rd ed.). Churchill Livingstone, New York.

Bohlman HH (1979). Acute fracture and dislocation of the cervical spine. JBJS 61A.1119-1123.

Bohlman HH, Emery SE (1988). The pathophysiology of cervical spondylosis and myelopathy. Spine 13.843-6.

Bolton PS, Stick PE, Lord RSA (1989). Failure of clinical tests to predict cerebral ischemia before neck manipulation. J Manip Physiol Thera 12.304-307.

Bongers PM, de Winter CR, Kompier MAJ, Hildebrandt VH (1993). Psychosocial factors at work and musculoskeletal disease. Scand J Work Environ Health 19.297-312.

Bono G, Antonaci F, Ghirmai S *et al.* (1998). The clinical profile of cervicogenic headache as it emerges from a study based on the early diagnostic criteria (Sjaastad *et al.* 1990). Functional Neurology 13.75-77.

Bono G, Antonaci F, Ghirmai S *et al.* (2000). Whiplash injuries: clinical picture and diagnostic work-up. Clin Exp Rheumatol 18.S19.S23-S28.

Boon A, Smith J (2002). Whiplash associated disorders: prognosis after injury. In: Eds Malanga GA, Nadler SF. *Whiplash*. Hanley & Belfus, Inc., Philadelphia.

Borchgrevink GE, Kaasa A, McDonagh D, Stiles TC, Haraldseth O, Lereim I (1998). Acute treatment of whiplash neck sprain injuries. A randomized trial of treatment during the first 14 days after a car accident. Spine 23.25-31.

Borghouts JAJ, Koes BW, Bouter LM (1998). The clinical course and prognostic factors of non-specific neck pain: a systematic review. Pain 77.1-13.

Borghouts JAJ, Koes BW, Vondeling H, Bouter LM (1999). Cost-of-illness of neck pain in The Netherlands in 1996. Pain 80.629-636.

Boswell MV, Singh V, Staats PS, Hirsch JA (2003a). Accuracy of precision diagnostic blocks in the diagnosis of chronic spinal ain of facet or zygapophyseal joint origin: A systematic review. Pain Physician 6.449-456.

Boswell MV, Hansen HC, Trescot AM, Hirsch JA (2003b). Epidural steroids in the management of chronic spinal pain and radiculopathy. Pain Physician 6.319-334.

Bovim G, Schrader H, Sand T (1994). Neck pain in the general population. Spine 19.1307-1309.

Brattberg G, Thorslund M, Wikman A (1989). The prevalence of pain in a general population. The results of a postal survey in a county of Sweden. Pain 37.215-222.

Brismee JM, Gilbert K, Isom K *et al.* (2004). Rate of false positive using the Cyriax Release Test for thoracic outlet syndrome in an asymptomatic population. J Man Manip Thera 12.73-81.

Brison RJ, Hartling L, Pickett W (2000). A prospective study of acceleration-extension injuries following rear-end motor vehicle collisions. J Musculoskeletal Pain 8.97-113.

Brodsky AE (1985). Cervical angina. Spine 10.699-709.

Bronfort G, Assendelft WJJ, Evans R, Haas M, Bouter L (2001). Efficacy of spinal manipulation for chronic headache: a systematic review. J Manip Physiol Thera 24.457-466.

Bronfort G, Haas M, Evans RL, Bouter LM (2004). Efficacy of spinal manipulation and mobilization for low back pain and neck pain: a systematic review and best evidence synthesis. Spine Journal 4.335-356.

Brookfield S (2000). Clinical reasoning and generic thinking skills. In: Eds J Higgs, M Jones. *Clinical Reasoning in the Health Professions*. Butterworth Heinemann, Oxford.

Brown CW, Deffer PA, Akmakjian J, Donaldson DH, Brugman JL (1992). The natural history of thoracic disc herniation. Spine 17.S97-S102.

Buckwalter JA, Woo SLY, Goldberg VM *et al.* (1993). Soft-tissue aging and musculoskeletal function. JBJS 75A.1533-1548.

Bunketorp L, Stener-Victorin E, Carlsson J (2005). Neck pain and disability following motor vehicle accidents – a cohort study. Eur Spine J 14.84-89.

Buntinx F, Knockaert D, Bruyninckx R, de Blaey N, Aerts M, Knottnerus JA, Delooz H (2001). Chest pain in general practice or in the hospital emergency department: is it the same? Family Practice 18.586-589.

Burton AK (1981). Back pain in osteopathic practice. Rheum Rehabil 20.239-246.

Busch E, Wilson PR (1989). Atlanto-occipital and atlanto-axial injections in the treatment of headache and neck pain. Reg Anesth 14.S2.45.

Bush K, Chaudhuri R, Hillier S, Penny J (1997). The pathomorphological changes that accompany the resolution of cervical radiculopathy. A prospective study with repeat magnetic resonance imaging. Spine 22.183-187.

Butler D (1991). *Mobilisation of the Nervous System*. Churchill Livingstone, Melbourne.

Butler D (2000). *The Sensitive Nervous System*. Noigroup Publications, Adelaide.

Carey PF (1995). A suggested protocol for the examination and treatment of the cervical spine: managing the risk. J Can Chir Ass 39.35-39.

Carlsson CA, Nachemson A (2000). Surgical treatment of neck pain. In: Eds Nachemson AL, Jonsson E. *Neck and Back Pain. The Scientific Evidence of Causes, Diagnosis, and Treatment*. Lippincott Williams & Wilkins, Philadelphia.

Carragee EJ (1997). Pyogenic vertebral osteomyelitis. JBJS 79A.874-880.

Carrico TJ, Mehrhof AI, Cohen IK (1984). Biology of wound healing. Surg Clinics Nth Am 64.721-733.

Cassidy EM, Tomkins E, Hardiman O, O'Keane V (2003). Factors associated with burden of primary headache in a speciality clinic. Headache 43.638-644.

Cattrysse E, Swinkels RAHM, Oostendorp RAB, Duquet W (1997). Upper cervical instability: are clinical tests reliable? Manual Therapy 2.91-97.

Chen J, Solinger AB, Poncet JF, Lantz CA (1999). Meta-analysis of normative cervical motion. Spine 24.1571-1578.

Chen TY (2000). The clinical presentation of uppermost cervical disc protrusion. Spine 25.439-442.

Chester JB (1991). Whiplash, postural control, and the inner ear. Spine 16.716-720.

Chestnut JL (2004). The stroke issue: paucity of valid data, plethora of unsubstantiated conjecture. J Manip Physio Ther 27.368-372.

Childs JD, Fritz JM, Piva SR, Erhard RE (2003). Clinical decision making in the identification of patients likely to benefit from spinal manipulation: a traditional versus an evidence-based approach. JOSPT 33.259-272.

Childs JD, Fritz JM, Piva SR, Whitman JM (2004a). Proposal of a classification system for patients with neck pain. JOSPT 34.686-700.

Childs JD, Fritz JM, Flynn TW *et al*. (2004b). A clinical prediction rule to identify patients with low back pain most likely to benefit from spinal manipulation: a validation study. Ann Intern Med 141.920-928.

Chiu TTW, Lo SK (2002). Evaluation of cervical range of motion and isometric neck muscle strength: reliability and validity. Clinical Rehabilitation 16.851-858.

Chou LH, Lenrow DA (2002). Cervicogenic headache. Review article. Pain Physician 5.215-225.

Christensen HW, Vach W, Vach K *et al*. (2002). Palpation of the upper thoracic study: an observer reliability study. J Manipulative Physiol Ther 25.285-292.

Christensen N, Jones MA, Carr J (2002). Clinical reasoning in orthopaedic manual therapy.
(3rd ed.). Churchill Livingstone, New York.

Clare HA, Adams R, Maher CG (2004a). Reliability of the McKenzie spinal pain classification using patient assessment forms. Physiotherapy 90.114-119.

Clare HA, Adams R, Maher CG (2004b). A systematic review of efficacy of McKenzie therapy for spinal pain. Aus J Physio 50.209-216.

Clare H, Adams R, Maher CG (2004c). Reliability of McKenzie classification of patients with cervical or lumbar pain. J Manipulative Physiol Ther 28.122-127.

Clark CR (1991). Degenerative conditions of the spine. In: Ed. Frymoyer JW. *The Adult Spine: Principles and Practice*, Raven Press, New York.

Cloward RB (1959). Cervical discography. A contribution to the etiology and mechanism of neck, shoulder and arm pain. Annals of Surgery 150.1052-1064.

Colledge NR, Barr-Hamilton RM, Lewis SJ, Sellar RJ, Wilson JA (1996). Evaluation of investigations to diagnose the cause of dizziness in elderly people: a community based controlled study. BMJ 313.788-792.

Collins D (1956). Paget's disease of bone: incidence and subclinical forms. Lancet 2.51-54.

Comeaux Z, Eland D, Chila A, Pheley A, Tate M (2001). Measurement challenges in physical diagnosis: refining inter-rater palpation, perception and communication. J Bodywork Movement Therapies 5.245-253.

Conaty JP, Mongan ES (1981). Cervical fusion in rheumatoid arthritis. J Bone Joint Surg 63A.1218-1227.

Connell MD, Wiesel SW (1992). Natural history and pathogenesis of cervical disk diseases. Orth Clin Nth Am 23.369-380.

Connor PM, Darden BV (1993). Cervical discography complications and clinical efficacy. Spine 18.2035-2038.

Cote P, Kreitz BG, Cassidy JD, Thiel H (1996). The validity of the extension-rotation test as a clinical screening procedure before neck manipulation: a secondary analysis. J Manip Physiol Thera 19.159-164.

Cote P, Cassidy JD, Carroll L (1998). The Saskatchewan health and back pain survey. The prevalence of neck pain and related disability in Saskatchewan adults. Spine 15.1689-1698.

Cote P, Cassidy JD, Carroll L (2000). The factors associated with neck pain and its related disability in the Saskatchewan population. Spine 25.1109-1117.

Cote P, Cassidy JD, Carroll L (2001). The treatment of neck and low back pain. Who seeks care? Who goes where? Medical Care 39.956-967.

Cote P, Cassidy JD, Carroll LJ, Kristman V (2004). The annual incidence and course of neck pain in the general population: a population-based cohort study. Pain 112.267-273.

Cousins M (1994). Acute and postoperative pain. In: Eds Wall PD, Melzack R. *Textbook of Pain* (3rd ed.). Churchill Livingstone, Edinburgh.

Crandal PH, Batzdorf U (1966). Cervical spondylotic myelopathy. J Neurosurg 25.57-66.

Croft PR, Lewis M, Papageorgiou AC *et al.* (2001). Risk factors for neck pain: a longitudinal study in the general population. Pain 93.317-325.

CSAG (1994). *Clinical Standards Advisory Group: Back Pain*. HMSO, London.

Cummings SR, Melton LJ (2002). Epidemiology and outcomes of osteoporotic fractures. Lancet 359.1761-1767.

Currier BL, Heller JG, Eismont FJ (1998). Cervical spinal infections. In: Eds The Cervical Spine Research Society. *The Cervical Spine* (3rd ed.). Lippincott-Raven, Philadelphia.

Cyriax J (1982). *Textbook of Orthopaedic Medicine* (8th ed.). Bailliere Tindall, London.

Dabbs V, Lauretti W (1995). A risk assessment of cervical manipulation vs NSAIDs for the treatment of neck pain. J Manip Physiol Ther 18.530-536.

Daffner SD, Hilibrand AS, Hanscom BS, Brislin BT, Vaccaro AR, Albert TJ (2003). Impact of neck and arm pain on overall health status. Spine 28.2030-2035.

Dahlof C, Linde M (2001). One-year prevalence of migraine in Sweden: a population-based study in adults. Cephalalgia 2001.664-671.

Dalton M, Coutts A (1994). The effect of age on cervical posture in a normal population. In: Eds Boyling JD, Palastanga N. *Grieve's Modern Manual Therapy* (2nd ed.). Churchill Livingstone, Edinburgh.

Dalton PA, Jull GA (1989). The distribution and characteristics of neck-arm pain in patients with and without a neurological deficit. Aus J Physio 35.3-8.

D'Andrea G, Granella F, Verdilli F (2002). Migraine with aura triggered by orgasm. Cephalalgia 22.485-486.

Dartigues JF, Henry P, Puymirat E, Commenges D, Peytour P, Gagnon M (1988). Prevalence and risk factors of recurrent cervical pain syndrome in a working population. Neuroepidemiology 7.99-105.

Davis SJ, Teresi LM, Bradley WG, Ziemba MA, Bloze AE (1991). Cervical spine hyperextension injuries: MR findings. Radiology 180.245-251.

Daw H, Markman M (2000). Epidural spinal cord compression in cancer patients: diagnosis and management. Cleveland Clinic Journal of Medicine 67.497, 501-504.

Deans GT, Magalliard JN, Kerr M, Rutherford WH (1987). Neck sprain – a major cause of disability following car accidents. Injury 18.10-12.

DeBoer KF, Harmon R, Tuttle CD, Wallace H (1985). Reliability study of detection of somatic dysfunctions in the cervical spine. J Manip Physiol Thera 8.9-16.

Deltoff MN (2001). Diagnostic imaging of the cranio-cervical region. In: Ed. Vernon H. *The Cranio-Cervical Syndrome. Mechanisms, Assessment and Treatment*. Butterworth Heinemann, Oxford.

Derby R, Chen Y, Lee SH, Seo KS, Kim BJ (2004). Non-surgical interventional treatment of cervical and thoracic radiculopathies. Pain Physician 7.389-394.

Derrick LJ, Chesworth BM (1992). Post-motor vehicle accident alar ligament laxity. JOSPT 16.6-11.

Deyo RA, Rainville J, Kent DL (1992). What can the history and physical examination tell us about low back pain? JAMA 268.70-765.

Dieck GS, Kelsey JL, Goel VK, Panjabi MM, Walter SD, Laprade MH (1985). An epidemiological study of the relationship between postural asymmetry in the teen years and subsequent back and neck pain. Spine 10.872-877.

Di Fabio RP (1999). Manipulation of the cervical spine: risks and benefits. Physical Therapy 79.50-65.

Dionne C, Bybee R (2003). Interrater reliability of McKenzie assessment of patients with cervical pain. The McKenzie Institute 8th International Conference, Rome, September 2003.

