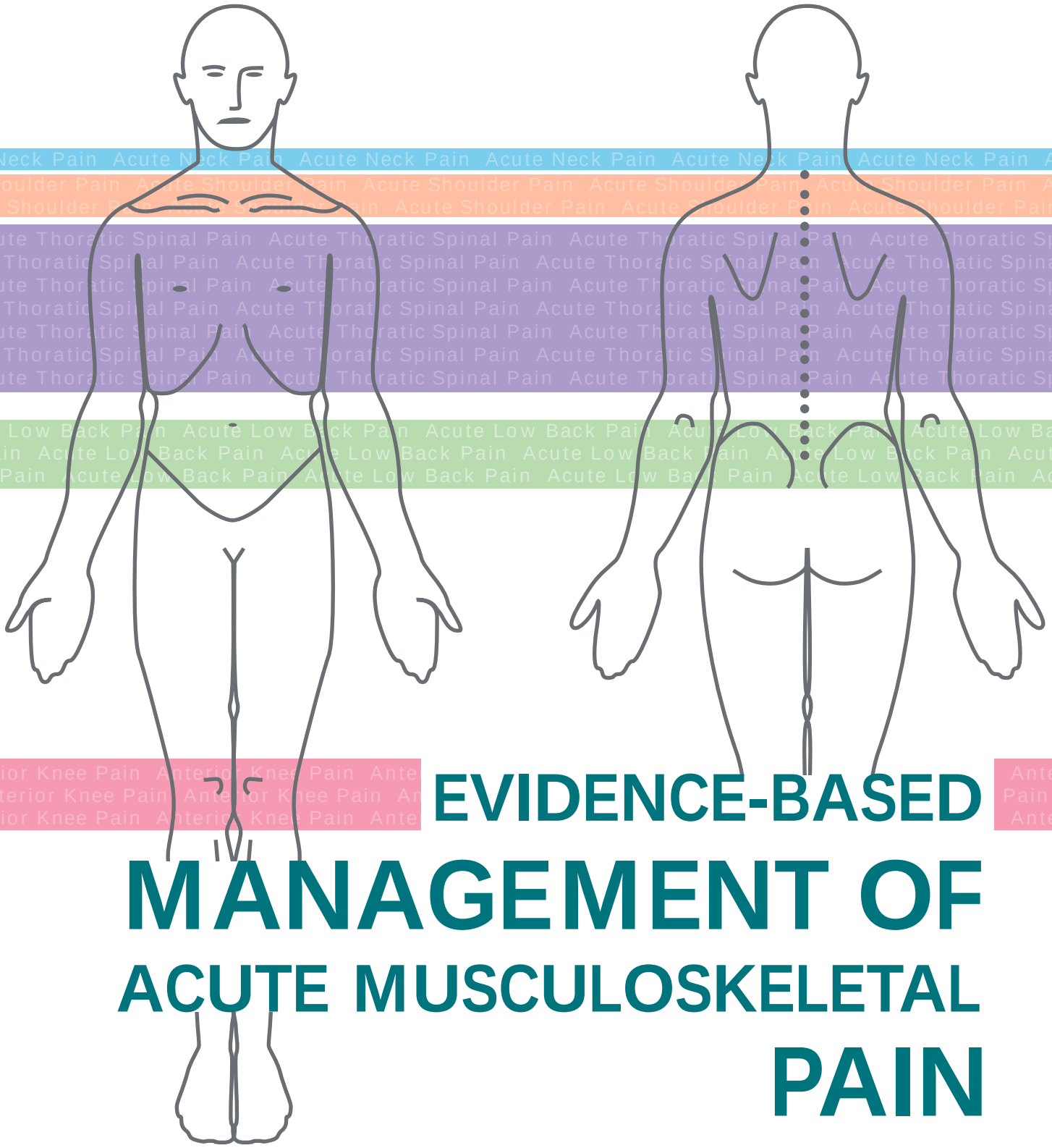

EVIDENCE-BASED MANAGEMENT OF ACUTE MUSCULOSKELETAL PAIN

Australian Acute Musculoskeletal Pain Guidelines Group

Australian Government
National Health and Medical Research Council

APPROVED 5 JUNE 2003

BY THE NATIONAL HEALTH AND MEDICAL RESEARCH COUNCIL

EVIDENCE-BASED MANAGEMENT OF ACUTE MUSCULOSKELETAL PAIN

Australian Acute Musculoskeletal Pain Guidelines Group

AUSTRALIAN ACADEMIC PRESS PTY LTD
Brisbane

PARTICIPATION

The work is a joint initiative of the University of Queensland and the Commonwealth Department of Health and Ageing. The evidence review was undertaken by a multi-disciplinary group. The following organisations participated in the review and have approved the contents:

- Australian and New Zealand College of Anaesthetists, Faculty of Pain Medicine
- Australian Osteopathic Association
- Australian Rheumatology Association
- Australian Physiotherapy Association
- Chiropractic and Osteopathic College of Australasia
- Chiropractors' Association of Australia
- Consumers' Health Forum of Australia
- Royal Australian College of General Practitioners

DISCLAIMER

Every attempt has been made to locate the most recent scientific evidence. Judgment is necessary when applying evidence in a clinical setting. It is important to note that weak evidence does not necessarily mean that a practice is unadvisable, but may reflect the insufficiency of evidence or the limitations of scientific investigation.

This document is intended as a guide to practice. The ultimate decision of how to proceed rests with the clinician and the patient and depends on individual circumstances and beliefs (NHMRC 1999).

First published in December 2003 by
Australian Academic Press Pty. Ltd.
32 Jeays Street
Bowen Hills QLD 4006
Australia
www.australianacademicpress.com.au

All responsibility for editorial matter rests with Australian Acute Musculoskeletal Pain Guidelines Group.
Any views or opinions expressed are therefore not necessarily those of Australian Academic Press.

National Library of Australia Cataloguing-in-Publication data:

Evidence-based management of acute musculoskeletal pain.

Bibliography.
For medical professionals.
ISBN 1 875378 49 9.

1. Musculoskeletal system — Diseases — Treatment.
2. Evidence-based medicine. 3. Pain — Treatment. I.
Australian Acute Musculoskeletal Pain Guidelines Group.

616.7

Cover and text design by Andrea Rinarelli of Australian Academic Press, Brisbane.
Typeset in Adobe Garamond by Australian Academic Press, Brisbane.

Contents

List of Tables

List of Figures

About the Group

Australian Acute Musculoskeletal Pain Guidelines Group

Executive Committee

Professor Peter Brooks, Chair	Executive Dean, Faculty of Health Sciences, University of Queensland
Associate Professor Lyn March	Consultant Rheumatologist and Clinical Epidemiologist; Associate Professor in Medicine and Public Health, University of Sydney; Senior Staff Specialist, Royal North Shore Hospital
Professor Nikolai Bogduk	Director, Newcastle Bone and Joint Institute, Royal Newcastle Hospital
Professor Nick Bellamy	Director, Centre of National Research on Disability and Rehabilitation Medicine, University of Queensland

Project Management

Ms Natalie Spearing	Project Manager, Faculty of Health Sciences, University of Queensland
Ms Megan Fraser	Project Assistant, Faculty of Health Sciences, University of Queensland

Review Groups

Acute Low Back Pain

Associate Professor Lyn March	Consultant Rheumatologist and Clinical Epidemiologist; Associate Professor in Medicine and Public Health, University of Sydney; Senior Staff Specialist, Royal North Shore Hospital
Dr Lyndal Trevena	General Practice, University of Sydney Department of General Practice
Mr Simon French	Chiropractor, Chiropractic and Osteopathic College of Australasia
Ms Trudy Rebbeck	Musculoskeletal Physiotherapist, Australian Physiotherapy Association
Dr Fiona Blyth	Medical Epidemiologist, Pain Management and Research Centre, Royal North Shore Hospital
Professor Nick Bellamy	Director, Centre of National Research on Disability and Rehabilitation Medicine, University of Queensland
Ms Rebecca Coghlan	Consumer Representative, Consumers' Health Forum of Australia
Dr Nick Penney	Osteopath, Australian Osteopathic Association
Dr Hanish Bagga	PhD Fellow, Institute of Bone and Joint Research, University of Sydney

Acute Thoracic Spinal Pain

Dr Michael Yelland	General Practice, Musculoskeletal Medicine Australasian Faculty of Musculoskeletal Medicine
Dr Keith Charlton	Chiropractor
Associate Professor Gwendolen Jull	Physiotherapist, Australian Physiotherapy Association
Professor Nick Bellamy	Director, Centre of National Research on Disability and Rehabilitation Medicine, University of Queensland
Professor Peter Brooks	Executive Dean, Faculty of Health Sciences, University of Queensland

Acute Neck Pain

Professor Nikolai Bogduk	Professor of Pain Medicine; Director, Newcastle Bone and Joint Institute, Royal Newcastle Hospital
Dr Philip Bolton	Chiropractor, Head of Human Physiology, University of Newcastle
Associate Professor Gwendolen Jull	Physiotherapist, Australian Physiotherapy Association
Professor Nick Bellamy	Director, Centre of National Research on Disability and Rehabilitation Medicine, University of Queensland

Dr Phillip Giles	General Practice, Musculoskeletal Medicine Australasian Faculty of Musculoskeletal Medicine
Associate Professor Les Barnsley	Rheumatologist, Australian Rheumatology Association
Acute Shoulder Pain	
Dr Wade King	Pain Medicine Specialist, Newcastle Pain Management and Research Group, Royal Newcastle Hospital
Associate Professor Rachelle Buchbinder	Rheumatologist, Australian Rheumatology Association
Associate Professor Sally Green	Physiotherapist, Australian Physiotherapy Association
Dr Scott Masters	General Practice, Musculoskeletal Medicine Australasian Faculty of Musculoskeletal Medicine
Dr Henry Pollard	Chiropractor, Chiropractic and Osteopathic College of Australasia
Dr Peter Nash	Rheumatology, Director Rheumatology Research Unit, Nambour Hospital
Professor Nick Bellamy	Director, Centre of National Research on Disability and Rehabilitation Medicine, University of Queensland
Professor Peter Brooks	Executive Dean, Faculty of Health Sciences, University of Queensland
Dr Simon Bell	Orthopaedic Surgeon, Victoria
Anterior Knee Pain	
Dr David Vivian	General Practice, Musculoskeletal Medicine Australasian Faculty of Musculoskeletal Medicine
Associate Professor Lyn March	Consultant Rheumatologist and Clinical Epidemiologist; Associate Professor in Medicine and Public Health, University of Sydney; Senior Staff Specialist, Royal North Shore Hospital
Dr Sallie Cowan	Centre for Sports Medicine Research, School of Physiotherapy, University of Melbourne
Dr Kay Crossley	Centre for Sports Medicine Research, School of Physiotherapy, University of Melbourne
Professor Nick Bellamy	Director, Centre of National Research on Disability and Rehabilitation Medicine, University of Queensland
Dr Myles Coolican	Orthopaedic Surgeon, New South Wales
Matthew Burke	Postgraduate Medical School, Northern Clinical School, University of Sydney
Ryan Clark	Postgraduate Medical School, Northern Clinical School, University of Sydney

Steering Committee

Associate Professor Les Barnsley	Australian Rheumatology Association
Dr Philip Bolton	Chiropractors' Association of Australia
Associate Professor Rachelle Buchbinder	Australian Rheumatology Association
Ms Rebecca Coghlan	Consumers' Health Forum of Australia
Associate Professor Milton Cohen	Australian and New Zealand College of Anaesthetists, Faculty of Pain Medicine
Professor Paul Glasziou	Royal Australian College of General Practitioners
Associate Professor Sally Green	Australian Physiotherapy Association
Dr Newman Harris	Australian and New Zealand College of Anaesthetists, Faculty of Pain Medicine
Associate Professor Gwendolen Jull	Australian Physiotherapy Association
Ms Roz Lucas	Commonwealth Department of Health and Ageing
Dr Peter MacIsaac	Commonwealth Department of Health and Ageing
Professor John Murtagh	Royal Australian College of General Practitioners
Dr Nick Penney	Australian Osteopathic Association

Chapter 1

Executive Summary

➔ *This document is the outcome of a multi-disciplinary review of the scientific evidence for the diagnosis, prognosis and treatment of acute musculoskeletal pain. The evidence is summarised in the form of a management plan and key messages that may be used to inform practice. The aim in conducting an evidence review is to facilitate the integration of the best available evidence with clinical expertise and the values and beliefs of patients.*

The project was proposed and coordinated by Professor Peter Brooks, Executive Dean of the Faculty of Health Sciences, The University of Queensland. The guideline development process was overseen by a national steering committee and undertaken by multi-disciplinary review groups. Funding for the project was received from the Commonwealth Department of Health and Ageing.

The evidence review was conducted according to standards outlined by the National Health and Medical Research Council (NHMRC) (1999a) and in accordance with ideas expressed by the pioneer of evidence-based medicine, Dr Archie Cochrane (1977). Cochrane proposed the rationalisation of interventions (both diagnostic and therapeutic) to promote those with evidence of safety and effectiveness. To that end he suggested: promoting diagnostic tests likely to have a beneficial effect on prognosis, evaluating existing interventions to exclude those shown to be ineffective or dangerous, and determining the place of interventions when there is insufficient evidence of benefit.

Rationale

Pain and disability associated with musculoskeletal conditions represent a significant health burden in Australia. Musculoskeletal disorders (arthritis, and musculoskeletal conditions including osteoporosis) cost Australia in excess of 15 billion dollars per annum, including direct and indirect costs (Access Economics 2001a,b). This evidence review complements the government's acknowledgement of the importance of arthritis and musculoskeletal conditions and their designation of this field as Australia's 7th national health priority area. The project aligns with the international Bone and Joint Decade initiative, and its two major Australian partners, Osteoporosis Australia and Arthritis Australia.

Within this context, this review of the scientific evidence for the management of acute musculoskeletal pain aims to promote informed and effective management of such pain, empower consumers and advance understanding of acute musculoskeletal pain through identification of research needs.

Summaries of this document have been developed for clinicians and for patients to promote a collaborative approach to decision-making. This approach is particularly important when a range of management options exists, as patients will bear the consequences of decisions affecting their health (Charles et al. 1999). The summary documents are available at http://www.nhmrc.gov.au

Scope

- This document provides information on the management of acute pain, communication between clinicians and patients, and the diagnosis, prognosis and interventions for acute low back, thoracic spine, neck, shoulder and anterior knee pain.
- The document is concerned only with the management of acute episodes of pain (less than three months duration) that are not associated with specific diseases and serious conditions. Discussion of the management of specific conditions is beyond the scope of this document.
- Existing unpublished draft guidelines developed by the Australasian Faculty of Musculoskeletal Medicine formed the basis for the document. Multi-disciplinary groups undertook the work of updating the draft guidelines. Information on how the existing work was updated is provided in each topic.
- Where sufficient evidence has been available, recommendations have been made; however the aim of this work is to provide clinicians and patients with information to guide decisions rather than being prescriptive.
- This master document containing the review of evidence serves as the source for summary publications for clinicians and patients. Same-source information promotes partnership in decision-making and facilitates the provision of informed consent.
- This document is not intended to, nor should there be any implication that it would be used in a regulatory fashion to dictate practice.
- The results of economic evaluations and cost information are included, where possible, to promote consideration of the efficient distribution of resources.

- A research agenda has been generated to highlight knowledge gaps in this area.
- The evidence contained in this document is current to January 2003. Search dates are specified in each guideline topic.

Summary of Findings

A number of themes have emerged from this review of the diagnosis, prognosis and treatment of acute musculoskeletal pain, forming the basis of the management plan:

- An episode of acute musculoskeletal pain is of short duration (less than three months). Recurrent episodes of acute musculoskeletal pain may occur, and a few people will develop chronic pain. Early identification of people at risk of chronic pain facilitates early intervention.
- Clinical assessment comprising a history and physical examination is important to identify features of rare but serious causes of acute musculoskeletal pain. In the majority of the remaining cases it is not possible to determine the cause of acute musculoskeletal pain and a specific diagnosis is not required for effective management.
- Ancillary investigations are generally not indicated for acute musculoskeletal pain.
- Simple interventions (providing information, assurance and encouraging reasonable maintenance of activity) may be all that are required for the successful management of acute musculoskeletal pain. These interventions can be used in combination with other non-pharmacological and pharmacological treatments.
- People with acute musculoskeletal pain should be reviewed to evaluate progress and to check for latent features of serious conditions ('red flags') and psychosocial and occupational factors ('yellow flags') that may influence recovery.
- Management of acute musculoskeletal pain involves a partnership approach; a management plan should be developed by the clinician and the patient and tailored to suit individual needs.

Limitations of Findings

- The vast majority of studies located in the search were performed in tertiary settings; there are limitations to applying the findings to other settings.
- There is both a lack of evidence (i.e. few or no studies conducted) and a lack of high quality, generalisable results in this area. The absence of evidence does not mean that an intervention is not efficacious.
- Insufficient or conflicting evidence for an intervention does not mean there is no benefit. Clinical decisions should be made with knowledge of the existing evidence and consideration of individual needs.
- There are limitations to the results of some systematic reviews as some have attempted to pool data from heterogeneous interventions. Specific and uniformly applied definitions for treatment modalities are required.
- There are difficulties in both locating and comparing the results of different studies due to the wide variety of terms used to describe acute musculoskeletal pain.
- The use of a variety of outcome measures limits the ability to compare results between studies.
- Few articles draw a distinction between acute and chronic durations of pain in relation to interventions for musculoskeletal pain. When there was a lack of studies involving specifically 'acute' populations, systematic reviews comprising a mixture of studies on acute and chronic populations were included.
- The decision to restrict the update of the evidence on interventions to Level I and II studies (with the exception of the thoracic spinal pain guidelines) precluded the inclusion of the results of Level III and IV studies on treatment.
- The authors acknowledge that the levels of evidence used in these guidelines were developed to rank studies of interventions and may not adequately reflect the study quality for other question types (e.g. diagnosis and prognosis), where cross-sectional and cohort studies may be the design of choice. An asterix has been used to highlight this limitation to readers.

Summary of Key Messages: Acute Pain Management

	EVIDENCE LEVEL
Management Plan	
It is recommended that the clinician and patient develop a management plan for acute musculoskeletal pain comprising the elements of assessment, management and review: • Assessment — Conduct a history and physical examination to assess for the presence of serious conditions; ancillary investigations are not generally indicated unless features of serious conditions are identified. • Management — Provide information, assurance and advice to resume normal activity and discuss other options for pain management as needed. • Review — Reassess the pain and revise the management plan as required.	CONSENSUS: Steering Committee
Non-Pharmacological Interventions	
Simple interventions (providing information, assurance and encouraging reasonable maintenance of activity) may be used alone or in combination with other interventions for the successful management of acute musculoskeletal pain.	CONSENSUS: Steering Committee
Pharmacological Interventions	
Specific pharmacological interventions may be required to relieve pain; such agents can be used in conjunction with non-pharmacological interventions.	CONSENSUS: Steering Committee; NHMRC 1999b

Acute Pain Management continued

Paracetamol or other simple analgesics, administered regularly, are recommended for relief of mild to moderate acute musculoskeletal pain.	CONSENSUS: Steering Committee; NHMRC 1999b
Where paracetamol is insufficient for pain relief, a non-steroidal anti-inflammatory (NSAID) medication may be used, unless contraindicated.	CONSENSUS: Steering Committee; NHMRC 1999b
Oral opioids may be necessary to relieve severe musculoskeletal pain. It is preferable to administer a short-acting agent at regular intervals, rather than on a pain-contingent basis. Ongoing need for opioid analgesia is an indication for reassessment.	CONSENSUS: Steering Committee; NHMRC 1999b
Adjuvant agents such as anticonvulsants and antidepressants are not recommended in the management of acute musculoskeletal pain.	CONSENSUS: Steering Committee; NHMRC 1999b
Any benefits from muscle relaxants may be outweighed by their adverse effects, therefore they cannot be routinely recommended.	CONSENSUS: Steering Committee

Summary of Key Messages: Effective Communication

	EVIDENCE LEVEL
Clinicians should work with patients to develop a management plan so that patients know what to expect, and understand their role and responsibilities.	CONSENSUS: Steering Committee
Information should be conveyed in correct but neutral terms, avoiding alarming diagnostic labels; jargon should be avoided.	CONSENSUS: Steering Committee
Explanation is important to overcome inappropriate expectations, fears or mistaken beliefs that patients may have about their condition or its management.	CONSENSUS: Steering Committee
Printed materials and models may be useful for communicating concepts.	CONSENSUS: Steering Committee
Clinicians should adapt their method of communication to meet the needs and abilities of each patient.	CONSENSUS: Steering Committee
Clinicians should check that information that has been provided has been understood; barriers to understanding should be explored and addressed.	CONSENSUS: Steering Committee

Summary of Key Messages: Acute Low Back Pain

DIAGNOSIS	EVIDENCE LEVEL
Aetiology and Prevalence	
The majority (approximately 95% of cases) of acute low back pain is non-specific; serious conditions are rare causes of acute low back pain.	*LEVEL I, III: Deyo et al. 1992; Suarez-Almazor et al. 1997; Hollingworth et al. 2002
Common findings in patients with low back pain (e.g. osteoarthritis, lumbar spondylosis, spinal canal stenosis) also occur in asymptomatic people; hence, such conditions may not be the cause of the pain.	*LEVEL I, III: van Tulder et al. 1997a; Torgerson and Dotter 1976
History	
History enables screening for features of serious conditions; however the reliability and validity of individual features in histories have low diagnostic significance.	*LEVEL III-2: Deyo et al. 1992; van den Hoogen et al. 1995
Physical Examination	
Clinical signs detected during physical and psychosocial assessment must be interpreted cautiously as many tests lack reliability and validity.	*LEVEL III-2: LeBoeuf-Yde et al. 2002; Truchon and Fillion 2000; Knutson 2002; Waddell et al. 1980; Deyo et al. 1992
A full neurological examination is warranted in the presence of lower limb pain and other neurological symptoms.	*LEVEL IV: Waddell et al. 1982; McCombe et al. 1989

Acute Low Back Pain continued

Ancillary Investigations	
Plain xrays of the lumbar spine are not routinely recommended in acute non-specific low back pain as they are of limited diagnostic value and no benefits in physical function, pain or disability are observed.	*LEVEL III-2: Suarez-Almazor et al. 1997; Hollingworth et al. 2002; Kendrick et al. 2001; Kerry et al. 2002
Appropriate investigations are indicated in cases of acute low back pain when alerting features ('red flags') of serious conditions are present.	*LEVEL III-2: Deyo and Diehl 1986
Terminology	
A specific patho-anatomic diagnosis is not necessary for effective management of acute non-specific low back pain.	CONSENSUS: Steering Committee
Terms to describe acute low back pain with no identifiable pathology include 'lumbar spinal pain of unknown origin' or 'somatic lumbar spinal pain'.	*LEVEL IV: Merskey and Bogduk 1994
PROGNOSIS	**EVIDENCE LEVEL**
The majority of people with a short duration of symptoms upon presentation with low back pain recover within three months; however milder symptoms often persist.	*LEVEL III-2: Croft and Rigby 1994; Schiottz-Christensen et al. 1999
Recurrences of acute low back pain are not uncommon.	*LEVEL III-3: van den Hoogen et al. 1998; Hurley et al. 2001a
Psychosocial and occupational factors ('yellow flags') appear to be associated with progression from acute to chronic pain; such factors should be assessed early to facilitate intervention.	*LEVEL III-2: Linton 2001; Pincus et al. 2002; Truchon and Fillion 2000
INTERVENTIONS	**EVIDENCE LEVEL**
Evidence of Benefit	
Advice to Stay Active (Activation) — Advice to stay active provides a small beneficial effect on pain, rate of recovery and function compared to bed rest and compared to a specific exercise regime in mixed populations with low back pain. Advice to stay active reduces sick leave compared to bed rest in mixed populations with low back pain.	LEVEL I, II: Based on systematic reviews (Waddell et al. 1997; Hagen et al. 2002; Hilde et al. 2002) and one additional study (Rozenberg et al. 2002)
Heat Wrap Therapy — Continuous low level heat wrap therapy reduces pain, stiffness and disability extending for three to four days compared with paracetamol, NSAIDs or placebo alone during the first 48 hours of acute low back pain. (This treatment is not routinely available in Australia).	LEVEL II: Based on one study (Nadler et al. 2002)
Patient Information (Printed) — Novel or 'activity-focused' printed information plus similar verbal advice provided by a clinician is more effective compared to traditional brochures or no printed information in acute low back pain. Printed information provided through the mail is less likely to have an effect on pain, disability and sick leave compared to information provided in person. Behavioural therapy interventions are more effective than printed information for preventing long-term disability in mixed populations.	LEVEL II: Based on controlled trials (Cherkin et al. 1996; Cherkin et al. 1998; Burton et al. 1999; Hazard et al. 2000; Roberts et al. 2002; Linton and Andersson 2000)
Conflicting Evidence	
Muscle Relaxants — There is conflicting evidence that muscle relaxants are effective compared to placebo in acute low back pain. There is insufficient evidence to determine whether muscle relaxants are more or less effective compared to NSAIDs for acute low back pain. Drowsiness, dizziness and dependency are common adverse effects of muscle relaxants.	LEVEL I: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b) that found numerous RCTs

Acute Low Back Pain continued

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) — There is conflicting evidence that oral and injectable NSAIDs are effective versus placebo or no treatment for acute low back pain. NSAIDs have a similar effect compared to opioid analgesics, combined paracetamol-opioid analgesics and to each other in their effect on acute low back pain. There is insufficient evidence that NSAIDs are more effective when compared to muscle relaxants and anti-anxiety agents in acute low back pain. NSAIDs are less effective in reducing pain than heat wrap therapy in the first three to four days of acute low back pain. Serious adverse effects of NSAIDs include gastrointestinal complications (e.g. bleeding, perforation).	LEVEL I, II: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b; van Tulder et al. 2002f; Koes et al. 1997) and numerous RCTs (Amlie et al. 1987; Basmajian 1989; Postacchini et al. 1988; Lacey et al. 1984; Nadler et al. 2002)
Spinal Manipulation — There is conflicting evidence that spinal manipulation provides pain relief compared to placebo in the first two to four weeks of acute low back pain. There is insufficient evidence that spinal manipulation is more or less effective than other conservative treatments for acute low back pain. Adverse effects of spinal manipulation are rare but potentially serious.	LEVEL I, II: Based on systematic reviews (van Tulder et al. 1997b; Bigos et al. 1994; Koes et al. 1996; Mohseni-Bandpei et al. 1998; Shekelle et al. 1992) and one RCT (Hsieh et al. 2002) LEVEL IV: Based on reviews of case studies (Haldeman and Rubinstein 1992; Assendelft et al. 1996; Stevinson and Ernst 2002)
Insufficient Evidence	
Acupuncture — There is insufficient evidence that acupuncture (dry-needling) is effective compared to injection therapy in acute low back pain. Adverse effects of acupuncture are rare but potentially serious.	LEVEL I: Based on a systematic review (van Tulder et al. 2002a) and one study (Garvey et al. 1989)
Analgesics, Compound and Opioid — There are no randomised controlled trials investigating the efficacy of opioids and compound analgesics in acute low back pain. There is evidence that the effect of opioid or compound analgesics is similar to NSAIDs for treatment of acute low back pain. In general, opioids and compound analgesics have a substantially increased risk of side effects compared with paracetamol alone.	No Level I or II evidence LEVEL I: Based on systematic reviews (van Tulder et al. 1997b; Bigos et al. 1994) and RCTs (Brown et al. 1986; Videman et al. 1984; Palangio et al. 2002)
Analgesics, Simple — There are no randomised controlled trials assessing the effectiveness of simple analgesics in acute low back pain. There is insufficient evidence for the effectiveness of simple analgesics versus NSAIDs in acute low back pain. Paracetamol is less effective than heat wrap therapy in acute low back pain. There is insufficient evidence for the effect of paracetamol compared to electroacupuncture in mixed populations with low back pain.	No Level I or II evidence LEVEL I, II: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b) of studies by Milgrom et al. 1993; Wiesel et al. 1980; Hackett et al. 1988
Back Exercises — McKenzie therapy provides similar pain and function outcomes compared to usual care in acute low back pain. There is conflicting evidence for the efficacy of back exercises in reducing pain and disability compared to other active and inactive treatments in mixed populations with low back pain. McKenzie therapy reduces pain and sick leave compared to one back school session, results in similar global improvement compared to manipulation and provision of an educational booklet and provides better functional and pain outcomes compared to flexion exercises in mixed populations with low back pain. Lateral multifidus muscle exercises reduce recurrences of low back pain compared to usual care in mixed populations with low back pain.	LEVEL I, II: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b; van Tulder et al. 2002d) of multiple controlled studies

Acute Low Back Pain continued

Back School — There is insufficient evidence that back school is more effective in reducing pain compared to active and passive therapies and to placebo in acute low back pain. There is insufficient evidence that back school is more effective in reducing pain compared to placebo and other treatments in mixed populations with low back pain.	LEVEL I, II: Based on systematic reviews (van Tulder et al. 1997b; van Tulder et al. 2002b) and an RCT by Hsieh et al. (2002)
Bed Rest — There is insufficient evidence that bed rest is more effective compared to advice to stay active, back exercises, spinal manipulation, non-steroidal anti-inflammatory drugs or no treatment in mixed populations with low back pain. There is conflicting evidence that bed rest increases disability and rate of recovery compared to staying active in mixed populations with low back pain. Bedrest for longer than two days increases the amount of sick leave compared to early resumption of normal activity in acute low back pain. There is evidence that prolonged bed rest is harmful.	LEVEL I, II: Based on systematic reviews (van Tulder et al. 1997b; Hagen et al. 2002) and an RCT (Rozenberg et al. 2002)
Cognitive Behavioural Therapy — Cognitive behavioural therapy reduces general disability in the long term compared to traditional care in mixed with populations back pain. Group cognitive behavioural therapy sessions may reduce sick leave and health care utilisation in the long term compared to general educational information in mixed populations with back pain. While cognitive behavioural strategies are often included as part of specific interventions for acute low back pain such as exercise and activity restoration, there are no studies on this approach as a single intervention.	LEVEL I: Based on systematic reviews (Turner 1996; van Tulder et al. 2002e) LEVEL II: Based on studies by Linton and Andersson (2000) and Linton and Ryberg (2001) No Level I or II studies
Electromyographic Biofeedback — There are no controlled studies testing the effectiveness of electromyographic biofeedback in acute low back pain.	No Level I or II evidence
Injection Therapy — There is insufficient evidence demonstrating the effectiveness of injection therapy (facet joint, epidural or soft tissue) in the treatment of acute low back pain. Adverse effects of injection therapy are rare but serious.	LEVEL I, II: Based on systematic reviews (Nelemans et al. 2002; Watts and Silagy 1995; Koes et al. 1999) and an RCT (Garvey et al. 1989)
Lumbar Supports — There are no controlled studies on the effect of lumbar supports in acute low back pain. There is insufficient evidence that lumbar supports are effective in reducing pain compared to spinal manipulation, exercises, massage, TENS and simple analgesia in mixed populations with low back pain.	No Level I or II evidence LEVEL I: Based on two systematic reviews (van Tulder et al. 2002c; Bigos et al. 1994)
Massage — There are no controlled studies for massage therapy in acute low back pain. Massage is superior to placebo (sham laser) and acupuncture in mixed populations with low back pain. Massage provides similar effect to back schools (involving exercise and education), corsets and TENS in mixed populations with low back pain. There is conflicting evidence of the effect of massage compared to manipulation and education in mixed populations with low back pain.	No Level I or II evidence LEVEL I, II: Based on systematic reviews (Furlan et al. 2002; Ernst 1999) and RCTs (Cherkin et al. 2001; Preyde 2000)
Multi-Disciplinary Treatment in the Workplace — There are no controlled studies on the effect of multi-disciplinary treatment in the workplace in acute low back pain. Multi-disciplinary treatment in the workplace improves return to work and subjective disability compared to usual care in mixed populations with low back pain.	No Level I or II evidence LEVEL I, II: Based on a systematic review (Karjailanen et al. 2002) and RCTs (Loisel et al. 1997; Lindstrom 1992a,b)
Topical Treatment — There is insufficient evidence for the effectiveness of spiroflar homeopathic gel or cremol capsici for treatment of acute low back pain.	LEVEL II: Based on one RCT (Stam et al. 2001)