Disla E, Rhim HR, Reddy A, Karten I, Taranta A (1994). Costochondritis. A prospective analysis in an emergency department setting. Arch Intern Med 154.2466-2469.

Dodick DW, Rozen TD, Goadsby PJ, Silberstein SD (2000). Cluster headache. Cephalalgia 20.787-803.

Dolan P, Adams MA, Hutton WC (1988). Commonly adopted postures and their effect on the lumbar spine. Spine 13.197-201.

Donatelli R, Owens-Burkhart H (1981). Effects of immobilization on the extensibility of periarticular connective tissue. JOSPT 3.67-72.

Donelson R, Silva G, Murphy K (1990). Centralisation phenomenon. Its usefulness in evaluating and treating referred pain. Spine 15.211 -213.

Donelson R, Grant W, Kamps C, Medcalf R (1991). Pain response to sagittal end-range spinal motion. A prospective, randomised, multicentered trial. Spine 16.S206-S212.

Donelson R, Grant W, Kamps C, Richman P (1997). Cervical and referred pain response to repeated end-range testing: a prospective, randomised trial. North American Spine Society, New York.

Downs SE (1990). Bronchogenic carcinoma presenting as neuromusculoskeletal pain. J Manip Physiol Thera 13.221-224.

Dreyfuss P, Tibiletti C, Dreyer SJ (1994a). Thoracic zygapophyseal joint pain patterns. A study in normal volunteers. Spine 19.807-811.

Dreyfuss P, Michaelsen M, Fletcher D (1994b). Atlanto-occipital and lateral atlanto-axial joint pain patterns. Spine 19.1125-11 31.

Dreyfuss P, Tibiletti C, Dreyer S, Sobel J (1994c). Thoracic zygapophyseal joint pain: a review and description of an intra-articular block. Pain Digest 4.46-54.

Dubner R (1991). Neuronal plasticity and pain following peripheral tissue inflammation or nerve injury. In: Eds Bond MR, Charlton JE, Woolf CJ. *Proceedings of the Sixth World Congress on Pain*. Elsevier Publishers BV., Philadelphia..

Dunne J (2001). Pre-manipulative testing: predicting risk or pretending to? Aus J Physio 47.165.

Durr HR, Wegener B, Krodel A, Muller PE, Jansson V, Refior HJ (2002). Multiple myeloma surgery of the spine. Spine 27.320-326.

Dvorak J, Froehlich D, Penning L, Baumgartner H, Panjabi MM (1988). Functional radiographic diagnosis of the cervical spine: flexion/extension. Spine 13.748-755.

Dwyer A, Aprill C, Bogduk N (1990). Cervical zygapophyseal joint pain patterns II: A study in normal volunteers. Spine 15.453-457.

Edmondston SJ (2004). Clinical biomechanics of the thoracic spine including the ribcage. In: Eds Boyling JD, Jull GA. *Grieve's Modern Manual Therapy* (3rd ed.). Churchill Livingstone, Edinburgh.

Edwards CC, Riew KD, Anderson PA, Hillibrand AS, Vaccaro AF (2003). Cervical myelopathy: current diagnostic and treatment strategies. Spine Journal 3.68-81.

Elvey RL (1986). Treatment of arm pain associated with abnormal brachial plexus tension. Aus J Physio 32.225-230.

Enwemeka CS (1989). Inflammation, cellularity, and fibrillogenesis in regenerating tendon: implications for tendon rehabilitation. Physical Therapy 69.816-825.

Epstein JA, Epstein BS, Lavine LS, Caarras R, Rosenthal AD (1978). Cervical myeloradiculopathy caused by arthrotic hypertrophy of the posterior facets and laminae. J Neurosurg 49.387-392.

Ernst E (2004). Cerebrovascular complications associated with spinal manipulation. Phys Ther Rev 9.5-15.

Errico TJ, Stecker S, Kostuik JP (1997). Thoracic pain syndromes. In: Ed. Frymoyer JW. *The Adult Spine: Principles and Practice* (2nd ed.). Lippincott-Raven, Philadelphia.

Evans P (1980). The healing process at cellular level: a review. Physiotherapy 66.256-259.

Famularo G, Polchi S, Tarroni P (2002). Headache as a presenting symptom of acute myocardial infarction. Headache 42.1025-1028.

Farmer JC, Wisneski RJ (1994). Cervical spine nerve root compression. An analysis of neuroforaminal pressures with varying head and arm positions. Spine 19.1850-1855.

Feinstein B, Langton JBK, Jameson RM, Schiller F (1954). Experiments on referred pain from deep somatic structures. J Bone Joint Surg 36A.981-988.

Ferguson RJ, Caplan LR (1985). Cervical spondylotic myelopathy. Neurol Clin 3.373-382.

Ferrari R, Russell AS (1997). Editorial. The whiplash syndrome – common sense revisited. J Rheum 24.618-623.

Ferrari R, Russell AS (1999). Epidemiology of whiplash: an international dilemma. Ann Rheum Dis 58.1-5.

Findlay GF, Eisenstein S (2000). Surgical treatment of diseases and trauma of the thoracic spine. In: Eds Giles LGF, Singer KP. *Clinical Anatomy and Management of Thoracic Spine Pain*. Butterworth Heinemann, Oxford.

Fishbain DA, Cutler R, Cole B, Rosomoff HL, Rosomoff RS (2001). International Headache Society headache diagnostic patterns in pain facility patients. Clin J Pain 17.78-93.

Fjellner A, Bexander C, Faleij R, Strender LE (1999). Interexaminer reliability in physical examination of the cervical spine. J Manip Physiol Thera 22.511-516.

Fouyas IP, Statham PFX, Sandercock PAG (2002). Cochrane review on the role of surgery in cervical spondylotic radiculomyelopathy. Spine 27.736-747.

Frank C, Woo SLY, Amiel D, Harwood F, Gomez M, Akeson W (1983). Medial collateral ligament healing. A multidisciplinary assessment in rabbits. Am J Sports Med 11.379-389.

Freeman MD, Croft AC, Rossignol AM (1998). "Whiplash associated disorders: redefining whiplash and its management" by the Quebec Task Force. A critical evaluation. Spine 23.1043-1049.

Freeman MD, Croft AC, Rossignol AM, Weaver DS, Reiser M (1999). A review and methodological critique of the literature refuting whiplash syndrome. Spine 24.86-98.

Friedenberg ZB, Miller WT (1963). Degenerative disc disease of the cervical spine. A comparative study of asymptomatic and symptomatic patients. JBJS 45A.1171-1178.

Fritz JM, Delitto A, Erhard RE (2003). Comparison of classification-based physical therapy with therapy based on clinical practice guidelines for patients with acute low back pain. A randomized clinical trial. Spine 28.1363-1372.

Fujiwara K, Owaki H, Fujimto M, Yonenobu K, Ochi T (2000). A long-term follow-up study of cervical lesions in rheumatoid arthritis. J Spinal Dis 13.519-526.

Furman JM, Whitney SL (2000). Central causes of dizziness. Physical Therapy 80.179-187.

Galasko CSB, Murray PM, Pitcher M *et al.* (1993). Neck sprains after road traffic accidents: a modern epidemic. Injury 24.155-157.

Galea MP (2002). Neuroanatomy of the nociceptive system. In: Eds Strong J, Unruh AM, Wright A, Baxter GD. Pain. *A Textbook for Therapists*. Churchill Livingstone, Edinburgh.

Gallai V, Sarchielli P, Alberti A *et al.* (2002). Application of the 1988 International Headache Society diagnostic criteria in nine Italian headache centers using a computerized structured record. Headache 42.1016-1024.

Gamsa A (1994a). The role psychological factors in chronic pain. I. A half century of study. Pain 57.5-15.

Gamsa A (1994b). The role psychological factors in chronic pain. II. A critical appraisal. Pain 57.17-29.

Gargan MF, Bannister GC (1990). Long-term prognosis of soft-tissue injuries of the neck. JBJS 72B.901-903.

Gargan M, Bannister G, Main C, Hollis S (1997). The behavioural response to whiplash injury. JBJS 79B.523-526.

Gatterman MI (1998). Chiropractic management of neck pain of mechanical origin. In: Eds Giles LGF, Singer KP. *The Clinical Anatomy and Management of Back Pain Series. Volume 3: Clinical Anatomy and Management of Cervical Spine Pain*. Butterworth Heinemann, Oxford.

George SZ, Fritz JM, Erhard RE (2001). A comparison of fear-avoidance beliefs in patients with lumbar spine pain and cervical spine pain. Spine 26.2139-2145.

Gerber C, Galantay RV, Hersche O (1998). The pattern of pain produced by irritation of the acromioclavicular joint and the subacromial space. J Shoulder Elbow Surg 7.352-355.

Gergoudis R, Barnes RW (1980). Thoracic outlet arterial compression: prevalence in normal persons. Angiology 31.538-541.

Giacobetti FB, Vaccaro AR, Bos-Giacobetti MA *et al.* (1997). Vertebral artery occlusion associated with cervical spine trauma. A prospective analysis. Spine 22.188-192.

Gibson T, Bogduk N, MacPherson J, McIntosh A (2000). Crash characteristics of whiplash associated chronic neck pain. J Musculoskeletal Pain 8.87-95.

Gillard J, Perez-Cousin M, Hachulla E *et al.* (2001). Diagnosing thoracic outlet syndrome: contribution of provocative tests, ultrasonography, electrophysiology, and helical computed tomography in 48 patients. Joint Bone Spine 68.416-424.

Gobel H (2001). Classification of headaches. Cephalalgia 21.770-773.

Gooch L, Lee HB, Twomey LT (1991). In vivo creep of the cervical spine. Proceedings of the 7th Biennial Conference of the Manipulative Physiotherapists of Australia, Blue Mountains, New South Wales, November 27-30, 1991.

Gordan SJ, Trott P, Grimmer KA (2002). Waking cervical pain and stiffness, headache, scapular or arm pain: gender and age effects. Aus J Physio 48.9-15.

Gore DR, Sepic SB (1984). Anterior cervical fusion for degenerated or protruded discs. A review of 146 patients. Spine 9.667-671.

Gore DR, Sepic SB, Gardner GM (1986). Roentgenographic findings of the cervical spine in asymptomatic people. Spine 11.521-524.

Gore DR, Sepic SB, Gardner GM, Murray MP (1987). Neck pain: a long-term follow-up of 205 patients. Spine 12.1-5.

Graat HCA, van Rhijn LW, Schrander-Stumpel CTRM, van Ooij A (2002). Classical Scheuermann disease in male monozygotic twins. Spine 27.E485-E487.

Grant R (1994a). Vertebral artery concerns: premanipulative testing of the cervical spine. In: Ed. Grant R. *Physical Therapy of the Cervical and Thoracic Spine* (2nd ed.). Churchill Livingstone, New York.

Grant R (1994b). Vertebral artery insufficiency: a clinical protocol for pre-manipulative testing of the cervical spine. In: Eds Boyling JD, Palastanga N. *Grieve's Modern Manual Therapy* (2nd ed.). Churchill Livingstone, Edinburgh.

Grant R (2002). Premanipulative testing of the cervical spine – reappraisal and update. In: Ed. Grant R. *Physical Therapy of the Cervical and Thoracic Spine* (3rd ed.). Churchill Livingstone, New York.

Gray JAM (1997). *Evidence-based Healthcare. How to Make Health Policy and Management Decisions*. Churchill Livingstone, New York.

Grayson MF (1987). Horner's syndrome after manipulation of the neck. Br Med J 295.1381-1382.

Gregorious FK, Estrin T, Crandall PH (1976). Cervical spondylotic radiculopathy and myelopathy. Arch Neurol 33.618-625.

Gregory PL, Biswas AC, Batt ME (2002). Musculoskeletal problems of the chest wall in athletes. Sports Med 32.235-250.

Grieco A, Molteni G, de Vito G, Sias N (1998). Epidemiology of musculoskeletal disorders due to biomechanical load. Ergonomics 41.1253-1260.

Griegel-Morris P, Larson K, Mueller-Klaus K, Oatis CA (1992). Incidence of common postural abnormalities in the cervical, shoulder, and thoracic regions and their association with pain in two age groups of healthy subjects. Physical Therapy 72.425-431.

Grieve GP (1991). *Mobilisation of the Spine* (5th ed.). Churchill Livingstone, Edinburgh.

Grieve GP (1994). Thoracic musculoskeletal problems. In: Eds Boyling JD, Palastanga N. *Grieve's Modern Manual Therapy*. Churchill Livingstone, Edinburgh.

Grimmer K (1996). The relationship between cervical resting posture and neck pain. Physiotherapy 82.45-51.

Groen GJ, Baljet B, Drukker J (1988). The innervation of the spinal dura mater: anatomy and clinical implications. Acta Neurochirurgica 92.39-46.

Groen GJ, Baljet B, Drukker J (1990). Nerves and nerve plexuses of the human vertebral column. Am J Anatomy 188.282-296.

Gross AR, Kay TM (2001). Guidelines for pre-manipulative testing of the cervical spine – an appraisal. Aus J Physio 47.166-167.

Gross AR, Kay T, Hondras M *et al.* (2002). Manual therapy for mechanical neck disorders: a systematic review. Manual Therapy 7.131-149.

Gross AR, Hoving JL, Haines TA *et al.* (2004). A Cochrane review of manipulation and mobilization for mechanical neck disorders. Spine 29.1541-1548.

Grubb SA, Kelly CK (2000). Cervical discography: Clinical implications from 12 years of experience. Spine 25.1382-1389.

Guez M, Hildingsson C, Stegmayr B, Toolanen G (2003). Chronic neck pain of traumatic and non-traumatic origin. A population-based study. Acta Orthop Scand 74.576-579.

Hagen KB, Kvien TK, Bjorndal A (1997a). Musculoskeletal pain and quality of life in patients with noninflamatory joint pain compared to rheumatoid arthritis: a population survey. J Rheumatol 24.1703-1709.

Hagen KB, Harms-Ringdahl K, Enger NO, Hedenstad R, Morten H (1997b). Relationship between subjective neck disorders and cervical spine mobility and motion-related pain in male machine operators. Spine 22.15011-1507.

Hagen K, Zwart JA, Vatten L, Stovner LJ, Bovim G (2000). Prevalence of migraine and non-migrainous headache – head-HUNT, a large population-based study. Cephalalgia 20.900-906.

Haldeman S, Kohlbeck FJ, McGregor M (1999). Risk factors and precipitating neck movements causing vertebrobasilar artery dissection after cervical trauma and spinal manipulation. Spine 24.785-794.

Haldeman S, Dagenais S (2001). Cervicogenic headaches: a critical review. Spine Journal 1.31-46.

Haldeman S, Kohlbeck FJ, McGregor M (2002). Unpredictability of cerebrovascular ischemia associated with cervical spine manipulation. A review of 64 cases after cervical spine manipulation. Spine 27.49-55.

Haldorsen EMH, Grasdal AL, Skouen JS, Risa AE, Kronholm K, Ursin H (2002). Is there a right treatment for a particular patient group? Comparison of ordinary treatment, light multidisciplinary treatment, and extensive multidisciplinary treatment for long-term sick-listed employees with musculoskeletal pain. Pain 95.49-63.

Hall MC (1965). *Luschka's Joint*. Thomas, Springfield.

Hanten WP, Olson SL, Russell JL, Lucio RM, Campbell AH (2000). Total head excursion and resting head posture: normal and patient comparisons. Arch Phys Med Rehab 81.62-66.

Hanten WP, Olson SL, Ludwig GM (2002). Reliability of manual mobility testing of the upper cervical spine in subjects with cervicogenic headache. J Manual Manip Thera 10. 76-82.

Harder S, Veilleux M, Suissa S (1998). The effect of socio-demographic and crash-related factors on the prognosis of whiplash. J Clin Epidemiol 51.377-384.

Hardy MA (1989). The biology of scar formation. Physical Therapy 69.1014-1024.

Hargreaves C, Cooper C, Kidd BL, Ellis R, Cawley MID (1989). Frozen shoulder and cervical spine disease. Br J Rheum 28.78-79.

Harms-Ringdahl K (1986). On assessment of shoulder exercise and load-elicited pain in the cervical spine. Scand J Rehab Med S14.1-40.

Harrison DD, Harrison SO, Croft AC, Harrison DE, Troyanovich SJ (1999). Sitting biomechanics part 1: review of the literature. J Manip Physiological Therapeutics 22.594-609.

Hartling L, Brison RJ, Ardern C, Pickett W (2001). Prognostic value of the Quebec classification of whiplash associated disorders. Spine 26.36-41.

Hasse LA, Ritchey N, Smith R (2002). Predicting the number of headache visits by type of patient seen in family practice. Headache 42.738-746.

Hasvold T, Johnsen R (1993). Headache and neck or shoulder pain – frequent and disabling complaints in the general population. Scand J Prim Health Care 11.219-224.

Haughie LJ, Fiebert IM, Roach KE (1995). Relationship of forward head posture and cervical backward bending to neck pain. J Manual Manip Thera 3.91-97.

Henderson CM, Hennessy RG, Shuey HM, Shackelford EG (1983). Posterior-lateral foraminotomy as an exclusive operative technique for cervical radiculopathy: a review of 846 consecutively operated cases. Neurosurg 13.504-511.

Herkowitz HN, Rothman RH (1984). Subacute instability of the cervical spine. Spine 9.348-357.

Hermann KM, Reese CS (2001). Relationships among selected measures of impairment, functional limitation, and disability inpatients with cervical spine disorders. Physical Therapy 811.903-914.

Hildingsson C, Toolanen G (1990). Outcome after soft-tissue injury of the cervical spine. A prospective study of 93 car-accident victims. Acta Orthop Scand 61.357-359.

Hill J, Lewis M, Papageorgiou AC, Dziedzic K, Croft P (2004). Predicting persistent neck pain. A 1-year follow-up of a population cohort. Spine 29.1648-1654.

Hilton RC, Ball J, Benn RT (1976). Vertebral end-plate lesions (Schmorl's nodes) in the dorsolumbar spine. Ann Rheum Dis 35.127-132.

Hilton RC, Ball J (1984). Vertebral rim lesions in the dorsolumbar spine. Ann Rheum Dis 43.302-307.

Hinkley HJ, Drysdale IP (1995). Audit of 1000 patients attending the clinic of the British College of Naturopathy and Osteopathy. Br Osteo J 16.17-22.

Ho KH, Ong BKC (2003). A community-based study of headache diagnosis and prevalence in Singapore. Cephalalgia 23.6-13.

Hohl M (1974). Soft-tissue injuries of the neck in automobile accidents. JBJS 56A.1675-1682.

Honet JC, Ellenberg MR (2003). What you always wanted to know about the history and physical examination of neck pain but were afraid to ask. Phys Med Rehab Clin Nth Am 14.473-491.

Horneij E, Hemborg B, Johnsson B, Ekdahl C (2002). Clinical tests on impairment level related to low back pain: a study of test reliability. J Rehabil Med 34.176-182.

Hove B, Gyldensted C (1990). Cervical analgesic joint arthrography. Neuroradiology 32.456-459.

Hoving JL, Gross AR, Gasner D *et al.* (2001). A critical appraisal of review articles on the effectiveness of conservative treatment for neck pain. Spine 26.196-205.

Hoving JL, de Vet HCW, Twisk JWR *et al.* (2004). Prognostic factors for neck pain in general practice. Pain 110.639-645.

Huang JH, Zager EL (2004). Thoracic outlet syndrome. Neurosurgery 55.897-903.

Hubka MJ, Phelan SP (1994). Interexaminer reliability of palpation for cervical spine tenderness. J Manip Physiol Thera 17.591-595.

Huler RJ (1997). Thoracolumbar spine fracture. In: Ed. Frymoyer JW. *The adult spine: principles and practice* (2nd ed.). Lippincott-Raven, Philadelphia.

Hunter G (1994). Specific soft tissue mobilisation in the treatment of soft tissue lesions. Physiotherapy 80.15-21.

Hurwitz EL, Aker PD, Adams AH, Meeker WC, Shekelle PG (1996). Manipulation and mobilization of the cervical spine. A systematic review of the literature. Spine 21.1746-1760.

Iai H, Moriya H, Goto S, Takahashi K, Yamagata M, Tamaki T (1993). Three-dimensional motion analysis of the upper cervical spine during axial rotation. Spine 18.2388-2392.

Ide J, Kataoka Y, Yamaga M, Kitmura T, Takagi K (2003). Compression and stretching of the brachial plexus in thoracic outlet syndrome: correlations between neuroradiographic findings and symptoms and signs produced by provocation manoeuvres. J Hand Surg 28B.218-223.

Ignatius YTS, Yee TY, Yan LT (1993). Self-reported musculoskeletal problems amongst typists and possible risk factors. J Human Ergol 22.83-93.

Inman VT, Saunders JBCM (1947). Anatomicophysiological aspects of injuries to the intervertebral disc. JBJS 29.461-475.

International Headache Society (IHS) 1988. Headache Classification Committee of the International Headache Society. Classification and diagnostic criteria of headache disorders, cranial neuralgias and facial pain. Cephalalgia 8.S7.1-96.

Irnich D, Behrens N, Molzen H *et al.* (2001). Randomised trial of acupuncture compared with conventional massage and "sham" laser acupuncture for treatment of chronic neck pain. BMJ 322.1574-1578.

Irnich D, Behrens N, Gleditsch JM *et al.* (2002). Immediate effects of dry needling and acupuncture at distant points in chronic neck pain: results of a randomised, double-blind, sham-controlled crossover trial. Pain 99.83-89.

Ishii T, Mukai Y, Hosono N *et al.* (2004). Kinematics of the subaxial cervical spine in rotation *in vivo* three-dimensional analysis. Spine 29.2826-2831.

Isu T, Iwasaki Y, Miyasaka K, Abe H, Tashiro K, Ito T (1986). A reappraisal of the diagnosis in cervical disc disease: the posterior longitudinal ligament perforated or not. Neuroradiology 28.215-220.

Jensen MV, Tuchsen F, Orhede E (1996). Prolapsed cervical intervertebral disc in male professional drivers in Denmark, 1981-1990. A longitudinal study of hospitalizations. Spine 21.2352-2355.

Johnson MI (1997). The physiology of the sensory dimensions of clinical pain. Physiotherapy 83.526-536.

Jones M, Butler D (1991). Clinical reasoning. In: Butler DS, *Mobilisation of the Nervous System*. Churchill Livingstone, Melbourne.

Jones MA (1992). Clinical reasoning in manual therapy. Physical Therapy 72.875-884.

Jones MA, Christensen N, Carr J (1994). Clinical reasoning in orthopaedic manual therapy. In: Ed. Grant R. *Physical Therapy of the Cervical and Thoracic Spine* (2nd ed.). Churchill Livingstone, New York.

Jones MA, Rivett DA (2004). Introduction to clinical reasoning. In: Eds Jones MA, Rivett DA. *Clinical Reasoning for Manual Therapists*. Butterworth Heinemann, Edinburgh.

Jones N (1998). Surgical management of neck pain of mechanical origin. In: Eds Giles LGF, Singer KP. *Clinical Anatomy and Management of Cervical Spine Pain*. Butterworth Heinemann, Oxford.

Jonsson H, Cesarini K, Sahlstedt B, Rauschning W (1994). Findings and outcome in whiplash-type neck distortions. Spine 19.2733-2743.

Jordan A, Mehlsen J, Ostergaard K (1997). A comparison of physical characteristics between patients seeking treatment for neck pain and age-matched healthy people. J Manipulative and Physiol Ther 20.468-475.

Jull G, Bogduk N, Marsland A (1988). The accuracy of manual diagnosis for zygapophysial joint pain syndromes. Med J Aus 148.233-236.

Jull G, Zito G, Trott P, Potter H, Shirley D, Richardson C (1997). Inter-examiner reliability to detect painful upper cervical joint dysfunction. Aus J Physio 43.125-129.

Jull GA (2002). Management of cervicogenic headaches. In: Ed. Grant R. *Physical Therapy of the Cervical and Thoracic Spine* (3rd ed.). Churchill Livingstone, New York.

Jull GA, Niere KR (2004). The cervical spine and headache. In: Eds Boyling JD, Jull GA. *Grieve's Modern Manual Therapy* (3rd ed.). Churchill Livingstone, Edinburgh.

Kadanka Z, Mares M, Bednarik J *et al.* (2002). Approaches to spondylotic cervical myelopathy. Conservative versus surgical results in a 3-year follow-up study. Spine 27.2205-2211.

Kalfas IH (2000). Laminectomy for thoracic spinal canal stenosis. Neurosurg Focus 9.1-3.

Kamwendo K, Linton SJ (1991). A controlled study of the effect of neck school in medical secretaries. Scand J Rehabil Med 23.143-152.

Kanis JA, Pitt FA (1992). Epidemiology of osteoporosis. Bone 13.S7-15.

Karjalainen K, Malmivaara A, van Tulder M *et al.* (2001). Multidisciplinary biopsychosocial rehabilitation for neck and shoulder pain among working age adults. Spine 26.174-181.

Kauppi M, Hakala M (1994). Prevalence of cervical spine subluxations and dislocations in a community-based rheumatoid arthritis population. Scand J Rheum 23.133-136.

Keegan JJ (1953). Alterations of the lumbar curve related to posture and seating. JBJS 35A.589-603.

Kellgren JH (1939). On the distribution of pain arising from deep segmental somatic structures with charts of segmental pain areas. Clin Sci 4.35-46.

Kelsey JL, Githens PB, Walter SD *et al.* (1984). An epidemiological study of acute prolapsed cervical intervertebral disc. JBJS 66A.907-914.

Kenneally M, Rubenach H, Elvey R (1988). The upper limb tension test: the SLR of the arm. In: Ed. Grant R. *Physical Therapy of the Cervical and Thoracic Spine*. Churchill Livingstone, New York.

Kerry R (2005). Haemodynamics and pain: the role of VBI testing. Physiotherapy Research Society Scientific Meeting. Nottingham, UK, April, 2005.

Kilbom A, Persson J, Jonsson BG (1986). Disorders of the cervicobrachial region among female workers in the electronic industry. Int J Ind Ergonomics 1.37-47.

King V (1998). Irritable bowel syndrome: a case for musculoskeletal assessment. PhD Thesis, Loughborough University, Loughborough. In: Sparkes V (2004). Abdominal pain of musculoskeletal origin. In: Eds Boyling JD, Jull GA. *Grieve's Modern Manual Therapy* (3rd ed.). Churchill Livingstone, Edinburgh.

Kitagawa T, Fujiwara A, Kobayashi N, Saiki K, Tamai K, Saotome K (2004). Morphologic changes in the cervical neural foramen due to flexion and extension. Spine 29.2821-2825.

Kjellman GV, Skargren EI, Oberg BE (1999). A critical analysis of randomised clinical trials on neck pain and treatment efficacy. A review of the literature. Scand J Rehab Med 31.1 39-152.

Kjellman G, Oberg B, Hensing G, Alexanderson (2001). A 12-year follow-up of subjects initially sicklisted with neck/shoulder or low back diagnoses. Physio Research Int 6.52-63.

Kjellman G, Oberg B (2002). A randomized clinical trial comparing general exercise. McKenzie treatment and a control group in patients with neck pain. J Rehabil Med 34.183-190.

Kjellman G, Skargren E, Oberg B (2002). Prognostic factors for perceived pain and function at one-year follow-up in primary care patients with neck pain. Disability Rehab 24.364-370.

Kjellman G, Oberg B (2004). A comparative study of objective outcomes after three different treatments in patients with neck pain. A prospective randomized clinical trial. International Forum for Primary Care Research in Low Back Pain. Edmonton, Alberta, October, 2004.

Klaber Moffett JA, Richardson PH (1995). The influence of psychological variables on the development and perception of musculoskeletal pain. Physio Theory & Pract 11.3-11.

Kobayashi N, Asamoto S, Doi H, Ikeda Y, Matusmoto K (2003). Spontaneous regression of herniated cervical disc. Spine Journal 3.171-173.

Kokobun S, Tanaka Y (1995). Types of cervical disc herniation and relation to myelopathy and radiculopathy. J Back Musculoskeletal Rehab 5.145-154.