Acute Low Back Pain continued

Traction — There are no controlled studies on the effect of traction for acute low back pain. There is insufficient evidence that traction is effective compared to placebo and compared to other treatments in mixed populations with low back pain. Adverse effects from traction have been reported, including reduced muscle tone, bone demineralisation, thrombophlebitis.	No Level I or II evidence LEVEL I: Based on systematic reviews (van der Heijden et al. 1995; van Tulder et al. 1997b)
Transcutaneous Electrical Nerve Stimulation — There are no controlled studies on the effect of TENS in acute low back pain. There is insufficient evidence for the effectiveness of TENS compared to exercises, back books, massage, corset use and simple analgesia in mixed populations with low back pain.	No Level I or II evidence LEVEL I, II: Based on a systematic review (van Tulder et al. 1997b) and additional studies (Pengel et al. 2002; Hurley et al. 2001b)
Cost Effectiveness — Published data is very limited; however there is some evidence that advice to maintain usual activities, provision of an education booklet and community-based exercises appear to be cost effective first line interventions for acute low back pain.	LEVEL II: Malmivaara et al. 1995; Cherkin et al. 1998; Moffet et al. 1999

Summary of Key Messages: Acute Thoracic Spinal Pain

DIAGNOSIS	EVIDENCE LEVEL
Aetiology and Prevalence	
Pain may be referred to the upper thoracic spine from visceral structures and cervical spinal structures or arise in the thoracic interspinous ligaments, paravertebral muscles and zygapophyseal joints	*LEVEL IV: Kelley 1997; Dwyer et al. 1990; Aprill et al. 1990; Fukui et al. 1996; Feinstein et al. 1954; Kellgren et al. 1939; Hockaday and Whitty 1967; Cloward 1959; Kellgren 1939; Dreyfuss et al. 1994
Men and women aged over 60 are at risk for spontaneous osteoporotic fractures of the thoracic spine; extent of vertebral deformity and multiple fractures appear linked with pain intensity.	*LEVEL IV: Ross et al. 1994; Patel et al. 1991; Huang et al. 1994
Clinicians should be alert to the potential for rare, serious conditions presenting as acute thoracic spinal pain; however most cases of thoracic spinal pain are of mechanical origin.	*LEVEL IV: Deyo and Diehl. 1988
History	
History serves to differentiate sources of acute thoracic spinal pain to identify features of potentially serious conditions; however it carries little diagnostic weight.	CONSENSUS: Flynn 1996; Kenna and Murtagh 1989; Corrigan and Maitland 1988
Physical Examination	
The reliability of palpation for tenderness of the thoracic spine is good but its validity is unknown.	*LEVEL IV: Christensen et al. 2002
The reliability of motion palpation of the thoracic spine is marginal.	*LEVEL IV: Love et al. 1987; Christensen et al. 2002
Following blunt trauma, a negative clinical examination in the presence of a clear sensorium makes a thoracic spinal fracture unlikely.	*LEVEL IV: Durham et al. 1995; Samuels and Kerstein 1993
Despite the absence of supportive, scientific data on the utility of physical examination of the thoracic spine, such examination provides an important opportunity to identify features of serious conditions.	*LEVEL IV: Deyo et al. 1988; Malawaski et al. 1991; Durham et al. 1995; Samuels and Kerstein 1993
Ancillary Investigations	
In the absence of trauma, plain radiography is of limited use in defining the cause of pain.	*LEVEL IV: Wood et al. 1995; Nathan 1962; Crawford and Singer 1995
Fractures are more likely to occur in people over age 60 with a history of blunt trauma; a lower threshold for investigation is warranted in this group.	*LEVEL IV: Frankel et al. 1994; Durham et al. 1995; Meldon and Moettus. 1995; Samuels and Kerstein 1993

Acute Thoracic Spinal Pain continued

In the presence of trauma, xray of the thoracolumbar spine is not indicated in those who are awake, alert and have no clinical evidence of injury; however those with equivocal or positive clinical findings or with an altered level of consciousness should undergo thoracolumbar spine evaluation.	*LEVEL IV: Samuels and Kerstein 1993; Durham et al. 1995
CT scanning is only indicated for the evaluation of the neural canal and posterior elements of the thoracic spine when fractures have been detected with plain films.	*LEVEL IV: Keene et al. 1982
There is no research to inform ancillary investigations for acute thoracic spinal pain; investigations should be selected on the basis of clinical features suggesting the presence of serious conditions.	CONSENSUS: Steering Committee
Terminology	
The appropriate labels for non-specific 'mechanical' thoracic spinal pain are 'thoracic spinal pain of unknown origin' or 'somatic thoracic spinal pain'.	CONSENSUS: Merskey and Bogduk 1994
PROGNOSIS	**EVIDENCE LEVEL**
There is a lack of published data on the natural history and influence of prognostic risk factors for acute thoracic spinal pain.	NO EVIDENCE
INTERVENTIONS	**EVIDENCE LEVEL**
Evidence of Benefit	
Spinal Manipulation — There is evidence from one small study that spinal manipulation is effective compared to placebo in thoracic spinal pain.	LEVEL II: Schiller 2001

Summary of Key Messages: Acute Neck Pain

DIAGNOSIS	EVIDENCE LEVEL
Aetiology and Prevalence	
Acute neck pain is most commonly idiopathic or attributed to a whiplash accident; serious causes of acute neck pain are rare (< 1%).	*LEVEL III-3: Based on cross-sectional and prospective radiological surveys (Heller et al. 1983; Johnson and Lucas 1997)
Degenerative changes, osteoarthrosis or spondylosis of the neck are neither causes nor risk factors for idiopathic neck pain.	*LEVEL III: Based on epidemiological and radiological surveys (van der Donk et al. 1991; Fridenberg and Miller 1963)
The most consistent determinant of idiopathic neck pain is the social nature of the work environment; occupation and stress at work are weakly associated risk factors.	*LEVEL III: Based on multiple epidemiological surveys (Makela et al. 1991; Kamwendo et al. 1991a; Linton and Kamwendo 1989; Vasseljen et al. 1995; Fredriksson et al. 2002; Ariens et al. 2001)
Involvement in a motor vehicle accident is not a risk factor for developing neck pain; however individuals who experience neck pain soon after such an event are more likely to develop chronic neck pain.	*LEVEL III: Based on a prospective epidemiological study (Berglund et al. 2000)
History	
Attention should be paid to the intensity of pain because regardless of its cause, severe pain is a prognostic risk factor for chronicity and patients with severe pain may require special or more concerted interventions.	CONSENSUS: Review Group and Steering Committee
The hallmarks of serious causes of acute neck pain are to be found in the nature and mode of pain onset, its intensity and alerting features.	CONSENSUS: Review Group and Steering Committee
Eliciting a history aids the identification of potentially threatening and serious causes of acute neck pain and distinguishes them from non-threatening causes.	CONSENSUS: Review Group and Steering Committee

Acute Neck Pain continued

Physical Examination	
Physical examination does not provide a patho-anatomic diagnosis of acute idiopathic or whiplash-associated neck pain as clinical tests have poor reliability and lack validity.	*LEVEL III: Gross et al. 1996; Fjellner et al. 1999; Smedmark et al. 2000; Nansel et al. 1989; De Boer et al. 1985; Mior et al. 1985; Youdas et al. 1991; Viikari-Juntura 1987
Despite limitations, physical examination is an opportunity to identify features of potentially serious conditions.	CONSENSUS: Review Group and Steering Committee
Tenderness and restricted cervical range of movement correlate well with the presence of neck pain, confirming a local cause for the pain.	*LEVEL III: Sandmark and Nisell 1995
Ancillary Investigations	
Plain radiography is not indicated for the investigation of acute neck pain in the absence of a history of trauma, or in the absence of clinical features of a possible serious disorder.	*LEVEL III: Based on radiological surveys (Heller et al. 1983; Johnson and Lucas 1997; Hoffman et al. 2000)
In symptomatic patients with a history of trauma, radiography is indicated according the Canadian C-Spine Rule.	*LEVEL III: Based on a large epidemiological survey (Stiell et al. 2001)
CT is indicated only when: plain films are positive, suspicious or inadequate; plain films are normal but neurological signs or symptoms are present; screening films suggest injury at the occiput to C2 levels; there is severe head injury; there is severe injury with signs of lower cranial nerve injury, or pain and tenderness in the sub-occipital region.	CONSENSUS: Based on published consensus views (El Khoury et al. 1995; Kathol 1997)
Acute neck pain in conjunction with features alerting to the possibility of a serious underlying condition is an indication for MRI.	CONSENSUS: Consensus view (El Khoury et al. 1995)
Terminology	
Except for serious conditions, precise identification of the cause of neck pain is unnecessary.	CONSENSUS: Review Group and Steering Committee
Once serious causes have been recognised or excluded, terms to describe acute neck pain can be either ‘acute idiopathic neck pain’ or ‘acute whiplash-associated neck pain’.	CONSENSUS: Review Group and Steering Committee
PROGNOSIS	**EVIDENCE LEVEL**
Approximately 40% of patients recover fully from acute idiopathic neck pain, approximately 30% continue to have mild symptoms and 30% of patients continue to have moderate or severe symptoms.	*LEVEL III: Based on retrospective surveys (Gore et al. 1987; Lees and Turner 1963)
Approximately 56% of patients fully recover within three months from onset of acute whiplash-associated neck pain, 80% recover fully within one or two years; 15–40% continue to have symptoms and 5% are severely affected.	*LEVEL III, LEVEL IV: Based on prospective studies (Radanov et al. 1995; Kasch et al. 2001) and other studies with limitations (Brison et al. 2000)
Psychosocial factors are not determinants of chronicity in whiplash-associated neck pain.	*LEVEL III: Radanov et al. 1991; Borchgrevink et al. 1997
Risk factors for chronicity of following whiplash-associated neck pain are older age at time of injury, severity of initial symptoms, past history of headache or head injury.	*LEVEL III: Based on prospective studies (Radanov and Sturzenegger 1996; Suissa et al. 2001)
INTERVENTIONS	**EVIDENCE LEVEL**
Evidence of Benefit	
Advice to Stay Active (Activation) — Encouraging resumption of normal activities and movement of the neck is more effective compared to a collar and rest for acute neck pain.	LEVEL I, II: Based on systematic reviews (Spitzer et al. 1995; Verhagen et al. 2002) and a controlled trial (Borchgrevink et al. 1998)

Acute Neck Pain continued

Exercises — Gentle neck exercises commenced early post-injury are more effective compared to rest and analgesia or information and a collar in acute neck pain. Exercises performed at home are as effective for neck pain as tailored outpatient treatments at two months and appear to be more effective at two years after treatment.	LEVEL II: Based on controlled trials for short-term data (McKinney et al. 1989; Rosenfeld et al. 2000) and a blinded prospective randomised trial for long-term data, with limitations (McKinney 1989)
Multi-Modal Therapy — Multi-modal (combined) treatments inclusive of cervical passive mobilisation in combination with specific exercise alone or specific exercise with other modalities are more effective for acute neck pain in the short term compared to rest, collar use and single modality approaches.	LEVEL I, II: Based on a systematic review (Gross et al. 2002c) and two randomised controlled trials (Bonk et al. 2000; Hoving et al. 2002)
Pulsed Electromagnetic Therapy (PEMT) — Pulsed electromagnetic therapy reduces pain intensity compared to placebo in the short term but is no different to placebo at 12 weeks for acute neck pain.	LEVEL I: Based on systematic reviews (Gross et al. 2002b; Kjellman et al. 1999) of two controlled trials (Foley-Nolan et al. 1990, 1992)
Insufficient Evidence	
Acupuncture — There are no randomised controlled studies on the effect of acupuncture or infrared acupuncture in the treatment of acute neck pain. There is conflicting evidence that acupuncture is more effective compared to placebo and other treatments for neck pain in mixed populations.	No Level I or II evidence LEVEL I: Based on systematic reviews (White and Ernst 1999; Harms-Ringdahl and Nachemson 2000; Gross et al. 2002b; Smith et al. 2000)
Analgesics, Opioid — Opioids may be used, however there are no randomised controlled studies of its effectiveness for acute neck pain. In general, opioid and compound analgesics have a substantially increased risk of side effects compared with paracetamol alone.	No Level I or II evidence LEVEL I: Based on a systematic review not specific to neck pain (de Craen et al. 1996)
Analgesics, Simple — Simple analgesics may be used to treat mild to moderate pain however there is insufficient evidence that paracetamol is more effective than placebo, natural history or other measures for relieving acute neck pain.	No Level I or II evidence
Cervical Manipulation — There are no randomised controlled trials investigating the effect of cervical manipulation in the treatment of acute neck pain. Adverse effects of cervical manipulation are rare but potentially serious.	No Level I or II evidence LEVEL I: Based on systematic reviews (Hurwitz et al. 1996; Gross et al. 2002c)
Cervical Passive Mobilisation — There are no randomised controlled studies on the effect of cervical passive mobilisation compared to natural history or placebo in the treatment of acute neck pain.	No Level I or II evidence
Electrotherapy — There is insufficient evidence that electrotherapy is effective compared to no treatment in acute neck pain.	LEVEL I: Based on a systematic review (Verhagen et al. 2002) that identified two controlled trials with limitations (Fialka et al. 1989; Hendriks and Horgan 1996)
Gymnastics — There are no randomised controlled trials on the effect of gymnastics for acute neck pain. Gymnastics may be no more effective than natural history in mixed populations.	No Level I or II evidence LEVEL I: Based on a systematic review (Kjellman et al. 1999) that identified one controlled trial involving mixed populations (Takala et al. 1994)
Microbreaks — There is insufficient evidence that taking regular breaks from computer work is more effective compared to irregular breaks for preventing acute neck pain.	LEVEL II: Based on one controlled study with limitations (McLean et al. 2001)

Acute Neck Pain continued

Multi-Disciplinary Biopsychosocial Rehabilitation — There are no randomised controlled studies investigating the effect of multi-disciplinary treatment in acute neck pain.	No Level I or II evidence
There is insufficient evidence that multi-disciplinary treatment is effective compared to other interventions for reducing neck pain in mixed populations.	LEVEL I, II: Based on a systematic review (Karjalainen et al. 2002) that identified two controlled trials and two subsequent trials that all involved mixed populations
Muscle Relaxants — There are no randomised controlled trials investigating the efficacy of muscle relaxants for the treatment of acute neck pain.	No Level I or II evidence
Muscle relaxants are no more effective than placebo for neck pain in mixed populations.	LEVEL I, II: Based on a systematic review (Aker et al. 1996) of two studies plus one additional study, all involving mixed populations
Drowsiness, dizziness and dependency are common adverse effects of muscle relaxants.	LEVEL I: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997)
Neck School — There are no randomised controlled trials on the effect of neck school for acute neck pain.	No Level I or II evidence
Neck school appears no more effective than no treatment for neck pain in mixed populations.	LEVEL II: Based on one controlled trial (Kamwendo and Linton 1991) involving a mixed population
Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) — There are no randomised controlled trials on the effectiveness of NSAIDs for acute neck pain.	No Level I or II evidence
There is evidence that NSAIDs are no more effective than placebo ultrasound for neck pain in mixed populations.	LEVEL I: Based on a systematic review (Aker et al. 1996) that located two studies involving mixed populations
Serious adverse effects of NSAIDs include gastrointestinal complications. (e.g. bleeding, perforation)	LEVEL I: Based on systematic reviews (Bigos et al. 1994; Henry et al. 1996)
Patient Education — There are no randomised controlled trials investigating the effect of patient education as a single strategy in the treatment of acute neck pain.	No Level I or II evidence
Spray and Stretch Therapy — There are no randomised controlled trials investigating the effect of spray and stretch therapy in acute neck pain.	No Level I or II evidence
Spray and stretch therapy appears no more effective than placebo for neck pain in mixed populations.	LEVEL I: Based on one study reported in abstract form (Snow et al. 1992) cited in three systematic reviews (Aker et al. 1996; Harms-Ringdahl and Nachemson 2000; Gross et al. 2002b)
Traction — There are no randomised controlled trials investigating the effectiveness of traction for acute neck pain.	No Level I or II evidence
In mixed populations, there is evidence that traction is of no benefit compared to a range of other interventions for neck pain.	LEVEL I: Based on systematic reviews (Aker et al. 1996; Harms-Ringdahl and Nachemson 2000; Verhagen et al. 2002; van der Heijden et al. 1995; Gross et al. 2002b) of five studies with limitations involving mixed populations
Transcutaneous Electrical Nerve Stimulation (TENS) — There is insufficient evidence of benefit from TENS compared to a collar or manual therapy in acute neck pain.	LEVEL I: Based on a systematic review (Gross et al. 2002b) that identified one controlled trial (Nordemar and Thorner 1981) with equivocal results

Acute Neck Pain continued

Evidence of No Benefit	
Collars — Soft collars are not effective for acute neck pain compared to advice to resume normal activity and other interventions.	LEVEL I, II: Based on a systematic review (Harms-Ringdahl and Nachemson 2000) and multiple controlled trials

Summary of Key Messages: Acute Shoulder Pain

DIAGNOSIS	EVIDENCE LEVEL
Aetiology and Prevalence	
Clinicians should be alert to the potential for rare, serious conditions (e.g. fracture/dislocation, tumour, infection, inflammatory arthropathies) presenting as acute shoulder pain.	*LEVEL IV: numerous case studies (Jones et al. 1994; Kaempffe 1995; Barlow and Newman 1994; Welch 1994; Linos et al. 1980)
Most cases of acute shoulder pain are of 'mechanical' origin and can be managed as acute regional pain.	*LEVEL III-2, III-3: Torstensen and Hollinshead 1999; Chandnani et al. 1992; Milgrom et al. 1995; Sher et al. 1995
Biological factors such as age, female gender, past history and response to repetitive physical tasks may contribute to the development of acute shoulder pain.	*LEVEL III-3: Jones et al. 1994; Cummings et al. 1995; Sambrook 1996; Ekberg et al. 1995; Skov et al. 1996
Psychosocial factors such as job dissatisfaction and work demands may contribute to the onset of acute shoulder pain.	*LEVEL III-2: Bergenudd et al. 1994; Ekberg et al. 1995; Marcus et al. 1996; Skov et al. 1996
History	
Information obtained from the history may alert to the presence of a serious condition as the underlying cause of acute shoulder pain.	CONSENSUS: Steering Committee
The reliability and validity of individual features in histories have low diagnostic significance; the history is to be interpreted with caution when choosing a course of action.	*LEVEL III-2: Nørregaard et al. 2002; Litaker et al. 2000
Physical Examination	
Findings of shoulder examination must be interpreted cautiously in light of the evidence of limited utility; no clinical test is both reliable and valid for any specific diagnostic entity.	*LEVEL III-2: Calis et al. 2000; MacDonald et al. 2000; Naredo et al. 2002; Itoi et al. 1999; Bennett 1998
Causes of acute shoulder pain cannot be diagnosed by clinical assessment; however, with the exception of serious conditions, satisfactory outcomes do not depend on precise identification of cause.	CONSENSUS: Steering Committee
Despite limitations, physical examination is an opportunity to identify features of potentially serious conditions.	*LEVEL III-2: Bamji et al. 1996; Liesdeck et al. 1997; de Winter et al. 1999; Pal et al. 2000; Nørregaard et al. 2002
Ancillary Investigations	
Imaging is not necessary unless there are alerting features of serious conditions; in the absence of alerting features, the diagnostic utility of imaging is minimal and the results are unlikely to improve management.	*LEVEL III: Numerous studies (Torstensen and Hollinshead 1999; Teefey et al. 2000a,b; Tempelhof et al. 1999; Milgrom et al. 1995; Chandnani et al. 1992; Sher et al. 1995; Sher et al. 1998; Blanchard et al. 1999a)
There is a need to educate consumers about the limitations of imaging and the risks of radiation exposure.	*LEVEL IV: Roebuck 1995

Acute Shoulder Pain continued

Terminology	
Terms to describe acute shoulder pain should summarise the discernible features of the condition to form the basis for a management plan.	CONSENSUS: World Health Organisation 1986; Merskey and Bogduk 1994
PROGNOSIS	**EVIDENCE LEVEL**
Approximately 50% of people with acute shoulder pain (treated conservatively) recover within six months; approximately 60% recover within 12 months.	*LEVEL III-2: Van der Windt et al. 1996; Winters et al. 1997b
Shoulder pain may recur even in those who appear to fully recover in the short term.	*LEVEL III-2: Croft et al. 1996
INTERVENTIONS	**EVIDENCE LEVEL**
Evidence of Benefit	
Corticosteroid Injection — Subacromical corticosteroid injection for acute shoulder pain may improve pain at four weeks compared to placebo but this benefit is not maintained at 12 weeks.	LEVEL I: Systematic review of RCTs of adults with acute shoulder pain (Adebajo et al. 1990, Vecchio et al. 1993); systematic review of steroid injections for shoulder pain (Buchbinder et al. 2002)
Exercises — Exercises may improve shoulder pain compared to placebo in people with rotator cuff disease in both the short and longer term.	LEVEL I: Systematic review of two RCTs (Ginn et al. 1997; Brox et al. 1997)
Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) — Topical and oral NSAIDs improve acute shoulder pain by a small to moderate degree for up to four weeks compared to placebo.	LEVEL I: Systematic review of three RCTs of adults with acute shoulder pain (Ginsberg and Famaey 1991; Mena et al. 1986; Adebajo et al. 1990)
Serious adverse effects of NSAIDs include gastrointestinal complications (e.g. bleeding, perforation)	LEVEL I: Based on a systematic review (Bigos et al. 1994)
Ultrasound — Therapeutic ultrasound may provide short-term pain relief in calcific tendonitis compared to placebo.	LEVEL I: Systematic review of one RCT in acute shoulder pain (Ebenbichler et al. 1999)
Conflicting Evidence	
Acupuncture — There is conflicting evidence of the effectiveness of acupuncture compared to placebo ultrasound for shoulder pain and function.	LEVEL I: Systematic review (Green et al. 2003) of two RCTs (Kleinhenz et al. 1999; Berry et al. 1980)
Insufficient Evidence	
Analgesics — There are no randomised controlled trials investigating the use of analgesics (paracetamol or compound analgesics) for acute or chronic shoulder pain.	No Level I or II evidence
Extracorporeal Shock Wave Treatment (ESWT) — There are no randomised controlled trials of Extracorporeal Shock Wave Treatment for acute shoulder pain.	No Level I or II evidence
Trials conducted in populations with chronic shoulder pain show conflicting results for ESWT compared with placebo.	LEVEL I: Buchbinder et al. 2003a (systematic review of four RCTs)
Manual Therapy — Shoulder joint mobilisation with combined treatments (hot packs, active exercise, stretching, soft tissue mobilisation and education) may improve acute shoulder pain in the short term compared to the combined treatments alone.	LEVEL I: Systematic review located one RCT of 14 patients (Conroy and Hayes 1998)
Oral Corticosteroids — There are no randomised controlled trials investigating the use of oral corticosteroids for acute shoulder pain.	No Level I or II evidence
Studies of mixed populations do not report significant benefit from oral corticosteroids compared with placebo or no treatment for adhesive capsulitis.	LEVEL I: Green et al. 1998 (systematic review of two RCTs with methodological limitations)

Acute Shoulder Pain continued

Suprascapular Nerve Blocks — There are no published studies investigating the value of suprascapular nerve blocks for acute shoulder pain. There is some evidence of short-term effect from suprascapular nerve blocks for chronic adhesive capsulitis and rotator cuff disease.	No Level I or II evidence LEVEL I: Buchbinder et al. 2003b (systematic review of three RCTs)
Surgery — There are no published randomised controlled trials investigating the effectiveness of surgery for acute shoulder pain although studies exist for chronic populations.	No Level I or II evidence
Transcutaneous Electrical Nerve Stimulation (TENS) — There is insufficient evidence for the use of TENS for acute shoulder pain.	LEVEL I: Systematic review of on RCT (Shehab and Adham 2000)

Summary of Key Messages: Anterior Knee Pain

DIAGNOSIS	EVIDENCE LEVEL
Aetiology and Prevalence	
'Patellofemoral pain' is a general term used to describe idiopathic pain arising from the anterior knee/patellofemoral region that is of otherwise unknown origin.	CONSENSUS: Steering Committee
Anterior knee pain is commonly idiopathic; serious causes are rare.	*LEVEL IV: Kaempffe 1995; Ferguson et al. 1997; Kaandorp et al. 1995
Intrinsic risk factors for knee pain may include female gender, knee anatomy, joint laxity, muscle imbalance and prior injury. Extrinsic risk factors include occupation, sport and obesity.	*LEVEL IV: Kujala et al. 2001; Reider et al. 1981a,b; Witvrouw et al. 2000; Tanaka et al. 1989; Cooper et al. 1994
History	
The history provides information on possible causes of anterior knee pain and assists the identification of serious underlying conditions	CONSENSUS: Steering Committee
Physical Examination	
Although examination techniques lack specificity for diagnosing knee disorders, physical examination may assist the identification of serious conditions underlying anterior knee pain.	*LEVEL III, IV: Daniel 1991; Cook et al. 2001; Cushnagan et al. 1990; Biedert and Warnke 2001
Ancillary Investigations	
Indications for plain radiography are a history of trauma and: qualification under one of the Knee Rules, or sudden onset of severe pain, or alerting features of a serious condition.	*LEVEL III, IV: Chapman-Jones et al. 1998; Petit et al. 2001; Stiell et al. 1996; Seaberg and Jackson 1994; Bauer et al. 1995
Suspected fracture in the presence of a normal plain radiograph is an indication for CT scan.	CONSENSUS: Steering Committee
The presence of alerting features of a serious condition is an indication for the use of MRI.	CONSENSUS: Steering Committee
Swelling or potential rupture of anterior knee structures are indications for the use of ultrasound.	*LEVEL IV: Bianchi et al. 1994
Terminology	
The term 'patellofemoral pain' describes anterior knee pain for which there is no specific identifiable cause; it refers to the probable anatomical site of origin and is synonymous with retropatellar and patellofemoral joint pain.	CONSENSUS: Steering Committee
PROGNOSIS	**EVIDENCE LEVEL**
Multiple studies on a range of populations show a trend towards improvement with time; however, anterior knee pain persists to some degree in the majority of people.	*LEVEL IV: Nimon et al. 1998; Milgrom et al. 1996

Anterior Knee Pain continued

INTERVENTIONS	EVIDENCE LEVEL
Evidence of Benefit	
Advice to Stay Active (Activation) — Maintenance of normal activity has a beneficial effect on patellofemoral pain compared to no treatment and to the use of patellofemoral orthoses.	LEVEL II: Finestone et al. 1993
Injection Therapy — There is evidence that injection therapy (treatment and placebo saline) is effective for the management of patellofemoral pain in the short term compared to no injection therapy.	LEVEL II: Kannus et al. 1992
Orthoses (Foot) — There is evidence that corrective foot orthoses in combination with quadriceps and hamstring exercises are effective compared to placebo insoles in women with patellofemoral pain.	LEVEL I: Based on a systematic review (Crossley et al. 2001) that located one RCT (Eng and Pierrynowski 1993)
Exercises — A six-week regimen of quadriceps muscle retraining, patellofemoral joint mobilisation, patellar taping and daily home exercises significantly reduces patellofemoral pain compared to placebo in the short term.	LEVEL II: Based on one RCT (Crossley et al. 2002)
Eccentric quadriceps exercises produce better functional outcomes compared to standard quadriceps strengthening exercises.	LEVEL I: Based on a systematic review (Crossley et al. 2001) of eight RCTs
Conflicting Evidence	
Orthoses (Patellofemoral) — There is conflicting evidence that patellofemoral orthoses are effective compared to other interventions and to no treatment for patellofemoral pain.	LEVEL I: Based on two systematic reviews (Crossley et al. 2001; D'hondt et al. 2002)
Insufficient Evidence	
Acupuncture — There are no randomised controlled studies evaluating the effect of acupuncture for relief of patellofemoral pain.	No Level I or II evidence
Analgesics (simple and opioid) — There are no randomised controlled studies of the effectiveness of paracetamol or opioids versus placebo in the treatment of patellofemoral pain.	No Level I or II evidence
Electrical Stimulation — There are no randomised controlled studies of the effectiveness of electrical stimulation of the quadriceps muscle for patellofemoral pain.	No Level I or II evidence
There is insufficient evidence that one form of electrical stimulation of the quadriceps muscle is superior to another for treating patellofemoral pain.	LEVEL II: Callaghan et al. (2001)
Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) — There are no randomised controlled studies of the effectiveness of NSAIDs versus placebo in the treatment of patellofemoral pain.	No Level I or II evidence
Different types of NSAIDs provide similar relief of patellofemoral pain after five days of use.	LEVEL II: Based on one RCT with limitations (Fulkerson and Folcik 1986)
Serious adverse effects of NSAIDs include gastrointestinal complications (e.g. bleeding, perforation).	LEVEL I: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 2002)
Patellar Taping — There is insufficient evidence that patellar taping alone is effective in relieving patellofemoral pain, however it may be a useful adjunct to exercise therapy programs.	LEVEL I, II: Based on two systematic reviews (Crossley et al. 2001; Harrison et al. 2001) and one subsequent RCT (Crossley et al. 2002)
Progressive Resistance Braces — There is insufficient evidence that progressive resistance braces are effective in relieving patellofemoral pain compared to no treatment (this treatment is not routinely available in Australia).	LEVEL I: Based on a systematic review (Crossley et al. 2001) that located one RCT (Timm 1998)
Therapeutic Ultrasound — There is insufficient evidence that therapeutic ultrasound is more effective compared to ice massage for the treatment of patellofemoral pain.	LEVEL I: Based on a Cochrane Review (Brosseau et al. 2002b) and two meta-analyses (Gam and Johannsen 1995; van der Windt et al. 1999)

Anterior Knee Pain continued

Evidence of No Benefit	
Laser Therapy — There is evidence that low-level laser therapy provides similar effect to sham laser in the management of patellofemoral pain.	LEVEL I: Based on a systematic review (Crossley et al. 2001) that identified one RCT (Rogvi-Hansen et al. 1991)

Note: * Indicative only. A higher rating of the level of evidence might apply (refer to the note in Chapter 1: Executive Summary, Limitations of Findings).

>References

Access Economics (2001a). The prevalence, cost and disease burden of Arthritis in Australia: 2001. A report commissioned by the Arthritis Foundation of Australia. Access Economics: Canberra.

Access Economics (2001b). The burden of brittle bones: costing Osteoporosis in Australia. A report prepared for Osteoporosis Australia. Access Economics: Canberra.

Charles C, Whelan T, Gafni A (1999). What do we mean by partnership in making decisions about treatment? British Medical Journal, 319: 780–782.

Cochrane AL (1977). Effectiveness and Efficiency. Random Reflections on Health Services. Cambridge University Press: Cambridge.

National Health and Medical Research Council (1999a). A guide to the development, implementation and evaluation of clinical practice guidelines. Canberra: AGPS.

National Health and Medical Research Council (1999b). Acute pain management: information for general practitioners. Commonwealth of Australia. Canberra.

Chapter 2

Acute Pain Management

➔ *This chapter contains information that is generic to the management of all people with acute musculoskeletal pain.*

This chapter is based on information contained in the National Health and Medical Research Council (NHMRC) guidelines for the management of acute pain (1999). No systematic search or appraisal of the literature was conducted. Experts in pain management were consulted and provided additional information for this section.

Pain

Pain is the most common reason for self-medication and entry into the health care system (Eccleston 2001). Pain, acute and chronic, is now appreciated in a biopsychosocial model (Engel 1977) that acknowledges the biological, psychological and social dimensions of the pain experience.

This model acknowledges that pain is not simply determined either by somatic factors or by factors 'outside' the body but rather is the end result of a disturbance in nociceptive function interacting with a person's experience of being. This is influenced in turn by interaction with people, objects and events in the outside world, including the family, the community and the environment. Thus whilst knowledge of nociception and pain from a traditional medical science aspect is essential to the understanding of pain, it cannot be divorced from knowledge of perception and pain from a psychosocial point of view.

Pain is an individual, multi-factorial experience influenced by culture, previous pain experience, belief, mood and ability to cope. Pain may be an indicator of tissue damage but may also be experienced in the absence of an identifiable cause. The degree of disability experienced in relation to the experience of pain varies; similarly there is individual variation in response to methods to alleviate pain (Eccleston 2001).

Effective pain relief is considered a human right derived from these principles (NHMRC 1999):

- Unrelieved severe pain has adverse psychological and physiological effects.
- Patients should be involved in the assessment and management of their pain.
- To be effective, pain treatment should be flexible and tailored to individual needs.
- Pain should be treated early; established, severe pain is more difficult to treat.
- It should be possible to reduce pain to a comfortable or tolerable level.