Kokobun S, Sakurai M, Tanaka Y (1996). Cartilaginous endplate in cervical disc herniation. Spine 21.190-195.

Kostuik JP, Heggeness MH (1997). Surgery of the osteoporotic spine. In: Ed. Frymoyer JW. *The adult spine: principles and practice* (2nd ed.). Lippincott-Raven, Philadelphia.

Kovach SG, Huslig EL (1984). Shoulder pain and Pancoast tumor: a diagnostic dilemma. J Manip Physiol Thera 7.25-31.

Kramer J (1990). *Intervertebral Disk Disease. Causes, Diagnosis, Treatment and Prophylaxis.* Thieme Medical Publishers, Inc., New York.

Kreig L (1993). Thoracic outlet syndrome: pathology and treatment. J Man Manip Thera 1.52-59.

Kroeling P, Gross A, Goldsmith CH, Houghton PE, Cervical overview group (2005). Electrotherapy for neck disorders. *The Cochrane Database of Systematic Reviews 2005*, Issue 2. Art. No: CD004251.pub3.

Krogsgaard MR, Wagn P, Bengtsson J (1998). Epidemiology of acute vertebral osteomyelitis in Denmark. Acta Orthop Scand 69.513-517.

Kunnasmaa KTT, Thiel HW (1994). Vertebral artery syndrome: a review of the literature. J Orthopaedic Med 16.17-20.

Kuslich SD, Ulstrom CL, Michael CJ (1991). The tissue origin of low back pain and sciatica: A report of pain response to tissue stimulation during operations on the lumbar spine using local anaesthesia. Orth Clin Nth Am 22.181-187.

Lahad A, Malter AD, Berg AO, Deyo RA (1994). The effectiveness of four interventions for the prevention of low back pain. JAMA 272.1286-1291.

Lampl C, Buzath A, Baumhackl U, Klinger D (2003). One-year prevalence of migraine in Austria: a nation-wide survey. Cephalalgia 23.280-286.

Lane JM, Riley EH, Wirganowicz PZ (1996). Osteoporosis: diagnosis and treatment. JBJS 78A.618-632.

Lanteri-Minet M, Auray JP, El Hasnaoui A *et al.* (2003). Prevalence and description of chronic daily headache in the general population in France. Pain 102.143-149.

Lawrence JS (1969). Disc degeneration. Its frequency and relationship to symptoms. Ann Rheum Dis 28.121-138.

Leak AM, Cooper J, Dyer S, Williams KA, Turner-Stokes L, Frank AO (1994). The Northwick Park neck pain questionnaire, devised to measure neck pain and disability. Br J Rheum 33.469-474.

Leclerc A, Niedhammer I, Landre MF, Ozguler A, Etore P, Pietri-Taleb F (1999). One-year predictive factors for various aspects of neck disorders. Spine 24.1455-1462.

Lee D (2002). Biomechanics of the thorax. In: Ed. Grant R. *Physical Therapy of the Cervical and Thoracic Spine* (3rd ed.). Churchill Livingstone, New York.

Lee H, Nicholson LL, Adams RD (2003). Cervical range of motion associations with subclinical neck pain. Spine 29.33-40.

Lee H, Nicholson LL, Adams RD (2005). Neck muscle endurance, self-report, and range of motion data from subjects with treated and untreated neck pain. J Manip Physiol Thera 28.25-32.

Lees F, Turner JWA (1963). Natural history and prognosis of cervical spondylosis. BMJ 1607-1610.

Lemire JJ, Mierau DR, Crawford CM, Dzus AK (1996). Scheuermann's juvenile kyphosis. J Manip Physiol Thera 19.195-201.

Lempert T, Gresty MA, Bronstein AM (1995). Fortnightly review: Benign positional vertigo: recognition and treatment. BMJ 311.489-491.

Lentell G, Kruse M, Chock B, Wilson K, Iwamoto M, Martin R (2002). Dimensions of the cervical neural foramina in resting and retracted positions using magnetic resonance imaging. JOSPT 32.380-390.

Leone M, D'Amico D, Grazzi L, Attanasio A, Bussone G (1998). Cervicogenic headache: a critical review of the current diagnostic criteria. Pain 78.1-5.

Lestini WF, Wiesel SW (1989). The pathogenesis of cervical spondylosis. Clin Orth & Rel Res 239.69-93.

Levine J, Taiwo Y (1994). Inflammatory pain. In: Eds Wall PD, Melzack R. *Textbook of Pain* (3rd ed.), 45-56. Churchill Livingstone, Edinburgh.

Levoska S, Keinanen-Kiukaanniemi S, Bloigu R (1993). Repeatability of measurement of tenderness in the neck-shoulder region by a dolorimeter and manual palpation. Clin J Pain 9.229-235.

Licht PB, Christensen HW, Hoilund-Carlsen PF (2000). Is there a role for premanipulative testing before cervical manipulation? J Manip Physiol Thera 23.175-179.

Licht PB, Christensen HW, Hoilund-Carlsen PF (2002). Carotid artery blood flow during premanipulative testing. J Manip Physiol Thera 25.568-572.

Lindgren KA (1993). Thoracic outlet syndrome with special reference to the first rib. Ann Chir Gynaecol 82.218-230.

Lindgren KA, Oksala I (1995). Long-term outcome of surgery for thoracic outlet syndrome. Am J Surgery 169.358-360.

Lindgren KA (1997). Conservative treatment of thoracic outlet syndrome: a 2-year follow-up. Arch Phys Med Rehabil 78.373-378.

Linton SJ (1996). Early interventions for the secondary prevention of chronic musculoskeletal pain. In: Ed. Campbell JN. *Pain 1996 – An Updated Review*. IASP, Seattle.

Linton SJ (1998). The socioeconomic impact of chronic back pain: is anyone benefiting? Pain 75.163-168.

Linton SJ, Hellsing AL, Hallden K (1998). A population-based study of spinal pain among 35-45-year-old individuals. Spine 23.1463-1475.

Linton SJ (2000). A review of psychological risk factors in back and neck pain. Spine 25.1148-1156.

Linton SJ, van Tulder MW (2000). Preventative interventions for back and neck pain. In: Eds Nachemson AL, Jonsson E. *Neck and Back Pain. The Scientific Evidence of Causes, Diagnosis, and Treatment*. Lippincott Williams & Wilkins, Philadelphia.

Lock C, Allgar V, Jones K, Marples G, Chandler C, Dawson P (1999). Prevalence of back, neck and shoulder problems in the inner city: implications for the provision of physiotherapy services in primary healthcare. Physio Res Int 4.161-168.

Lofgren H, Johansen F, Skogar O, Levander B (2003). Reduced pain after surgery for cervical disc protrusion / stenosis: a 2 year clinical follow-up. Disability & Rehab 25.1033-1043.

Logue V (1952). Thoracic intervertebral disc prolapse with spinal cord compression. J Neurol Neurosurg Psychiat 15.227-241.

Long A, Donelson R, Fung T (2004). Does it matter which exercise? A randomized control trial of exercise for low back pain. Spine 29.2593-2602.

References 

Lord SM, Barnsley L, Wallis BJ, Bogduk N (1994). Third occipital nerve headache: a prevalence study. J Neurol Neurosurg & Psych 57.1187-1190.

Lord SM, Barnsley L, Wallis BJ, Bogduk N (1996a). Chronic cervical zygapophyseal joint pain after whiplash. A placebo-controlled prevalence study. Spine 21.1737-1745.

Lord SM, Barnsley L, Wallis BJ, McDonald GJ, Bogduk N (1996b). Percutaneous radio-frequency neurotomy for chronic cervical zygapophyseal-joint pain. New Eng J Med 335.1721-1726.

Lowe TG (1990). Current concepts review. Scheuermann disease. J Bone Jnt Surg 72A.940-945.

Lunsford LD, Bissonette DJ, Zorub DS (1980). Anterior surgery for cervical disc disease. Part 2: Treatment of cervical spondylotic myelopathy in 32 cases. J Neurosurg 53.12-19.

Mackinnon SE, Novak CB (2002). Thoracic outlet syndrome. Current Problems in Surgery 39.1070-1145.

Magarey ME, Rebbeck T, Coughlan B, Grimmer K, Rivett DA, Refshauge K (2004). Pre-manipulative testing of the cervical spine review, revision and new clinical guidelines. Manual Therapy 9.95-108.

Magee DJ, Oborn-Barrett E, Turner S, Fenning N (2000). A systematic overview of the effectiveness of physical therapy intervention on soft tissue neck injury following trauma. Physio Canada, Spring 2000.111-130.

Maghraoui AE, Bensabbah R, Bahiri R, Bezza A, Guedira N, Hajjaj-Hassouni N (2003). Cervical spine involvement in ankylosing spondylitis. Clinical Rheumatology 22.94-98.

Magnaes B (1982). Clinical recording of pressure on the spinal cord and cauda equina. Part 1: The spinal block infusion test: method and clinical studies. J Neurosurg 57.48-56.

Maigne JY, Deligne L (1994). Computed tomographic follow-up study of 21 cases of nonoperatively treated cervical intervertebral soft disc herniation. Spine 19.189-191.

Mailis-Gagnon A, Tepperman PS (2004). Comments on: Gross AR, Hoving JL, Goldsmith CH *et al.* Manipulation and mobilisation for mechanical neck disorders. Cochrane database Systematic Review 2004: Issue 1. In *Linkages*.

Maimaris C, Barnes MR, Allen MJ (1988). 'Whiplash injuries' of the neck: a retrospective study. Injury 19.393-396.

Mainardi F, Maggioni F, Dainese P, Zanchin G (2002). Spontaneous carotid artery dissection with cluster-like headache. Cephalalgia 22.557-559.

Maitland GD (1986). *Vertebral Manipulation* (5th ed.). Butterworths, London.

Makela M, Heliovaara M, Sievers K, Impivaara O, Knekt P, Aromaa A (1991). Prevalence, determinants, and consequences of chronic neck pain in Finland. Am J Epidemiology 134.1356-1367.

Makofsky HW (1994). Update on headache. Classification and the implications for orthopaedic physical therapists. J Man Manip Ther 2.7-10.

Malanga GA, Landes P, Nadler SF (2003). Provocative tests in cervical spine examination: historical basis and scientific analyses. Pain Physician 6.199-205.

Manchikanti L, Singh V, Rivera J, Pampati V (2002a). Prevalence of cervical facet joint pain in chronic neck pain. Pain Physician 5.243-249.

Manchikanti L, Singh V, Pampati V, Damron KS, Beyer CD, Barnhill RC (2002b). Is there correlation of facet joint pain in lumbar and cervical spine? An evaluation of prevalence in combined chronic low back and neck pain. Pain Physician 5.365-371.

Manchikanti L, Singh V, Pampati V, Beyer CD, Damron KS (2002c). Evaluation of the prevalence of facet joint pain in chronic thoracic pain. Pain Physician 5. 354-359.

Manchikanti L, Boswell MV, Singh V, Pampati V, Damron KS, Beyer CD (2004) Prevalence of facet joint pain in chronic spinal pain of cervical, thoracic, and lumbar regions. BMC Musculoskeletal Disorders 5.15.

Mann T, Refshauge KM (2001). Causes of complications from cervical manipulation. Aus J Physio 47.255-266.

Marchiori DM, Henderson CNR (1996). A cross-sectional study correlating cervical radiographic degenerative findings to pain and disability. Spine 21.2747-2752.

Matsumoto M, Fujimura Y, Suzuki N *et al.* (1998). MRI of cervical intervertebral discs in asymptomatic subjects. JBJS 80B.19-24.

Matthews JA (1969). Atlanto-axial subluxation in rheumatoid arthritis. Ann Rheum Dis 28.260-265.

Maxwell M (1996). Current physiotherapy treatment for whiplash injury to the neck. Br J Therapy & Rehab 3.391-395.

May S (2003). An outcome audit for musculoskeletal patients in primary care. Physio Theory Practice 19.189-198.

May S (2004a). An audit of mechanical diagnosis classification at multiple sites. McKenzie Institute (UK) Newsletter 12.3.22-31.

May S (2004b). The McKenzie classification system – an international prevalence survey (unpublished).

Mayou R (1989). Invited review: atypical chest pain. J Psychosomatic Res 33.393-4406.

Mayou R, Bryant B, Duthie R (1993). Psychiatric consequences of road traffic accidents. BMJ 307.647-651.

Mayou R, Bryant B, Forfar C, Clark D (1994). Non-cardiac chest pain and benign palpations in the cardiac clinic. Br Heart J 72.548-553.

Mayou R, Bryant B (1996). Outcome of 'whiplash' neck injury. Injury 27.617-623.

Mayou RA, Bass CM, Bryant BM (1999). Management of non-cardiac chest pain: from research to clinical practice. Heart 81.387-392.

McCallister P, Kaufman H (1994). Spinal epidural metastases: a common problem for the primary care physician. West Virginia Med J 90.101-105.

McCarthy C, Cushnaghan J, Dieppe P (1994). Osteoarthritis. In: Eds Wall PD, Melzack R. *Textbook of Pain* (3rd ed.). Churchill Livingstone, Edinburgh.

McClune T, Clarke R, Walker C, Burtt R (1998). Osteopathic management of cervical pain. In: Eds Giles LGF, Singer KP. *The Clinical Anatomy and Management of Back Pain Series. Volume 3: Clinical Anatomy and Management of Cervical Spine Pain*. Butterworth Heinemann, Oxford.

McGuirk B, King W, Govind J, Lowry J, Bogduk N (2001). Safety, efficacy, and cost effectiveness of evidence-based guidelines for the management of acute low back pain in primary care. Spine 26.2615-2622.

McKenzie K, Lin G, Tamir S (2004). Thoracic outlet syndrome. Part 1: a clinical review. J Am Chiro Assoc 41.17-24.

McKenzie RA (1981). *The Lumbar Spine: Mechanical Diagnosis & Therapy*. Spinal Publications, New Zealand.

McKenzie RA (1983). *Treat Your Own Neck*. Spinal Publications, New Zealand.

McKenzie RA (1990). *The Cervical & Thoracic Spine: Mechanical Diagnosis & Therapy*. Spinal Publications, New Zealand.

McKenzie R, May S (2000). *The Human Extremities: Mechanical Diagnosis & Therapy*. Spinal Publications, New Zealand.

McKenzie R, May S (2003). *The Lumbar Spine: Mechanical Diagnosis & Therapy*. Spinal Publications, New Zealand.

McKinney LA (1989). Early mobilisation and outcome in acute sprains of the neck. BMJ 299.1006-1008.

McKinney LA, Dornan JO, Ryan M (1989). The role of physiotherapy in the management of acute neck sprains following road-traffic accidents. Arch Emergency Med 6.27-33.

McLain RF (1994). Mechanoreceptor endings in human cervical facet joints. Spine 19.495-501.

Mealy K, Brennan H, Fenelon GCC (1986). Early mobilisation of acute whiplash injuries. BMJ 292.656-657.

Meijers KAE, van Beusekam GT, Luyendijk W, Duijfjes F (1974). Dislocation of the cervical spine with cord compression in rheumatoid arthritis. J Bone Joint Surg 56B.668.

Mellion LR, Ladeira C (2001). The herniated thoracic disc: a review of the literature. J Manual Manip Ther 9.154-163.

Meloche J, Bergeron Y, Bellavance A, *et al.* (1993). Painful intervertebral dysfunction: Robert Maigne's original contribution to headache of cervical origin. The Quebec Headache Study Group. Headache 33.328-334.

Melton LJ (1997). Epidemiology of spinal osteoporosis. Spine 22.2S-11S.

Melzack R, Wall P (1988). *The Challenge of Pain* (2nd). Penguin Books, London.

Mendel T, Wink CS, Zimny ML (1992). Neural elements in human cervical intervertebral discs. Spine 17.132-135.

Mercer SR, Jull GA (1996). Morphology of the cervical intervertebral disk. Implications for McKenzie's model of the disc derangement syndrome. Manual Therapy 1.76-81.

Mercer S, Bogduk N (1999). The ligaments and annulus fibrosus of human adult cervical intervertebral discs. Spine 24.619-628.

Mercer S (2004a). Comparative anatomy of the spinal disc. In: Eds Boyling JD, Jull GA. *Grieve's Modern Manual Therapy* (3rd ed.). Churchill Livingstone, Edinburgh.

Mercer S (2004b). Kinematics of the spine. In: Eds Boyling JD, Jull GA. *Grieve's Modern Manual Therapy* (3rd ed.). Churchill Livingstone, Edinburgh.

Merskey H, Bogduk N (Eds) (1994). *Classification of chronic pain. Descriptions of chronic pain syndromes and definitions of pain terms.* (2nd ed.). IASP Press, Seattle.

Michaeli A (1993). Reported occurrence and nature of complications following manipulative physiotherapy in South Africa. Aus J Physio 39.309-315.

Milne N (1993). Composite motion in cervical disc segments. Clin Biomech 8.193-202.

Mitchell J, Keene D, Dyson C, Harvey L, Pruvey C, Phillips R (2004). Is cervical spine rotation, as used in the standard vertebrobasilar insufficiency test, associated with a measurable change in intracranial vertebral artery blood flow? Manual Therapy 9.220-227.

Mitchell SL, Creed G, Thow M *et al.* (1999). *Physiotherapy Guidelines for the Management of Osteoporosis.* CSP, London.

Mochida K, Komori H, Okawa A, Muneta T, Haro H, Shinomiya K (1998). Regression of cervical disc herniation observed on magnetic resonance images. Spine 23.990-997.

Mongini F, Deregibus A, Raviola F, Mongini T (2003). Confirmation of the distinction between chronic migraine and chronic tension-type headache by the McGill Pain questionnaire. Headache 43.867-877.

Mooney V, Robertson J (1976). The facet syndrome. Clin Orth & Rel Res 115.149-156.

Moquin RR, Rosner MK, Cooper PB (2003). Combined anterior-posterior fusion with laterally placed threaded interbody cages and pedicle screws for Scheuermann kyphosis. Neurosurg Focus 14.1-5.

Mosdal C (1984). Cervical osteochondrosis and disc herniation. 18 years use of interbody fusion by Cloward's technique in 755 cases. Acta Neurochir 70.207-225.

Mosdal C, Overgaard J (1984). Lateral cervical facetectomy. The surgical pathology of radicular brachialgia. Acta Neurochir 70.199-205.

Nachemson A, Vingard E (2000). Assessment of patients with neck and back pain: a best-evidence synthesis. In: Eds Nachemson AL, Jonsson E. *Neck and Back Pain. The Scientific Evidence of Causes, Diagnosis, and Treatment.* Lippincott Williams & Wilkins, Philadelphia

Nakajima M, Hirayama K (1995). Midcervical central cord syndrome: numb and clumsy hands due to midline cervical disc protrusion at the C3-4 intervertebral level. J Neurol Neurosurg Psych 58.607-613.

Nansel DD, Peneff AL, Jansen RD, Cooperstein R (1989). Interexaminer concordance in detecting joint-play asymmetries in the cervical spines of otherwise asymptomatic subjects. J Manip Physiol Thera 12.428-433.

Narlawar RS, Shah JR, Pimple MK. Patkar DP, Patankar T, Castillo M (2002). Isolated tuberculosis of posterior elements of spine. MRI findings in 33 patients. Spine 27.275-281.

Nathan H (1962). Osteophytes of the vertebral column: an anatomical study of their development according to age, race and sex with considerations as to their etiology and sgnificance. J Bone Jnt Surg 44A.243-268.

Nederhand MJ, Ijzerman MJ, Hermens HJ, Baten CTM, Zilvold G (2000). Cervical muscle dysfunction in the chronic whiplash associated disorder grade II (WAD-II). Spine 25.1938-1943.

Nilsson N (1995). The prevalence of cervicogenic headache in a random population sample of 20-59-year-olds. Spine 20.1884-1888.

Nilsson S, Scheike M, Engblom D *et al.* (2003). Chest pain and ischaemic heart disease in primary care. Br J Gen Pract 53.378-382.

Norlander S, Nordgren B (1998). Clinical symptoms related to musculoskeletal neck-shoulder pain and mobility in the cervico-thoracic spine. Scand J Rehab Med 30.243-251.

Norris SH, Watts I (1983). The prognosis of neck injuries resulting from rear-end vehicle collisions. JBJS 65B.608-611.

Novak CB, Mackinnon SE, Patterson GA (1993). Evaluation of patients with thoracic outlet syndrome. J Hand Surg 18A.292-299.

Nuckley DJ, Konodi MA, Raynak GC, Ching RP, Mirza SK (2002). Neural space integrity of the lower cervical spine. Effect of normal range of motion. Spine 27.587-595.

Nurick S (1972a). The pathogenesis of the spinal cord disorder associated with cervical spondylosis. Brain 95.78-100.

Nurick S (1972b). The natural history and the results of surgical treatment of the spinal cord disorder associated with cervical spondylosis. Brain 95.101-108.

O'Brien Cousins S (1998). *Exercise, Aging & Health. Overcoming Barriers to an Active Old Age*. Brunner/Mazel, Philadelphia.

Odom GL, Finney W, Woodhall B (1958). Cervical disk lesions. JAMA 166.23-28.

O'Driscoll ML (1999). Chronic benign headache: causes and classification. Br J Ther Rehab 6.136-140.

Oh K, Chung CS, Lee SJ (2001). Orthostatic headache: an usual presentation of a skull base tumour. Cephalalgia 21.1000-1001.

O'Laoire SA, Thomas DGT (1983). Spinal cord compression due to prolapse of cervical intervertebral disc (herniation of nucleus pulposus). J Neurosurg 59.847-853.

Oliver J, Middleditch A (1991). Functional Anatomy of the Spine. Butterworth Heinemann, Oxford.

Olney DB, Marsden AK (1986). The effect of head restraints and seat belts on the incidence of neck injury in car accidents. Injury 17.365-367.

Ombregt L (2003). The thoracic spine – disorders of the thoracic spine: non-disc lesions. In: Ombregt L, Bisschop P, ter Veer HJ (2003). *A System of Orthopaedic Medicine* (2nd ed.). Churchill Livingstone, Edinburgh.

Ordway NR, Seymour RJ, Donelson RG, Hojnowski LS, Edwards WT (1999). Cervical flexion, extension, protrusion, and retraction. A radiographic segmental analysis. Spine 24.240-247.

O'Sullivan PB, Grahamslaw KM, Kendell M, Lapenskie SC, Moller NE, Richards KV (2002). The effect of different standing and sitting postures on trunk muscle activity in a pain-free population. Spine 27.1238-1244.

Paramar HV, Raymakers R (1993). Neck injuries from rear impact road traffic accidents: prognosis in persons seeking compensation. Injury 24.75-78.

Rasmussen BK, Jensen R, Schroll M, Olesen J (1991). Epidemiology of headache in a general population: a prevalence study. J Clin Epidemiol 44.1147-1157.

Rasmussen BK (2001). Epidemiology of headache. Cephalalgia 21.774-777.

Rasmussen C, Rechter L, Schmidt I, Hansen VK, Therkelsen K (2001). The association of the involvement of financial compensation with the outcome of cervicobrachial pain that is treated conservatively. Rheumatology 40.552-554.

Ravichandran G, Frankel HL (1981). Paraplegia due to intervertebral disc lesions: a review of 57 operated cases. Paraplegia 19.133-139.

Rayan G, Jensen C (1995). Thoracic outlet syndrome: provocative examination manoeuvres in a typical population. J Shld Elb Surg 4.113-117.

Rayan GM (1998). Thoracic outlet syndrome. J Shld Elb Surg 7.440-451.

Refshauge KM, Goodsell M, Lee M (1994). The relationship between surface contour and vertebral body measures of upper spine curvature. Spine 19.2180-2185.

Refshauge K, Bolst LB, Goodsell M (1995). The relationship between cervicothoracic posture and the presence of pain. J Manual & Manipulative Therapy 3.21-24.

Refshauge KM, Parry S, Shirley D, Larsen D, Rivett DA, Boland R (2002). Professional responsibility in relation to cervical spine manipulation. Aus J Physio 48.171-179.

Reitman CA, Mauro KM, Nguyen L, Ziegler JM, Hipp JA (2004). Intervertebral motion between flexion and extension in asymptomatic individuals. Spine 29.2832-2843.

Rekola KE, Keinanen-Kiukaanniemi S, Takala J (1993). Use of primary health services in sparsely populated country districts by patients with musculoskeletal symptoms: consultations with a physician. J Epidemiology Comm Health 47.153-157.

Rekola KE, Levoska S, Takala J, Keinanen-Kiukaanniemi S (1997). Patients with neck and shoulder complaints and multiple musculoskeletal symptoms – a prospective study. J Rheum 24.2424-2428.

Resnik L, Jensen GM (2003). Using clinical outcomes to explore the theory of expert practice in physical therapy. Physical Therapy 83.1090-1106.

Riddle DL, Rothstein JM (1993). Intertester reliability of McKenzie's classifications of the syndrome types present in patients with low back pain. Spine 18.1333-1344.

Riddle DL, Stratford PW (1998). Use of generic versus region-specific functional status measures on patients with cervical spine disorders. Physical Therapy 78.951-963.

Rivett DA, Milburn P (1997). Complications arising from spinal manipulative therapy in New Zealand. Physiotherapy 83.626-632.

Rivett DA, Reid D (1998). Risk of stroke for cervical spine manipulation in New Zealand. NZ J Physio 26.14-17.

Rivett DA, Sharples KJ, Milburn PD (1999). Effect of pre-manipulative tests on vertebral artery and carotid artery blood flow: a pilot study. J Manip Physiol Thera 22.368-375.

Rivett DA (2001). A valid pre-manipulative screening tool is needed. Aus J Physio 47.166.

Rivett DA (2004). The vertebral artery and vertebrobasilar insufficiency. In: Eds Boyling JD, Jull GA. *Grieve's Modern Manual Therapy* (3rd ed.). Churchill Livingstone, Edinburgh.

Rivett DA, Jones MA (2004). Improving clinical reasoning in manual therapy. In: Eds Jones MA, Rivett DA, *Clinical Reasoning for Manual Therapists*. Butterworth Heinemann, Edinburgh.

Robertson A, Branfoot T, Barlow IF, Giannoudis PV (2002). Spinal injury patterns resulting from car and motorcycle accidents. Spine 27.2825-2830.

Ronnen HR, de Korte PJ, Brink PRG, *et al.* (1996). Acute whiplash injury: is there a role for MG imaging? – A prospective study of 100 patients. Radiology 201.93-96.

Rosenfeld M, Gunnarsson R, Borenstein P (2000). Early intervention in whiplash associated disorders. A comparison of two treatment protocols. Spine 25.1782-1787.

Rosenfeld M, Seferiadis A, Carlsson J, Gunnarsson R (2003). Active intervention in patients with whiplash associated disorders improves long-term prognosis. Spine 22.2491-2498.

Rosomoff HL, Fishbain D, Rosomoff RS (1992). Chronic cervical pain: radiculopathy or brachialgia. Noninterventional treatment. Spine 17.S362-S366.

Roth DA (1976). Cervical analgesic discography. JAMA 235.1713-1714.

Russell T (1989). Thoracic intervertebral disc protrusion: experience of 67 cases and review of the literature. Br J Neurosurg 3.153-160.

Rydevik B, Brown MD, Lundborg G (1984). Pathoanatomy and pathophysiology of nerve root compression. Spine 9.7-15.

Saada J, Song S, Breidahl WH (2000). Developmental anomalies of the thoracic region. In: Giles LGF, Singer KP. *Clinical Anatomy and Management of Thoracic Spine Pain*. Butterworth Heinemann, Oxford.

Saal JS, Saal JA, Yurth EF (1996). Nonoperative management of herniated cervical disc with radiculopathy. Spine 21.1877-1883.

Sackett DL, Richardson WS, Rosenberg W, Haynes RB (1997). *Evidence-Based Medicine. How to Practice and Teach EBM*. Churchill Livingstone, New York.

Sackett DL, Haynes RB (2002) Evidence base of clinical diagnosis: The architecture of diagnostic research. British Medical Journal 324.539-541.