Acute Pain

The term 'acute pain' refers to pain that has been present for less than three months (Bonica 1953; Merskey 1979). Successful management of pain in the acute phase is essential to prevent transition to chronic pain, which presents a significant individual, social and financial burden. Chronic pain is pain that has been present for longer than three months (Merskey and Bogduk 1994).

The NHMRC (1999) cites a number of misconceptions about the management of acute pain, including a lack of understanding of the pharmacokinetics of analgesics, mistaken beliefs about addiction, poor knowledge of dosage requirements, concerns about side effects and the concept that pain is not harmful.

Factors Influencing the Progression from Acute to Chronic Pain

Individuals vary in their potential to develop chronic pain. A combination of behaviours, beliefs and emotions may be involved in the transition from acute to chronic pain (Linton 2002). When pain is unrelieved over time, or if there are recurrent episodes of pain, persistent pain may develop.

The development of chronic pain is likely to be the result of small, cumulative changes in lifestyle that have been made to cope with acute musculoskeletal pain (Linton 2002). The intensity, duration and character of the pain influence the psychosocial response and the psychosocial response in turn influences the course of events.

There is strong evidence that psychosocial factors at work (i.e. occupational factors) are tied to the development of chronic pain. Job satisfaction may protect against the progression from acute low back pain to chronic low back pain. It is essential to identify those at risk of developing chronic pain and to intervene early to prevent this occurrence.

Pain Assessment

Pain History

The elements of a pain history (Figure 2.1) provide information that can alert to the presence of a serious underlying condition. It is important to note that in the absence of a serious cause for the pain (e.g. fracture), it is not necessary to obtain a specific patho-anatomic diagnosis to manage acute musculoskeletal pain effectively.

Site

The anatomical site where the person feels the pain may or may not be the site of origin as in the case of referred pain. The clinician should ask which part hurts the most and whether the pain started there or elsewhere.

Distribution

The regions in which pain is felt should be described. Even a person who initially complains of 'pain all over' can usually describe distinct region(s) of pain (possibly large and

Pain History

Site
Distribution
Quality
Duration
Temporal factors
Intensity
Aggravating factors
Relieving factors
Impact on activities of daily living
Associated symptoms
Onset
Previous similar symptoms
Previous treatment
Current treatment

Figure 2.1
Elements of a pain history.

overlapping). Having the patient draw their pain focus and radiation on a pain diagram (Figure 2.2) clarifies its distribution and can act as a baseline from which to assess response to treatment and changes in pain patterns.

Quality
The quality of pain may be described in different ways. Somatic pain is usually deep, dull and aching. Radicular pain is mostly sharp and 'electric' or 'shooting'. Neuropathic pain is often 'burning'. Visceral pain is dull at first but sharp when lining tissues such as the peritoneum become involved.

Duration
By convention, pain present for less than three months is described as 'acute' pain. Chronic pain refers to pain present for greater than three months duration. Pain duration will affect pain management.

Temporal Factors
Pain may be constant or intermittent. If pain is constant the history should elicit whether its intensity varies. If pain is intermittent, the history should elicit its pattern in relation to time of day, activity and duration.

Intensity
The intensity of pain reflects the impact of the experience, not necessarily the degree of nociception. Even though pain is essentially subjective (Merskey and Bogduk 1994) it is important to assess the intensity of the pain. Simple tools can be used to assess pain at the initial and follow-up visits to evaluate progress. There is good correlation between the various types of scales (Jensen at al. 1986). The Numerical Rating Scale is suitable for many clinical situations because it is simple to apply (refer to Figures 2.3, 2.4, 2.5).

Aggravating and Relieving Factors
Aggravating factors include those that precipitate or worsen pain. Relieving factors are those that alleviate, reduce or abolish pain. People who say that nothing eases the pain can be asked about the posture in which they are least uncomfortable.

Impact on Activities of Daily Living and Sleep
The effects of pain on activities of daily living (ADL) determine associated disabilities and handicaps (WHO 1986).

Pain Diagram

Please describe the pain problem:

Please indicate with an 'x' on these figures where your main pain is. Shade any area where your pain spreads. Please number (2,3,4 etc) any other areas where you have pain.

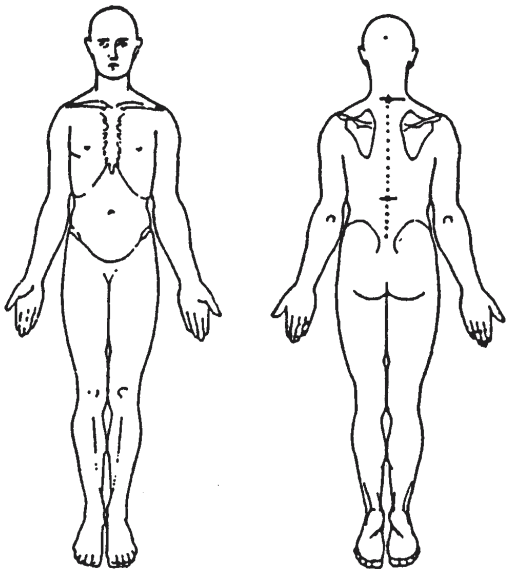

Figure 2.2
Pain diagram. Based on National Health and Medical Research Council (1999). Acute Pain Management: Scientific Evidence. Commonwealth of Australia: Canberra.

Identifying such effects gives the clinician an idea of the impact of pain on the patient's lifestyle. The effect of pain on sleep should be specifically sought; sleep deprivation is a powerful amplifier of the pain experience.

Associated Symptoms
These include any symptom apparently associated with the painful condition, in contrast to symptoms associated with other conditions the person may also have.

Onset (Precipitating Event)
The first appearance of the pain and the circumstances in which it started should be assessed. The clinician should distinguish between an event that may have aggravated rather than precipitated the pain.

Previous Similar Symptoms
Previous experience of similar symptoms suggests a recurrent condition.

Previous Action to Relieve Pain
All measures used for the condition before (and their effectiveness) should be noted. Unwanted effects associated with past treatment should also be recorded. Information on how each intervention was applied can be helpful, as treatment 'failures' may be due to misapplication rather than to true failure of effect.

Current Action to Relieve Pain
All forms of treatment in current use should be noted. The clinician should ask about the use of physical interventions, including self-applied measures, all passive treatments, and all

Figure 2.3

Categorical pain rating scale. Based on National Health and Medical Research Council (1999). Acute Pain Management: Scientific Evidence. Commonwealth of Australia: Canberra.

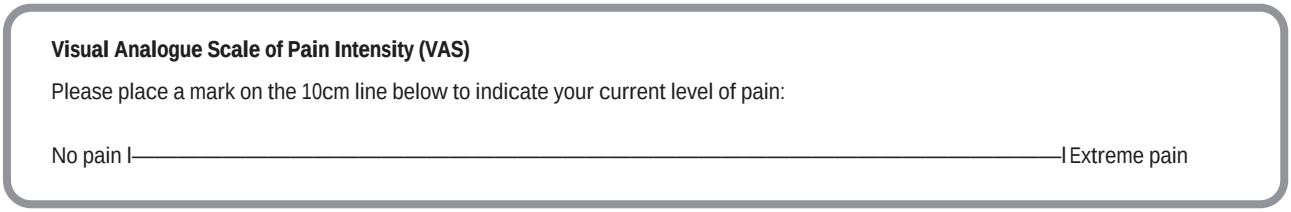

Figure 2.4

Visual analogue scale. Based on National Health and Medical Research Council (1999). Acute Pain Management: Scientific Evidence. Commonwealth of Australia: Canberra.

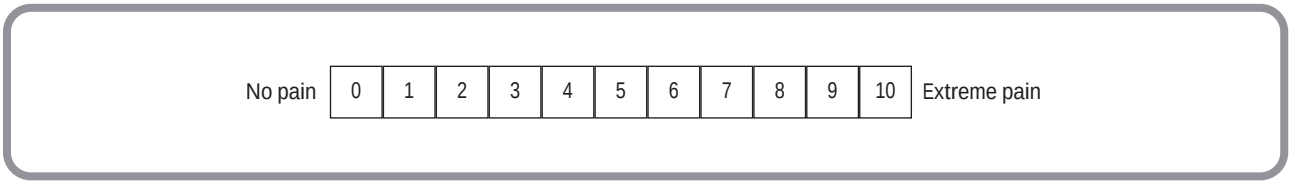

Figure 2.5

Ten point numerical rating scale (NRS). Based on National Health and Medical Research Council (1999). Acute Pain Management: Scientific Evidence. Commonwealth of Australia: Canberra.

substances whether prescribed or otherwise that the person is taking or applying, with an appraisal of the helpfulness of each.

Other History

Social and Occupational History

The social and occupational history provides information on the personal, social and environmental context. It may include information on close relationships, domicile, occupation (with details of work tasks), present and previous employment, sources of income, education, occupational and other qualifications, and leisure interests.

Psychosocial History

Elicitation of psychosocial history is aimed at understanding the pain (Engel 1977) and identifying any significant psychosocial issues that may place the person at risk of developing chronic disability. The aspects to be explored include: general affect, understanding of and reaction to the painful condition, associated fears, relevant cognitions and beliefs (personal and socio-cultural), and coping strategies used in relation to the painful condition.

Intercurrent Conditions

The history of intercurrent conditions should be elicited and note taken of any symptom or condition that may have a bearing on the pain problem.

Past and Current Medical History

The patient's medical history should be explored and note taken of any condition that may have a bearing on the pain condition. All forms of treatment in current use for other conditions should be ascertained and particular note taken of any that may have a bearing on the pain condition or its treatment.

Systems Review

Information can be obtained on past or present symptoms from each system of the body to assess for conditions that may influence the pain condition.

'Red Flags' and 'Yellow Flags'

'Red Flags'

The term 'red flags' refers to clinical (i.e. physical) features that may alert to the presence of serious but relatively uncommon conditions or diseases requiring urgent evaluation. Such conditions include tumours, infection, fractures and neurological damage. Screening for serious conditions occurs as part of the history and physical examination and should occur at the initial assessment and subsequent visits. Alerting features of serious conditions are covered in detail in the specific guideline topics.

'Yellow Flags'

The term 'yellow flags' was introduced to identify psychosocial and occupational factors that may increase the risk of chronicity in people presenting with acute low back pain. The New Zealand Guidelines Group (www.nzgg.org.nz) developed guidelines for assessing 'yellow flags' in acute low back pain (1998), outlining factors that should be assessed, particularly when progress is slower than expected. The presence of such factors is a prompt for further detailed assessment and early intervention. The areas to evaluate include:

- attitudes and beliefs about pain
- behaviours

- compensation issues
- diagnostic and treatment issues
- emotions
- family
- work

'Red flags' and 'yellow flags' are not mutually exclusive and intervention may be required for both clinical and psychosocial risk factors.

Pain Management

Von Korff (1999) demonstrated that people in pain want to: know what the problem is; be reassured that it is not serious; be relieved of their pain; and receive information. People in pain want advice regarding the management of their pain, including non-pharmacological and pharmacological interventions. They also want advice on how to return to normal activity.

Patients may lack current knowledge of interventions for pain management. For instance, they may believe that xrays will determine the cause of their pain and that bed rest is indicated. It is important to satisfy the need for knowledge, alleviate fear and to focus on preventing disability due to pain (Main 2002). The use of a preventive approach to shape behaviour is best done at the initial visit. This is particularly important in acute musculoskeletal pain, which may recur.

The following is a suggested framework to manage acute musculoskeletal pain:

1. Elicit a pain history in a biopsychosocial context.
2. Assess for clinical features ('red flags') of serious conditions including serious systemic illness, fracture, tumour and infection. If such features are present, further investigation or referral is warranted.
3. Assess for the presence of psychosocial and occupational factors ('yellow flags') that may affect the presentation of acute pain, response to treatment and influence the risk of progression to chronic pain.
4. Provide information on the prognosis of acute musculoskeletal pain and discuss options for pain management (pharmacological, non-pharmacological and activity).
5. Develop a management plan in conjunction with the patient, fostering a cooperative environment and reinforcing the importance of self-management.

Pain Management Plan

The management plan (Figure 2.6) should be tailored to meet the needs of each patient, taking their preferences and abilities into account. It is important to ensure that the patient understands what is involved to facilitate their participation.

Management plans are designed to assist progress through an episode of acute pain and the return to normal function. The plan should include actions that the consumer and clinician may take in the event of an exacerbation or recurrence of pain or slow progress to recovery. The plan should enable the individual to take responsibility for his or her own rehabilitation (bearing in mind that some people will require greater levels of support and assistance) or to seek help from a clinician if necessary.

There are three phases of the management plan:

- Assessment
- Management
- Review

Assessment

- A history and physical examination are conducted to assess whether clinical features of serious conditions ('red flags') are present and to identify psychosocial and occupational factors ('yellow flags') that may influence recovery.
- Ancillary investigations are not generally indicated unless features of serious conditions are identified.
- In cases where features of serious conditions are present, an alternative plan of management is required.

Management

- Provide information — consumers seek an explanation and information about the nature of their pain. The clinician should use effective communication techniques and use appropriate terms to describe acute musculoskeletal pain.
- Provide assurance — the natural history of acute musculoskeletal pain is generally favourable; thus, epidemiological data serves as the basis for assurance that recovery can be expected. Information on the prognosis and the provision of assurance is an integral part of the management plan.
- Provide advice to remain active — activity should be encouraged; resumption of normal activity should occur as soon as possible. For each of the conditions covered by these guidelines, activation is a seminal intervention for restoring function and avoiding disability.
- Discuss other options for pain management including the addition of non-pharmacological and pharmacological interventions to the management plan to assist return to normal activity. A combination of measures may be used. The clinician should provide information on the options available, what they are designed to achieve and describe potential risks and benefits. It is important not to overstate the power of interventions to avoid unrealistic expectations. It is also important to avoid the assumption that consumers expect medication each time they visit. On the contrary, many do not want their consultation ended prematurely by the writing of a prescription.

Review

- Prescription of a single, one-step intervention is unlikely to be successful. The plan may be iterative, requiring small amendments or major changes. On subsequent visits, the clinician should enquire whether the plan has been satisfactory and explore questions, concerns and possible alternatives as required. Further explanation and assurance can be provided.
- Ongoing review provides an important opportunity to assess for features of serious conditions and psychosocial factors that may not have been evident on previous visits and to intervene as required.
- Review also demonstrates concern that progress has been made. This is particularly important when there was intense pain and distress at the initial presentation. The need for further visits can be discussed at each consultation.

Key Messages

> It is recommended that the clinician and patient develop a management plan for acute musculoskeletal pain comprising the elements of assessment, management and review. (CONSENSUS)

- Assessment — Conduct a history and physical examination to assess for the presence of serious conditions; ancillary investiga-

tions are not generally indicated unless features of serious conditions are identified.

- Management — Provide information, assurance and advice to resume normal activity and discuss other options for pain management as needed.
- Review — Reassess the pain and revise the management plan as required.

Interventions for Acute Musculoskeletal Pain

In addition to initial interventions such as providing information, assurance and advice to maintain reasonable activity levels, other options (non-pharmacological and pharmacological) exist for the management of acute musculoskeletal pain.

Non-pharmacological Interventions

Evidence for the effectiveness of a range of additional non-pharmacological (i.e. not involving medication) interventions for people with acute musculoskeletal pain is provided in the specific guideline topics. These include active, passive and behavioural therapies. Non-pharmacological interventions may be used in conjunction with pharmacological interventions (NHMRC 1999).

Key Message

Simple interventions (providing information, assurance and encouraging reasonable maintenance of activity) may be used alone or in combination with other interventions for the successful management of acute musculoskeletal pain. (CONSENSUS)

Pharmacological Interventions

Simple Analgesics (Non-Opioid)

Paracetamol is considered an effective medication for mild to moderate pain and can be used in conjunction with opioids to manage more severe pain.

Generally, paracetamol has few side effects. Paracetamol is contraindicated for people with liver dysfunction. It can be used when NSAIDs are contraindicated. Patients should be warned of the risk of liver damage with the combination of alcohol and paracetamol.

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)

NSAIDs are considered effective in the management of mild to moderate pain. Concurrent use of opioids and NSAIDs may provide more effective analgesia than either of the drug classes alone. They may also reduce the side effects of opioid medications (NHMRC 1999).

The adverse effects of NSAIDs are potentially serious and all people cannot use them. NSAID use may result in gastro intestinal bleeding, renal dysfunction (particularly in older people), NSAID-induced asthma and impaired blood clotting. It is imperative that contraindications are identified and respected (e.g. asthma, peptic ulcer) (NHMRC 1999).

More recently, Cox-2 selective NSAIDs have become available. Evidence for their efficacy in a number of rheumatological disorders has been demonstrated. Currently they are not subsidised for acute musculoskeletal pain in Australia.

Key Messages

> Specific pharmacological interventions may be required to relieve pain; such agents can be used in conjunction with interventions. (CONSENSUS)

> Paracetamol or other simple analgesics administered regularly are recommended for relief of mild to moderate acute musculoskeletal pain. (CONSENSUS)

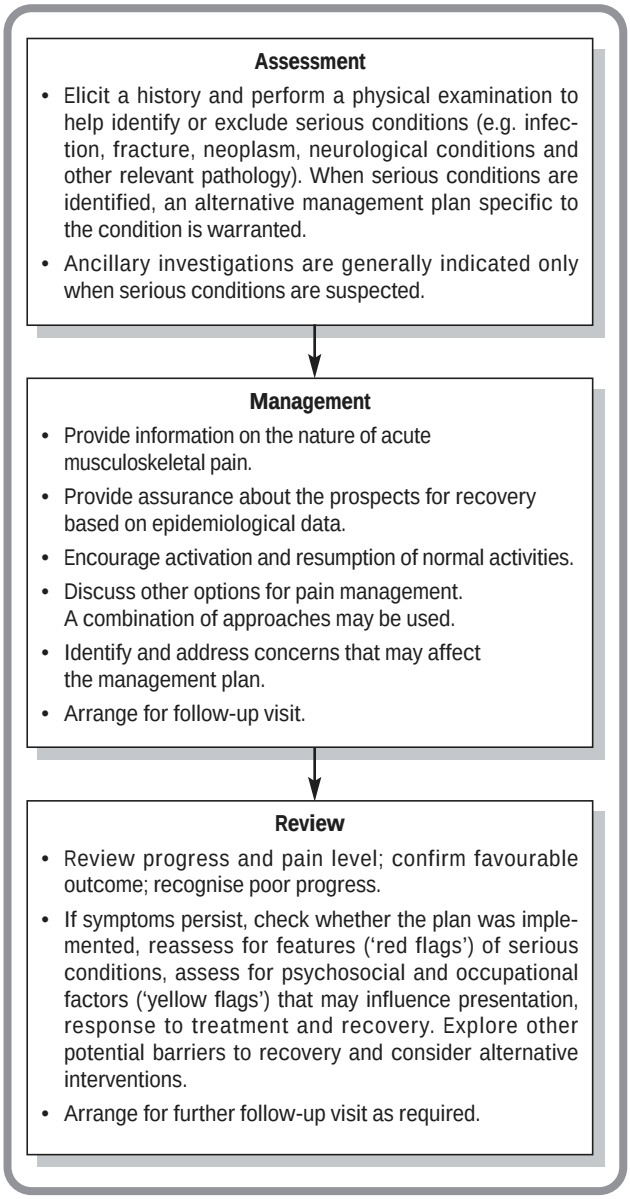

Figure 2.6

Management plan for acute musculoskeletal pain

> Where paracetamol is insufficient for pain relief, a non-steroidal anti-inflammatory (NSAID) medication may be used, unless contraindicated. (CONSENSUS)

Opioid Analgesics

Opioid analgesics bind to opioid receptors both within and outside the central nervous system and are used for management of severe pain.

All opioid medications have the potential to cause side effects including constipation, urinary retention, sedation, respiratory depression, nausea and vomiting. Titration of medication should occur to optimise the response to the analgesic and to minimise side effects. The following points are highlighted in the NHRMC (1999) acute pain guidelines:

- True allergy to opioids is uncommon; people may have side effects that are mistakenly referred to as 'allergies'.

- There is no evidence that the use of opioids for the treatment of severe acute pain leads to dependence on, or addiction to, opioid medications.
- The dosage should be tailored to each individual and the need for pain relief considered of greater importance than adhering strictly to a specific dose interval.

Key Message

Oral opioids may be necessary to relieve severe musculoskeletal pain. It is preferable to administer a short-acting agent at regular intervals, rather than on a pain-contingent basis. Ongoing need for opioid analgesia is an indication for reassessment. (CONSENSUS)

Muscle Relaxants

Muscle relaxants have the potential for side effects and show some short-term benefit in studies for low back pain. (Bigos et al. 1994; van Tulder et al. 1997).

Adjuvant Agents

There is no evidence to support the use of adjuvant agents, including antidepressants, anticonvulsants and oral corticosteroids, in the treatment of acute musculoskeletal pain.

Key Messages

> Any benefits from muscle relaxants may be outweighed by their adverse effects, therefore they cannot be routinely recommended. (CONSENSUS, LEVEL I)

> Adjuvant agents such as anticonvulsants and antidepressants are not recommended in the management of acute musculoskeletal pain. (CONSENSUS)

>References

Bigos S, Bowyer O, Braen G, et al. (1994). Acute low back problems in adults. Clinical Practice Guideline no.14. AHCPR Publication No. 95-0642. Agency for Health Care Policy and Research, Public Health Service, US, Department of Health: Rockville MD.

Bonica JJ (1953). The Management of Pain. Lea and Febiger: Philadelphia.

Eccleston C (2001). Role of psychology in pain management. British Journal of Anaesthesia, 87: 144–152.

Engel G (1977). The need for a new medical model: a challenge for biomedicine. Science, 196: 129–136.

Jensen MP, Karoly P, Braver S (1986). The measurement of clinical pain intensity: a comparison of six methods. Pain, Oct; 27: 117–26.

Linton SJ (2002). Why does chronic pain develop? A behavioural approach. In: Linton SJ (ed). Pain Research and Clinical Management, Vol 12. Elsevier Science: Amsterdam.

Main CJ (2002). Concepts of treatment and prevention in musculoskeletal disorders. In: Linton SJ (ed). Pain Research and Clinical Management, Vol 12. Elsevier Science: Amsterdam.

Merskey H (1979). Pain terms: a list with definitions and notes on usage recommended by the IASP Subcommittee on Taxonomy. Pain, 6: 249–252.

Merskey H, Bogduk N (eds) (1994). Classification of Chronic Pain. Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms (2nd Edition). IASP Press: Seattle. p 210.

National Health and Medical Research Council (1999). Acute Pain Management: Scientific Evidence. Commonwealth of Australia: Canberra.

New Zealand Guidelines Group (1998). Guide to Assessing Psychosocial Yellow Flags in Acute Low Back Pain: Risk Factors for Long-Term Disability and Work Loss. Auckland, New Zealand.

van Tulder MW, Koes BW, Bouter LM (1997). Conservative treatment of acute and chronic non-specific low back pain: a systematic review of randomised controlled trials of the most common interventions. Spine, 22: 2128–2156.

Von Korff M (1999). Pain management in primary care: an individualised stepped / care approach. In: Gatchel DJ,Turk DC (eds). Psychosocial Factors in Pain. Guilford Press: New York. Pp. 360–373.

World Health Organisation (1986). International Classification of Impairments, Disabilities and Handicaps. WHO: Geneva.

Chapter 3

Effective Communication

➔ *This chapter contains information that is generic to the management of all people with acute musculoskeletal pain.*

This chapter was developed by the steering committee and the key messages have been developed through consensus. A systematic process was not undertaken to search the literature. The studies included were nominated by individuals involved in the project and underwent critical appraisal.

For details of the Summary Table refer to Appendix E: Tables of Included and Excluded Studies.

Communication

All consultations involve the exchange of information between a clinician and a patient. Effective communication of information is fundamental to the success of any management plan.

Information is gathered from the patient initially as part of the clinical assessment, enabling the clinician to formulate a working diagnosis. After the assessment it is important for the clinician to communicate their findings to the patient and where possible provide an explanation of the possible causes of the pain. 'Two-way' communication should be encouraged so that all issues of concern are raised, a management plan is developed and the respective roles and responsibilities are clear in relation to implementing the plan.

Key Message

Clinicians should work with patients to develop a management plan so that patients know what to expect, and understand their role and responsibilities. (Consensus)

Medical Terminology

In most cases of acute musculoskeletal pain, the cause is non-specific and non-threatening. In labeling or naming the condition, the clinician should take care to use neutral terms. Such terms are provided in the individual topics covered in this document.

Clinicians should convey their explanation in words that will be understood, avoiding the use of alarming, inappropriate or incorrect terms that may be misconstrued. Jargon should be avoided.

Key Message

Information should be conveyed in correct but neutral terms, avoiding alarming diagnostic labels; jargon should be avoided. (Consensus)

Learning Methods

People learn in different ways. Some perceive new concepts readily through hearing them explained and forming mental impressions based on the words used to describe them. Others learn more easily through seeing images and developing understanding based on visual perception. The clinician should be sensitive to these differences, be prepared to use a variety of educational techniques and be able to adapt their communication method to suit the needs of individuals.

Printed material, such as diagrams, can be useful for communication of concepts. Brochures or leaflets are rarely effective if simply handed out to a patient, but can be used to reinforce what the clinician has communicated personally.

Anatomical models facilitate visual perception and the appreciation of spatial relationships. They are particularly useful for demonstrating the location of body parts affected by pain, what the cause of pain might be, and how the plan of management can be designed to promote recovery.

Key Messages

> Explanation is important to overcome inappropriate expectations, fears or mistaken beliefs that patients may have about their condition or its management. (Consensus)

> Printed materials and models may be useful for communicating concepts. (Consensus)

Factors Affecting Communication

People may present with fears, beliefs and misunderstandings about their problem, its cause and how they should respond to pain. Clinicians need to take these factors into account when providing information and ensure that any information provided has been understood. Barriers to understanding should be identified. These may include educational level, cultural/ethnic background and language barriers. If the patient appears unconvinced by an explanation, or harbours fears, further exploration is required and a sensitive approach taken to addressing these issues.

Key Messages

> Clinicians should adapt their method of communication to meet the needs and abilities of each patient. (Consensus)

> Clinicians should check that any information provided has been understood; barriers to understanding should be explored and addressed. (Consensus)

Evidence Review

It is logical that clinicians and patients should strive to understand each other, that clinicians should avoid the use of intimidating jargon and misleading diagnostic labels and that patients have a need to be supported. These elements of effective communication are based largely on concept validity and face validity, however there is some evidence for these practices contained in the literature on low back pain.

Studies of the treatment of subacute low back pain have demonstrated that significant improvements in the number of patients with back pain returning to work can be achieved by providing an explanation, assurance and encouragement to remain active, with no other intervention (Indahl et al. 1995, 1998). A non-randomised study of acute low back pain found that good outcomes can be achieved by focusing on the fears of

patients and showing them how to undertake their own rehabilitation with a minimum use of passive interventions (McGuirk et al. 2001).

A study by Burton et al. (1999) compared the use of a novel educational booklet encouraging patients to be active, assuring them that nothing was seriously wrong with their back, encouraging them to have a positive attitude, and discussing their involvement and responsibilities, with a traditional booklet describing spinal injury and damage, and advising against activity if in pain. A clinically important improvement in fear belief scores was achieved at two weeks and sustained for up to twelve months in the group receiving the novel educational booklet.

Explanations can be a simple and effective substitute for automatically ordering investigations. A controlled study assessed the impact of a brief (5 minute) educational intervention for patients eligible for lumbar spine films (Deyo et al. 1987). At follow-up, the proportion of people in the educational group who believed that xrays were necessary fell only slightly, but was substantially and significantly less (44% vs 73%) than in the control group. Fewer of those in the educational group underwent radiography after the study, but there were no significant differences in patient satisfaction and no serious diagnoses were missed.

Abenheim et al. (1995) investigated the prognostic consequences of making an initial diagnosis of work-related back injury. A chart review revealed that approximately 9% of workers were given a specific diagnosis. Older workers were more likely to be given a specific diagnosis and overall the group receiving a specific diagnosis was 4.9 times more likely to develop chronic pain than workers in whom pain was described as 'non-specific'. The results reflect the possibility that labeling of older workers (> 55 years) is more likely to result in chronicity compared with younger workers with non-specific pain. While this may reflect accurate diagnosis of more harmful and chronic conditions, given that precise diagnosis of back problems in the absence of fracture or tumour lacks sensitivity, it is likely that the labeling contributed to the psychosocial aspects of pain perception that are associated with chronicity. The study highlights the importance of effective, non-emotive communication with patients with back pain, particularly in occupational settings.

Failing to review patients creates the illusion that if they do not return they must have recovered. An observational study revealed that this is not the case (Croft et al. 1998). For one year the study followed people with acute low back pain who consulted their general practitioner but subsequently did not return. Rather then having recovered, approximately 75% of patients still had problems; they had simply stopped going to their general practitioner.

This evidence pertains explicitly to low back pain and should be evaluated carefully in relation to other acute musculoskeletal pain problems.

Research Priorities

- Further evaluation is required to determine the most effective and acceptable ways to convey messages to patients regarding their musculoskeletal pain and management.
- There may be value in further study of electronic and telephone contact for improving adherence to management plans and their effects on patient outcomes.

>References

Abenheim L, Rossignol M, Gobeille D, Bonvalot Y, Fines P, Scott S (1995). The prognostic consequences in the making of the initial diagnosis of work related back injuries. Spine, 20: 791–795.

Burton AK, Waddell G, Tillotson M, Summerton N (1999). Information and advice to patients with back pain can have a positive effect. Spine, 24: 2484–2491.

Croft PR, Macfarlane GJ, Papageorgiou AC, Thomas E, Silman AJ (1998). Outcome of low back pain in general practice: a prospective study. British Medical Journal, 316: 1356–1359.

Deyo RA, Diehl AK, Rosenthal M (1987). Reducing roentgenography use: can patient expectations be altered? Archives of Internal Medicine, 147: 141–145.

Indahl A, Haldorsen EH, Holm S, Reikeras O, Ursin H (1998). Five-year follow-up study of a controlled clinical trial using light mobilization and an informative approach to low back pain. Spine, 23: 2625–2630.

Indahl A, Velund L, Reikeraas O (1995). Good prognosis for low back pain when left untampered: a randomised clinical trial. Spine, 20: 473–477.

McGuirk B, King W, Govind J, Lowry J, Bogduk N (2001). The safety, efficacy, and cost-effectiveness of evidence-based guidelines for the management of acute low back pain in primary care. Spine, 26: 2615–2622.

Chapter 4

Acute Low Back Pain

➔ *This document was developed by a multi-disciplinary group to provide the evidence for the management of acute low back pain.*

Low back pain is common in developed countries affecting approximately 70% of the adult population (Deyo et al. 1992) at some stage during their life. Episodes of low back pain lasting more than two weeks have a cumulative life-time prevalence of 14% (Deyo and Tsui-Wu 1987). The cause of pain is non-specific in about 95% of people presenting with acute low back pain; serious conditions are rare (Suarez-Almazor et al. 1997; Hollingworth et al. 2002). The condition is generally self-limiting.

In Australia, back problems are the most frequently seen musculoskeletal condition in general practice and the seventh most common reason for seeking care (AIHW 2000).

The aim in the management of acute low back pain is to:

- *identify potentially serious causes of acute low back pain (< 5%)*
- *promote effective self-management of symptoms through the provision of timely and appropriate advice*
- *maximise functional status and minimise disability*

Chronic low back pain is a well-documented disabling condition costly to both individuals and society (Waddell 1992).

Definition of Acute Low Back Pain

Definitions of 'acute' and 'subacute' durations of pain vary in the literature, but for the purposes of these guidelines:

- Acute pain refers to an episode of pain present for less than three months; it does not refer to the severity or quality of pain.
- Subacute pain is an episode of pain with a duration of more than five weeks (van Tulder et al. 1997a) but less than three months.
- Chronic pain is defined as an episode of pain that has persisted for longer than three months (Merskey and Bogduk 1994).