Salemi G, Savettieri G, Meneghini F *et al.* (1996). Prevalence of cervical spondylotic radiculopathy: a door-to-door survey in a Sicilian municipality. Acta Neurol Scand 93.184-188.

Sampath P, Bendebba M, Davis JD, Ducker T (1999). Outcome in patients with cervical radiculopathy. Prospective, multicenter study with independent clinical review. Spine 24.591-597.

Sanchez MC, Arenillas JIC, Gutierrez DA, Alonso JLG, Alvarez JDP (1983). Cervical radiculopathy: a rare symptom of giant cell arteritis. Arth & Rhem 26.207-209.

Sanin LC, Mathew NT, Bellmeyer LR, Ali S (1994). The International Headache Society (IHS) headache classification as applied to a headache clinic population. Cephalalgia 14.443-446.

Sarig-Bahat H (2003). Evidence for exercise therapy in mechanical neck disorders. Manual Therapy 8.10-20.

Schaberg J, Gainor BJ (1985). A profile of metastatic carcinoma of the spine. Spine 10.19-20.

Schellhas KP, Pollei SR, Dorwart RH (1994). Thoracic discography. Spine 19.2103-2109.

Schellhas KP, Smith MD, Gundry CR, Pollei SR (1996). Cervical discogenic pain. Prospective correlation of magnetic resonance imaging and discography in asymptomatic subjects and pain sufferers. Spine 21.300-312.

Schenk R, Dukhon I, Guyer Z, Sommers M, Venning A, Zalewski K (2002). An evidence-based algorithm for examination of the cervical spine. Orthopaedic Practice 14.9-11.

Schenker M, Kay SPJ (2001). Mechanical neuropthay at the thoracic outlet and associated pain syndrome. Current Orthopaedics 15.264-274.

Scher AI, Stewart WF, Lipton RB (1999). Migraine and headache: a meta-analytic approach. In: Eds Crombie IK, Croft PR, Linton SJ, LeResche L, von Korff M. *Epidemiology of Pain*. IASP Press, Seattle.

Schneider G (1989). Restricted shoulder movement: capsular contracture or cervical referral – a clinical study. Aus J Physio 35.97-100.

Scholten-Peeters GGM, Bekkering GE, Verhagen AP *et al.* (2002). Clinical practice guideline for the physiotherapy of patients with whiplash associated disorders. Spine 27.412-422.

Scholten-Peeters GGM, Verhagen AP, Bekkering GE *et al.* (2003). Prognostic factors of whiplash associated disorders: a systematic review of prospective cohort studies. Pain 104.303-322.

Schrader H, Obeliene D, Bovim G *et al.* (1996). Natural evolution of late whiplash syndrome outside the medicolegal context. Lancet 347.1207-1211.

Scott A, Khan KM, Roberts CR, Cook JL, Duronio V (2004). What do we mean by the term "inflammation"? A contemporary basic science update for sports medicine. Br J Sports Med 38.372-380.

Seffinger MA, Najm WI, Mishra SI *et al.* (2004). Reliability of spinal palpation for diagnosis of back and neck pain. A systematic review of the literature. Spine 29.E413-E425.

Shaw DA, Cartlidge NE (1976). Cervical myelopathy in rheumatoid arthritis. Acta Neurol Belg 76.279-286.

Shelokov AP (1991). Evaluation, diagnosis and initial treatment of cervical disc disease. Spine State Art Rev 5.167-176.

Sherk HH (1978). Atlantoaxial instability and acquired basilar invagination in rheumatoid arthritis. Orthop Clin North Am 9.1053-1063.

Shinomiya K, Komori H, Matsuoka T, Mutoh N, Furuya K (1994). Neuroradiologic and electrophysiologic assessment of cervical spondylotic amytrophy. Spine 19.21-25.

Shorland S (1998). Management of chronic pain following whiplash injuries. In: Ed. Gifford L. *Topical Issues in Pain 1*. NOI Press, Falmouth.

Siddall PJ, Cousins MJ (1997). Spine Update. Spinal pain mechanisms. Spine 22.98-104.

Silbert PL, Mokri B, Schievink WI (1995). Headache and neck pain in spontaneous internal carotid and vertebral artery dissections. Neurology 45.1517-1522.

Sim J, Waterfield J (1997). Validity, reliability and responsiveness in the assessment of pain. Physio Theory & Practice 13.23-37.

Sinaki M, Mikkelsen BA (1984). Postmenopausal spinal osteoporosis: flexion versus extension exercises. Arch Phys Med Rehabil 65.593-596.

Singer KP (1994). Anatomy and biomechanics of the thoracolumbar junction. In: Eds Boyling JD, Palastanga N. *Grieve's Modern Manual Therapy*. Churchill Livingstone, Edinburgh.

Singer KP (2000). Pathology of the thoracic spine. In: Giles LGF, Singer KP. *Clinical Anatomy and Management of Thoracic Spine Pain*. Butterworth Heinemann, Oxford.

Singer KP, Edmondston SJ (2000). Introduction: the enigma of the thoracic spine. In: Giles LGF, Singer KP. *Clinical Anatomy and Management of Thoracic Spine Pain*. Butterworth Heinemann, Oxford.

Singer KP, Goh S (2000). Anatomy of the thoracic spine. In: Giles LGF, Singer KP. *Clinical Anatomy and Management of Thoracic Spine Pain*. Butterworth Heinemann, Oxford.

Singer KP (2004). The spine and the effects of ageing. In: Eds Boyling JD, Jull GA. *Grieve's Modern Manual Therapy* (3rd ed.). Churchill Livingstone, Edinburgh.

Singer KP, Boyle JJW, Fazey P (2004). Comparative anatomy of the zygapophyseal joints. In: Eds Boyling JD, Jull GA. *Grieve's Modern Manual Therapy* (3rd ed.) Churchill Livingstone, Edinburgh.

Singounas EG, Kypriades EM, Kellerman AJ, Garvan N (1992). Thoracic disc herniation. Analysis of 14 cases and review of the literature. Acta Neurochir 116.49-52.

Sjaastad O, Saunte C, Hovdahl H *et al.* (1983). "Cervicogenic" headache. An hypothesis. Cephalalgia 3.249-256.

Sjaastad O, Fredriksen TA, Pfaffenrath V (1990). Cervicogenic headache: diagnostic criteria. Headache 30.725-726.

Sjaastad O, Bovim G (1991). Cervicogenic headache. The differentiation from common migraine. An overview. Functional Neurology 6.93-100.

Sjaastad O, Fredriksen TA, Pfaffenrath V (1998). Cervicogenic headache: diagnostic criteria. The Cervicogenic Headache International Study Group. Headache 38.442-445.

Sjaastad O, Bakketeig LS (2002). Exertional headache. 1. Vaga study of headache epidemiology. Cephalalgia 22.784-790.

Slipman CW, Plastaras CT, Palmitier RA, Huston CW, Sterenfeld EB (1998). Symptom provocation of fluoroscopically guided cervical nerve root stimulation. Spine 23.2235-2242.

Slipman CW, Patel RK, Botwin K, Huston C, Zhang L, Lenrow D, Garvan C (2003). Epidemiology of spine tumors presenting to musculoskeletal physiatrists. Archives of Physical Medicine and Rehabilitation 84.492-495.

Slipman CW, Plastaras C, Patel R *et al.* (2005). Provocative cervical discography symptom mapping. Spine Journal 5.381-388.

Smedmark V, Wallin M, Arvidsson I (2000). Inter-examiner reliability in assessing passive intervertebral motion of the cervical spine. Manual Therapy 5.97-101.

Smith GW (1959). The normal cervical discogram, with clinical observations. AJR 81.1006-1010.

Smith R (2000). Metabolic disorders of the spine. In: Eds Bartley R, Coffey P. *Management of Low Back Pain in Primary Care*. Butterworth Heinemann, Oxford.

Smyth MJ, Wright V (1958). Sciatica and the intervertebral disc. An experimental study. JBJS 40A.1401-1418.

Soderlund A, Olerud C, Lindberg P (2000). Acute whiplash associated disorders (WAD): the effects of early mobilization and prognostic factors in long-term symptomatology. Clin Rehab 14.457-467.

Sparkes V (2004). Abdominal pain of musculoskeletal origin. In: Eds Boyling JD, Jull GA. *Grieve's Modern Manual Therapy* (3rd ed.). Churchill Livingstone, Edinburgh.

Spengler DM, Kirsh MM, Kauffer H (1973). Orthopaedic aspects and early diagnosis of superior sulcus tumor of lung (Pancoast). JBJS 55A.1645-1650.

Spierings ELH (2002). Acute and chronic hypertensive headache and hypertensive encephalopathy. Cephalalgia 22.313-316.

Spitzer WO, LeBlanc FE, Dupuis M *et al.* (1987). *Scientific approach to the activity assessment and management of activity-related spinal disorders.* Spine 12.7.S1-S55.

Spitzer WO, Skovron ML, Salmi LR *et al.* (1995). *Scientific monograph of the Quebec Task Force on whiplash associated disorders: redefining "whiplash" and its management.* Spine 20.S1-S73.

Squires B, Gargan MF, Bannister GC (1996). Soft-tissue injuries of the cervical spine. 15-year follow-up. JBJS 78B.955-957.

Sterling M, Kenardy J, Jull G, Vincenzino B (2003a). The development of psychological changes following whiplash injury. Pain 106.481-489.

Sterling M, Jull G, Vincenzino B, Kenardy J, Darnell R (2003b). Development of motor system dysfunction following whiplash injury. Pain 103.65-73.

Sterling M (2004). A proposed new classification system for whiplash associated disorders – implications for assessment and management. Manual Therapy 9.60-70.

Stevinson C, Honan W, Cooke B, Ernst E (2001). Neurological complications of cervical spinal manipulation. J Royal Soc Med 94.107-110.

Stiell IG, Wells GA, Vandemheen KL *et al.* (2001). The Canadian C-spine rule for radiography in alert and stable trauma patients. JAMA 286.1841-1848.

Stiell IG, Clement CM, McKnight RD *et al.* (2003). The Canadian C-spine rule versus the NEXUS low-risk criteria in patients with trauma. New Eng J Med 349.2510-2518.

Stokes IAF (2000). Biomechanics of the thoracic spine and ribcage. In: Giles LGF, Singer KP. *Clinical Anatomy and Management of Thoracic Spine Pain.* Butterworth Heinemann, Oxford.

Stovner LJ (1996). The nosologic status of the whiplash syndrome: a critical review based on a methodological approach. Spine 21.2735-2746.

Streiner DL, Norman GR (1996). *PDQ Epidemiology* (2nd ed) Mosby, St Louis.

Streiner DL, Norman GR (2003). *Health Measurement Scales – a practical guide to their development and use* (3rd ed.). Oxford University Press, Oxford.

Strender LE, Lundin M, Nell K (1997). Interexaminer reliability in physical examination of the neck. J Manip Physiol Thera 20.516-520.

Strong J (2002). Chronic pain problems. In: Eds Strong J, Unruh AM, Wright A, Baxter GD. *Pain. A Textbook for Therapists*. Churchill Livingstone, Edinburgh.

Swinkels RAHM, Oostendorp RAB (1996). Upper cervical instability: fact or fiction. J Manip Physiol Thera 19.185-194.

Takala J, Sievers K, Klaukka T (1982). Rheumatic symptoms in the middle-aged population in south-western Finland. Scand J Rheum (Suppl) 47.15-29.

Tanaka N, Fujimoto Y, An HS, Ikuta Y, Yasuda M (2000). The anatomic relation among nerve roots, intervertebral foramina, and intervertebral discs of the cervical spine. Spine 25.286-291.

Taylor AJ, Kerry R (2004). Vascular syndromes presenting as pain of spinal origin. In: Eds Boyling JD, Jull GA. *Grieve's Modern Manual Therapy* (3rd ed.). Churchill Livingstone, Edinburgh.

Taylor AJ, Kerry R (2005). Neck pain and headache as a result of internal carotid artery dissection: implications for manual therapists. Manual Therapy 10.73-77.

Taylor JR, Twomey LT (1993). Acute injuries to cervical joints. An autopsy study of neck sprain. Spine 18.1115-1122.

Taylor JR, Twomey L (2002). Functional and applied anatomy of the cervical spine. In: Ed. Grant R. *Physical Therapy of the Cervical and Thoracic Spine*. Churchill Livingstone, New York.

Teasell RW, Shapiro AP (2002). The clinical picture of whiplash injuries. In: Eds Malanga GA, Nadler SF. *Whiplash*. Hanley & Belfus, Philadelphia.

Teresi LM, Lufkin RB, Reicher MA *et al.* (1987). Asymptomatic degenerative disk disease and spondylosis of the cervical spine: MR imaging. Radiology 164.83-88.

Terrett AGJ (1998). Contraindications to cervical spine manipulation. In: Eds Giles LGF, Singer KP. *The Clinical Anatomy and Management of Back Pain Series. Volume 3: Clinical Anatomy and Management of Cervical Spine Pain*. Butterworth Heinemann, Oxford.

Terry W, Higgs J (1993). Educational programmes to develop clinical reasoning skills. Aus J Physio 39.47-51.

Thiel H, Wallace K, Donat J, Yong-Hing K (1994). Effect of various head and neck positions on vertebral artery blood flow. Clin Biomech 9.105-110.

Thiel H, Rix G (2005). Is it time to stop functional pre-manipulation testing of the cervical spine? Manual Therapy 10.154-158.

Thomas J (1990). Road traffic accidents before and after seatbelt legislation – study in a district general hospital. J Royal Soc Med 83.79-81.

Threlkeld AJ, Currier DP (1988). Osteoarthritis. Effects on synovial joint tissues. Physical Therapy 68.364-370.

Thurston RC, Keefe FJ, Bradley L, Krishnan KRR, Caldwell DS (2001). Chest pain in the absence of coronary artery disease: a biopsychosocial perspective. Pain 93.95-100.

Tong HC, Haig AJ, Yamakawa K (2002). The Spurling Test and cervical radiculopathy. Spine 27.156-159.

Trott PH, Pearcy MJ, Ruston SA, Fulton I, Brien C (1996). Three-dimensional analysis of active cervical motion: the effect of age and gender. Clin Biomech 11.201-206.

Trott PH (2002). Management of selected cervical syndromes. In: Ed. Grant R. *Physical Therapy of the Cervical and Thoracic Spine*. Churchill Livingstone, New York.

Turner JA (1996). Educational and behavioural interventions for back pain in primary care. Spine 21.2851-2859.

Turner PA (2000). Osteoporosis – its causes and prevention: an update. Physio Theory Pract 16.135-149.

Twomey LT, Taylor JR, Taylor MM (1989). Unsuspected damage to lumbar zygapophyseal (facet) joints after motor-vehicle accidents. Med J Aust 151.210-217.

Twomey LT, Taylor JR (1993). The whiplash syndrome: pathology and physical treatment. J Manip Manual Ther 1.26-29.

Twomey LT, Taylor JR (1994). Factors influencing ranges of movement in the spine. In: Eds Boyling JD, Palastanga N. *Grieve's Modern Manual Therapy* (2nd ed.). Churchill Livingstone, Edinburgh.

Uhrenholt L, Grunnet-Nilsson N, Hartvigsen J (2002). Cervical spine lesions after road traffic accidents. A systematic review. Spine 27.1934-1941.

Uitvlught G, Indenbaum S (1988). Clinical assessment of atlantoaxial instability using the Sharp-Purser test. Arth & Rheum 31.370-374.

Unruh AM, Henriksson C (2002). Psychological, environmental and behavioural dimensions of the pain experience. In: Eds Strong J, Unruh AM, Wright A, Baxter GD. *Pain. A Textbook for Therapists*. Churchill Livingstone, Edinburgh.

Unruh AM, Strong J, Wright A (2002). Introduction to pain. In: Eds Strong J, Unruh AM, Wright A, Baxter GD. *Pain. A Textbook for Therapists*. Churchill Livingstone, Edinburgh.

Urwin M, Symmons D, Allison T *et al.* (1998). Estimating the burden of musculoskeletal disorders in the community: the comparative prevalence of symptoms at different anatomical sites, and the relation to social deprivation. Ann Rheum Dis 57.649-655.

Valencia F (1994). Clinical anatomy and biomechanics of the thoracic spine. In: Eds Boyling JD, Palastanga N. *Grieve's Modern Manual Therapy*. Churchill Livingstone, Edinburgh.

van der Donk J, Schouten JSAG, Passchier J, van Romunde LKJ, Valkenburg HA (1991). The association of neck pain with radiological abnormalities of the cervical spine and personality traits in a general population. J Rheum 18.1884-1889.

van der Windt DAWM, Koes BW, Boeke AJP, Deville W, de Jong BA, Bouter LM (1996). Shoulder disorders in general practice: prognostic indicators of outcome. Br J Gen Pract 46.519-523.

van Mameren H, Drukker J, Sanches H, Beursgens J (1990). Cervical spine motion in the sagittal plane (I). Range of motion of actually performed movements, an X-ray cinematographic study. Eur J Morphology 28.47-68.

van Suijlekom HA, de Vet HCW, van den Berg SGM, Weber WEJ (1999). Interobserver reliability of diagnostic criteria for cervicogenic headache. Cephalalgia 19.817-823.

van Suijlekom HA, de Vet HCW, van den Berg SGM, Weber WEJ (2000). Interobserver reliability in physical examination of the cervical spine in patients with headache. Headache 40.581-586.

van Tulder MW, Koes BW, Bouter LM (1995). A cost-of-illness study of back pain in The Netherlands. Pain 62.233-240.

van Wijmen PM (1994). The use of repeated movements in the McKenzie method of spinal examination. In: Eds Boyling JD, Palastanga N. *Grieve's Modern Manual Therapy* (2nd ed.). Churchill Livingstone, Edinburgh.

Vargo MM, Flood KM (1990). Pancoast tumor presenting as cervical radiculopathy. Arch Phys Med Rehab 71.606-609.

Vassilouthis J, Kalovithouris A, Papandreou A, Tegos S (1989). The symptomatic incompetent cervical intervertebral disc. Neurosurgery 25.232-239.

Vazquez-Barquero A, Ibanez FJ, Herrera S, Izquierdo JM, Berciano J, Pascual J (1994). Isolated headache as the presenting clinical manifestation of intracranial tumors: a prospective study. Cephalalgia 14.270-272.

Vernon H, Mior S (1991). The Neck Disability Index: a study of reliability and validity. J Manipulative Physiol Ther 14.409-415.

Videman T (1987). Connective tissue and immobilisation. Clin Orth & Rel Res 221.26-32.

Viikari-Juntura E (1987). Interexaminer reliability of observations in physical examinations of the neck. Physical Therapy 67.1526-1532.

Viikari-Juntura E, Porras M, Laasonen EM (1989). Validity of clinical tests in the diagnosis of root compression in cervical disc disease. Spine 14.253-257.

Viikari-Juntura E, Vuori J, Silverstein BA, Kalimo R, Kuosma E, Videman T (1991). A life-long prospective study on the role of psychosocial factors in neck-shoulder and low-back pain. Spine 16.1056-1061.

Vincent MB, Luna RA (1999). Cervicogenic headache: a comparison with migraine and tension-type headache. Cephalalgia 19.S25.11-16.

Vingard E, Nachemson A (2000). Work-related influences on neck and back pain. In: Eds Nachemson AL, Jonsson E. *Neck and Back Pain. The Scientific Evidence of Causes, Diagnosis, and Treatment*. Lippincott Williams & Wilkins, Philadelphia.

Vlaeyen JWS, Linton SJ (2000). Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. Pain 85.317-332.

Waddell G (2004). *The Back Pain Revolution* (2nd ed.). Churchill Livingstone, Edinburgh.

Wainner RS, Gill H (2000). Diagnosis and nonoperative management of cervical radiculopathy. J Orth Sports Phys Ther 30.728-744.

Wainner RS, Fritz JM, Irrgang JJ, Boninger ML, Delitto A, Alison S (2003). Reliability and diagnostic accuracy of the clinical examination and patient self-report measures for cervical radiculopathy. Spine 28.52-62.

Wallis BJ, Lord SM, Barnsley L, Bogduk N (1996). Pain and psychologic symptoms of Australian patients with whiplash. Spine 21.804-810.

Wallis BJ, Lord SM, Bogduk N (1997). Resolution of psychological distress of whiplash patients following treatment by radiofrequency neurotomy: a randomised, double-blind, placebo-controlled trial. Pain 73.15-22.

Walmsley RP, Kimber P, Culham E (1996). The effect of initial head position on active cervical axial rotation range of motion in two age populations. Spine 21.2435-2442.

Walton KA, Buono LM (2003). Horner syndrome. Curr Opin Ophthalmol 14.357-63.

Wang WT, Olson SL, Campbell AH, Hanten WP, Gleeson PB (2003). Effectiveness of physical therapy for patients with neck pain: an indiviualized approach using a clinical decision-making algorithm. Am J Phys Med Rehabil 82.203-218.

Warrens AN, Heaton JM (1987). Thoracic outlet compression syndrome: the lack of reliability of its clinical assessment. Ann Royal Coll Surg Eng 69.203-204.

Watkinson A, Gargan MF, Bannister GC (1991). Prognostic factors in soft tissue injuries of the cervical spine. Injury 22.307-309.

Watson DH (1994). Cervical headache: an investigation of upper cervical head postures and upper cervical flexor muscle performance. In: Eds Boyling JD, Palastanga N. *Grieve's Modern Manual Therapy* (2nd ed.). Churchill Livingstone, Edinburgh.

Webb R, Brammah T, Lunt M, Urwin M, Allison T, Symmons D (2003). Prevalence and predictors of intense, chronic, and disabling neck and back pain in the UK in the general population. Spine 28.1195-1202.

Weinfeld RM, Olson PN, Maki DD *et al.* (1997). The prevalence of diffuse idiopathic skeletal hyperstosis (DISH) in two large American Midwest metropolitan hospital populations. Skel Radiol 26.222-225.

Weinstein JN, McLain RF (1987). Primary tumors of the spine. Spine 12.843-851.

Welch M, Fletcher P, Piper C (1995). A statistical package for clinical audit analysis: a pilot study at the European School of Osteopathy. Br Osteo J 15.21-27.

Wells P (1982). Cervical dysfunction and shoulder problems. Physiotherapy 68.66-73.

Werneke M, Hart DL, Cook D (1999). A descriptive study of the centralization phenomenon. Spine 24.676-683.

Westaway MD, Stratford PW, Binkley JM (1998). The patient-specific functional scale: validation of its use in persons with neck dysfunction. JOSPT 27.331-338.

Westaway MD, Stratford P, Symons BN (2003). False-negative extension/rotation pre-manipulative screening test on a patient with an atretic and hypoplastic vertebral artery. Manual Therapy 8.120-127.

Westerling D, Jonsson BG (1980). Pain from the neck-shoulder region and sick leave. Scand J Soc Med 8.131-136.

Wheeler AH, Goolkasian P, Baird AC, Darden BV (1999). Development of the Neck Pain and Disability Scale. Spine 24.1290-1294.

Whitcomb DC, Martin SP, Schoen RE, Jho HD (1995). Chronic abdominal pain caused by thoracic disc herniation. Am J Gastroentology 90.835-837.

White AA, Analysis of the mechanics of the thoracic spine in man. Acta Ortho Scand S127.1-105.

White AA, Panjabi MM (1978a). *Clinical Biomechanics of the Spine*. J.B. Lippincott Company, Philadelphia.

White AA, Panjabi MM (1978b). The basic kinematics of the human spine. A review of past and current knowledge. Spine 3.12-20.

White AR, Ernst E (1999). A systematic review of randomised controlled trials of acupuncture for neck pain. Rheumatology 38.143-147.

White P, Lewith G, Prescott P, Conway J (2004). Acupuncture versus placebo for the treatment of chronic mechanical neck pain. Ann Intern Med 141.911-919.

Whitecloud TS, Seago RA (1987). Cervical discogenic syndrome. Results of operative intervention in patients with positive discography. Spine 12.313-316.

Wilke A, Wolf U, Lageard P, Griss P (2000). Case report. Thoracic disc herniation a diagnostic challenge. Manual Therapy 5.181-184.

Williams PL, Warwick R, Dyson M *et al.* (1980) *Gray's Anatomy* (36th ed.). Churchill Livingstone, Edinburgh.

Wilson PR (1987). Thoracic facet joint syndrome – a clinical entity? Pain S4.S87.

Witte MB, Barbul A (1997). General principles of wound healing. Surg Clinics Nth Am 77.509-528.

Wolff I, van Croonenborg JJ, Kemper HC, Kostense PJ, Twisk JW (1999). The effect of exercise training programs on bone mass: a meta-analysis of published controlled trials in pre- and postmenopausal women. Osteoporosis International 9.1-12.

Wolsko PM, Eisenberg DM, Davis RB, Kessler R, Phillips RS (2003). Patterns and perceptions of care for treatment of back and neck pain. Results of a national survey. Spine 28.292-298.

Wood KB, Garvey TA, Gundry C, Heithoff KB (1995). Thoracic MRI evaluation of asymptomatic individuals. J Bone Jnt Surg 77A.1634-1638.

Wood KB, Blair JM, Aepple DM *et al.* (1997). The natural history of asymptomatic thoracic disc herniations. Spine 22.525-530.

Wood KB, Schellhas KP, Garvey TA, Aeppli D (1999). Thoracic discography in healthy individuals. A controlled prospective study of MRI and discography in asymptomatic and symptomatic individuals. Spine 24.1548-1555.

Woolf CJ (1991). Generation of acute pain: central mechanisms. Br Med Bull 47.523-533.

Woolf CJ, Bennett GJ, Doherty M *et al.* (1998). Towards a mechanism-based classification of pain? Pain 77.227-229.

Worth DR (1994). Movements of the head and neck. In: Eds Boyling JD, Palastanga N. *Grieve's Modern Manual Therapy* (2nd ed.). Churchill Livingstone, Edinburgh.

Wright A (2002). Neurophysiology of pain and pain modulation. In: Eds Strong J, Unruh AM, Wright A, Baxter GD. *Pain. A Textbook for Therapists*. Churchill Livingstone, Edinburgh.

Wrisley DDM, Sparto PJ, Whitney SL, Furman JM (2000). Cervicogenic dizziness: a review of diagnosis and treatment. JOSPT 30.755-766.

Yacoub M, Hupert C (1980). Shoulder pain as an early symptom of Pancoast tumor. J Med Soc New Jersey 77.583-586.

Yamazaki S, Kokobun, Ishii Y, Tanaka Y (2003). Courses of cervical disc herniation causing myelopathy or radiculopathy. An analysis based on computered tomographic discograms. Spine 28.1171-1175.

Yoo JU, Zou D, Edwards WT, Bayley J, Yuan HA (1992). Effect of cervical spine motion on the neuroforaminal dimensions of human cervical spine. Spine 17.1131-1136.

Yoshimatsu H, Nagata K, Goto H *et al.* (2001). Conservative treatment for cervical spondylotic myelopathy: prediction of treatment effects by multivariate analysis. Spine Journal 1.269-273.

Youdas JW, Garrett TR, Suman VJ, Bogard CL, Hallman HO, Carey JR (1992). Normal range of motion of the cervical spine: an initial goniometric study. Physical Therapy 72.770-780.

Young S, O'Laoire S (1987). Cervical disc prolapse in the elderly: an easily overlooked, reversible cause of spinal cord compression. Br J Neurosurg 1.93-98.

Young, WF (2000) Cervical spondylotic myelopathy: a common cause of spinal cord dysfunction in older persons. American Family Physician. 62.1064-1070, 1073.

Yu YL, Woo E, Huang CY (1987). Cervical spondylotic myelopathy and radiculopathy. Acta Neurol Scand 75.367-373.

Zaina C, Grant R, Johnson C, Dansie B, Taylor J, Spyropolous (2003). The effect of cervical rotation on blood flow in the contralateral vertebral artery. Manual Therapy 8.103-109.

Zeidman SM, Ducker TB (1998) Evaluation of patients with cervical spine lesions. In: *The Cervical Spine* (3rd ed.). The Cervical Spine Research Society Editorial Committee. Lippincott-Raven, Philadelphia.

Zusman M (1992). Central nervous system contribution to mechanically produced motor and sensory responses. Aus J Physio 38.245-255.