The International Association for the Study of Pain (IASP) adopted a topographic basis for the definition of acute low back pain (Merskey and Bogduk 1994). The IASP recognises different forms of spinal pain: lumbar spinal pain, sacral spinal pain, or lumbosacral pain, as constituting low back pain. These definitions explicitly locate the pain as perceived in the lumbar and/or sacral regions of the spine, which collectively cover the following regions:

- superiorly, by an imaginary transverse line through the tip of the last thoracic spinous process
- inferiorly, by an imaginary transverse line through the posterior sacrococcygeal joints
- laterally, by vertical lines tangential to the lateral borders of the lumbar erectores spinae, continuing to imaginary lines passing through the posterior superior and posterior inferior iliac spines

Scope

These guidelines describe the diagnosis and treatment of acute non-specific low back pain. The following are beyond the scope of this document:

- serious conditions including infection, neoplasm, fracture
- neuropathic conditions including radicular pain (i.e. 'sciatica')
- other specific conditions such as degenerative disc disease, osteoarthritis, spinal canal stenosis and inflammatory conditions such as ankylosing spondylitis
- loin pain (pain perceived over the posterior region of the trunk but lateral to the erector spinae muscles)
- gluteal pain (pain in a sector centred on the greater trochanter and spanning from the posterior inferior iliac spine to the anterior superior iliac spine)
- thoracic spinal pain
- somatic referred pain, visceral referred pain
- serious underlying conditions including aortic aneurysm, pelvic disease, retroperitoneal disease, Paget's disease, hyperparathyroidism

Guideline Development Process

Evaluation of Existing Guidelines

Guidelines developed by other groups were evaluated to determine whether an existing guideline could be adapted for use in the Australian context. Other countries have produced national clinical practice guidelines for low back pain. In a recent qualitative review, Koes et al. (2001) identified 11 guidelines published in English, Dutch and German (the original draft version of these Australian guidelines was included in this review). The guidelines overlap in their target audiences, development methods, evidence base and recommendations. There is considerable consistency in recommendations across guidelines regarding diagnostic strategies and therapeutic interventions.

The decision was made to proceed with updating the draft version of the existing Australian guidelines, developed by Professor Nikolai Bogduk. This work was developed using a process of conventional literature review. A multi-disciplinary group was formed to update the existing document, which was first evaluated using the AGREE (2001) criteria for clinical practice guidelines.

Updating Existing Guidelines

The update of the existing work involved a review of the evidence on acute low back pain conducted by a multi-disciplinary group (refer to Chapter 9: Process Report). Group members had the opportunity to evaluate the literature forming the basis of the existing guidelines, review the interpretation of the literature, nominate additional articles to undergo the appraisal process or request that an article be re-appraised.

A systematic process was used to identify new studies on the diagnosis, prognosis and interventions for acute low back pain in line with current standards for guideline development (NHMRC 1999a). Studies were appraised against selection criteria and those meeting the criteria for inclusion were used to update the existing text of the guidelines.

The most recent Clinical Evidence text (2002) was used as the basis for the update of information on the effectiveness of interventions. Studies cited in Clinical Evidence were checked against the selection criteria; details of these individual studies are not recorded. Additional studies published subsequent to the search date in Clinical Evidence were sought to determine whether there was a need to update the conclusions outlined in Clinical Evidence. These studies were critically appraised.

For details of the Tables of Included and Excluded Studies refer to Appendix E: Tables of Included and Excluded Studies. Studies that were previously described in the existing guidelines have not undergone appraisal and are not described in these tables.

Study Selection Criteria

The chart below is an outline of the criteria used to identify, select and appraise new studies on acute low back pain.

Search Strategy

Sensitive searches were performed of electronic databases. Searches were limited to adults, humans, and articles published in English in peer-reviewed journals. Where available,

Study Selection Criteria

DIAGNOSIS	
The sections on Aetiology and Prevalence, History, Examination and Investigations comprise information from the existing draft (developed by conventional literature review) combined and updated with relevant articles located and appraised according to the following inclusion and exclusion criteria:	
Inclusion criteria	Systematic reviews, cross-sectional studies, case series, case reports Adults
Exclusion criteria	Chronic pain Children and adolescents Neuropathic pain, somatic referred pain, visceral referred pain, radicular pain, loin and gluteal pain, osteoarthritis, sciatica, degenerative joint and disc disease, aortic aneurysm, pelvic disease, retroperitoneal disease, Paget's disease, hyperparathyroidism, osteomyelitis, infection, neoplasm, fracture, low back pain associated with pregnancy, ankylosing spondylitis Aetiological risk factors
PROGNOSIS	
Information from the existing draft was combined and updated with relevant articles located and appraised independently by two reviewers according to the following inclusion and exclusion criteria:	
Inclusion criteria	Systematic reviews, cohort studies Adults
Exclusion criteria	Chronic pain Children and adolescents Low back pain associated with pregnancy, neuropathic pain, somatic referred pain, visceral referred pain, radicular pain, loin and gluteal pain, osteoarthritis, sciatica, degenerative joint and disc disease, aortic aneurysm, pelvic disease, retroperitoneal disease, Paget's disease, hyperparathyroidism, osteomyelitis, infection, neoplasm, fracture, ankylosing spondylitis

Study Selection Criteria continued

INTERVENTIONS	
Information from the existing draft was updated with information from Clinical Evidence and relevant articles located and appraised independently by two reviewers according to the following inclusion and exclusion criteria. In cases where no evidence was available on interventions specifically for acute low back pain, studies containing mixed populations (acute and chronic low back pain) were considered in the review:	
Inclusion criteria	Systematic reviews, randomised controlled trials Adults Articles describing cost effectiveness of interventions
Exclusion criteria	Chronic pain Low back pain associated with pregnancy, neuropathic pain, somatic referred pain, visceral referred pain, radicular pain, loin and gluteal pain, osteoarthritis, sciatica, degenerative joint and disc disease, aortic aneurysm, pelvic disease, retroperitoneal disease, Paget's disease, hyperparathyroidism, osteomyelitis, infection, neoplasm, fracture, ankylosing spondylitis Primary prevention of low back pain

methodological filters were used. There were no hand searches conducted.

Searches for information on diagnosis and prognosis of low back pain were conducted from the years 1998 to 2002 taking into account the date when the original guidelines were developed.

Searches for articles on interventions were conducted for the years 2001 to 2002, taking into account the search date (October 2001) used in the Clinical Evidence text (2002).

Additional articles not identified by the database search, those located in the reference lists of retrieved articles and other articles identified by content experts were also submitted to the appraisal process.

The following databases were searched in August 2002:

- PubMed (Clinical Queries)
- CINAHL
- EMBASE — Physical and Rehabilitation Medicine
- The Cochrane Library, 2002, Issue 2

Access to CHIROLARS/MANTIS and PEDro was unavailable for this review.

Search Terms

- Low back pain .exp
- Back pain .exp
- Diagnosis .exp
- Pain assessment
- Drug therapy
- Clinical trial
- Aetiology
- Prognosis .exp
- Controlled trial
- Randomised
- Therapies .exp
- Systematic review .tw
- Patient .tw
- Consumer .tw

Research Agenda for Acute Low Back Pain

Research should be aimed at optimising the uptake of evidence-based guidelines by clinicians and consumers.

All new interventions for acute low back pain need to be tested in well-designed randomised controlled trials (RCT) with 'advice to avoid bed rest and maintain usual activities' as the appropriate comparator.

This review identified the need for research on the following interventions, testing them in well-designed RCTs with 'advice to avoid bed rest and maintain usual activities' as the appropriate comparator:

- Temperature treatments, ice, heat
- Topical NSAIDs
- Head to head comparator trials between Cox-2 NSAIDs, traditional NSAIDs, paracetamol and opioid analgesics, and between these medications and placebo for acute low back pain
- McKenzie therapy and other specific physical regimens
- Multi-disciplinary treatment (e.g. non-occupational settings, programmatic approaches to delivering multi-disciplinary care)
- Counselling and cognitive behavioural therapy
- Spinal manipulation (with and without prior xray)
- Massage (and placebo-controlled trials of massage therapy as mono-therapy and in combination with other modalities)
- TENS in patients not responding to early advice to resume normal activities
- Optimum combinations of therapies

International standardisation of definitions of intervention strategies and consistent outcome measures is strongly recommended.

Intervention studies addressing clinical and psychosocial predictors should be conducted early in the subacute phase with adequate follow up to assess for prevention of chronicity.

Further research into secondary prevention of low back pain. Cost effectiveness analysis and evidence of harm should be incorporated into future intervention studies for acute and subacute low back pain.

Summary of Key Messages: Acute Pain Management

	EVIDENCE LEVEL
Management Plan	
It is recommended that the clinician and patient develop a management plan for acute musculoskeletal pain comprising the elements of assessment, management and review: • Assessment — Conduct a history and physical examination to assess for the presence of serious conditions; ancillary investigations are not generally indicated unless features of serious conditions are identified. • Management — Provide information, assurance and advice to resume normal activity and discuss other options for pain management as needed. • Review — Reassess the pain and revise the management plan as required.	CONSENSUS: Steering Committee
Non-Pharmacological Interventions	
Simple interventions (providing information, assurance and encouraging reasonable maintenance of activity) may be used alone or in combination with other interventions for the successful management of acute musculoskeletal pain.	CONSENSUS: Steering Committee
Pharmacological Interventions	
Specific pharmacological interventions may be required to relieve pain; such agents can be used in conjunction with non-pharmacological interventions.	CONSENSUS: Steering Committee; NHMRC 1999b
Paracetamol or other simple analgesics, administered regularly, are recommended for relief of mild to moderate acute musculoskeletal pain.	CONSENSUS: Steering Committee; NHMRC 1999b
Where paracetamol is insufficient for pain relief, a non-steroidal anti-inflammatory (NSAID) medication may be used, unless contraindicated.	CONSENSUS: Steering Committee; NHMRC 1999b
Oral opioids may be necessary to relieve severe musculoskeletal pain. It is preferable to administer a short-acting agent at regular intervals, rather than on a pain-contingent basis. Ongoing need for opioid analgesia is an indication for reassessment.	CONSENSUS: Steering Committee; NHMRC 1999b
Adjuvant agents such as anticonvulsants and antidepressants are not recommended in the management of acute musculoskeletal pain.	CONSENSUS: Steering Committee; NHMRC 1999b
Any benefits from muscle relaxants may be outweighed by their adverse effects, therefore they cannot be routinely recommended.	CONSENSUS: Steering Committee

Summary of Key Messages: Effective Communication

	EVIDENCE LEVEL
Clinicians should work with patients to develop a management plan so that patients know what to expect, and understand their role and responsibilities.	CONSENSUS: Steering Committee
Information should be conveyed in correct but neutral terms, avoiding alarming diagnostic labels; jargon should be avoided.	CONSENSUS: Steering Committee
Explanation is important to overcome inappropriate expectations, fears or mistaken beliefs that patients may have about their condition or its management.	CONSENSUS: Steering Committee
Printed materials and models may be useful for communicating concepts.	CONSENSUS: Steering Committee
Clinicians should adapt their method of communication to meet the needs and abilities of each patient.	CONSENSUS: Steering Committee
Clinicians should check that information that has been provided has been understood; barriers to understanding should be explored and addressed.	CONSENSUS: Steering Committee

Summary of Key Messages: Acute Low Back Pain

DIAGNOSIS	EVIDENCE LEVEL
Aetiology and Prevalence	
The majority (approximately 95% of cases) of acute low back pain is non-specific; serious conditions are rare causes of acute low back pain.	*LEVEL I, III: Deyo et al. 1992; Suarez-Almazor et al. 1997; Hollingworth et al. 2002
Common findings in patients with low back pain (e.g. osteoarthritis, lumbar spondylosis, spinal canal stenosis) also occur in asymptomatic people; hence, such conditions may not be the cause of the pain.	*LEVEL I, III: van Tulder et al. 1997a; Torgerson and Dotter 1976
History	
History enables screening for features of serious conditions; however the reliability and validity of individual features in histories have low diagnostic significance.	*LEVEL III-2: Deyo et al. 1992; van den Hoogen et al. 1995
Physical Examination	
Clinical signs detected during physical and psychosocial assessment must be interpreted cautiously as many tests lack reliability and validity.	*LEVEL III-2: LeBoeuf-Yde et al. 2002; Truchon and Fillion 2000; Knutson 2002; Waddell et al. 1980; Deyo et al. 1992
A full neurological examination is warranted in the presence of lower limb pain and other neurological symptoms.	*LEVEL IV: Waddell et al. 1982; McCombe et al. 1989
Ancillary Investigations	
Plain xrays of the lumbar spine are not routinely recommended in acute non-specific low back pain as they are of limited diagnostic value and no benefits in physical function, pain or disability are observed.	*LEVEL III-2: Suarez-Almazor et al. 1997; Hollingworth et al. 2002; Kendrick et al. 2001; Kerry et al. 2002
Appropriate investigations are indicated in cases of acute low back pain when alerting features ('red flags') of serious conditions are present.	*LEVEL III-2: Deyo and Diehl 1986
Terminology	
A specific patho-anatomic diagnosis is not necessary for effective management of acute non-specific low back pain.	CONSENSUS: Steering Committee
Terms to describe acute low back pain with no identifiable pathology include 'lumbar spinal pain of unknown origin' or 'somatic lumbar spinal pain'.	*LEVEL IV: Merskey and Bogduk 1994
PROGNOSIS	**EVIDENCE LEVEL**
The majority of people with a short duration of symptoms upon presentation with low back pain recover within three months; however milder symptoms often persist.	*LEVEL III-2: Croft and Rigby 1994; Schiottz-Christensen et al. 1999
Recurrences of acute low back pain are not uncommon.	*LEVEL III-3: van den Hoogen et al. 1998; Hurley et al. 2001a
Psychosocial and occupational factors ('yellow flags') appear to be associated with progression from acute to chronic pain; such factors should be assessed early to facilitate intervention.	*LEVEL III-2: Linton 2001; Pincus et al. 2002; Truchon and Fillion 2000
INTERVENTIONS	**EVIDENCE LEVEL**
Evidence of Benefit	
Advice to Stay Active (Activation) — Advice to stay active provides a small beneficial effect on pain, rate of recovery and function compared to bed rest and compared to a specific exercise regime in mixed populations with low back pain. Advice to stay active reduces sick leave compared to bed rest in mixed populations with low back pain.	LEVEL I, II: Based on systematic reviews (Waddell et al. 1997; Hagen et al. 2002; Hilde et al. 2002) and one additional study (Rozenberg et al. 2002)

Acute Low Back Pain continued

Heat Wrap Therapy — Continuous low level heat wrap therapy reduces pain, stiffness and disability extending for three to four days compared with paracetamol, NSAIDs or placebo alone during the first 48 hours of acute low back pain. (This treatment is not routinely available in Australia).	LEVEL II: Based on one study (Nadler et al. 2002)
Patient Information (Printed) — Novel or 'activity-focused' printed information plus similar verbal advice provided by a clinician is more effective compared to traditional brochures or no printed information in acute low back pain. Printed information provided through the mail is less likely to have an effect on pain, disability and sick leave compared to information provided in person. Behavioural therapy interventions are more effective than printed information for preventing long-term disability in mixed populations.	LEVEL II: Based on controlled trials (Cherkin et al. 1996; Cherkin et al. 1998; Burton et al. 1999; Hazard et al. 2000; Roberts et al. 2002; Linton and Andersson 2000)
Conflicting Evidence	
Muscle Relaxants — There is conflicting evidence that muscle relaxants are effective compared to placebo in acute low back pain. There is insufficient evidence to determine whether muscle relaxants are more or less effective compared to NSAIDs for acute low back pain. Drowsiness, dizziness and dependency are common adverse effects of muscle relaxants.	LEVEL I: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b) that found numerous RCTs
Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) — There is conflicting evidence that oral and injectable NSAIDs are effective versus placebo or no treatment for acute low back pain. NSAIDs have a similar effect compared to opioid analgesics, combined paracetamol-opioid analgesics and to each other in their effect on acute low back pain. There is insufficient evidence that NSAIDs are more effective when compared to muscle relaxants and anti-anxiety agents in acute low back pain. NSAIDs are less effective in reducing pain than heat wrap therapy in the first three to four days of acute low back pain. Serious adverse effects of NSAIDs include gastrointestinal complications (e.g. bleeding, perforation).	LEVEL I, II: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b; van Tulder et al. 2002f; Koes et al. 1997) and numerous RCTs (Amlie et al. 1987; Basmajian 1989; Postacchini et al. 1988; Lacey et al. 1984; Nadler et al. 2002)
Spinal Manipulation — There is conflicting evidence that spinal manipulation provides pain relief compared to placebo in the first two to four weeks of acute low back pain. There is insufficient evidence that spinal manipulation is more or less effective than other conservative treatments for acute low back pain. Adverse effects of spinal manipulation are rare but potentially serious.	LEVEL I, II: Based on systematic reviews (van Tulder et al. 1997b; Bigos et al. 1994; Koes et al. 1996; Mohseni-Bandpei et al. 1998; Shekelle et al. 1992) and one RCT (Hsieh et al. 2002) LEVEL IV: Based on reviews of case studies (Haldeman and Rubinstein 1992; Assendelft et al. 1996; Stevinson and Ernst 2002)
Insufficient Evidence	
Acupuncture — There is insufficient evidence that acupuncture (dry-needling) is effective compared to injection therapy in acute low back pain. Adverse effects of acupuncture are rare but potentially serious.	LEVEL I: Based on a systematic review (van Tulder et al. 2002a) and one study (Garvey et al. 1989)
Analgesics, Compound and Opioid — There are no randomised controlled trials investigating the efficacy of opioids and compound analgesics in acute low back pain. There is evidence that the effect of opioid or compound analgesics is similar to NSAIDs for treatment of acute low back pain. In general, opioids and compound analgesics have a substantially increased risk of side effects compared with paracetamol alone.	No Level I or II evidence LEVEL I: Based on systematic reviews (van Tulder et al. 1997b; Bigos et al. 1994) and RCTs (Brown et al. 1986; Videman et al. 1984; Palangio et al. 2002)

Acute Low Back Pain continued

Analgesics, Simple — There are no randomised controlled trials assessing the effectiveness of simple analgesics in acute low back pain. There is insufficient evidence for the effectiveness of simple analgesics versus NSAIDs in acute low back pain. Paracetamol is less effective than heat wrap therapy in acute low back pain. There is insufficient evidence for the effect of paracetamol compared to electroacupuncture in mixed populations with low back pain.	No Level I or II evidence LEVEL I, II: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b) of studies by Milgrom et al. 1993; Wiesel et al. 1980; Hackett et al. 1988
Back Exercises — McKenzie therapy provides similar pain and function outcomes compared to usual care in acute low back pain. There is conflicting evidence for the efficacy of back exercises in reducing pain and disability compared to other active and inactive treatments in mixed populations with low back pain. McKenzie therapy reduces pain and sick leave compared to one back school session, results in similar global improvement compared to manipulation and provision of an educational booklet and provides better functional and pain outcomes compared to flexion exercises in mixed populations with low back pain. Lateral multifidus muscle exercises reduce recurrences of low back pain compared to usual care in mixed populations with low back pain.	LEVEL I, II: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b; van Tulder et al. 2002d) of multiple controlled studies
Back School — There is insufficient evidence that back school is more effective in reducing pain compared to active and passive therapies and to placebo in acute low back pain. There is insufficient evidence that back school is more effective in reducing pain compared to placebo and other treatments in mixed populations with low back pain.	LEVEL I, II: Based on systematic reviews (van Tulder et al. 1997b; van Tulder et al. 2002b) and an RCT by Hsieh et al. (2002)
Bed Rest — There is insufficient evidence that bed rest is more effective compared to advice to stay active, back exercises, spinal manipulation, non-steroidal anti-inflammatory drugs or no treatment in mixed populations with low back pain. There is conflicting evidence that bed rest increases disability and rate of recovery compared to staying active in mixed populations with low back pain. Bedrest for longer than two days increases the amount of sick leave compared to early resumption of normal activity in acute low back pain. There is evidence that prolonged bed rest is harmful.	LEVEL I, II: Based on systematic reviews (van Tulder et al. 1997b; Hagen et al. 2002) and an RCT (Rozenberg et al. 2002)
Cognitive Behavioural Therapy — Cognitive behavioural therapy reduces general disability in the long term compared to traditional care in mixed with populations back pain. Group cognitive behavioural therapy sessions may reduce sick leave and health care utilisation in the long term compared to general educational information in mixed populations with back pain. While cognitive behavioural strategies are often included as part of specific interventions for acute low back pain such as exercise and activity restoration, there are no studies on this approach as a single intervention.	LEVEL I: Based on systematic reviews (Turner 1996; van Tulder et al. 2002e) LEVEL II: Based on studies by Linton and Andersson (2000) and Linton and Ryberg (2001) No Level I or II studies
Electromyographic Biofeedback — There are no controlled studies testing the effectiveness of electromyographic biofeedback in acute low back pain.	No Level I or II evidence
Injection Therapy — There is insufficient evidence demonstrating the effectiveness of injection therapy (facet joint, epidural or soft tissue) in the treatment of acute low back pain. Adverse effects of injection therapy are rare but serious.	LEVEL I, II: Based on systematic reviews (Nelemans et al. 2002; Watts and Silagy 1995; Koes et al. 1999) and an RCT (Garvey et al. 1989)
Lumbar Supports — There are no controlled studies on the effect of lumbar supports in acute low back pain. There is insufficient evidence that lumbar supports are effective in reducing pain compared to spinal manipulation, exercises, massage, TENS and simple analgesia in mixed populations with low back pain.	No Level I or II evidence LEVEL I: Based on two systematic reviews (van Tulder et al. 2002c; Bigos et al. 1994)

Acute Low Back Pain continued

Massage — There are no controlled studies for massage therapy in acute low back pain. Massage is superior to placebo (sham laser) and acupuncture in mixed populations with low back pain. Massage provides similar effect to back schools (involving exercise and education), corsets and TENS in mixed populations with low back pain. There is conflicting evidence of the effect of massage compared to manipulation and education in mixed populations with low back pain.	No Level I or II evidence LEVEL I, II: Based on systematic reviews (Furlan et al. 2002; Ernst 1999) and RCTs (Cherkin et al. 2001; Preyde 2000)
Multi-Disciplinary Treatment in the Workplace — There are no controlled studies on the effect of multi-disciplinary treatment in the workplace in acute low back pain. Multi-disciplinary treatment in the workplace improves return to work and subjective disability compared to usual care in mixed populations with low back pain.	No Level I or II evidence LEVEL I, II: Based on a systematic review (Karjailanen et al. 2002) and RCTs (Loisel et al. 1997; Lindstrom 1992a,b)
Topical Treatment — There is insufficient evidence for the effectiveness of spiroflar homeopathic gel or cremol capsici for treatment of acute low back pain.	LEVEL II: Based on one RCT (Stam et al. 2001)
Traction — There are no controlled studies on the effect of traction for acute low back pain. There is insufficient evidence that traction is effective compared to placebo and compared to other treatments in mixed populations with low back pain. Adverse effects from traction have been reported, including reduced muscle tone, bone demineralisation, thrombophlebitis.	No Level I or II evidence LEVEL I: Based on systematic reviews (van der Heijden et al. 1995; van Tulder et al. 1997b)
Transcutaneous Electrical Nerve Stimulation — There are no controlled studies on the effect of TENS in acute low back pain. There is insufficient evidence for the effectiveness of TENS compared to exercises, back books, massage, corset use and simple analgesia in mixed populations with low back pain.	No Level I or II evidence LEVEL I, II: Based on a systematic review (van Tulder et al. 1997b) and additional studies (Pengel et al. 2002; Hurley et al. 2001b)
Cost Effectiveness — Published data is very limited; however there is some evidence that advice to maintain usual activities, provision of an education booklet and community-based exercises appear to be cost effective first line interventions for acute low back pain.	LEVEL II: Malmivaara et al. 1995; Cherkin et al. 1998; Moffet et al. 1999

Note: * Indicative only. A higher rating of the level of evidence might apply (refer to Chapter 1: Executive Summary, Limitations of Findings).

DIAGNOSIS

>Aetiology and Prevalence

Acute low back pain has many possible sources, including all diseases, injuries and other impairments that invoke nociceptive mechanisms in the region. Table 4.1 outlines some of the possible causes of acute low back pain, however pain does not always correlate with the presence of a particular condition.

With the exception of conditions posing a serious threat to health, identification of a specific cause is not a precondition for effective management of acute low back pain (Bogduk and McGuirk 2002).

Conditions Associated with Acute Low Back Pain: Radiological Findings

Table 4.2 shows the prevalence of conditions identified in patients presenting with acute low back pain based on radiological findings. Data were obtained from prospective studies of patients with acute low back pain referred for lumbar radiography from primary care (Suarez-Almazor et al. 1997; Hollingworth et al. 2002). The table demonstrates that the findings were 'normal' or showed degenerative changes in the vast majority (95%) of cases. The information demonstrates that serious conditions are an infrequent source of acute low back pain.

Non-Specific Low Back Pain

The majority of cases of acute low back pain are non-specific, with earlier reports citing 85% (Deyo et al. 1992). Two more recent studies (Suarez-Almazor et al. 1997; Hollingworth et al. 2002) found no change (approximately 40%) or only minor degenerative changes (approximately 55%) among people referred for xray from primary care settings, suggesting that as many as 95% of cases may be non-specific.

Fractures

The prevalence of compression fracture in primary care ranges from 3 to 5% (Deyo et al. 1992; Suarez-Almazor et al. 1997; Hollingworth et al. 2002).

In the general population, significant fractures presenting as back pain occur chiefly in patients with a history of major trauma (Scavone et al. 1981a). Fractures may occur among the elderly and among corticosteroid users in cases of minor trauma.

Table 4.1

Conditions Associated with the Presence of Acute Low Back Pain

Serious conditions	Fracture (traumatic and osteoporotic)
	Tumour: primary (myeloma, tumours of bone, cartilage, neuronal and muscle tissue); secondary (prostate, breast, lung, thyroid, kidney, gastrointestinal, melanoma)
	Infection (osteomyelitis, epidural abscess)
Neuropathic conditions	Nerve root entrapment, sciatica, radicular pain
Mechanical conditions	Sprains, strains, tears of muscle fascia, ligament, joint or disc
Visceral conditions	Arising from abdominal structures, pelvic viscera, renal tract (infection, renal calculi)
	Pancreatitis
	Aortic aneurysm, retroperitoneal disease
Other conditions	Metabolic bone disease (Paget's disease, hyperparathyroidism)
	Osteoarthritis, degenerative joint disease, ankylosing spondylitis, enthesopathy, myositis, disc disruption, discitis

Table 4.2

Radiological Findings in Patients with Low Back Pain in Primary Care

Lumbar Radiography Findings	Hollingworth et al. 2002		Suarez-Almazor et al. 1997	
	N	%	*N*	%
Normal findings	855	40.8	44	38.9
Minor degenerative changes and other findings	1100	52.5	64	56.6
Fracture	100	4.8	4	3.5
Infection	4	0.2	—	—
Tumour	15	0.7	1	0.9
Inflammatory disorders	12	0.6	—	—
Total	**2086**	**99.6%**	**113**	**99.9%**

Note: Based on data from Suarez-Almazer et al. (1997) and Hollingworth et al. (2002).

Infection

Of those presenting in primary care settings with back pain, less than 0.5% have a spinal infection (Deyo et al. 1992; Suarez-Almazor et al. 1997; Hollingworth et al. 2002).

Tumours

Less than 1% of patients presenting to primary care with low back pain will have spinal tumours (Deyo et al. 1992; Suarez-Almazor et al. 1997; Hollingworth et al. 2002). The majority who prove to have cancer are elderly (Deyo and Diehl 1988).

Key Message

The majority (approximately 95% of cases) of acute low back pain is non-specific; serious conditions are rare causes of acute low back pain. (*Level I, III)

Ankylosing Spondylitis

Ankylosing spondylitis and other inflammatory disorders affect 0.3–0.9% of the population presenting with acute low back pain (Deyo et al. 1992; Suarez-Almazor 1997; Hollingworth et al. 2002; Gran 1985).

Degenerative Spinal Conditions

Spondylosis (Table 4.3), disc degeneration (Table 4.4), facet degeneration and osteoarthritis occur frequently in asymptomatic individuals. The correlation between pain and the presence of these conditions on radiographs is low with relative risks less than or equal to 2.5 (Torgerson and Dotter 1976; van Tulder et al. 1997a).

Key Message

Common findings in patients with low back pain (e.g. osteoarthritis, lumbar spondylosis, spinal canal stenosis) also occur in asymptomatic people; hence, such conditions may not be the cause of the pain. (*Level I, III)

>History

While there is a range of methods to assess the history of acute low back pain, the reliability and validity of history taking has not been demonstrated. Despite this, eliciting a history is an important part of the clinical assessment as it facilitates the identification of serious underlying conditions.

Features of serious conditions can manifest over time; it is important to reassess for signs and symptoms of serious conditions during subsequent visits.

Pain History

Site

It is necessary to establish that the presenting pain is low back pain and not pain in another region (e.g. loin, gluteal pain). However, formal studies have shown that two observers readily disagree on this question (McCombe et al. 1989). If there is more than one pain site, a separate history should be taken of each.

Distribution

Low back pain may be referred to the lower limb girdle, the lower limb, the groin or perineum. Pain may be experienced in

Table 4.3

Prevalence of Spondylosis in Asymptomatic Individuals and Patients with Lumbar Spinal Pain

Age	Asymptomatic			Symptomatic		
	N	*n*	%	*N*	*n*	%
All	217	102	47%	387	208	57%
40–49	64		22%			34%
50–59			49%			54%
60–69	69		74%			73%

Note: *N* = total number of patients surveyed; *n* = number affected. The relationship between spondylosis and symptoms is not significant statistically.
Based on data from Torgerson and Dotter 1976.

Table 4.4

Prevalence of Disc Degeneration in Asymptomatic Individuals and Patients with Lumbar Spinal Pain

Age	Asymptomatic			Symptomatic		
	N	*n*	%	*N*	*n*	%
All	217	48	22%	387	218	56%
40–49	64	4	6%	146	70	48%
60–69	69	33	48%	78	48	62%

Note: *N* = total number of patients surveyed; *n* = number affected. The relationship between disc degeneration and symptoms is significant ($p < 0.05$) on a $\chi 2$ test.
Based on data from Torgerson and Dotter 1976.

the low back and any of these regions. It is important to be aware that more than one condition may be present.

It is possible for a disorder of the lumbar spine to produce both somatic referred pain and radicular pain. For example, a disrupted disc may cause spinal pain and referred pain, but an associated prolapse may be an indicator of radicular pain.

The clinical distinction between radicular pain and somatic referred pain lies in its distribution and behaviour. Pain distal to the knee may not necessarily be radicular pain. Somatic pain from the lumbar zygapophyseal joints (Mooney and Robertson 1976; Fairbank et al. 1981; Fukui et al. 1997) can be referred distal to the knee.

Quality

Radicular pain tends to be shooting, lancinating or electric in quality (Smyth and Wright 1959), whereas somatic referred pain is typically a dull, deep ache or pressure-like in quality (Kellgren 1939; Feinstein et al. 1954). Distinctive qualitative features of the pain may suggest whether it is somatic in nature, radicular in nature or both. An appreciation of the quality of pain experienced and its topographic distribution guides management of the patient.

Radicular Pain ('Sciatica') and Somatic Referred Pain

Low back pain should not be confused with or regarded as synonymous with radicular pain ('sciatica'). Whilst back pain and radicular pain may occur together, their causes and mechanisms differ (Bogduk and McGuirk 2002) (refer to Table 4.5). The management of radicular pain is outside the scope of this guideline.

Radicular pain relates explicitly to pain felt in the lower limb; it is evoked by stimulation of the nerve roots or dorsal root ganglion of a spinal nerve (Merskey and Bogduk 1994). Radicular pain should not be confused with somatic referred pain, defined by Merskey and Bogduk (1994) as pain perceived in a region innervated by nerves or branches of nerves other than those that innervate the primary source of pain, where that source lies in one of the tissues or structures of the body wall (soma) or limbs.

It has been generally considered that pain radiating below the knee was radicular pain (i.e. representing nerve root pathology). However, a recent study (O'Neill et al. 2002) has shown that disc stimulation alone may cause referred pain into the distal extremity. Thus, pain that radiates below the knee cannot be considered to be specific for nerve root pathology. This should be taken into consideration to avoid unnecessary investigation and treatment.

A description of burning pain that is often a feature of neuropathic pain (i.e. pain resulting from a disease or injury to a nerve as opposed to pain from musculoskeletal tissues) is difficult to interpret. Deep, burning pain in the absence of any other feature, distribution or quality is not necessarily neuropathic pain. Burning sensations in the skin imply a neuropathic mechanism that may include a radicular or other neuropathic process.

Duration

It is important to establish the duration of pain (i.e. acute, subacute, chronic) as the evidence base for management options varies depending on pain duration. While duration does not carry diagnostic significance, it does have prognostic significance.

Frequency

Low back pain may wax and wane, but does not exhibit periodicity that is of diagnostic significance. Frequency is more likely to be a function of aggravating factors than an index of the cause or mechanism of pain.

Intensity

The severity of low back pain carries little diagnostic or prognostic weight. There are no valid guidelines by which to assess the clinical significance of very severe pain.

It is helpful to record the severity of the pain, at baseline and subsequently, using a quantitative assessment to provide an indication of whether or not the pain is improving or altering in severity over time (refer to Chapter 2: Acute Pain Management).

Table 4.5

Comparison of Somatic Referred and Radicular Pain

Characteristic	Somatic Referred Pain	Radicular Pain
Origin of pain	Due to spread of pain from deep spinal tissues (including muscles and spinal disc)	Due to chemical or mechanical irritation of nerves
Pain site	Back pain worse than leg pain, which may be bilateral	Unilateral leg pain worse than back pain
Pain distribution	Pain concentrates proximally in the buttock and thigh, but may spread below the knee	Pain concentrates distally, running into the lower limb, usually extending below the knee
Pain quality	Deep, dull, aching, expanding pressure-like quality	Sharp shooting electric quality, often deep and superficial
Pain location	Location is vague, varies over time, distribution ill-defined	Pain runs along defined narrow band in dermatome distribution
Paraesthesia	Poorly defined paraesthesia may be present	Numbness and paraesthesia in dermatomal distribution
Reflexes and motor strength	Normal reflexes and power (if these are abnormal, further assessment is indicated)	Reflexes may be reduced or absent; motor weakness may be present

Note: Adapted from Royal Australian College of General Practitioners (2002). Evidence-Based Primary Care Handbook on Acute Low Back Pain. RACGP: Victoria.

Onset

No particular cause of low back pain has a characteristic time of onset. Morning stiffness is said to be a feature of ankylosing spondylitis, but while this feature has a high to moderate sensitivity, its specificity is moderate to low, and its positive likelihood ratio is only 1.5 (Calin et al. 1977) or 1.6 (Gran 1985). A slow onset at less than 30 years of age, male gender, and improvement with exercise are early warning signs. For a more comprehensive discussion of how to establish a diagnosis of ankylosing spondylitis, see Gran (1985).

Spontaneous pain of an explosive onset should raise concerns of a spontaneous fracture or an infection. Recent history of penetrating injury in the form of a surgical or dental procedure, catheterisation or cannulation, a wound, or self-injection constitutes an alerting feature for possible osteomyelitis, epidural abscess or discitis. Low back pain that persists at night or disturbs sleep is also cause for concern.

Sudden onset of low back pain in association with trauma or minor trauma in the elderly or those on corticosteroids should alert the clinician to the possibility of fracture. This type of presentation is the only indication for plain xray of the lumbar spine.

Activities of Daily Living

It is important to evaluate the impact of pain on the patient's daily activities. The clinician should identify the main occupational, domestic and recreational activities and assess whether the acute low back pain is affecting activities such as dressing, driving, sitting, standing and sleeping.

Aggravating Factors

Regardless of whether there is a non-specific or specific cause, particular movements or activities may aggravate pain in the low back. Listing aggravating factors provides a description of the consumer and their problem, and foreshadows the assessment of disability.

The absence of aggravating factors is significant. A consumer with back pain that is not aggravated by spinal movement warrants assessment for a cause of pain that refers pain to the spine. Abdominal aortic aneurysms can present in this way (El Farhan and Busuttil 1997).

Relieving Factors

It is useful for people to identify factors that relieve their pain. These may include a range of non-pharmacological and pharmacological interventions, and also certain postures or activities.

Clinical Features of Specific Conditions

The following clinical features may be associated with specific conditions. While there are no data to substantiate a relationship between particular precipitating factors and particular causes of low back pain, the presence of these features in conjunction with acute low back pain should prompt further investigation. The following list is a guide only; it is not exhaustive.

Visceral Conditions

- A history of vascular disease, the presence of cardiovascular risk factors or the absence of aggravating features warrants assessment for aortic aneurysm.
- Endocrine disorders that erode bone or stretch periosteum may present with spinal pain, but offer few, if any clues on history alone.
- Hyperparathyroidism and Paget's disease are possible occult causes of spinal pain.

Neurological Conditions

Neurological symptoms are not indicative of any particular cause of spinal pain. They are features that should be assessed and investigated in their own right apart from any complaint of spinal pain.

Inflammatory Arthropathies

- Psoriatic and similar rashes offer a cue towards the seronegative spondylarthropathies.
- Symptoms or a history of diarrhoea may be a cue towards the seronegative spondylarthropathies.
- Pain elsewhere warrants consideration of systemic rheumatic diseases.
- Morning stiffness warrants assessment for ankylosing spondylitis

Fracture

A history of major trauma should provoke suspicion of fracture (Scavone et al. 1981a). Minor trauma is not a risk factor for fractures unless the patient has osteoporosis and is over 50 years of age. The literature suggests that patients with osteoporotic fractures following minor trauma tend to be substantially older than this limit (Scavone et al. 1981b). Use of corticosteroids is another risk factor for osteoporosis.

Infection

The cardinal feature of systemic infection is fever. A history of sweats or night sweats requires consideration of osteomyelitis, discitis, epidural abscess and other infection.

Injury to the skin or mucous membranes increases the risk of infection. Possible risk factors include a recent history of medical or surgical procedures, the presence of invasive devices (e.g. catheters), injecting drug use and trauma.

Other risk factors for infection include occupational exposure (e.g. Brucellosis), travel and immunosuppression (e.g. exposure to Mycobacterium tuberculosis). Cutaneous infections may be a source of spinal infection.

Features or a history of urinary tract infection or haematuria warrant an assessment of the renal tract as a source of pain referred to the low back.

Tumour

Features that alert to the presence of tumours are weight loss, age, past history of cancer, failure to improve with therapy and prolonged pain; a past history of cancer is the strongest predictor (Deyo and Diehl 1988; van den Hoogen et al. 1995; Scavone et al. 1981b).

The strongest negative predictors are age less than 50, no past history of cancer, no weight loss and no failure to improve with therapy (van den Hoogen et al. 1995; Scavone et al. 1981b). Patients with this combination of features are unlikely to have cancer as the cause of their back pain.

A history of cough may warrant consideration of lung cancer as a risk factor for spinal metastases. In men, symptoms of urinary retention warrant assessment for prostate cancer.

Key Message

History enables screening for features of serious conditions; however the reliability and validity of individual features in histories have low diagnostic significance. (*Level III-2)

Alerting Features of Serious Conditions (see Table 4.6)

The features and risk factors associated with serious conditions such as malignancy, infection and fracture may be detected through an assessment of the history of the condition (refer to Table 4.6). While there are no data to substantiate a relationship between particular precipitating factors and particular causes of back pain, the presence of these features in conjunction with acute low back pain should prompt further investigation (Refer to Appendix C: Ancillary Investigations). The table is intended as a guide only.

>Physical Examination

In the presence of acute low back pain an examination may include physical and psychosocial elements.

Physical Assessment

Inspection

Inspection may reveal minor aberrations of shape or posture of the lumbar spine, such as a loss of lordosis or a list. In some studies, the reliability of detecting aberrations has been found to be good, with kappa scores of the order of 0.5 to 0.7 (Waddell et al. 1982); however, agreement is worse in other studies (Strender et al. 1997). There are no data to show that such features have any construct validity for diagnosis or any predictive validity concerning treatment.

Identifying major postural deformities such as scoliosis is important for the diagnosis of such deformities. However, there appears to be no direct relationship between a major deformity and any known source or cause of low back pain.

Palpation

Palpation can be used to identify hyperaesthesia. In some studies this has been found to be a common feature amongst patients with back pain (Glover 1960); but this feature is non-specific, offering no diagnostic information.

Studies have shown that two observers can agree on finding tenderness somewhere in the lumbar spine in patients with back pain, with kappa scores equal to 1.00 (Waddell et al. 1982). However, when the location of tenderness is specified, agreement falls and varies from site to site.

One site where kappa scores for tenderness are good is over the iliac crest superomedial to the posterior superior iliac spine (Njoo et al. 1995). However, the specificity of tenderness over

Table 4.6

Alerting Features ('Red Flags') of Serious Conditions Associated with Acute Low Back Pain

Feature or Risk Factor	Condition
Symptoms and signs of infection (e.g. fever)	Infection
Risk factors for infection (e.g. underlying disease process, immunosuppression, penetrating wound)	
History of trauma	Fracture
Minor trauma (if > 50 years, history of osteoporosis and taking corticosteroids)	
Past history of malignancy	Tumour
Age > 50 years	
Failure to improve with treatment	
Unexplained weight loss	
Pain at multiple sites	
Pain at rest	
Absence of aggravating features	Aortic aneurysm

this site is unknown. Bone tenderness over the lumbar spinous processes has been held to be an alerting sign of osseous disorders such as infection or neoplasm. The reliability of this has been shown to be good to very good, however this sign has poor specificity and offers a positive likelihood ratio of only 2.2 for infection (Deyo et al. 1992).

As a diagnosis, 'trigger point syndrome' lacks validity for there is no objective criterion standard. In the lumbar spine, the detection of trigger points in the erector spinae or quadratus lumborum has poor reliability, with kappa scores less than 0.4 (Nice et al. 1992; Njoo and van der Does 1994). The entity of 'muscle spasm' has no validity for there is no known neurophysiological correlate of this clinical sign (Andersson et al. 1989; Roland 1986). In formal studies, the reliability of muscle spasm as a finding has been too poor to report in terms of kappa scores (Waddell et al. 1982).

Leboeuf-Yde et al. (2002) aimed to evaluate the prevalence of positive motion-palpation findings (fixations with spontaneous pain response) and to determine their sensitivity and specificity for detecting self-reported back pain. Fourteen percent of the study population reported low back pain of unclear duration; 43% had at least one lumbar spine fixation. They reported a sensitivity and specificity of 54% and 77% respectively for the ability of the abnormal clinical examination to detect those with current low back pain. This would yield a positive predictive value (PPV) of only 27.5% and a likelihood ratio of 2.3. Overall, the authors concluded that assessment of motion palpation did not help differentiate people with and without low back pain.

Range of Motion

A range of simple tests for range of motion of the lumbar spine exists, some of which are more reliable than others. Their clinical importance and validity remains uncertain.

Gross limitations of range of motion of the lumbar spine can be reliably detected by inspection, although the kappa scores for limited flexion are better than for limited lateral flexion. Use of a goniometer may offer greater precision in measuring range of motion, but the probability of an inter-examiner difference of 5° is 0.59; the probability of a difference of 10° is 0.28; and the probability of a 15° difference is as high as 0.11 (Mayer et al. 1995). Consequently, inter-examiner variation erodes any precision in measurement offered by a goniometer.

While limited range of motion may be common in the presence of low back pain, there is no evidence of a relationship to any specific cause and limited evidence that it predicts or influences recovery (but these effects are small).

Intervertebral Motion

It has been proposed that symptomatic lumbar spinal segments can be identified by careful examination of intersegmental motion. One study (Phillips and Twomey 1996) claimed a good correlation between the findings on manual examination and the results of diagnostic spinal blocks, but the nature of the blocks or their results was not described. Furthermore, the reliability of examination was poor, with kappa scores ranging from minus 0.15 to 0.32.

Other studies have indicated agreement among physiotherapists as to whether an L5-S1 or an L4-5 segment is hypomobile, but doctors were unable to agree on this feature (Strender et al. 1997). However, when estimates of intersegmental stiffness were compared, agreement was poor (Maher and Adams 1994).

Leg Length Asymmetry

Knutson (2002) evaluated the relationship between a number of clinical measures including leg length alignment asymmetry (LLA) and self-reported back pain among 74 volunteers. Overall, 51% of these volunteers had some leg length asymmetry; 82% reported having had back pain. The authors reported a significant association between LLA and back pain (current and recurrent) with sensitivity of 65%, specificity of 71% and a positive predictive value (PPV) of 88% for postural leg-length inequality detecting back pain. However, this yields a likelihood ratio (LR) of only 2.2, limiting its utility as a diagnostic test.

McKenzie

The McKenzie method of spinal assessment maintains that discogenic pain can be diagnosed on the basis of whether or not the pain 'centralises' upon certain movements of the lumbar spine (i.e. the extent of radiation of pain into the lower limb retracts) (Donelson et al. 1997). The reliability of McKenzie examination differs amongst observers. Some have found poor reliability (Riddle and Rothstein 1993) but others have found good reliability (Donelson et al. 1997) and have argued that expert training is critical. The validity of McKenzie examination has been tested against discography as a criterion standard and the correlation between findings is statistically significant. As a diagnostic test McKenzie examination is only marginally effective, offering modest likelihood ratios of 1.6–2.4 (Bogduk and Lord 1997).

Sacroiliac Joint

The sacroiliac joint is considered to be a source of low back pain and a number of physical tests have been developed. When these tests were evaluated they were found to be reliable, with kappa scores of the order of 0.8, but they lacked validity and have poor predictive value (LR ~1.0) (Dreyfuss et al. 1996). The tests may be positive in some 25% of individuals who have no pain (Dreyfuss et al. 1994).

Psychosocial Assessment

Perhaps the best-known example of an operationally defined clinical observation measure is that of Waddell et al. (1980). This entails performing a series of physical examinations on the patient that are not expected to aggravate pain (refer to Figure 4.1). To the extent that the patient reports pain on a yes/no basis for each of seven procedures, the clinician may

Predictors of Chronicity: Waddell's Non-organic Signs

- Superficial tenderness, non-anatomic tenderness
- Pain reaction to simulation tests for axial loading
- Pain reaction to simulated rotation
- Effect of distraction during examination (straight leg raising test)
- Regional sensory disturbance
- Regional weakness in non-anatomic distributions
- Over-reaction during examination (overt pain behaviour — grimacing, sighing, guarding, bracing, rubbing)

Figure 4.1

Waddell's physical signs: predictors of chronicity. Based on Main and Waddell 1998.

conclude whether the pain is non-organically based. They propose that in such cases, more detailed psychological assessment is required to clarify the basis of the pain behaviours (Waddell et al. 1980).

Unfortunately, some clinicians appear to have taken this test as a measure of malingering on the part of the patient. Main and Waddell (1998) have been strenuous in their attempts to rectify this impression and have pointed to a number of possible explanations for high scores with this measure; principally anxiety or fear. They counsel against excessive reliance on this test as a measure of psychosocial factors in clinical examinations.

Key Message

Clinical signs detected during physical and psychosocial assessment must be interpreted cautiously as many tests lack reliability and validity. (*Level III-2)

Neurological Assessment

A full neurological examination is warranted in a person presenting with lower limb pain in association with low back pain and any other neurological symptoms. Having the patient walk on their heels and toes can rapidly assess integrity of the L5 and S1 myotomes. Integrity of the sensory roots of L1 and S2 can be assessed by touch in the centres of the respective dermatomes. Studies have shown that neurological examination in patients with and without radiculopathy is quite reliable, with kappa scores in excess of 0.6 (Waddell et al. 1982; McCombe et al. 1989).

Key Message

A full neurological examination is warranted in the presence of lower limb pain and other neurological symptoms. (*Level IV)

>Ancillary Investigations

When a serious condition is suspected, appropriate investigations should be undertaken (refer to Appendix C: Ancillary Investigations). However, as serious conditions are rare causes of acute low back pain (approximately 5% of cases) (Suarez-Almazor et al. 1997; Hollingworth et al. 2002), ancillary investigations are usually unnecessary.

Imaging

Plain Radiography

Xrays reveal bone and may provide a crude image of some soft-tissues; they do not show pain. In patients with acute low back pain, lumbar radiographs are typically normal or show only spondylosis. In the published literature, the incidence of normal radiographs ranges from 21% in medical centre settings (Scavone et al. 1981b; Frazier et al. 1989) to 38% in emergency departments (Reinus et al. 1998), and 37% (Halpin et al. 1991) or 43% in primary care (Deyo and Diehl 1986). Two more recent studies (Suarez-Almazor et al. 1997; Hollingworth et al. 2002) found no change (approximately 40%) or only minor degenerative changes (approximately 55%) among people referred for xray from primary care settings, suggesting that as many as 95% of cases may be non-specific.

Kendrick et al. (2001) performed a randomised controlled trial of patients with low back pain present for at least six weeks. Patients were randomised to have a plain xray (n = 210; 168 underwent xray) or no xray (n = 211; 27 underwent xray). At three months the xray group had more pain (74% vs 65%; the number needed to harm = 11), disability and medical attention (53% vs 30%; the number needed to xray to cause a visit to the doctor = 4) than the control group. By nine months this difference was no longer significant but 65% of the xray group and 57% of the control group still had pain. Despite no benefit in outcomes, more than 80% overall said they would choose to have an xray; patients in the xray group reported being more satisfied with their medical care. The six-week entry criteria put these patients into a prolonged symptom group where many clinicians would be considering further investigation such as xray, however, this study suggests that the xray does not have a positive influence on outcome and should not be routinely recommended.

Kerry et al. (2002) conducted a randomised trial of xray (n = 65) versus no xray (n = 76) among patients presenting with acute low back pain to family health services in the United Kingdom. The xray group had higher mental health scores (74 vs 65) at six weeks and one year (77 vs 70) than the no-xray group. While this reached statistical significance (adjusted $p < 0.05$), the clinical significance of the less than ten point difference on a 100-point maximum scale was not discussed. There were no differences in any of the other seven domains of the SF-36, nor in the Roland and Morris disability scale or the HAD (Hospital Anxiety and Depression) scale. The authors concluded that there were no benefits in physical functioning, pain or disability associated with referring a patient for lumbar spine xray following their first presentation. They warned that the small advantage in mental health should be balanced against the exposure to radiation. Cost was not mentioned but should also be considered in decision-making.

Apart from unnecessary exposure to radiation, normal films may create a false sense of security. Lumbar spine radiographs may be false-negative in up to 41% of patients with known vertebral cancer (Frazier et al. 1989). Radiological evidence of vertebral osteomyelitis does not appear before two to eight weeks of evolution of the disease and a normal xray does not exclude the diagnosis of spinal infection (Waldvogel and Vasey 1980).

Key Message

Plain xrays of the lumbar spine are not routinely recommended in acute non-specific low back pain as they are of limited diagnostic value and no benefits in physical function, pain or disability are observed. (*LEVEL III-2)

Computed Tomography

Computed tomography (CT) scans are of limited value in the investigation of low back pain of unknown or unsuspected origin as the most frequently seen abnormalities are also common in people with no symptoms (Wiesel 1986). Even in the context of serious conditions, the role of CT is restricted to the confirmation of pathology otherwise indicated by history, clinical examination or other imaging tests. Additionally, there is variation in the interpretation of abnormalities among observers (Wiesel 1986). See Table 4.7.

Magnetic Resonance Imaging

Because of its high cost, the use of magnetic resonance imaging (MRI) cannot be justified for the screening of acute low back pain. Utilisation reviews demonstrate the low yield of serious conditions identified using MRI (Kitchener et al. 1986; Sorby 1989).

Table 4.7

Prevalence of Abnormalities on CT Scan in a Population of Asymptomatic Individuals Aged Between 21 and 80 Years

	N	Herniated Nucleus Propulsus	Degenerative Joint Disease	Spinal Stenosis
Age < 40	21–24	19.5%	0%	0%
Age > 40	24–27	26.9%	10.4%	3.4%

Note: Based on data from Wiesel et al. 1986.

Table 4.8

Prevalence of Abnormalities on MRI Scans of 67 Asymptomatic People

	N	Herniated Nucleus Propulsus	Disc Bulge	Spinal Stenosis	Disc Degeneration
All ages	67	24%	—	4%	—
Age 20–39	35	20%	54%	—	34%
Age 40–59	18	22%	—	—	—
Age 60–80	14	36%	79%	21%	92%

Note: Based on data from Boden et al. 1990.

MRI may be useful or should be considered if alerting features of cancer and infection are present on clinical assessment (refer Appendix C: Ancillary Investigations).

Validity

As with plain xray and CT-scan, it is common to identify conditions such as herniated discs, disc bulges, spinal stenosis, disc degeneration and spondylosis by MRI. These conditions occur quite frequently in asymptomatic individuals and are associated with age (Jensen et al. 1994; Boden et al. 1990). See Table 4.8.

Other Investigations

The presence of alerting features for serious conditions is an indication for ancillary investigations as outlined in Appendix C: Ancillary Investigations.

Key Message

Appropriate investigations are indicated in cases of acute low back pain when alerting features ('red flags') of serious conditions are present. (*Level III-2)

>Terminology

The evidence shows that symptoms and physical signs do not correlate sufficiently for definitive diagnosis of acute low back pain where serious conditions do not exist (Bogduk and McGuirk 2002). In the absence of any features of a specific condition it is difficult (and unnecessary) to determine the exact cause of pain in order for the pain to be managed effectively.

Key Message

A specific patho-anatomic diagnosis is not necessary for effective management of acute non-specific low back pain. (Consensus)

Diagnostic Terms

There are a variety of terms used by clinicians to define 'low back pain'. It is important to strive for consistency in terms to describe both the duration of pain (i.e. acute, subacute, chronic) and the anatomical location of pain.

The following definitions developed by the International Association for the Study of Pain (IASP) are based on anatomical topography (Merskey and Bogduk 1994). The taxonomy refers to different forms of spinal pain:

Lumbar Spinal Pain is pain perceived as arising anywhere within a region bounded superiorly by an imaginary transverse line through the tip of the last thoracic spinous process, inferiorly by an imaginary transverse line through the tip of the first sacral spinous process and laterally by vertical lines tangential to the lateral borders of the lumbar erectores spinae.

Sacral Spinal Pain is pain perceived as arising from anywhere with a region bounded superiorly by an imaginary transverse line through the tip of the first sacral spinous process, inferiorly by an imaginary transverse line through the posterior sacrococcygeal joints and laterally by imaginary lines passing through the posterior superior and posterior inferior iliac spines.

For pain overlapping between the lumbar and sacral regions, the IASP has developed the following definition:

Lumbosacral Pain is pain perceived as arising from a region encompassing or centred over the lower third of the lumbar region as described above and the upper third of the sacral region as described above.

Lumbar spinal pain, sacral spinal pain, lumbosacral pain or any combinations constitute what colloquially might be referred to as 'low back pain'. These definitions explicitly locate the pain as perceived in the lumbar and/or sacral regions of the spine. Terms that might be applied to a patient presenting with non-specific acute low back pain are 'lumbar spinal pain of unknown origin' or 'somatic lumbar spinal pain'.

Key Message

Terms to describe acute low back pain with no identifiable pathology include 'lumbar spinal pain of unknown origin' or 'somatic lumbar spinal pain'. (*Level IV)

PROGNOSIS

Systematic reviews and additional primary studies were used to develop this section, however the studies included in the systematic reviews have not been individually assessed.

Natural History

There are conflicting data from studies on the natural history of acute low back pain which may be partly explained by variations in symptom duration at inclusion and length of follow up.

Estimates range from 90% with complete recovery at two weeks from an episode of acute low back pain in a primary care cohort with pain for less than 72 hours at presentation (Coste and Rigby 1994) to only 27% completely better at a three month follow up among another primary care cohort with a mean pain duration of three weeks at inclusion to the study (Croft and Rigby 1994). The latter cohort was followed for a period of 12 months and while more than 90% had stopped seeking medical care for their back pain by three months, only 25% stated that they were completely re-covered (i.e. no pain and no disability) at 12 months (Croft et al. 1998). Thus, ceasing medical care does not necessarily mean the patient is symptom free or has returned to full function.

Schiottz-Christensen et al. (1999) performed a prospective cohort study in general practice in Denmark of 524 patients with low back pain of less than two weeks duration. Of those on sick leave for their low back pain, 50% returned to work by eight weeks and 98% by 12 months, although approximately 15% had taken further time off work during the 12 month follow up period and 46% were not completely recovered. No objective factors at the first visit predicted prognosis, whereas the general practitioner's global impression of the likelihood of developing chronic low back pain and those having a positive Straight Leg Raising test predicted more sick leave days in the first month. There were no other differences. While it would appear that many continue to have some back pain, most can still perform work-related activities and no longer seek medical care.

Australian data are available from the usual care arm of a non-randomised study among patients in primary care with median duration of low back pain of 2.1 weeks. Forty-nine percent had completely recovered at three months, 64% at six months and 56% at 12 months (McGuirk et al. 2001).

van den Hoogen et al. (1998) conducted a study of 443 patients including 342 with an onset of pain in the preceding seven weeks. The median time to recovery was seven weeks (interquartile range: 3–16 weeks); 70% still had pain at four weeks, 48% at eight weeks, 35% at 12 weeks and 10% at 12 months. Approximately 76% of patients had a recurrence of pain. The median number of relapses was two (interquartile range: 1–3), with a median time to relapse of seven weeks (interquartile range: 5–12) and a median duration of three weeks for the first relapse, two weeks for the second and third and one week for the fourth. Thus, recurrences of low back pain were not uncommon in the acute phase and appear to diminish in duration with each episode.

Key Messages

> The majority of people with a short duration of symptoms upon presentation with low back pain recover within three months; however milder symptoms often persist. (*Level III-2)

> Recurrences of acute low back pain are not uncommon. (*Level III-3)

Prognostic Risk Factors

While numerous studies were identified, very few met sound methodological criteria for prognostic studies. However, there was consistent evidence across multiple studies involving different populations and different measures for the role of psychosocial factors in progression to chronic pain. Consensus on which measures are optimal for particular psychosocial constructs and which combination of measures is optimal to provide adequate coverage of relevant psychosocial constructs has not been established.

Psychosocial Predictors of Chronicity

Three relevant systematic reviews were located focusing on: an evaluation of the evidence for psychosocial factors as predictors of chronicity/disability in low back pain (Pincus et al. 2002); an evaluation of the role of psychosocial workplace factors on back pain (Linton 2001); and an evaluation of biopsychosocial risk factors for low back pain (Truchon and Fillion 2000). All three reviews highlighted a lack of agreement in study definitions and inclusion criteria.

The review by Pincus et al. (2002) was the only review to formally assess study quality (methodological quality, quality of measurement of psychosocial factors and quality of statistical analysis) using multiple independent reviewers and presented quantitative findings (e.g. effect sizes, odds ratios) for all included studies. The scope of this review was appropriate for primary care settings as it was not limited to prognostic factors in occupational settings.

Linton (2001) defined criteria for the strength of evidence relating to each factor included for review. Nine of the 21 studies included in Linton's review involved samples with 'non-chronic back pain'; there was a mix of occupational and general population samples. In contrast, Truchon and Fillion (2000) provided no information about the overall strength of evidence for factors included in the review. Occupational and general population samples in the studies were reviewed as well as acute and subacute pain populations.

All three studies used similar and appropriate search strategies and only included prospective studies. There was only limited overlap in the studies included in all three reviews even though searches were conducted for similar time frames. Despite the differences between reviews, all three found consistent evidence across multiple studies involving different populations and different measures for the role of psychosocial factors in progression to chronic pain.

Pincus et al. (2002) based their results on six studies of high or acceptable quality. They found it difficult to differentiate between psychosocial distress, depressive symptoms and depressive mood and these were considered as a composite measure of 'distress'. Two high quality studies and two acceptable studies identified 'distress' as a significant predictor of unfavourable outcome independent of pain and function at baseline. They described a moderate effect size (~ 0.4) that was similar across studies and an Odds Ratio (OR) of ~ 3.0; this was greater than the effect sizes found for 'physical clinical' factors measured in the same study populations. Somatisation had one high quality and one acceptable study that found it to be a predictive factor of unfavourable outcome (effect size varying from 0.2 and 0.6 at one year and 0.9 at two years). Other factors that had been reported by others to be predictors did not appear to be independent in these analyses including Personality MMPI — Hysteria Subscale, praying/hoping/catastrophising or passive coping strategies and fear avoidance beliefs. This was at least partly due to a lack of relevant studies of acceptable quality relating to some of these psychosocial constructs (e.g. fear avoidance).

Truchon and Fillion (2000) identified preoccupation with own health, negative attitudes and outlook and passive coping strategies as useful predictors of chronicity. In general, psychosocial variables, when included, were more powerful predictors than clinical ones. The following factors were considered to have some potential in predicting chronicity, but more studies are required: locus of control; work environment;

job satisfaction; compensation/litigation process; family situation; personality type.

Evidence for Occupational Factors

Two systematic reviews focusing on psychosocial factors in occupational settings both concluded that psychosocial factors are important in the prognosis for return to work after onset of low back pain.

Linton's review (2001) identified 11 different psychosocial factors; six met *a priori* criteria for strong evidence (> 75% of studies agree, from three or more prospective studies) in relation to future back pain and disability. These were: job satisfaction (13 of 14 studies); monotonous work (4 of 6 studies); work relations (5 of 6); self-rated work demands (3 studies); self-reported stress (3 studies); and perceived ability to work (3 studies). There was moderate evidence (> 50% of studies agree, from two or more prospective studies) for work pace (2 studies), control (2 studies), perceived emotional effort at work (2 studies) and the belief that work is dangerous (2 studies).

McIntosh et al. (2000a) examined predictors for receiving sickness/compensation benefits three months after claiming for occupation-related low back pain injury in Canadian workers recruited from rehabilitational programs. Having three or more positive Waddell non-organic signs was one of the significant predictors of length of time on benefits. These signs measure behavioural responses to clinical examination and are used as an indicator of the need for more detailed psychological assessment (Main and Waddell 1998).

Fritz et al. (2000) evaluated a screening tool for its ability to predict return to work after an episode of acute low back pain. The Non-Organic Symptom Score based on Waddell's symptoms and signs was not found to be useful in this small study.

Fritz et al. (2001) found that a high score on a Fear Avoidance Beliefs Scale was an independent predictor of poor outcomes at four weeks (more disability on Oswestry scores, less likely to return to work) in a work-based clinical trial for low back pain. Numerous other factors including pain ratings, physical activity levels and work activity were not predictors.

Shaw et al. (2001) conducted a systematic review of predictors of chronicity following occupational low back pain. Twenty-two studies met their inclusion criteria; all studies had subjects with pain for less than six months. There was some overlap with some of the systematic reviews already discussed. They presented no individual study quality review, nor any raw data to calculate size of effect, but reported the following predictors of poor outcome (defined as days lost from work, not returning to work and remaining on workers' compensation): prior episodes, personal stress and severity of pain. Functional impact, radicular findings, delayed reporting, lack of support at work, shorter job tenure and heavier occupations without light duties were all reported as predictors of lack of return to work. They recommended the use of behavioural approaches to pain and disability, improved communication between employers and low back pain sufferers and the specification of return to work accommodations.

Fransen et al. (2002) published a more recent study in an occupational setting. This was a prospective study of 854 people with low back pain receiving workers' compensation. The subjects had a high rate of previous back trouble and current prevalence of radiating leg pain, thus the results may not be generalisable to all low back pain sufferers. At three month follow up 24% were still receiving compensation. The strongest predictor of remaining on compensation was a high distress score on the General Health Questionnaire (OR 2.8; 95%CI 2,3.9). Other risk factors included obesity, presence of severe radiating leg pain and baseline disability scores as measured on the Oswestry scale. This study found that some work activities, namely not having light duties available and needing to lift for more than 75% of the working day, were risk factors. Other studies have failed to identify work-related activities as risk factors. Job satisfaction, psychosocial factors in the work place (Work Apgar) and external locus of control were not risk factors in this cohort.

Abenheim et al. (1995) conducted a retrospective chart review of a random sample of 2147 workers compensated for back and neck injury and found that workers receiving a specific diagnosis were almost five times more likely to develop chronic pain and require prolonged compensated absences from work. Older workers given a specific diagnosis for their back injury were ten times more likely to progress to chronic pain than younger non-specific back pain subjects. While this may reflect accurate diagnosis of more harmful and chronic conditions, given that precise diagnosis of back problems in the absence of fracture or tumour lacks sensitivity it is likely that the labelling contributed to the psychological aspects of pain perception that are associated with chronicity. They highlight the importance of effective, non-emotive comuniation with patients with back pain, particularly in occupational settings.

Krause et al. (2001) conducted a retrospective study of a claimant cohort for acute low back pain. They found that a heavy work index and more severe injury predicted lack of return to work and those with an employment history of more than 12 months prior to injury were more likely to return to work.

A prospective cohort study by Infante-Rivard and Lortie (1996) followed an inception cohort of workers after their first compensated episode of acute low back pain. The results demonstrated that people are more likely to return to work if they are younger, had no disc involvement, received early intervention (within 30 days of the accident), had good flexion at baseline, had been employed for over two years and were allowed to take unscheduled breaks. This study didn't examine the contribution of psychosocial or other functional factors.