Zusman M (1994). The meaning of mechanically produced responses. Aus J Physio 40.35-39.

Zwart JA (1997). Neck mobility in different headache disorders. Headache 37.6-11.

# Glossary of Terms



### Centralisation

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

### Derangement syndrome

Rapid and lasting changes in pain intensity and location, sometimes over a few minutes or a few days, and mechanical presentation can occur in this syndrome with the performance of movements or the adoption of sustained postures. Loading strategies produce a decrease, abolition or centralisation of symptoms. Opposite loading strategies may cause production, worsening or peripheralisation of symptoms if prolonged over sufficient time. A distinguishing set of characteristics is found during the history-taking and physical examination. The conceptual model involves internal articular displacement that causes a disturbance in the joint, which produces pain and impairment.

### Directional preference

See 'Mechanically determined directional preference'.

### Distal symptoms

These are the symptoms located farthest down the arm; these may be radicular or somatic referred pain, or paraesthesia. During the evaluation of symptomatic responses to mechanical loading, the most distal symptoms are closely monitored. Movements that decrease or abolish these symptoms are prescribed, whilst movements that increase or produce them are avoided.

### Dysfunction syndrome

Pain from the dysfunction syndrome is caused by mechanical deformation of structurally impaired soft tissues. This abnormal tissue may be the product of previous trauma or degenerative processes and the development of imperfect repair. Contraction, scarring, adherence, adaptive shortening or imperfect repair tissue become the source of

symptoms and functional impairment. Pain is felt when the abnormal tissue is loaded. A distinguishing set of characteristics is found during the history-taking and physical examination. In spinal dysfunction pain is consistently produced at restricted end-range and abates once the loading is released. Dysfunction may affect contractile, peri-articular or neural structures, with the latter two occurring in the spine.

### Extension principle

This principle of treatment encompasses procedures, both patient- and therapist-generated, that produce extension of the cervical or thoracic spine. In a posterior derangement these will be used to abolish, decrease or centralise symptoms. In an extension dysfunction, the extension principle is used for remodelling.

### Flexion principle

This principle of treatment encompasses procedures, both patient- and therapist-generated, that produce flexion of the cervical or thoracic spine. In an anterior derangement these will be used to abolish, decrease or centralise symptoms. In a flexion or ANR dysfunction, the flexion principle is used for remodelling.

### Force alternatives

A change in the manner in which a force may be applied during the exploration of loading strategies; for instance, alternative start positions (sitting or lying), force directions (sagittal, lateral flexion or rotation), dynamic (repeated movements) or static forces (sustained positions), or with the addition of traction.

### Force progressions

An increase of forces within a treatment principle. Within each principle of treatment direction (extension, flexion, lateral) there is a range of loading strategies that involve greater or more specific forces; for instance, sustained mid-range positions, end-range patient-generated movement, patient-generated force with clinician overpressure, clinician-generated force or repeated movements over several days. Force progressions are used to determine the correct directional preference and are used when lesser forces are not able to maintain improvements.

### 'Green flags'

'Green flags' are identified when repeated movements or sustained positions result in the centralisation, abolition or decrease of symptoms

Glossary of Terms


and/or an increase in range of movement and indicate the potential for good to excellent short- and long-term outcomes.

### Kappa

The kappa coefficient is commonly used in studies to address the reliability of two testers to come to the same conclusion about a test. It takes account of the fact that there is a 50% probability of chance agreement even if random judgements are made. It reports a numerical value, with 1.00 being perfect agreement and 0.00 for agreement no better than chance. Negative values imply that agreement is worse than what would be expected by chance alone.

**Guide to kappa values** (Altman 1991)

| Kappa value | Strength of agreement |
|---|---|
| <0.20 | *Poor* |
| 0.21-0.40 | *Fair* |
| 0.41-0.60 | *Moderate* |
| 0.61-0.80 | *Good* |
| 0.81-1.00 | *Very good* |

### Lateral compartment

Describes the compartment of the intervertebral segment that is compressed with lateral forces. The lateral compartment becomes relevant if lateral forces influence the patient's symptoms.

### Relevant lateral component

This refers to patients with derangement who have unilateral or asymmetrical symptoms that do not improve with sagittal plane forces. When the lateral component is relevant, asymmetrical forces are necessary to achieve centralisation or decrease of symptoms.

### Lateral principle

This principle of treatment encompasses procedures, both patient- and therapist-generated, that produce an asymmetrical force on the cervical or thoracic spine. In postero-lateral or antero-lateral derangement these are used to abolish, decrease or centralise symptoms.

### Loading strategies

Describes the applied movements, positions or loads required to stress particular structures, and may be dynamic or static – dynamic would be a repeated movement; static, a sustained posture. The significant loading strategies, postures and repeated movements are those that alter symptoms.

### Mechanical presentation

The outward manifestations of a musculoskeletal problem, such as deformity, loss of movement range, velocity of movement or movement deviations. Very important in re-assessment of treatment efficacy.

### Mechanical response

Change in mechanical presentation; for instance, an increase or decrease in range of movement in response to a particular loading strategy.

### Mechanical syndromes

Refers to the three mechanical syndromes as described by McKenzie – derangement, dysfunction and posture, which describe the majority of non-specific spinal problems.

### Mechanically determined directional preference

The phenomenon of preference for postures or movement in one direction is a characteristic of the derangement syndrome and is determined by mechanical evaluation. It describes the situation when postures or movements in one direction decrease, abolish or centralise symptoms and often increase a limitation of movement. Postures or movements in the opposite direction often cause these symptoms and signs to worsen. This does not always occur, and may be a product of the length of exposure to provocative loading.

### Non-mechanical factors

Factors that are non-mechanical in nature that may influence a patient's experience of pain. For instance, in the acute phase of a problem the pain-generating mechanism may be primarily inflammatory. In the chronic stage various non-mechanical factors, such as central or peripheral sensitisation or psychosocial factors, may influence pain modulation.

### Pain

*Acute pain*

Pain of recent onset of less than seven days. Will include some with pain of an inflammatory nature, but many will have pain of a mechanical nature due to derangement.

*Sub-acute*

Pain that lasts between seven days and seven weeks. In some this may represent an interface between inflammatory and mechanical pain, but again, mechanical factors are likely to predominate.

Glossary of Terms


*Chronic pain*
Pain that lasts for longer than seven weeks. In the majority this will be mechanical in nature, and non-mechanical in a minority.

*Chronic pain states*
Pain of long duration in which non-mechanical factors are important in pain maintenance. These factors may relate to peripheral or central sensitisation or psychosocial factors, such as fear-avoidance, etc. Symptoms are often widespread and aggravated by all activity, and patients display exaggerated pain behaviour and mistaken beliefs about movement and pain.

*Chemical or inflammatory pain*
Pain mediated by the inflammatory chemicals released following tissue damage or due to systemic pathology, such as ankylosing spondylitis.

*Mechanical pain*
Pain resulting from mechanical deformation of tissues. This occurs with abnormal stresses on normal tissues, as in the postural syndrome, and normal stresses on abnormal tissues, such as occurs in derangement and dysfunction.

*Constant pain*
Constant pain describes symptoms that are present throughout the patient's waking day, without any respite even though it may vary in intensity. This may be chemical or mechanical in origin, and may also exist in chronic pain states.

*Intermittent pain*
This describes pain that comes and goes during the course of the day. Commonly this relates to intermittent mechanical deformation that results in pain. Pain may be momentary or appear and linger for varying amounts of time, but does at some point in a day completely stop.

*Site and spread of pain*
The area in which pain is perceived in terms of the extent of referral into the limb. The most distal site of pain is important to monitor regarding centralisation and peripheralisation. This information provides important information during assessment and re-assessment of the symptomatic presentation. The degree of pain referral is a description of symptom severity. Management of derangements is described as for central and symmetrical or unilateral and asymmetrical symptoms, with the latter further divided between pain above or below the elbow.

*Severity of pain*
This information provides important information during assessment and re-assessment of the symptomatic presentation. Either the patient is asked on a one-to-ten scale the intensity of the pain on different occasions or in retrospect asked to compare present pain to when they first attended.

### Peripheralisation

Peripheralisation describes the phenomenon when pain emanating from the spine, although not necessarily felt in it, *spreads* distally into or further down the limb. This is the reverse of centralisation. In response to repeated movements or a sustained posture, if pain is produced and remains in the limb, spreads distally or increases distally, that loading strategy should be avoided. The phenomenon only occurs in the derangement syndrome. The temporary production of distal pain with end-range movement that does not worsen is not peripheralisation, as this response may occur with an adherent nerve root.

### Postural syndrome

Mechanical deformation of normal soft tissues arising from prolonged postural stresses, affecting any articular structures and resulting in pain. A distinguishing set of characteristics is found during the history-taking and physical examination. If prolonged sitting produces pain, it is abolished by posture correction. Range is full and pain-free, and repeated movements have no effect.

### 'Red Flags'

This refers to features of the history-taking that may indicate serious spinal pathology, such as cancer, cord compression or fracture. If possible 'red flag' pathology is suspected, further mechanical therapy is contraindicated and the patient should be referred to a specialist.

### Reliability

This is the characteristic of a test or measuring tool to give the same answer in different situations. Intertester reliability examines the degree of agreement between different clinicians on the same occasion; intratester reliability examines the degree of reliability of a single tester on different occasions. Results are presented in several ways: as percentile agreement, correlation coefficients or kappa values.

### Sensitivity

This is a characteristic of a clinical test used to diagnose a problem. The sensitivity is the ability of the test to be positive in all who have the problem. When a test is 100% sensitive, it is able to detect all who have the condition of interest. The sensitivity is the true positive rate. When sensitivity is extremely high (>0.95 or 95%), a negative test response rules out that disease. Poor sensitivity indicates a test that fails to identify many of those with the disease of interest.

### Specificity

This is a characteristic of a clinical test used to diagnose a problem. The specificity is the ability of a test not to be positive in those who do not have the problem; it is thus the true negative rate. When a test is 100% specific, it identifies all those who do not have the condition of interest. When specificity is extremely high (>0.95 or 95%), a positive test result gives a definite positive diagnosis. Poor specificity indicates a test that fails to exclude many individuals without the disease of interest.

### Stage of condition

All musculoskeletal conditions can be anywhere on the continuum from acute to sub-acute to chronic. These stages are often of more significance to management than a structural diagnosis.

### Standardised terms

These are used to make consistent descriptions of symptomatic responses to different loading strategies to judge their value for self-treatment. The description of symptoms during and *after* loading is significant in determining the management strategy to be applied. These are the words used to describe symptom response during the physical examination.

*During loading:*

| | |
|---|---|
| *Increase* | Symptoms already present are increased in intensity. |
| *Decrease* | Symptoms already present are decreased in intensity. |
| *Produce* | Movement or loading creates symptoms that were not present prior to the test. |
| *Abolish* | Movement or loading abolishes symptoms that were present prior to the test. |
| *Better* | Symptoms produce on movement, decrease on repetition. |

*Centralise* Movement or loading abolishes the most distal symptoms.

*Peripheralise* Movement or loading produces more distal symptoms.

*End-range pain* This is pain that only appears at end-range of movement, disappears once end-range is released, and in which the range does not rapidly change. In end-range pain due to derangement, increased force reduces symptoms, whilst in end-range pain due to dysfunction increased force increases symptoms.

*Pain during movement* Pain is produced during the range of movement, but then subsides or remains when the individual moves further into the range of movement. In the three mechanical syndromes in the spine, this only occurs in derangements.

*After loading:*

*Worse* Symptoms produced or increased with movement or loading remain aggravated following the test.

*Not Worse* Symptoms produced or increased with movement or loading return to baseline following the test.

*Better* Symptoms decreased or abolished with movement or loading remain improved after testing.

*Not Better* Symptoms decreased or abolished with movement or loading return to baseline after testing.

*Centralised* Distal symptoms abolished by movement or loading remain abolished after testing.

*Peripheralised* Distal symptoms produced during movement or loading remain after testing.

*No Effect* Movement or loading has no effect on symptoms during or after testing.

### State of tissues

This describes the different conditions in which tissues could be found. They may be normal or abnormal. Abnormal tissues may be injured, healing, scarred or contracted, with healing suspended, hypersensitive to normal loading due to changes in the nervous system, degenerated or painful due to derangements.

Glossary of Terms


### 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 in decisions concerning management.

### Symptomatic presentation

This describes the details of the patient's complaints, and can be assessed and re-assessed regarding site, intermittency/constancy, diurnal variation, severity, consequent analgesic/NSAID consumption and self-reported functional disability. This is very important in re-assessment of treatment efficacy.

### Symptomatic response

The behaviour of pain in response to a particular loading strategy, for instance centralisation, peripheralisation, worse or better.

### Traffic Light Guide

Identification of patient's responses to loading strategies, using standardised terminology, determines the appropriateness of a management direction. If the patient remains worse afterwards this is a 'red light' to that procedure; if the patient remains better this is a 'green light' for that exercise; if there is no change, an 'amber light'; a force progression or force alternative *may* be required. An 'amber' response is also a 'green light' in the presence of a dysfunction.

### Treatment principle

The treatment principle describes the direction of movement used in management; they are termed extension, flexion or lateral. Each principle of treatment contains patient- and clinician-generated force progressions. In a derangement the treatment principle is determined by the direction that causes a decrease, abolition or centralisation of pain. In a dysfunction the treatment principle is determined by the direction that reproduces the relevant symptom.

### Validity

This is the ability of a test to diagnose or measure what it is intended to diagnose or measure. There are various dimensions of validity, but criterion validity is critical to the accuracy of a diagnosis. This is the ability of a test to determine the presence or absence of a particular pathology. The value of a test is judged by its ability to diagnose the pathology compared to a 'gold standard'. The validity of the

'gold standard' is meant to be about 100%. Validity is measured by sensitivity and specificity.

### 'Yellow flags'

Term used to describe psychosocial risk factors for developing or perpetuating long-term disability or sick leave as a consequence of musculoskeletal symptoms. They include factors such as the attitudes and beliefs of the patient about their problem, their behavioural responses to it, compensation issues, inappropriate health care advice, information or treatment, emotions such as depression, anxiety and fear of movement, and relations with family and work.

# Index

## U



## V



## W

## X



## Z