Evidence for Clinical Factors

Truchon and Fillion (2000) concluded in their review that clinical factors were only weak predictors of long-term outcome from acute low back pain. Six studies found that the severity of the diagnosis as determined by medical examination and medical imaging did not predict chronic disability or functional status. Four out of 5 studies found clinical tests may account for some of the variance in function. For example, one study found that the presence of pain on Straight Leg Raising (SLR) explained 7% of the variance in the Roland Morris Questionnaire. In this study clinical variables accounted for 10% while psychosocial variables accounted for 47% of variance. Another study found the clinical tests of lateral mobility, finger-floor distance and Achilles reflexes to correctly predict 67% of the workers likely to be absent from work 12 months later. The strongest clinical factor was a prior history of low back pain.

A small prospective study by Pulliam et al. (2001) published since the reviews also supports the role of psychosocial factors as predictor variables. The authors established a prospective cohort of patients from orthopaedic practices in Texas who had been seen for acute low back pain within 10 weeks of their injury. Four hundred and twenty-five were screened with the Dallas Back Pain Questionnaire and the MMPI Scale 3. Only 57 completed the additional questionnaires and interviews, reducing the generalisability of the

results. Affective disorder, anxiety, somatoform disorder or substance abuse were independently significant (OR 6.9). There was an unusual result with Axis II disorders (character pathology) that showed an unexpected inverse relationship with high-risk status in these models (Pulliam et al. 2001).

Werneke and Hart (2001) reported findings from a cohort (*n* = 223) of consecutive patients followed for 12 months after discharge from physical therapy rehabilitation services for low back pain. The mean duration of pain at entry was 13.3 days (SD 9.6 days). A number (30.7%) also had leg pain, therefore did not meet our study selection criteria. Loss to follow-up was 16%. All had had a dynamic assessment using the McKenzie protocol at the completion of seven sessions. Those patients who still had a non-centralised pattern of pain (22.7%) were three times more likely to have high pain intensity 12 months later (OR 3.0; 95%CI 1.4, 6.4), almost ten times more likely not to return to work (OR 9.4; 95%CI 3.4, 26.0), were more likely to report activity interference at home (OR 5.2; 95%CI 2.4, 11.3) and were more likely to continue to receive health care related to their low back pain (OR 4.4; 95%CI 2.0,10.1). The level of pain at the beginning of treatment, perceived disability at discharge and fear of work activities were not independent predictors of the outcomes in these analyses.

Hurley et al. (2001a) recruited a consecutive cohort (*n* = 118) from the Northern Ireland National Health Service with 100% uptake rate and 76% follow-up at 12 months. All completed a screening questionnaire (ALBPSQ) designed to be a Biopsychosocial Risk Profile for Chronicity. At baseline, 56% had had pain for less than three months (thus almost 50% had chronic pain and did not meet our inclusion criteria). Forty-five percent had had pain in the previous 12 months and 83.1% had a recurrence of low back pain within the twelve month period. Results were analysed in two main groups: Low ALBPSQ (< 112) or High ALBPSQ (> 112). They presented figures of 53.5% for sensitivity and 60% for specificity for recurrence of pain and 100% sensitivity and 61.5% specificity for work loss. The ALBPSQ correctly classified all cases involving sickness absence. Given that the Likelihood Ratio (LR) for a positive test was only 1.3 and for a negative test was 0.8, it did not support its use as a screening questionnaire for recurrent back pain. The ALBPSQ did not predict response to treatment.

Key Message

Psychosocial and occupational factors ('yellow flags') appear to be associated with progression from acute to chronic pain; such factors should be assessed early to facilitate intervention. (*Level III-2)

INTERVENTIONS

Although there are many forms of therapy for low back pain, there are only a limited number of randomised controlled trials (RCTs) testing the effectiveness of interventions for acute, non-specific low back pain. No RCTs were located for a number of interventions (e.g. intermittent heat, ice, ultrasound, hydrotherapy, short wave diathermy, Pilates, Feldenkrais, Alexander technique). Studies may exist that test these and other interventions on patients with chronic low back pain and low back pain associated with specific conditions (e.g. sciatica, osteoarthritis).

It is important to note that a lack of evidence (i.e. insufficient evidence) does not mean that a particular intervention has no place in the management of acute low back pain, however, it is preferable to employ interventions for which there is evidence of benefit, where appropriate. Management decisions should be based upon knowledge of the existing evidence, consideration of individual patient needs and clinical judgment.

The criteria formulated to categorise the following interventions are described in Chapter 1: Executive Summary. The levels of evidence are described in Chapter 9: Process Report.

Adverse effects have not specifically been investigated during this review, however information has been included in the text where adverse effects have been described in the cited material.

Evidence of Benefit

Advice to Stay Active (Activation)

The vast majority of studies investigating the effect of resuming normal activity involve populations with mixed durations of pain (acute and chronic) or patients with specific conditions (e.g. sciatica).

Clinical Evidence (2002) identified one Cochane Review (Hagen et al. 2002; last updated 2002) and one other systematic review (Waddell et al. 1997).

The Hagen et al. (2002) and Waddell et al. (1997) reviews included 11 RCTs. Five of these involved co-interventions (Indahl et al. 1995; Lindequist et al. 1984; Lindstrom et al. 1992a,b; Linton et al. 1993) and one involved patients with sciatica only (Vroomen et al. 1999). One study (Wiesel et al. 1980) met the criteria for this update, comparing bedrest to ambulation and to the use of analgesics (non-steroidal anti-inflammatory drugs and paracetamol) in male army recruits. They concluded that bedrest reduced pain compared to ambulation. However, the study was rated as low quality in the systematic reviews and it is unlikely that the results can be generalised to primary care settings.

The remaining four studies involved patients with mixed duration of pain (acute and chronic) with and without radiating pain. Two (Philips et al. 1991; Malmivaara et al. 1995) reported pain outcomes for groups receiving advice to stay active or advice to rest in bed. They found no significant difference in pain intensity in the short term (< 3 weeks) between the groups, however Malmivaara et al. (1995) reported a small but statistically significant reduction in pain intensity (0.8 points on an 11-point scale) in the stay active group in the intermediate term (> 3–12 weeks). Both studies found evidence of a faster rate of recovery in the stay active group. Malmivaara et al. (1995) concluded that advice to stay active also had a small beneficial effect on functional status and sick leave compared to two days bed rest and compared to a specific exercise regime comprised of staying active plus hourly back extension and lateral bending movements. The third study (Fordyce et al. 1986) compared analgesics, exercises and activity provided either on a time-contingent or pain-contingent (i.e. 'let pain be your guide') basis. They concluded that there was less likelihood of progressing to chronic pain in the behaviour-contingent group (this study is discussed in the section on 'Cognitive Behavioural Therapy'). The Wilkinson (1995) study found no significant difference in functional status or duration of sick leave between bed rest for two days versus staying active after one week; no pain outcomes were reported.

The Hagen et al. (2002) review pooled the results of two studies (Malmivaara et al. 1995; Vroomen et al. 1999) comparing advice to remain active and bedrest. Hagen et al. (2001) subsequently reported that there was no difference in pain intensity at less than three weeks (standardised mean deviation 0.03; 95%CI –0.20, 0.26) and at three to 12 weeks (0.20; 95%CI –0.03, 0.43).

A randomised controlled trial by Hagen et al. (2000) measured the return to full work duties at three, six and twelve month follow up periods. The treatment group was given advice to stay active and education on how to self-manage their pain compared to usual care in the control group. While pain outcomes were not measured, more people in the intervention group had returned to work at each of the follow up periods and the amount of sick leave was less than in the control group ($p = 0.0002$).

There is no evidence that remaining active (i.e. continuing with normal daily routines within the limits of pain) is harmful.

Additional Evidence

A Cochrane Review by Hilde et al. (2002; last updated 2001) reviewed advice to stay active as a single treatment for acute low back pain and sciatica. Four RCTs ($n = 491$) were included in the review (Wiesel et al. 1980; Wilkinson 1995; Malmivaara et al. 1995; Vroomen et al. 1999), overlapping with the 11 RCTs identified by Hagen et al. (2002) and Waddell et al. (1997). The reviewers concluded that while there was no major difference between the effects of advice to stay active compared to bed rest (particularly in the short term), the potential for side effects from long-term bed rest bears consideration.

Rozenberg et al. (2002) concluded that there was no difference in pain outcomes between normal activity and four days of bed rest. A reduction in sick leave was substantiated in the active group in this study (86% versus 52%; $p < 0.0001$), however there were methodological limitations. Approximately 60% of the patients had a history of low back pain.

Key Messages

> Advice to stay active provides a small beneficial effect on pain, rate of recovery and function compared to bed rest and compared to a specific exercise regime in mixed populations with low back pain. (Level I, II)

> Advice to stay active reduces sick leave compared to bed rest in mixed populations with low back pain. (Level I, II)

Heat Wrap Therapy

Nadler et al. (2002) conducted an RCT comparing continuous low-level heat wrap therapy to ibuprofen, acetaminophen, an oral placebo and an unheated back wrap. The heat wrap therapy consisted of a device that wraps around the lumbar region, heats to 40°C and maintains this temperature continuously for eight hours. Pain relief and decreased disability were more significant in those that wore the heated back wrap compared to all other groups. The follow up for this study was only four days; long lasting benefit of continuous heat therapy has not been established.

One participant reported minor redness from the heat wrap, which resolved spontaneously within an hour of removal. No other heat wrap-specific adverse effects were reported. This treatment is not routinely available in Australia.

No RCTs were located that assessed the efficacy of heat therapy used for intermittent periods (e.g. hot water bottle).

Key Message

Continuous low level heat wrap therapy reduces pain, stiffness and disability extending for 3–4 days compared with paracetamol, NSAIDs or placebo alone during the first 48 hours of acute low back pain. This treatment is not routinely available in Australia. (Level II)

Patient Information (Printed)

Printed information includes booklets, leaflets and brochures that provide evidence-based advice on the aetiology and natural history of acute low back pain, along with reassurance and advice to stay active. Such information can be used to supplement verbal advice provided by clinicians.

Five RCTs investigated the effect of printed consumer information on acute low back pain (Cherkin et al. 1996; Cherkin et al. 1998; Burton et al. 1999; Hazard et al. 2000; Roberts et al. 2002). The format and mode of administration of the interventions in these studies varied considerably.

Three RCTs (Cherkin et al. 1996; Cherkin et al. 1998; Hazard et al. 2000) found that the posting of printed information versus no information to patients showed no effect on pain, disability or duration of sick leave. When compared to Mackenzie exercises and spinal manipulation, posted printed information was less effective in reducing pain and disability (Cherkin et al. 1998). However, two RCTs (Burton et al. 1999; Roberts et al. 2002) using doctor-provided positively framed information about staying active along with verbal advice showed improved knowledge ($p = 0.006$), behaviour ($p = 0.009$), fear-avoidance beliefs, pain and disability scores compared with no additional printed information or a traditional 'passively framed' brochure.

Little et al. (2001) compared the effects of a detailed booklet on self-management of low back pain versus verbal advice to take regular exercise versus a combination of booklet and advice versus no intervention. At one week there was some benefit to either giving verbal advice or a booklet, but not using a combination of these methods. However, at three weeks there was no significant difference between the groups. No harms were reported with this intervention.

An RCT by Linton and Andersson (2000) compared the provision of an educational pamphlet versus a weekly, more extensive information package versus six two-hour group sessions of CBT focusing on activation coping strategies. The duration of pain was not defined, however subjects were described as having acute or subacute spinal pain and less than three months of sick leave in the past year. At one year, there were no between group differences in pain outcomes. However, sick leave and health care utilisation were both significantly lower in the CBT group compared to those receiving printed information. The population comprised a mix of acute and chronic pain.

Key Messages

> Novel or 'activity-focused' printed information plus similar verbal advice provided by a clinician is more effective compared to traditional brochures or no printed information in acute low back pain. (Level II)

> Printed information provided through the mail is less likely to have an effect on pain, disability and sick leave compared to information provided in person. (Level II)

> Behavioural therapy interventions are more effective than printed information for preventing long-term disability in mixed populations. (Level II)

Conflicting Evidence

Muscle Relaxants

Muscle relaxants are a diverse group of drugs acting in a variety of ways at the neuromuscular junction or directly on skeletal muscle to reduce muscle spasm.

There have been a number of studies conducted on the efficacy of muscle relaxants in acute low back pain. Based on the results of two systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b), Clinical Evidence (2002) concludes that

muscle relaxants versus placebo reduce pain and muscle tension and increase mobility. The outcomes for one muscle relaxant over another were not significantly different.

van Tulder et al. (1997b) identified 14 RCTs in their review of muscle relaxants for acute, non-specific low back pain, rating eight as high quality. Of these, five (Hindle 1972; Baratta 1982; Berry and Hutchinson 1988; Casale 1988; Dapas et al. 1985) compared muscle relaxants to placebo; all reported better pain relief from the muscle relaxant. Three studies (Boyles et al. 1983; Middleton 1984; Rollings et al. 1983) compared different types of muscle relaxants and all reported no differences in pain outcomes. The review concluded there is evidence that muscle relaxants are more effective than placebo and they are equally effective in treating acute low back pain.

Bigos et al. (1994) identified three additional RCTs (Arbus et al. 1990; Klinger et al. 1988; Basmajian 1989) comparing a muscle relaxant to placebo. Two studies (Arbus et al. 1990; Klinger et al. 1988) involving mixed populations reported favourable results for muscle relaxants. However, Basmajian (1989), in a study involving acute, non-specific low back pain, reported no difference in outcomes. Basmajian (1989) also compared a muscle relaxant combined with an NSAID versus muscle relaxant and NSAID separately and reported no difference between muscle relaxants and NSAIDs. The method of randomisation was not described in this study.

The Bigos et al. (1994) review concluded that muscle relaxants are likely to be more effective than placebo but there is insufficient evidence to determine whether muscle relaxants are more or less effective than NSAIDs or whether combination therapy offers a synergistic effect.

Adverse effects of muscle relaxants are common, including drowsiness, dizziness and dyspepsia. Dependency has been reported after one week of use (Bigos et al. 1994; van Tulder et al. 1997b).

Additional Evidence

Browning et al. (2001) describes a meta-analysis of cyclobenzaprine compared with placebo, reporting that cyclobenzaprine substantially improves local pain (59% reduction; days 1–4) and global symptoms (Odds Ratio = 4.7; days 1–4) compared with placebo. The effect declined considerably after the first week and was associated with a 25% increase in side effects such as drowsiness, dry mouth and dizziness compared to placebo (53% cyclobenzaprine versus 28% placebo). Cyclobenzaprine is not currently available in Australia.

Key Messages

- > There is conflicting evidence that muscle relaxants are effective compared to placebo in acute low back pain. (Level I)
- > There is insufficient evidence to determine whether muscle relaxants are more or less effective compared to NSAIDs for acute low back pain. (Level I)
- > Drowsiness, dizziness and dependency are common adverse effects of muscle relaxants. (Level I)

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)

Non-steroidal anti-inflammatory drugs (NSAIDs) are commonly administered by oral, topical or intramuscular routes. As a drug class they are thought to act through inhibiting prostaglandin production. Many oral and topical NSAIDs are available without prescription.

Clinical Evidence (2002) reports four systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b; van Tulder et al. 2002f; Koes et al. 1997) and three RCTs (Pohjolainen et al. 2000; Laws 1994; Chok et al. 1999), concluding there is a significant increase in global improvement after one week from NSAIDs versus placebo (pooled Relative Risk 1.24; 95%CI 1.10, 1.41) and a reduction in the amount of additional analgesic required (pooled RR 1.29; 95%CI 1.05, 1.57). No studies on the efficacy of topical NSAIDs were located.

Many of the RCTs included in these reviews involved populations with chronic low back pain or a mix of acute and chronic low back pain, sciatica and spinal degenerative disease. Various modes of drug administration were also included. Fifteen studies in these reviews appear to meet the criteria for this update (Amlie et al. 1987; Basmajian 1989; Bakshi et al. 1994; Borenstein et al. 1990; Brown et al. 1986; Colberg et al. 1996; Hosie 1993; Lacey et al. 1984; Milgrom et al. 1993; Postacchini et al. 1988; Sweetman et al. 1987; Szpalski and Hayez 1994; Videman et al. 1984; Orava 1986; Wiesel et al. 1980).

Five studies (Amlie et al. 1987; Basmajian 1989; Lacey et al. 1984; Postacchini et al. 1988; Milgrom et al. 1993) measured oral NSAIDs against placebo or no treatment. Amlie et al. (1987) demonstrated that oral NSAIDs reduced pain at three days but were no different to placebo at seven days in 282 patients. Lacey et al. (1984) included a subgroup analysis of patients with acute back strain or sacro-iliac pain of less than three days duration. They reported significant improvement in moderate pain at one week ($p < 0.001$) in the group receiving two weeks of oral NSAID (piroxicam) treatment compared to placebo. Postacchini et al. (1988) reported greater improvement (a combined pain, disability and mobility score) following 10–14 days of oral NSAID use compared to placebo and other treatments at two months follow up, however the result was not statistically significant. Basmajian (1989) reported no difference between oral NSAID and placebo at two, four and seven days. Milgrom et al. (1993) compared oral NSAID with no treatment in male military recruits with acute low back pain and reported no significant differences between the groups after ten weeks. These two trials have methodological limitations. One additional study (Szpalski and Hayez 1994) reported significantly better pain relief at day eight in a group receiving an initial dose of injected NSAID followed by oral NSAIDs compared to a group receiving placebo injection and oral medication.

Four studies (Bakshi et al. 1994; Hosie 1993; Orava 1986; Colberg et al. 1996) compared NSAID versus NSAID. No significant difference in pain intensity was reported between oral diclofenac and piroxicam (Bakshi et al. 1994), oral ibuprofen and felbinac foam (Hosie 1993) or oral diflunisal and indomethacin (Orava 1986). Colberg et al. (1996) reported greater efficacy with meloxicam compared to diclofenac, however the meloxicam was delivered intravenously compared to intramuscular administration of diclofenac.

Three studies compared NSAIDs with analgesics (Brown et al. 1986; Videman et al. 1984; Wiesel et al. 1980). Brown et al. (1996) compared oral diflunisal with acetaminophen plus codeine and found no significant differences in pain, although more people in the diflunisal group experienced side effects. Videman et al. (1984) compared oral diflunisal with an oral opioid analgesic (meptazinol) and found similar improvements in pain intensity and similar side effects in the two groups. Wiesel et al. (1980), in a study on male military recruits, compared aspirin versus phenylbutazone

versus acetaminophen and found no significant differences in the mean number of days to full activity.

NSAIDs were compared to muscle relaxants in two studies (Basmajian 1989; Borenstein et al. 1990). Basmajian (1989) compared diflunisal versus a combination of diflunisal and cyclobenzaprine versus cyclobenzaprine alone versus placebo. After four days, the group receiving combination therapy showed significant improvement (based on total distribution) compared to the others. Borenstein et al. (1990) compared naproxen with a combination of naproxen plus cyclobenzaprine. Pain resolved more quickly in the group receiving combination therapy however more patients suffered drug side effects.

Sweetman et al. (1987) compared mefenamic acid versus a combination of anti-anxiety agent plus acetaminophen (paracetamol) versus a combination of anti-anxiety agent plus aspirin. More patients in the combination acetaminophen plus anti-anxiety agent group reported no pain at day one (28% and 25% respectively) compared to the NSAID group (17%). The differences between groups at day seven were negligible.

Adverse effects including gastrointestinal bleeding and perforation, tiredness and dizziness can occur to varying degrees with the use of NSAIDs and appear to be dose-related (Bigos et al. 1994; Henry et al. 1996).

Additional Evidence

Nadler et al. (2002) reported that pain was significantly worse in the first four days for patients on oral NSAIDs compared with those on heat wrap therapy (see 'Heat Wrap Therapy'). The mean score was 0.93 points lower in the NSAID group using a six point verbal rating scale for pain ($p = 0.0001$). Nadler et al. (2002) did not report the comparison between NSAIDs and the placebo arm of the trial.

Key Messages

- There is conflicting evidence that oral and injectable NSAIDs are effective versus placebo or no treatment for acute low back pain. (Level I)
- NSAIDs have a similar effect compared to opioid analgesics, combined paracetamol-opioid analgesics and to each other in their effect on acute low back pain. (Level I)
- There is insufficient evidence that NSAIDs are more effective when compared to muscle relaxants and anti-anxiety agents in acute low back pain. (Level I)
- NSAIDs are less effective in reducing pain than heat wrap therapy in the first 3–4 days of acute low back pain. (Level II)
- Serious adverse effects of NSAIDs include gastrointestinal complications (e.g. bleeding, perforation). (Level I)

Spinal Manipulation

Spinal manipulation is a form of manual therapy involving the movement of a spinal joint to the end of its voluntary range of motion followed by application of a single high-velocity, low amplitude thrust. It is distinct from other forms of manual therapy such as spinal mobilisation, which comprises the passive application of repetitive, rhythmical, low velocity movements applied within the joint range of motion. An attempt has been made to refer only to those studies involving spinal manipulation in this update.

The evidence for the efficacy of spinal manipulation is inconclusive due to methodological limitations in the majority of trials conducted to date and the use of different spinal manipulation techniques.

Clinical Evidence (2002) reports six systematic reviews (Evans and Richards 1996; van Tulder et al. 1997b; Shekelle et al. 1992; Koes et al. 1996; Mohseni-Bandpei. et al. 1998; Bigos et al. 1994) on spinal manipulation. With the exception of the Evans and Richards (1996) review, these systematic reviews have been obtained and assessed for this update.

van Tulder et al. (1997b) included 16 RCTs in their review, ascribing quality scores between 22 and 51 out of 100. Two studies were rated as high quality (MacDonald and Bell 1990; Sanders et al. 1990) although the latter was removed from the van Tulder et al. (1997b) analysis due to a follow up period of only 30 minutes. Eleven of the remaining 15 studies reported positive results for spinal manipulation. Four studies (Bergquist-Ullman and Larsson 1977; Glover et al. 1974; Postacchini et al. 1988; Wreje et al. 1992) compared spinal manipulation to placebo and three were positive for manipulation. Fourteen of the RCTs compared manipulation with other interventions (massage, analgesic, NSAID, shortwave diathermy, exercises). The authors concluded that there is limited evidence that manipulation is more effective than placebo and no evidence that manipulation is more or less effective than other treatments because of conflicting results and methodological limitations.

The review by Shekelle et al. (1992) defined acute low back pain as less than three weeks duration and included nine RCTs of acute low back pain without sciatica. Shekelle et al. (1992) rated the methodological quality of the studies; scores ranged from 28-56 out of 100. Hadler et al. (1987) and MacDonald and Bell (1990) earned the two highest quality scores of 56 and 53 respectively; both involved patients with pain of between two and four weeks duration. Hadler et al. (1987) compared manipulation with mobilisation (as sham manipulation) and reported a more rapid reduction in pain scores in the manipulation group ($p < 0.03$). MacDonald and Bell (1990) compared manipulation plus back exercises plus instructions versus back exercises plus instructions and reported a greater improvement on a disability index at one week for the treatment group ($p < 0.04$). A meta-analysis was performed on the remaining seven studies as they used a similar outcome measure (Coyer and Curwin 1955; Bergquist-Ullman and Larsson 1977; Farrell and Twomey 1982; Godfrey et al. 1984; Rasmussen 1979; Waterworth and Hunter 1985; Mathews et al. 1987). The results showed the probability of recovery increased by 0.17 (95% probability limits, 0.07, 0.28) at two to three weeks after commencing manipulation treatment. The authors concluded that spinal manipulation hastens recovery and provides relief from acute uncomplicated low-back pain in patients with between two to four weeks of symptoms. However, the long-term effects of manipulation in preventing the development of chronic low back pain or in preventing recurrences of acute low back pain are unknown. A number of the included studies contained mixed populations or did not fully describe the participants.

Koes et al. (1996) defined acute low back pain as pain of less than six weeks duration. Three studies of spinal manipulation compared to placebo are described (Sanders et al. 1990; Bergquist-Ullman and Larsson 1977; Glover et al. 1974) with conflicting results. The Sanders et al. (1990) study was excluded because the results were not clinically relevant. Twelve RCTs comparing manipulation with other treatments including massage, exercises, short wave diathermy, back school, analgesics, infrared heat and NSAIDs were described;

all were included in the review by Shekelle et al. (1992) with the exception of two studies. Helliwell and Cunliffe (1987) reported no significant difference between manipulation versus analgesic use (n = 14). Delitto et al. (1992) compared mobilisation of the sacroiliac joint with flexion exercises and reported better function in the manipulation group at three and five days (n = 24). The majority of these studies contain mixed acute and chronic populations, describe specific conditions or do not provided details of the participants. Many have methodological limitations.

Mohseni-Bandpei et al. (1998) conducted a review of studies of spinal manipulative therapy published between 1985 and 1997. Acute low back pain was defined as pain for less than 12 weeks. The authors identified 25 RCTs that met their criteria; 12 of these involved acute/subacute populations and two provided acute low back pain subgroup analyses. Five of the 14 RCTs compared manipulation to placebo therapy (Gibson et al. 1985; Rupert et al. 1985; Postacchini et al. 1988; Wreje et al. 1992; Sanders et al. 1990). With the exception of the Gibson et al. study (1985), the authors report that four of the five studies are positive for manipulation. However, the result of the Sanders et al. (1990) study was not deemed clinically relevant, the Wreje et al. (1992) study found no significant difference in pain outcomes and the review authors noted that the Rupert et al. (1985) study gave inadequate consideration to the nature of the placebo. Only one study (Postacchini et al. 1988) reports outcomes (positive) for patients with acute, non-specific low back pain; it is likely that the other studies included patients with radiating pain and other specific conditions. All of the studies have been previously described in the reviews by Koes et al. (1996), van Tulder et al. (1997b) and Shekelle et al. (1992).

Bigos et al. (1994) identified 12 RCTs; all have been discussed in previous reviews with the exception of Brodin (1984), however this trial is not described in the text of this review.

Clinical Evidence (2002) provided an unreferenced statement that serious complications from spinal manipulation for low back pain are rare. In order to verify this, three published literature reviews of case studies were obtained (Haldeman and Rubinstein 1992; Stevinson and Ernst 2002; Assendelft et al. 1996). From these studies it is concluded that serious complications (i.e. cauda equina syndrome) from spinal manipulation are rare when a qualified practitioner performs the procedure after assessing for potential contraindications.

Additional Evidence

Pengel et al. (2002) reviewed interventions for subacute low back pain; two studies investigated the effects of spinal manipulation on patients with non-specific pain of between three weeks and six months duration. The first by Hsieh et al. (1992) compared the effects of manipulation, massage, corset use and TENS on disability and reported a significant difference between the manipulation and TENS groups ($p < 0.05$). Andersson et al. (1999) found no difference in pain and other outcome measures between groups receiving spinal manipulation or usual care.

A randomised controlled trial (Hsieh et al. 2002) was located that assessed the efficacy of manipulation compared to other manual therapies and back school (200 subjects). This RCT found that manipulation was not superior to the other therapies for acute low back pain of between three weeks and six months duration.

Key Messages

> There is conflicting evidence that spinal manipulation provides pain relief compared to placebo in the first two to four weeks of acute low back pain. (Level I)

> There is insufficient evidence that spinal manipulation is more or less effective than other conservative treatments for acute low back pain. (Level I)

> Adverse effects of spinal manipulation are rare but potentially serious. (Level IV)

Insufficient Evidence

Acupuncture

Clinical Evidence (2002) cited two reviews on acupuncture. A Cochrane Review by van Tulder et al. (2002a; last updated 1999) located 11 RCTs on acupuncture, however only one met the criteria for this update (Garvey et al. 1989). A meta-analysis conducted by Ernst and White (1998) included 12 RCTs; all except the Garvey et al. (1989) study involved chronic low back pain or were not published in English.

Garvey et al. (1989) conducted a double-blind study to evaluate trigger point injection therapy in patients with non-radiating low back pain. The duration of pain is not specifically reported, however it appears to be acute. Four groups (n = 63) were randomised to receive either injection with lignocaine, injection with lignocaine combined with steroid, a single dry needle-stick (acupuncture) or acupressure using a plastic needle guard following vapocoolant spray. Subjective reports of pain improvement were higher following acupuncture or acupressure compared to injection therapy (63% reporting improvement versus 42%) at two weeks. However, the result was not statistically significant (p = 0.093). Side effects of acupuncture (haematoma formation) were reported in the study.

No additional systematic reviews or randomised controlled trials on the effects of acupuncture on acute, non-specific low back pain were located. Adverse effects are rare but potentially serious, including infection, pneumothorax and visceral trauma (Ernst and White 1997).

Key Messages

> There is insufficient evidence that accupuncture (dry-needling) is effective compared to injection therapy in acute low back pain. (Level I)

> Adverse effects of accupuncture are rare but potentially serious. (Level I)

Analgesics (Compound and Opioid)

Weak opioids alone and in combination with paracetamol are available in Australia with and without prescription. There are no placebo-controlled trials for the use of compound analgesics in acute, non-specific low back pain.

Clinical Evidence (2002) reports on paracetamol and opioids as a group via two systematic reviews (van Tulder et al. 1997b; Bigos et al. 1994). Of the six studies cited in the van Tulder et al. (1997b) review, two studies investigating the effect of compound and narcotic analgesics met the criteria for this update. A study by Videman et al. (1984) compared an opioid analgesic (meptazinol) to an NSAID (diflunisal) in patients with acute low back pain. No significant difference in pain was found after three weeks of treatment. Brown et al. (1986) compared a combination of paracetamol and codeine with diflunisal and found no significant difference in pain, but more side effects associated with the combined therapy.

Another study cited in Bigos et al. (1994) evaluated the effect of diflunisal compared to acetaminophen combined with codeine in people with soft tissue injury and found no significant difference in pain outcomes at two weeks (Muncie et al. 1986).

A systematic review not specific to acute low back pain (de Craen et al. 1996) reported on 29 RCTs with a pooled 5% reduction in pain with compound analgesia compared with paracetamol alone but a substantial increase in side effects with multiple doses of compound analgesics compared with multi-dose paracetamol alone (OR = 2.5; 95%CI 1.5, 4.2). The most commonly reported adverse effects were nausea, dizziness, vomiting, constipation and drowsiness. Given the lack of convincing evidence for the efficacy of compound analgesia versus NSAIDs in reducing acute, non-specific low back pain, the risk of harm associated with using paracetamol or NSAIDs or opioid/compound analgesics must be considered for the individual patient.

Additional Evidence

Palangio et al. (2002) conducted a study of patients with moderate to severe acute low back pain comparing combined paracetamol and oxycodone versus combined ibuprofen and hydrocodone. There was no difference in the additional benefits or harms between the groups.

Key Messages

> There are no randomised controlled trials investigating the efficacy of opioids and compound analgesics in acute low back pain. (No Level I or II studies)

> There is evidence that the effect of opioid or compound analgesics is similar to NSAIDs for treatment of acute low back pain. (Level I, II)

> In general, opioids and compound analgesics have a substantially increased risk of side effects compared with paracetamol alone. (Level I)

Analgesics (Simple)

Simple analgesics (i.e. paracetamol/acetaminophen) are widely available without prescription to patients with acute low back pain. Most trials report on the effect of a regular weight-appropriate dose of these agents rather than on their use as required regimen. Paracetamol overdose is associated with liver damage; the drug is safe if taken according to appropriate dose for weight.

Two systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b) were cited in Clinical Evidence (2002). No placebo-controlled RCTs on the efficacy of simple analgesics in the treatment of acute, non-specific low back pain were located. However, their effectiveness in treating other types of musculoskeletal pain is reportedly comparable to non-steroidal anti-inflammatory drugs (Bradley et al. 1991 cited in Deyo 1996). Three RCTs were located that compared use of a simple analgesic with another treatment.

Two studies (Wiesel et al. 1980; Milgrom et al. 1993) involved male military recruits and were considered low quality in the reviews. Wiesel et al. (1980) compared two NSAIDs (oral aspirin and phenylbutazone) with acetaminophen and found no significant differences in the mean number of days to full activity. Milgrom et al. (1993) compared oral ibuprofen versus paracetamol versus no drug treatment and found no significant difference between the groups. At ten weeks follow up, 67% of the NSAID group, 54% of the paracetamol group and 82% of the no drug group had fully recovered. This was a small trial (n = 70) with methodological limitations.

Hackett et al. (1988) conducted a study comparing electro-acupuncture with paracetamol. The population includes patients with acute low back pain with and without pain radiation. Electroacupuncture was more effective in reducing pain compared with paracetamol at six weeks follow up, but not at one or two weeks.

Paracetamol overdose is associated with liver damage.

Additional Evidence

Nadler et al. (2002) compared paracetamol against continuous low level heat wrap therapy for treatment of acute, non-specific low back pain. The authors concluded that paracetamol was less effective in reducing pain in the first four days than continuous low-level heat wrap therapy (see Heat Wrap Therapy). Heat wraps have not been evaluated in combination with analgesics and the specific heat device used in the trial is not widely available. There was no report of the paracetamol versus placebo arm of the trial.

Key Messages

> There are no randomised controlled trials assessing the effectiveness of simple analgesics in acute low back pain. (No Level I or II studies)

> There is insufficient evidence for the effectiveness of simple analgesics versus NSAIDs in acute low back pain. (Level I)

> Paracetamol is less effective than heat wrap therapy in acute low back pain. (Level II)

> There is insufficient evidence for the effect of paracetamol compared to electroacupuncture in mixed populations with low back pain (Level I)

Back Exercises

A variety of exercises are advocated for people with low back pain, including stretching, back flexion and extension exercises, endurance (aerobic) training and strengthening exercises or combinations of these (and other) exercises. Exercise programs differ in their content, delivery and therapeutic objectives. Studies of the effectiveness of exercises differ in their study populations, outcome measures, exercise regimes, treatment and control groups and length of follow up. These issues make it difficult to draw conclusions on the effectiveness of exercises for acute, non-specific low back pain.

Clinical Evidence (2002) reported on five systematic reviews (Bigos et al. 1994; Evans and Richards 1996; van Tulder et al. 1997b; Faas 1996; and a Cochrane-style review by van Tulder et al. 2000) and two RCTs on back exercises versus conservative or inactive treatments (Chok et al. 1999; Hides et al. 1996). All of these studies were obtained and reviewed except Evans and Richards (1996).

Bigos et al. (1994) identified six RCTs on exercise for acute low back pain (Evans et al. 1987; Lindstrom et al. 1992a,b; Stankovic and Johnell 1990; Coxhead et al. 1981; Davies et al. 1979; Zylbergold and Piper 1981). The Coxhead et al. (1981) study involved patients with sciatica and the remaining five studies either involved acute pain with and without radiation, did not describe this aspect or involved a mix of acute and chronic pain durations. Evans et al. (1987) compared four days bed rest versus four days bed rest, exercises and education versus exercise and education versus no treatment in patients with acute low back pain with and without pain radiation (n = 242). They reported no significant differences in pain or

mobility. Lindstrom et al. (1992a,b) also involved patients with acute low back pain with and without pain radiation, comparing usual care with exercises (aerobic and back strengthening). They reported significantly less sick leave in the exercise group at six and 12 weeks and no differences in functional status after one year (n = 103). Stankovic and Johnell (1990) involved patients with less than four weeks of low back pain in a study comparing one session of back school with McKenzie exercises to restore or maintain lumbar lordosis. They reported significantly less pain at three weeks and one year, fewer recurrences and less absence from work in the exercise group. Davies et al. (1979) showed no significant differences in improvement at two and four weeks between short-wave diathermy (SWD) plus extension exercises versus isometric flexion plus SWD versus SWD alone in a mixed population. Zylbergold and Piper (1981) compared home care instructions versus exercises plus heat versus manual therapy and found no significant difference in pain intensity at one month in a population with lumbar disc disease.

In addition to the RCTs identified by Bigos et al. (1994), van Tulder et al. (1997b) identified seven additional studies (Faas et al. 1993; Malmivaara et al. 1995; Waterworth and Hunter 1985; Nwuga 1982; Nwuga and Nwuga 1985; Farrell and Twomey 1982; Delitto et al. 1993). These studies involved mixed populations (mixed acute and chronic pain or pain with and without radiation) or women only (Nwuga 1982; Nwuga and Nwuga 1985).

The Cochrane Review by van Tulder et al. (2002d, last updated 2000) located only one study (Underwood and Morgan 1998) involving patients with acute low back pain without pain radiation out of 12 RCTs on exercises for acute low back pain. Four of the 12 RCTs were rated by van Tulder et al. (2002d) as high quality (Cherkin et al. 1998; Malmivaara et al. 1995; Faas et al. 1993; Nwuga and Nwuga 1985). Exercises were compared to usual care, manual therapy, back school and NSAIDs in eight studies. There were no differences reported in the studies comparing exercises with usual care (Fass et al. 1993; Seferlis et al. 1998; Underwood and Morgan 1998) or NSAID use (Waterworth and Hunter 1985). Two (Farrell and Twomey 1982; Nwuga 1982) out of five studies comparing exercises with manipulation reported lower recovery and less improvement in the exercise group. The remaining three studies found no significant difference between the groups. Four studies compared exercises with a placebo consisting of inactive treatment such as bedrest (Malmivaara et al. 1995; Gilbert et al. 1995), placebo ultrasound (Faas et al. 1993) and printed educational material (Cherkin et al. 1998). Three studies reported no differences in pain outcomes between groups and a third reported a better outcome for the placebo group (bedrest).

A number of the studies compared extension and flexion exercises to other therapies and to each other. Five of the studies tested extension exercises conducted according to the McKenzie principles. Three compared McKenzie therapy to other treatments; one high quality study (Cherkin et al. 1998) found no difference in global improvement between McKenzie therapy versus manipulation or an educational booklet. Two studies (Stankovic and Johnell 1990; Underwood and Morgan 1998) reported less pain following McKenzie exercises compared to one session of back school and no significant differences in pain or function outcomes between McKenzie exercises and usual care, respectively (the Underwood and Morgan study was the only one involving patients with acute, non-specific low back pain). Malmivaara et al. (1995), who did not conduct back extension exercises according to McKenzie principles, reported significantly better pain outcomes for the control group (who maintained ordinary activity) compared to the exercise group and to the bedrest (two days) group at three and 12 weeks. Williams flexion and McKenzie extension exercises were compared in two studies (Nwuga and Nwuga 1985; Delitto et al. 1993). Nwuga and Nwuga (1985) reported significantly better pain outcomes for McKenzie exercises and Delitto et al. (1993) reported better functional outcomes from McKenzie therapy.

The review by Faas (1996) involved RCTs on exercises for low back pain published between 1991 and 1995. Eleven studies were included; four of these involved acute low back pain (Faas et al. 1993; Malmivaara et al. 1995; Stankovic and Johnell 1990; Delitto et al. 1993) and one involved subacute low back pain (Lindstrom et al. 1992a,b). Their results are described previously in the summary of the van Tulder et al. (2002d) and Bigos et al. (1994) reviews.

Hides et al. (1996) evaluated the effect of an exercise regime on recovery of the multifidus muscle following an initial episode of acute low back pain (n = 39), on the basis that exercises of the multifidus and transversus abdominis muscles have been shown to reduce pain in patients with chronic low back pain. The study compared specific, localised exercises designed to restore the stabilising function of the multifidus muscle plus usual care versus usual care alone. At 10 weeks, they reported recovery of the multifidus muscle in the group receiving exercise therapy (p = 0.0001); such recovery was not spontaneous in the usual care group. Other outcome measures (pain, disability, range of motion) were not significantly different between the groups at four weeks.

Chok et al. (1999) evaluated the effect of trunk extensor endurance training on patients with acute low back pain with and without pain radiation. They compared exercises plus hot packs plus back care advice with hot packs plus back care advice (n = 54). At three weeks, the exercise group had less pain and better function ($p < 0.05$); at six weeks there were no differences between the groups.

There is no evidence of harm from back exercises.

Additional Evidence

Hides et al. (2001) reported the long-term effects of specific exercises following an initial acute episode of low back pain. They concluded that one year after treatment with lateral multifidus exercises plus usual care, the recurrence rate of low back pain was 30% compared to 84% for the usual care group ($p < 0.001$). At three years post-treatment, the recurrence rates were 35% and 75% respectively ($p < 0.01$).

Key Messages

- McKenzie therapy provides similar pain and function outcomes compared to usual care in acute low back pain. (Level I)
- There is conflicting evidence for the efficacy of back exercises in reducing pain and disability compared to other active and inactive treatments in mixed populations with low back pain. (Level I)
- McKenzie therapy reduces pain and sick leave compared to one back school session, results in similar global improvement compared to manipulation and provision of an educational booklet and provides better functional and pain outcomes compared to flexion exercises in mixed populations with low back pain. (Level I)
- Lateral multifidus muscle exercises reduce recurrences of low back pain compared to usual care in mixed populations with low back pain. (Level II)

Back School

Back schools may provide education, skills and exercises. The content of back schools appears to vary widely; most are led by a trained therapist. Cost effectiveness analyses have not been included in RCTs on back school.

Clinical Evidence (2002) identified one Cochrane Review (van Tulder et al. 2002b, last updated May 1999). Of the 15 RCTs included, two met the criteria for this update; both involved patients with acute low back pain excluding sciatica and other specific conditions (Leclaire et al. 1996; Postacchini et al. 1988). Leclaire et al. (1996) compared usual care (daily hot/cold, massage, ultrasound, TENS, exercises) to usual care plus 90 minutes of back school at zero, one and eight weeks. The back school group performed the exercises better and had greater knowledge however there was no difference in pain, functional disability, time off work or the number or duration of recurrences after one year between the groups. Postacchini et al. (1988) compared back school to spinal manipulation, NSAID, physical therapy, bedrest and placebo for patients with a mean duration of 15 days of low back pain without pain radiation. At three weeks, the manipulation group showed the greatest improvement on a combined pain, disability and mobility score (subjective and objective). At two and six months, there was no difference between the groups.

Four other studies in the van Tulder et al. (2002b) review (Stankovic and Johnell 1990; Lindequist et al. 1984; Bergquist-Ullman and Larsson 1977; Berwick et al. 1989) had mixed populations (acute and chronic pain durations with and without pain radiation). Stankovic and Johnell (1990) tested back school (one lesson) versus McKenzie therapy and reported less pain in the McKenzie therapy group at three and 52 weeks. Bergquist-Ullman and Larsson (1977) compared Swedish back school including four sessions over two weeks versus combined physical therapy versus placebo and found no difference in pain between back school and physical therapy at three and six weeks but significantly less sick leave in the back school group than the placebo group. Lindequist et al. (1984) compared back school with advice plus analgesia as required and reported no significant difference at one, three and six weeks. Berwick et al. (1989) compared usual care versus one four hour back school session versus the back school session plus encouragement strategies. At three, six, 12 and 18 months there was no measurable effect on pain and function in the groups receiving back school compared to usual care.

Additional Evidence

van Tulder et al. (1997b) conducted a systematic review identifying four studies (Bergquist-Ullman and Larsson 1977; Stankovic and Johnell 1990,1995; Lindequist et al. 1984; Morrison et al. 1988). The first three of these studies are covered in other reviews. The Morrison et al. (1988) study compared back school including education and exercise with a control group, however the control group was not described and nor was the duration and nature of the low back pain. The authors report significant improvement in physical strength and mobility compared to the control group.

Hsieh et al. (2002) compared back school to myofascial therapy, joint manipulation and combined myofascial therapy and joint manipulation in 200 subjects with low back pain of between three weeks and six months duration. The study found that back school was no more effective than the three manual treatments at three weeks and six months follow up.

Key Messages

- There is insufficient evidence that back school is more effective in reducing pain compared to active and passive therapies and to placebo in acute low back pain. (Level I)
- There is insufficient evidence that back school is more effective in reducing pain compared to placebo and other treatments in mixed populations with low back pain (Level I, Level II)

Bed Rest

Clinical Evidence (2002) found no evidence that bed rest is better for low back pain, but there is evidence from six systematic reviews that it may be worse than no treatment, advice to stay active, back exercises, physical therapy, spinal manipulation or non-steroidal anti-inflammatory drugs (Bigos et al. 1994; Koes and van den Hoogen 1994; Evans and Richards 1996; Waddell et al. 1997; van Tulder et al. 1997b; Hagen et al. 2002).

The systematic reviews by Koes and van den Hoogen (1994) and Evans and Richards (1996) could not be obtained. The remaining four reviews identified eleven RCTs, ten involving mixed populations. Seven studies (Gilbert et al. 1985; Deyo et al. 1986; Postacchini et al. 1988; Szpalski and Hayez 1992; Wilkinson 1995; Malmivaara et al. 1995; Wiesel et al. 1980) that meet the criteria for this update are described here. Those excluded from this update involved populations with sciatica only (Vroomen et al. 1999; Coomes 1961), a study in which cointerventions were used differently in the comparison groups (Rupert et al. 1985) and a study that compared the effects of traction rather than bed rest (Pal et al. 1986).

Two of the seven studies included for this review compared bed rest to staying active (Malmivaara et al. 1995; Wilkinson 1995). The study by Malmivaara et al. (1995) compared two days of bed rest versus back mobilizing exercises versus advice to maintain ordinary activity as tolerated. Small improvements in pain intensity were seen in the group maintaining activity levels; these were not clinically significant. The study found that function, rate of recovery and sick leave were significantly improved at three and 12 weeks in the stay active group. Wilkinson (1995) found no significant difference in function or rate of recovery between two days of bed rest versus staying active at one week and one month follow up.

Four of the seven studies compared bed rest to other treatments. Gilbert et al. (1985) compared four days bed rest versus four days bed rest plus exercises and education versus exercise and education versus no treatment. Malmivaara et al. (1995) compared two days of bed rest versus ordinary activity plus back mobilising exercises versus ordinary activity in patients with acute low back pain with and without radiation. No significant differences were found in pain or function in either study. Postacchini et al. (1988) analysed a subgroup with acute, non-specific low back pain in a study comparing bed rest with manipulation, NSAIDs, physical therapy (light massage, analgesic currents and diathermy) and placebo and found no difference in pain and function scores. Wiesel et al. (1980) found that bedrest reduced pain by 60% compared to ambulation in a study of male army recruits. Pain was further reduced with concomitant use of analgesics.

Two studies compared durations of bed rest. Deyo et al. (1986) compared seven days with two days of bed rest. No significant differences were found in pain, function or rate of recovery at three and 12 weeks follow up, however, two days of bed rest resulted in significantly less sick leave than seven days of bed rest (p = 0.01). Szpalski and Hayez (1992) compared

the effects of seven days versus three days of bed rest and found no significant difference in pain after two days of follow up.

Adverse effects of bed rest, including joint stiffness, muscle wasting, loss of bone mineral density, decubitus ulcers and venous thromboembolism were reported by Waddell et al. (1997).

Additional Evidence

An additional RCT compared four days of bed rest versus advice to continue normal activity (Rozenberg et al. 2002). The study found no significant difference in pain intensity and functional disability at one week, one month and three months between four days of bed rest and advice to continue with normal activity. The population included those with acute low back pain (the majority with recurrent pain) with no radiation. However, these results are limited by non-blinding, the measurement of outcomes by the investigator, and lack of baseline equivalence between the groups. The study found evidence of potential harm, particularly if bed rest is prescribed for more than four days.

Key Messages

- There is insufficient evidence that bed rest is more effective compared to advice to stay active, back exercises, spinal manipulation, non-steroidal anti-inflammatory drugs or no treatment in mixed populations with low back pain. (Level I, II)
- There is conflicting evidence that bed rest increases disability and rate of recovery compared to staying active in mixed populations with low back pain. (Level I)
- Bedrest for longer than two days increases the amount of sick leave compared to early resumption of normal activity in acute low back pain. (Level I)
- There is evidence that prolonged bed rest is harmful. (Level I)

Cognitive Behavioural Therapy

A cognitive behavioural approach involves helping people achieve their desired goals through specifying the steps required and systematically reinforcing progress. It is critical that the client and therapist work in partnership with shared responsibilities. This approach is often incorporated with exercise and activity restoration interventions (Indahl et al. 1995; Lindstrom et al. 1992; Maher et al. 1999). More complex cases are likely to require cognitive behavioural therapy (CBT), which is a more sophisticated and specialised application of this approach.

Clinical Evidence (2002) reports that CBT versus traditional care or electromyographic biofeedback reduces acute low back pain and disability, based on the results of systematic reviews (Bigos et al. 1994; Evans and Richards 1996; van Tulder et al. 1997b; Turner 1996; van Tulder et al. 2002e) and one RCT (Hasenbring et al. 1999). However, there is limited ability to generalise the findings of these studies to those with acute, non-specific low back pain as a variety of behavioural therapies were compared, a number of the systematic reviews did not differentiate between acute and chronic pain populations or specifically reviewed the chronic literature and some of the included studies involved people with specific conditions (i.e. sciatica). All of the systematic reviews except Evans and Richards (1996) were obtained and reviewed for this update.

The van Tulder et al. (2002e) Cochrane Review specifically addressed chronic low back pain. The van Tulder et al. (1997b) review identified one RCT (Fordyce et al. 1986) involving patients with acute low back pain, however it is unclear whether the pain was non-specific in nature. Groups were randomised to traditional symptom-contingent medical treatment (e.g. 'let pain be your guide') versus time-contingent interventions and restoration of activity independent of pain (referred to as 'behaviour therapy'). No benefit was seen at six weeks, but at 9-12 months the group who had received 'behaviour therapy' was 'less sick' overall, according to a sick-well index combining work, health service use, disability and activity. van Tulder et al. (1997b) concluded that there was no evidence on the effectiveness for CBT based on this one study. Hasenbring et al. (1999) involved a small number (n = 22) of patients with sciatica with a mean duration of symptoms of 13.5 weeks and radiologically proven disc prolapse, therefore it does not meet the inclusion criteria for this update.

The Turner review (1996) identified an additional study (Philips et al. 1991) involving patients with acute low back pain, however there was no description of the review process or study quality and it involved patients with acute neck pain. A symptom-contingent approach ('let pain guide') to returning to previous physical function was compared to graded reactivation, irrespective of pain. The effect of two counselling techniques was also compared; groups were subdivided to receive either behavioural or psychotherapy counselling. The authors concluded there was no significant difference in pain outcomes between the groups at six months, however there was a trend (non-significant) towards earlier return to normal and less risk of persistent pain in those receiving behavioural counselling. The authors also concluded that by three months post-injury, it was possible to predict those at risk of developing chronic pain (Philips et al. 1991).

The Bigos et al. review (1994) did not mention CBT specifically but includes the Fordyce et al. (1986) study in exercise interventions, concluding that patients improved faster when given specific quotas of exercises to perform.

Additional Evidence

The Royal College of General Practitioners' guideline for Acute Low Back Pain (Waddell et al. 2001) included the Fordyce et al. (1986) and Philips et al. (1991) studies in their trials of advice on activity for acute and subacute low back pain and concluded that there was no significant difference in pain at six months.

Two additional randomised controlled trials were identified from the same group evaluating CBT for the prevention of disability (Linton and Ryberg 2001; Linton and Andersson 2000).

Linton and Ryberg (2001) conducted a study involving 35–45 year olds in the general population who had experienced neck, thoracic or low back pain in the past 12 months. Subjects were randomised to receive usual care or six two-hour group sessions of CBT that focused on activation coping strategies. Outcomes were assessed by postal questionnaire at 12 months. The authors concluded that CBT 'produced a significant preventive effect with regard to disability'. However, the study had numerous limitations. The participants had recurrent spinal pain with pain scores of at least seven out of 10 and a history of at least four episodes in the past 12 months, thus limiting generalisability of the results to all cases of acute low back pain. In addition, the distribution of different pain sites between the two study groups was not presented, the study was not blinded, the analysis was not an intention-to-treat (48% of the CBT group dropped out before it started), the key outcome measure labelled as 'disability' was self-reported days of sick leave recalled over the past six months (and not validated) and there was little

difference between the two groups in the numerous psychological and physical measures.

In their study on the prevention of chronic spinal pain, Linton and Andersson (2000) compared an educational pamphlet versus a more extensive weekly package of information on managing pain versus weekly CBT sessions on coping strategies. The study population comprised patients with acute or subacute back and neck pain (duration not defined) with less than three months sick leave in the previous year. All patients perceived they were at risk of developing chronic pain. Sick leave and health care utilisation were the primary outcome measures and at one year, the risk of long-term sick leave was reduced nine-fold in the group receiving CBT compared to the information groups (Relative Risk, 9.3). There was also a significant reduction in physician and physical therapy visits ($p < 0.001$ and $p < 0.01$, respectively). Pain outcomes were not significantly different between the groups. The authors concluded that long-term disability in patients with unresolved acute and subacute pain could be prevented with a CBT intervention.

Firm conclusions cannot be drawn from these studies regarding the efficacy of CBT as an intervention for acute low back pain, however it may prevent chronicity; further evaluation is warranted.

Key Messages

> Cognitive behavioural therapy reduces general disability in the long term compared to traditional care in mixed populations with back pain. (Level I)

> Group cognitive behavioural therapy sessions may reduce sick leave and health care utilisation in the long term compared to general educational information in mixed populations with back pain. (Level II)

> While cognitive behavioural strategies are often included as part of specific interventions for acute low back pain such as exercise and activity restoration, there are no studies on this approach as a single intervention. (No Level I or II studies)

Electromyographic Biofeedback

Clinical Evidence (2002) located one randomised controlled trial (Hasenbring et al. 1999) reporting that risk factor based cognitive behavioural therapy was more effective in relieving pain and preventing chronicity compared to electromyographic (EMG) biofeedback. However, the study involved patients with sciatica, radiologically proven disc protrusion and high psychosocial risk factors and thus did not meet the criteria for this update. While there are several RCTs on the use of EMG biofeedback in patients with chronic low back pain and specific conditions, there are none on the use of this therapy in acute, non-specific low back pain.

Key Message

There are no controlled studies testing the effectiveness of electromyographic biofeedback in acute low back pain. (No Level I or II studies)

Injection Therapy

Five systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b; Nelemans et al. 2002; Koes et al. 1999; Watts and Silagy 1995) were cited in Clinical Evidence (2002). Studies on soft tissue, facet joint and epidural injections are reviewed.

Bigos et al. (1994) located six RCTs on local soft tissue injections (trigger point and ligamentous injections). These studies included predominantly chronic low back pain, did not specify pain duration, addressed a specific syndrome or did not report the duration of pain. Bigos et al. (1994) located five RCTs on facet joint injections that met their criteria; all involved pain of mixed acute and chronic duration or did not specify pain duration. The review identified nine RCTs on epidural injections of steroids, lidocaine and opioids; all involved patients with chronic pain, a mix of acute and chronic pain or specific conditions. Based on the mixed studies, Bigos et al. (1994) concluded there is insufficient evidence to support the use of injection therapy in acute, non-specific low back pain.

The Cochrane Review by Nelemans et al. (2002, last updated in 2001) of injection therapy for subacute and chronic low back pain also distinguished between three injection sites (soft tissue, facet joint and epidural). All of the 21 RCTs included in this review included patients with chronic pain or specific syndromes. The authors concluded that none of the studies produced convincing evidence for or against injection therapy in any site.

van Tulder et al. (1997b) identified one RCT (Mathews et al. 1987) on epidural injections. The study compared epidural steroids with subcutaneous lignocaine injections and found no difference at one month and a small benefit at three months. However, as all included patients had uniradicular neurological deficit, the study did not meet the criteria for this update. Similarly, the studies included in the reviews by Koes et al. (1999) and Watts and Silagy (1995) evaluated epidural steroid injections in patients with sciatica and chronic low back pain.

A review by Deyo (1996) on the use of drug therapy for low back pain identified 15 RCTs on the use of epidural steroid injection; all studies involved subjects with sciatica or chronic low back pain.

Adverse effects of injection therapy are infrequent but potentially serious including headache, fever, subdural penetration, epidural abscess and respiratory depression (Bigos et al. 1994; Nelemans et al. 2002; Koes et al. 1999).

Key Messages

> There is insufficient evidence demonstrating the effectiveness of injection therapy (facet joint, epidural or soft tissue) in the treatment of acute low back pain. (Level I, II)

> Adverse effects of injection therapy are rare but serious. (Level I)

Lumbar Supports

In the Cochrane Review by van Tulder et al. (2002c, last updated 2000) six RCTs tested the use of lumbar supports for the treatment of low back pain. Of these, three (Doran and Newell 1975; Hsieh et al. 1992; Valle-Jones et al. 1992) involved non-specific low back pain, however the duration of pain was a mixture of acute and chronic. One other study (Penrose et al. 1991) comparing lumbar supports with no intervention did not describe the duration of pain. No studies on acute, non-specific low back pain were located.

Doran and Newell (1975) compared use of a corset for three weeks versus manipulation twice weekly, therapy comprising any treatment except manipulation twice weekly and regular use of paracetamol ($n = 456$). Hsieh et al. (1992) compared use of a corset with metal stays for three weeks versus spinal manipulation three times a week, soft tissue massage three times a week and use of a TENS unit for eight hours a day ($n = 164$). Valle-Jones et al. (1992) compared use of an elasticated back support with advice on rest and lifestyle ($n = 216$).

Doran and Newell (1975) Hsieh et al. (1992) and reported no significant difference in pain outcomes between lumbar supports and other treatments. Doran and Newell (1975) found no difference in overall improvement between

interventions. Hsieh et al. (1992) found no difference in functional outcomes between groups at four weeks follow up. Valle-Jones et al. (1992) reported that use of lumbar supports made a significant difference in pain, overall improvement and the ability to return to work at three weeks compared to advice on rest and lifestyle, however there was no data on compliance.

Prolonged use of lumbar supports has been associated with harmful effects such as decreased muscle strength, however, there is no clear evidence that this applies specifically to low back pain. A false sense of security, skin irritation and general discomfort have been reported (Bigos et al. 1994).

Key Messages

> There are no controlled studies on the effect of lumbar supports in acute low back pain. (No Level I or II studies)

> There is insufficient evidence that lumbar supports are effective in reducing pain compared to spinal manipulation, exercises, massage, TENS and simple analgesia in mixed populations with low back pain. (Level I)

Massage

Massage is widely utilised as a form of therapy for acute and chronic low back pain and involves stroking or rubbing the soft-tissues with the hands or a mechanical device.

Clinical Evidence (2002) reported two systematic reviews (Furlan et al. 2000; Ernst 1999) that found no difference with massage versus spinal manipulation or transcutaneous electrical stimulation (TENS) in pain, functional status or mobility outcomes but a possible beneficial effect compared to placebo.

Three RCTs were included in both reviews; all had methodological flaws. The study by Godfrey et al. (1984) was the only one to specifically involve patients (n = 90) with acute low back pain (less than two weeks duration). The results showed that all three groups (massage, spinal manipulation and electrical stimulation) improved significantly from baseline and no differences were seen between them in the two to three week follow up period. Hoehler et al. (1981) included acute and chronic patients (n = 95) with no detail of pain radiation. The study compared spinal manipulation with soft tissue massage and found that manipulation was superior immediately after the end of the first session for pain and straight-leg raising but this effect was not maintained at the end of the treatment period. At follow up after three weeks there was no difference between groups; both had improved significantly from baseline. The Hsieh et al. (1992) study involved patients with subacute and chronic pain (three weeks to six months duration) who were treated for three weeks with spinal manipulation, corset, massage and TENS. There were no significant differences among groups in relation to pain, however the manipulation group demonstrated significantly better function scores compared with the massage group.

The Furlan et al. Cochrane Review (2000) reported two additional RCTs (Pope et al. 1994; Melzack et al. 1983). The Pope et al. study (1994) involved the same study population as the Hsieh et al. (1992) study. Melzack et al. (1983) compared TENS and massage in a purely chronic low back pain population. A study by Konrad et al. (1992) included in the Ernst review involved purely chronic low back pain.

No harms were reported.

Additional Evidence

The update of the Cochrane Review by Furlan et al. (2002) identified four additional RCTs (Cherkin et al. 2001; Franke et al. 2000; Hernandez-Reif et al. 2001; Preyde 2000). Both the Franke et al. (2000) and the Hernandez-Reif et al. (2001) studies involved chronic pain patients (duration more than one year and six months, respectively).

Cherkin et al. (2001) included participants with pain lasting from six weeks to more than a year (61%). Therapeutic massage, traditional Chinese acupuncture and a self-educational strategy involving a book and videotapes were compared. After 10 weeks of treatment the massage group had less severe symptoms (pain, numbness, tingling) and less dysfunction than the self-education group (p = 0.01, p < 0.001 respectively) and less dysfunction than the acupuncture group (p = .01). At one year, massage had a greater effect on symptoms (p = 0.002) and function (p = 0.051) compared to acupuncture, however there was no significant difference in symptoms or function compared to self-education at one year (p = 0.42; p = 0.97 respectively).

Preyde (2000) compared comprehensive massage therapy (a package comprising soft-tissue manipulation, stretching exercises and posture education) versus soft-tissue manipulation (massage) only versus stretching exercises and posture education versus sham laser therapy. The study involved patients with low back pain ranging from one week to 8 months duration (average 13 weeks duration). It does not specify whether patients had pain radiation, however those with significant pathology were excluded. This appears to be the only placebo-controlled trial evaluating massage as a monotherapy. Massage alone reduced pain more effectively compared to placebo (achieving statistical significance) and to exercise and education both immediately post-treatment and at one month. The combined massage, exercise and education group achieved significantly lower pain intensity and quality scores after treatment and at one month compared to the other groups (p < 0.001 and p = 0.006, respectively).

Furlan et al. (2002) state that it cannot be concluded that massage is effective for acute low back pain, but there is moderate evidence that massage improves pain intensity and pain quality in the subacute period (pain duration of four to 12 weeks) compared to placebo especially when combined with exercise. The review found that these effects were similar to the effects for exercise and manipulation.

Another RCT (Hsieh et al. 2002) that was not included in the Furlan et al. review (2002) assessed the efficacy of massage (myofascial therapy) versus joint manipulation versus combined massage and joint manipulation versus back school that comprised education and a supervised home exercise program. Subjects had a mix of acute, subacute and chronic low back pain (more than three weeks but less than six months). Allowed co-interventions included paracetamol and NSAIDs. The authors concluded that combined and monotherapies (massage with and without manipulation) are equally as effective and not superior to back school at three weeks and six months follow-up.

Key Messages

> There are no controlled studies for massage therapy in acute low back pain. (No Level I or II studies)

> Massage is superior to placebo (sham laser) and acupuncture in mixed populations with low back pain. (Level I, II)

> Massage provides similar effect to back schools (involving exercise and education), corsets and TENS in mixed populations with low back pain. (Level I, II)

> There is conflicting evidence of the effect of massage compared to manipulation and education in mixed populations with low back pain. (Level I, II)

Multi-disciplinary Treatment in the Workplace

Clinical Evidence (2002) reported that multi-disciplinary treatment including a visit to the workplace improved return to work compared with usual care. The conclusion was based on one Cochrane Review (Karjalainen et al. 2002, last updated September 1999) of two RCTs (Loisel et al. 1997; Lindstrom et al. 1992a,b) comparing physician consultation plus either a psychological, vocational or social intervention or a combination of these approaches in working age people with low back pain.

Lindstrom et al. (1992a,b) studied the effect of a graded activity program plus a workplace visit versus usual care in a group of factory workers who had been on eight weeks of sick leave for low back pain. There was no detail of the duration of low back pain. Loisel et al. (1997) compared occupational intervention versus a clinical intervention versus combined occupational and clinical intervetion versus usual care. The study comprised subjects with low back or thoracic spinal pain with four weeks of sick leave in the preceding year.

The review (Karjalainen et al. 2002) found evidence of positive effects on return to work (return to work was approximately 7–8 weeks earlier in the treatment group versus the control group) and significant improvement in subjective disability from the combined approaches (i.e. improvement of 10 points on the Oswestry scale in the Loisel study and 1.2 points on the Waddell et Main scale in the Lindstrom study). No major differences were identified in other outcome measures.

No harms were reported and the costs of these programs were not evaluated.

Key Messages

> There are no controlled studies on the effect of multi-disciplinary treatment in the workplace in acute low back pain (No Level I or II studies)

> Multi-disciplinary treatment in the workplace improves return to work and subjective disability compared to usual care in mixed populations with low back pain. (Level I, II)

Topical Treatments

One RCT (Stam et al. 2001) was located that evaluated the effect of spiroflar homeopathic gel compared with cremol capsici. There was no control group in this study and no significant difference in pain scores after one week of treatment. Harms of these treatments were not reported.

Key Message

There is insufficient evidence for the effectiveness of spiroflar homeopathic gel or cremol capsici for treatment of acute low back pain. (Level II)

Traction

Clinical Evidence (2002) found conflicting evidence on the effects of traction, citing studies by van der Heijden et al. (1995), Evans and Richards (1996) and van Tulder et al. (1997b). The Evans and Richards (1996) review could not be obtained.

The van der Heijden et al. (1995) review located 14 RCTs on the use of lumbar traction. None of these studies involved acute, non-specific populations; three included mixed populations (pain duration and radiation), however all had methodological flaws. Mathews et al. (1987) compared continuous traction with infrared heat and found no significant difference in patient global estimate of improvement at two weeks. Pal et al. (1986) compared continuous bed traction to placebo and found no significant difference in pain outcomes at three weeks. Larsson et al. (1980) compared the effect of traction combined with corset use and bedrest versus corset use and bedrest and found that there was a statistically significant ($p < 0.05$) improvement in pain in the group receiving traction at one week but not at three weeks. The majority (73%) of patients ($n = 82$) in this study had chronic pain and 89% had sciatica.

The van Tulder et al. (1997b) review did not identify additional studies on acute, non-specific low back pain or studies involving mixed populations.

Bigos et al. (1994) note that the potential harms of traction relate to prolonged bed rest (i.e. loss of muscle tone, thrombophlebitis and bone demineralisation).

Key Messages

> There are no controlled studies on the effect of traction for acute low back pain. (No Level I or II studies)

> There is insufficient evidence that traction is effective compared to placebo and compared to other treatments in mixed populations with low back pain. (Level I)

> Adverse effects from traction have been reported, including reduced muscle tone, bone demineralisation, thrombophlebitis. (Level I)

Transcutaneous Electrical Nerve Stimulation (TENS)

Transcutaneous electrical nerve stimulation (TENS) is a non-invasive therapy involving the delivery of electrical stimulation to peripheral nerves via surface electrodes (Milne et al. 2002). Clinical Evidence (2002) found insufficient evidence on the effects of TENS in the treatment of acute low back pain based on systematic reviews by Bigos et al. (1994), van Tulder et al. (1997b) and a Cochrane Review by Milne et al. (2002, last updated 2001). The latter is a review of trials involving chronic low back pain only and therefore has been excluded from this update.

Bigos et al. (1994) reported eight RCTs evaluating TENS. One of these studies involved patients with acute low back pain however there is no indication whether the pain is non-specific (Hackett et al. 1988). This study compared electroacupuncture (likened to TENS) and placebo analgesia versus placebo electroacupuncture and paracetamol ($n = 37$). Pain was significantly less ($p > 0.01$) at six weeks in the group receiving electroacupuncture compared to those receiving paracetamol. No harms were reported.

In addition to the Hackett et al. (1988) study, the van Tulder et al. review (1997b) included a study by Herman et al. (1994). This study included mainly male patients with acute low back pain; there was no description of whether the pain was non-specific. TENS plus exercises was compared with placebo electrical stimulation plus exercises on groups equivalent at baseline. At four weeks follow up, the control group had significantly less pain and disability indicating that TENS in addition to exercise did not result in better outcomes than exercise alone.

Additional Evidence

Hurley et al. (2001b) compared interferential therapy (IFT) placed in painful areas of the back plus a back book versus IFT in the spinal nerve area plus a back book versus a back book

only. A trend toward improvement in functional disability scores was noted in the group receiving IFT over the spinal nerve area compared with the other two groups (p = 0.030). However this study involved a mixture of patients with chronic and acute low back pain, a small sample size and the results were of borderline significance statistically.

A recent review by Pengel et al. (2002) identified the study by Herman et al. (1994) in addition to one study reported by both Hsieh et al. (1992) and Pope et al. (1994). Pengel et al. (2002) combined data from both studies to generate effect sizes for pain in relation to TENS versus massage (–0.3; 95%CI –0.8, 0.3) and TENS versus corset use (–0.2; 95%CI –0.8, 0.4). The pain duration in this study was a mixture of acute and chronic (three weeks to six months).

Key Messages

> There are no controlled studies on the effect of TENS in acute low back pain. (No Level I or II studies)

> There is insufficient evidence for the effectiveness of TENS compared to exercises, back books, massage, corset use and simple analgesia in mixed populations with low back pain. (Level I, II)

>Economic Implications

A search of the Cochrane Library revealed three randomised controlled trials that included a cost effectiveness analysis of interventions for acute low back pain. Malmivaara et al. (1995) compared three groups: rapid mobilisation and back extensions; usual activities avoiding bed rest; and bed rest. The costing analysis suggested that undertaking usual activities and avoiding bed rest was the most 'economical' approach, although the Cochrane reviewer suggested the results should be interpreted with caution as there were only 50 to 60 people per group, there was a large loss to follow up and the outcomes were self-reported.

Cherkin et al. (1998) compared the costs and benefits of an educational booklet with spinal manipulation (short lever, high velocity thrust directed at a manipulable lesion) and with McKenzie therapy. At four weeks the manipulation group had less pain and at 12 months there was very little difference between the three groups. It was concluded that as first line therapy, it was unlikely to be cost effective to refer for manipulation or McKenzie therapy.

Moffet et al. (1999) evaluated cost, preferences and clinical outcomes between a community-based exercise program (comprised of four therapist-led one hour exercise classes over four weeks, including education, stretching, strengthening) and usual primary care management. The exercise program was more clinically effective and more cost effective than usual care, with lower direct and indirect costs (as measured by days off work).

Key Message

Published data is very limited; however there is some evidence that advice to maintain usual activities, provision of an education booklet and community-based exercises appear to be cost effective first line interventions for acute low back pain. (Level II)

>References

Abenheim L, Rossignol M, Gobeille D, Bonvalot Y, Fines P, Scott S (1995). The prognostic consequences in the making of the initial diagnosis of work related back injuries. Spine, 20: 791–795.

AGREE Collaboration (2001). Appraisal of Guidelines for Research and Evaluation (AGREE) Instrument. URL: http://www.agree collaboration.org

Amlie E, Weber H, Holme I (1987). Treatment of acute low back pain with piroxicam: results of a double-blind placebo-controlled trial. Spine, 12: 473–476.

Andersson GB, Lucente T, Davis AM et al. (1999). A comparison of osteopathic spinal manipulation with standard care for patients with low back pain. New England Journal of Medicine, 341: 1426–1431.

Andersson G, Bogduk N et al. (1989). Muscle: clinical perspectives. In: Frymoyer JW, Gordon SL (eds). New Perspectives on Low Back Pain. American Academy of Orthopaedic Surgeons: Park Ridge, Illinois. pp 293–334.

Anonymous (2001a). Growing body of evidence on massage as a treatment for low back pain: recent studies and systematic reviews. BackLetter, 16: 51.

Anonymous (2001b). Opiates not a first option. BackLetter, 16: 63.

Arbus L, Fajadet B, Aubert D, Morre M, Goldberger E (1990). Activity of tetrazepam (myolostan) in low back pain: a double blind trial versus placebo. Clinical Trials Journal, 27: 258–267.

Assendelft WJ, Bouter LM, Knipschild PG (1996). Complications of spinal manipulation: a comprehensive review of the literature. Journal of Family Practice, May; 42: 475–480.

Australian Institute of Health and Welfare (2000). Australia's health 2000: the seventh biennial health report of the Australian Institute of Health and Welfare. AIHW: Canberra.

Bakshi R, Thumb N, Broll H, Klein G, Mayrhofer F, Rainer F, Singer F, Smolen J (1994). Treatment of acute lumbosacral back pain with diclofenac resinate: results of a double blind comparative trial versus piroxicam. Drug Investigation, 8: 288–293.

Barrata RR (1982). A double-blind study of cyclobenzaprine and placebo in the treatment of acute musculoskeletal conditions of the low back. Current Therapeutic Research, 32: 646–652.

Basmajian JV (1989). Acute back pain and spasm: a controlled multicentre trial of combined analgesic and antispasm agents. Spine, 14: 438–439.

BenDebba M, Torgerson WS, Boyd RJ, Dawson EG, Hardy RW, Robertson JT, Sypert GW, Watts C, Long DM (2002). Persistent low back pain and sciatica in the United States: treatment outcomes. Journal of Spinal Disorders, 15: 2–15.

Bergquist-Ullman M, Larsson U (1977). Acute low back pain in industry: a controlled prospective study with special reference to therapy and confounding factors. Acta Orthopedica Scandinavia, 170 (suppl): 111–117.

Berry H, Hutchinson DR (1988). Tizanidine and ibuprofen in acute low back pain: results of a double-blind multicentre study in general practice. Journal of Internal Medicine Research, 16: 83–91.

Berwick DM, Budman S, Feldstein M (1989). No clinical effect of back schools in an HMO. A randomised prospective trial. Spine, 14: 339–344.

Bigos S, Bowyer O, Braen G, et al. (1994). Acute low back problems in adults. Clinical Practice Guideline no.14. AHCPR Publication No. 95–0642. Agency for Health Care Policy and Research, Public Health Service, US, Department of Health: Rockville MD.

Blomberg S, Hallin G, Grann K, Berg E, Sennerby U (1994). Manual therapy with steroid injections — a new approach to treatment of low back pain: a controlled multicenter trial with an evaluation by orthopedic surgeons. Spine, 19: 569–577.

Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW (1990). Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. Journal of Bone and Joint Surgery, 72A: 403–408.

Bogduk N, Lord SM (1997). Commentary on: a prospective study of centralisation of lumbar and referred pain: a predictor of

symptomatic discs and anular competence. Pain Medicine Journal Club, 3: 246–248.

Bogduk N, McGuirk B (2002). Medical management of acute and chronic low back pain: an evidencebased approach. In: Pain Research and Clinical Management. Elsevier: Amsterdam.

Borenstein DG (2001). Epidemiology, etiology, diagnostic evaluation and treatment of low back pain. Current Opinion in Rheumatology, 13: 128–134.

Borenstein DG, Lacks S, Wiesel SW (1990). Cyclobenzaprine and naproxen versus naproxen alone in the treatment of acute low back pain and muscle spasm. Clinical Therapeutics, 12: 125–131.

Boyles WF, Glassman JM, Soyka JP (1983). Management of acute musculoskeletal conditions: thoracolumbar strain or sprain. A double-blind evaluation comparing the efficacy and safety of carisoprodol with diazepam. Today's Therapeutic Trends, 1: 1–16.

Bradley JD, Brandt KD, Katz BP, Kalsinki LA, Ryan SI (1991). Comparison of an anti-inflammatory dose of ibuprofen and acetaminophen in the treatment of patients with osteoarthritis of the knee. New England Journal of Medicine, 325: 87–91.

Brockow T, Dillner A, Franke E, Resch KL (2001). Analgesic effectiveness of subcutaneous carbon dioxide insufflations as an adjunct treatment in patients with non-specific neck or low back pain. Complementary Therapies in Medicine, 9: 64–76.

Brodin H (1984). Cervical pain and mobilization. International Journal of Rehabilitation Research, 7:190–191.

Brown FL, Bodison S, Dixon J, Davis W, Nowoslawski J (1986). Comparison of diflunisal and acetaminophen with codeine in the treatment of initial or recurrent acute low back pain. Clinical Therapeutics, 9 (suppl c): 52–58.

Browning R, Jackson JL, O'Malley PG (2001). Cyclobenzaprine and back pain: a meta-analysis. Archives of Internal Medicine, 161: 1613–1620.

Burton AK, Waddell G, Tillotson KM, Summerton N (1999). Information and advice to patients with back pain can have a positive effect. A randomized controlled trial of a novel educational booklet in primary care. Spine, 24: 2484–2491.

Calin A, Porta J, Fries JF, Schurman DJ (1977). Clinical history as a screening test for ankylosing spondylitis. Journal of the American Medical Association, 237: 2613–2614.

Cardon GM, De CD, De BM (2002). Back education efficacy in elementary schoolchildren: a 1-year follow-up study. Spine, 27: 299–305.

Carey TS, Garrett J, Jackman A, McLaughlin C, Fryer J, Smucker DR (1995). The outcomes and costs of care for acute low back pain among patients seen by primary care practitioners, chiropractors, and orthopaedic surgeons. New England Journal of Medicine, 333: 913–917.

Casale R (1988). Acute low back pain: symptomatic treatment with a muscle relaxant drug. Clinical Journal of Pain, 4: 81–88.

Cherkin DC, Deyo RA, Street JH, Hunt M, Barlow W. (1996) Pitfalls of patient education. Limited success of a program for back pain in primary care. Spine, 21: 345–55.

Cherkin DC, Deyo RA, Battie M, Street J, Barlow W (1998). A comparison of physical therapy, chiropractic manipulation, and provision of an educational booklet for the treatment of patients with low back pain. New England Journal of Medicine, 339: 1021–1029.

Cherkin DC, Eisenberg D, Sherman KJ, Barlow W, Kaptchuk TJ, Street J, Deyo RA (2001). Randomized trial comparing traditional Chinese medical acupuncture, therapeutic massage, and self-care education for chronic low back.pain. Archives of Internal Medicine, 161: 1081–1088.

Chok B, Lee R, Latimer J, Tan SB (1999). Endurance training of the trunk extensor muscles in people with subacute low back pain. Physical Therapy, 79: 1032–1042.

Chrubasik S, Kunzel O, Model A, Conradt C, Black A (2001). Treatment of low back pain with a herbal or synthetic antirheumatic: a randomised controlled study. Willow bark extract for low back pain. Rheumatology UK, 40: 1388–1393.

Clinical Evidence. June 2002; 7: 1018–1031. BMJ: London.

Colberg K, Hettich M, Sigmund R, Degner FL (1996). The efficacy and tolerability of an 8-day administration of intravenous and oral meloxicam: a comparison of intramuscular and oral diclofenac in patients with acute lumbago. Current Medical Research Opinion, 13: 363–377.

Coomes NE (1961). A comparison between epidural anaesthesia and bed rest in sciatica. British Medical Journal, Jan: 20–24.

Cooperstein R, Perle SM, Gatterman MI, Lantz C, Schneider MJ (2001). Chiropractic technique procedures for specific low back conditions: characterising the literature. Journal of Manipulative and Physiological Therapeutics, 24: 407–424.

Coste J, Delecoeuillerie G, Cohen de Lara A, Le Parc M, Paolaggi JB (1994). Clinical course and prognostic risk factors in acute low back pain: an inception cohort study in primary care practice. British Medical Journal, 308: 577–580.

Coyer AB, Curwin I (1955). Low back pain treated by manipulation. British Medical Journal, 1: 705–707.

Coxhead CE, Inskip H, Meade TW, North WRS, Troup JDG (1981). Multi-centre trial of physiotherapy in the management of sciatic symptoms. Lancet, 1: 1065–1068.

Croft PR, Rigby AS (1994). Socioeconomic influences on back problems in the community in Britain. Journal of Epidemiology and Community Health, Apr 48: 166–70.

Croft PR, Macfarlane GJ, Papageorgiou AC, Thomas E, Silman AJ (1998). Outcome of low back pain in general practice: a prospective study. British Medical Journal, 316: 1356–1359.

Dapas F, Hartmen SF, Martinez L, et al. (1985). Baclofen for the treatment of acute low back syndrome: a double-blind comparison with placebo. Spine, 10: 345–349.

Davies JR, Gibson T, Tester L (1979). The value of exercises in the treatment of low back pain. Rheumatology and Rehabilitation, 18: 243–247.

de Craen AJ, Di Giulio G, Lampe-Schoenmaeckers JE, Kessels AG, Kleijnen J (1996) Analgesic efficacy and safety of paracetamol-codeine combinations versus paracetamol alone: a systematic review. British Medical Journal, 313: 321–325.

Delitto A, Cibulka MT, Erhard RE, Bowling RW, Tenhula JH (1992). Evidence for use of an extension-mobilisatoin category in acute low back syndrome: a prescriptive validation pilot study. Physical Therapy, 73: 216–223.

Del-Mar CB, Glasziou PP, Spinks AB, Sanders SL (2001). Is laser treatment effective and safe for musculoskeletal pain? Medical Journal of Australia, 175: 169.

Deyo RA (1996). Drug therapy for back pain: which drugs help which patients? Spine, 21: 2840–2850.

Deyo RA, Diehl AK (1988). Cancer as a cause of back pain: frequency, clinical presentation and diagnostic strategies. Journal of General Internal Medicine, 3: 230–238.

Deyo RA, Diehl AK (1986). Lumbar spine films in primary care: current use and effects of selective ordering criteria. Journal of General Internal Medicine, 1: 20–25.

Deyo RA, Diehl AK, Rosenthal M (1986). How many days of bed rest for acute low back pain? New England Journal of Medicine, 1986, 315: 1064–1070.

Deyo RA, Rainville J, Kent DL (1992). What can the history and physical examination tell us about low back pain? Journal of the American Medical Association, 268: 760–765.

Deyo RA, Tsui-Wu YJ (1987). Descriptive epidemiology of low back pain and its related medical care in the United States. Spine, 12: 264–268.

Dionne CE, Von Km, Koepsell TD, Deyo RA, Barlow WE, Checkoway H (2001). Formal education and back pain: a review. Journal of Epidemiology and Community Health, 55: 455–468.

Donelson R, Aprill C, Medcalf R, Grant W (1997). A prospective study of centralisation of lumbar and referred pain. Spine, 33: 1115–1122.

Doran DML, Newell DJ (1975). Manipulation in treatment of low back pain: a multicentre study. British Medical Journal, 2: 161–164.

Dreyfuss P, Dreyer S, Griffin J, Hoffman J, Walsh N (1994). Positive sacroiliac screening tests in asymptomatic adults. Spine, 19: 1138–1143.

Dreyfuss P, Michaelsen M, Pauza K, McLarty J, Bogduk N (1996). The value of history and physical examination in diagnosing sacroiliac joint pain. Spine, 21: 2594–2602.

Drezner JA, Herring SA (2001). Managing low back pain. The Physician and Sportsmedicine, 29: 37–43.

El-Farhan N, Busuttil A (1997). Sudden unexpected deaths from ruptured abdominal aortic aneurysms. Journal of Clinical and Forensic Medicine, 4: 111–116

Ernst E (2000). Does spinal manipulation have specific treatment effects? Family Practice, 17: 554–556.

Ernst E (1999). Massage therapy for low back pain: a systematic review. Journal of Pain and Symptom Management, 17: 65–69.

Ernst E, Harkness E (2001). Spinal manipulation: a systematic review of sham-controlled double blind randomised clinical trials. Journal of Pain and Symptom Management, 22: 879.

Ernst E, White A (1997). Life-threatening adverse reactions after acupuncture? A systematic review. Pain, 71: 123–126.

Ernst E, White A (1998). Acupuncture for back pain: a meta-analysis of randomized controlled trials. Archives of Internal Medicine, 158; 20: 2235–2241.

Evans C, Gilbert JR, Taylor W, Hildebrand A (1987). A randomised controlled trial of flexion exercises, education, and bed rest for patients with acute low back pain. Physiotherapy Canada, 39: 96–101.

Evans G, Richards S (1996). Low Back Pain: An Evaluation of Therapeutic Interventions. Health Care Evaluation Unit, University of Bristol: Bristol.

Faas A (1996). Exercises: which ones are worth trying, for which patients and when? Spine, 21: 2874–2879.

Faas A, Chavannes AW, van Eijk JThM, Gubbels JW (1993). A randomised placebo-controlled trial of exercise therapy in patients with acute low back pain. Spine, 18: 1388–1395

Fairbank JCT, Park WM, McCall IW, O'Brien JP (1981). Apophyseal injections of local anaesthetic as a diagnostic aid in primary low back pain syndromes. Spine, 6: 598–605.

Farrell JP, Twomey LT (1982). Acute low back pain. Comparison of two conservative treatment approaches. Medical Journal of Australia, 1: 160–164.

Feinstein B, Langton JNK, Jameson RM, Schiller F (1954). Experiments on pain referred from deep somatic tissues. Journal of Bone and Joint Surgery, 35A: 981–987.

Fitz-Ritson D (2001). Lasers and their therapeutic application in chiropractic. Journal of the Canadian Chiropractic Association, 45: 26–34.

Fordyce WE, Brockway JA, Bergman JA, Spengler D (1986). Acute back pain: a control group comparison of behavioural vs traditional management methods. Journal of Behavioral Medicine, 9: 127–140.

Frank AO, DeSouza LH (2001). Conservative management of low back pain. International Journal of Clinical Practice, 55: 21–31.

Franke A, Gebauer S, Franke K, Brockow T (2000). Acupuncture massage vs Swedish massage and individual exercise vs group exercise inlow back pain sufferers — a randomised controlled clinical trial in a 2x2 factorial design. Forsch Komplementarmed Klass Naturheilkd, 7: 2860293

Fransen M, Woodward M, Norton R, Coggan C, Dawe M, Sheridan N (2002). Risk factors associated with the transition from acute to chronic occupational back pain. Spine 27: 92–98.

Frazier LM, Carey TS ,Lyles MF, Khayrallah MA, McGaghie WC (1989). Selective criteria may increase lumbosacral spine roentgenogram use in acute low-back pain. Archives of Internal Medicine, 149: 47–50.

Fritz JM, George SZ, Delitto A (2001). The role of fear-avoidance beliefs in acute low back pain: relationships with current and future disability and work status. Pain, 94: 7–15.

Fritz JM, Wainner RS, Hicks GE (2000). The use of nonorganic signs and symptoms as a screening tool for return to work in patients with acute low back pain. Spine, 25: 1925–1931.

Fukui S, Ohseto K, Shiotani M, Ohno K, Karasawa H, Naganuma Y (1997). Distribution of referred pain from the lumbar zygapophyseal joints and dorsal rami. Clinical Journal of Pain, 13: 303–307.

Furlan AD, Brosseau L, Imamura M, Irvin E (2002). Massage for low back pain. In: The Cochrane Library; Issue 2. Oxford: Update Software (updated 2002).

Garfin JM, Garfin SR (2002). A quick guide to exercise as acute therapy. Consultant, 42: 348–353.

Garvey A, Marks MR, Wiesel SW (1989). Prospective, randomized, double-blind evaluation of trigger-point injection therapy for low-back pain. Spine, 14: 962–964.

Gibson T, Grahame R, Harkenss J, Woo P, Blagrave P, Hills R (1985). Controlled comparison of shortwave diathermy with osteopathic treatment in non-specific low back pain. Lancet, 2: 1258–1261.

Gilbert JR, Taylor DW, Hildebrand A, Evans C (1985). Clinical trial of common treatments for low back pain in family practice. British Medical Journal, 291: 791–794.

Glomsrod B, Lonn JH, Soukup MG, Bo K, Larsen S (2001). Active back school, prophylactic management for low back pain: three year follow up of a randomised controlled trial. Journal of Rehabilitation Medicine, 33: 26–30.

Glover JR (1960). Back pain and hyperaesthesia. Lancet, 1: 1165–1168.

Glover JR, Morris JG, Khosla T (1974). Back pain: a randomised clinical trial of rotational manipulation of the trunk. British Journal of Industrial Medicine, 31: 59–64.

Godfrey CM, Morgan PP, Schatzker J (1984). A randomized trial of manipulation for low-back pain in a medical setting. Spine; 9: 301–304.

Gran JT (1985). An epidemiological survey of the signs and symptoms of ankylosing spondylitis. Clinical Rheumatology, 4: 161–169.

Hackett GI, Seddon D, Kaminski D (1988). Electroacupuncture compared with paracetamol for acute low back pain. Practitioner, 232: 163–164.

Hadler NM, Curtis P, Gilings DB, Stinnett S (1987). A benefit of spinal manipulation as adjunctive therapy for acute low back pain: a stratified controlled trial. Spine, 12: 703–705.

Hagen EM, Eriksen HR, Ursin H (2000). Does early intervention with a light mobilization program reduce long-term sick leave for low back pain? Spine, 25: 1973–1976.

Hagen KB, Hilde G, Jamtvedt G, Winnem MF (2002). Bed rest for acute low back pain and sciatica. In: The Cochrane Library, Issue 2. Oxford: Update Software. pp 83A: 789.

Hagen KB, Hilde G, Jamtvedt G, Winnem MF, Pfeifer BA (2001). Review: Bed rest is not effective for acute LBP or sciatica. Journal of Bone and Joint Surgery, 83: 789.

Haigh R (2001). The rehabilitation of spinal pain in the elderly patient. Reviews in Clinical Gerontology, 11: 277–283.

Haijiao W, Koti M, Smith FW, Wardlaw D (2001). Diagnosis of lumbosacral nerve root anomalies by magnetic resonance imaging. Journal of Spinal Disorders, 14: 143–149.

Haldeman S, Rubinstein SM (1992). Cauda equina syndrome in patients undergoing manipulation of the lumbar spine. Spine, 17: 1469–1473.

Halpin SFS, Yeoman L, Dundas DD (1991). Radiographic examination of the lumbar spine in a community hospital: an audit of current practice. British Medical Journal, 303: 813–815.

Hasenbring M, Ulrich HW, Hartmann M, Soyka D (1999). The efficacy of a risk factor-based cognitive behavioral intervention and electromyographic biofeedback in patients with acute sciatic pain: an attempt to prevent chronicity. Spine, 24: 2525–2535.

Hawk C, Azad A, Phongphua C, Long CR (1999). Preliminary study of the effects of a placebo chiropractic treatment with sham adjustments. Journal of Manipulative and Physiological Therapeutics, 22: 436–443.

Hazard RG, Reid S, Haugh LD, McFarlane G (2000). A controlled trial of an educational pamphlet to prevent disability after occupational low back injury. Spine, 25: 1419–23.

Helliwell PS, Cunliffe G (1987). Manipulation in low back pain. The Physician, April: 187–188.

Hemmila HM, Keinanen-Kiukaanniemi SM, Levoska S, Puska P (2002). Long-term effectiveness of bone-setting, light exercise therapy, and physiotherapy for prolonged back pain: a randomized controlled trial. Journal of Manipulative and Physiological Therapeutics, 25: 99–104.

Hemmila HM (2002). Quality of life and cost of care of back pain patients in Finnish general practice. Spine, 27: 647–653.

Henry D, Lim LLY, Rodriguez LAG, Gutthann SP, Carson JL et al. (1996). Variability in the risk of gastrointestinal complications with individual non-steroidal anti-inflammatory drugs: results of a collaborative meta-analysis. British Medical Journal, 312: 1563–1566.

Herman E, Williams R, Stratford P, Fargas-Babjak A, Trott M (1994). A randomised controlled trial of transcutaneous electrical nerve stimulation (CODETRON) to determine its benefits in a rehabilitation program for acute occupational low back pain. Spine, 19: 561–568.

Hernandez-Reif M, Field T, Krasnegor J, Theakston H (2001). Lower back pain is reduced and range of motion increased after massage therapy. International Journal of Neuroscience, 106: 131–145.

Hides JA, Jull GA, Richardson CA (2001). Long term effects of specific stabilizing exercises for first episode low back pain. Spine, 26: 243–248.

Hides JA, Richardson CA, Jull GA (1996). Multifidus muscle recovery is not automatic after resolution of acute first episode low back pain. Spine, 21: 2763–2769.

Hilde G, Hagen KB, Jamtvedt G, Winnem M (2002). Advice to stay active as a single treatment for low back pain and sciatica. In: The Cochrane Library; Issue 2. Oxford: Update Software (updated 2002).

Hindle TH (1972). Comparison of carisoprodol, butabarbital and placebo in treatment of the low back syndrome. Calif Med, 117: 7–11.

Hoehler FK, Tobis JS, Buerger AA (1981). Spinal mnipulation for low back pain. Journal of the American Medical Association, 245: 1835–1838.

Hollenberg GM, Beattie PF, Meyers SP, Weinberg EP, Adams MJ (2002). Stress reactions of the lumbar pars interarticularis: the development of a new MRI classification system. Spine, 27: 181–186.

Hollingworth W, Todd CJ, King H, Males T, Dixon AK, Karia KR, Kinmonth AL (2002). Primary care referrals for lumbar spine radiography: diagnostic yield and clinical guidelines. British Journal of General Practice, 52: 475–480.

Horneij E, Hemborg B, Jensen I, Ekdahl C (2001). No significant differences between intervention programs on neck, shoulder and low back pain: a prospective randomised study among home-care personnel. Journal of Rehabilitation Medicine, 33: 170–176.

Hosie GAC (1993). The topical NSAID, felbinac, versus oral ibuprofen: a comparison of efficacy in the treatment of acute lower back injury. British Journal of Clinical Research, 4: 5–17.

Hsieh CY, Adams AH, Tobis J, Hong CZ, Danielson C. Platt K, Hoehler F, Reinsch S, Rubel A (2002). Effectiveness of four conservative treatments for subacute low back pain: a randomised clinical trial. Spine, 27: 1142–1148.

Hsieh CYJ, Phillips RB, Adams AH, Pope MH (1992). Functional outcomes of low back pain: comparison of four treatment groups in a randomized controlled trial. Journal of Manipulative and Physiological Therapeutics, 15: 4–9.

Hudak PL, Cole DC, Frank JW (1998). Perspectives on prognosis of soft tissue musculoskeletal disorders. International Journal of Rehabilitation Research, 21: 29–40.

Hunter J (2001). Physical symptoms and signs and chronic pain. Clinical Journal of Pain, 17: S26–32.

Hurley DA, Dusoir TE, McDonough SM, Moore AP, Baxter GD (2001a). How effective is the acute low back pain screening questionnaire for predicting 1-year follow-up in patients with low back pain? Clinical Journal of Pain, 17: 256–263.

Hurley DA, Minder PM, McDonough SM, Walsh DM, Moore AP, Baxter DG (2001b). Interferential therapy electrode placement technique in acute low back pain: a preliminary investigation. Archives of Physical Medicine and Rehabilitation, 82: 485–493.

Hurwitz EL, Morgenstern H, Harber P, Kominski GF, Belin TR, Yu F, Adams AH, Kominsky GF (2002). Second prize: the effectiveness of physical modalities among patients with low back pain randomised to chiropractic care: findings from the UCLS low back pain study. Journal of Manipulative and Physiological Therapeutics, 25: 10–20.

Indahl A, Velund L, Reikeraas O (1995). Good prognosis for low back pain when left untampered: a randomized clinical trial. Spine, 20: 473–7.

Infante-Rivard C, Lortie M (1996). Prognostic factors for return to work after a first compensated episode of back pain. Occupational and Environmental Medicine, 53: 488–494.

Jacobson BH, Gemmell HA, Hayes BM, Altena TS (2002). Effectiveness of a selected bedding system on quality of sleep, low back pain, shoulder pain and spine stiffness. Journal of Manipulative and Physiological Therapeutics, 25: 88–92.

Jensen MC, Bran-Zawadzki MN, Obucjowski N, Modic MT, Malkasian D, Ross JS (1994). Magnetic resonance imaging of the lumbar spine in people without back pain. New England Journal of Medicine, 331: 69–73.

Johanning E (2000). Evaluation and management of occupational low back disorders. American Journal of Industrial Medicine, 37: 94–111.

Kaiser PO, Mattsson B, Marklund S, Wimo A (2001). The impact of psychosocial 'markers' on the outcome of rehab. Disability and Rehabilitation, 23: 430–435.

Karjalainen K, Malmivaara A, van Tulder M, Roine R, Jauhiainen M, Hurri H, Koes B (2002). Multi-disciplinary biopsychosocial rehabilitation for subacute low back pain among working age adults. In: The Cochrane Library; Issue 2. Oxford: Update Software.

Kellgren JH (1939). On the distribution of pain arising from deep somatic structures with charts of segmental pain areas. Clinical Science, 4: 35–46.

Kendrick D, Fielding K, Bentley E, Kerslake R, Miller P, Pringle M (2001). Radiography of the lumbar spine in primary care patients with low back pain: randomised controlled trial. BMJ, 322: 400–405.

Kerry S, Hilton S, Dundas D, Rink E, Oakeshott P (2002). Radiography for low back pain: a randomised controlled trial and observational study in primary care. The British Journal of General Practice, 52: 469–474.

Kilpikoski S, Airaksinene O, Kankaapaa M, Leminem P, Videman T, Alen M (2002). Interexaminer reliability of low back pain assessment using the McKenzie method. Spine, 27: E207–214.

Kitchener P, Houang M, Anderson B (1986). Utilisation review of magnetic resonance imaging: the Australian experience. Australian Clinical Review, September: 127–136.

Kjellman G, Oberg B, Hensing G, Alexanderson K (2001). A 12 year follow up of subjects initially sicklisted with neck/shoulder or low back pain diagnoses. Physiotherapy Research International, 6: 52–63.

Klinger NM, Wilson RR, Kanniainen CM, Wagenknecht KA, Re ON, Gold RH (1988). Intravenous orphenadrine for the treatment of lumbar paravertebral muscle strain. Current Therapeutic Research, 43; 2: 247–254.

Knutson GA (2002). Incidence of foot rotation, pelvic crest unleveling, and supine leg length alignment asymmetry and their relationship to self-reported back pain. Journal of Manipulative and Physiological Therapeutics, 25: 110E.

Koes BW, Assendelft WJJ, van der Heijden GJMG, Bouter LM (1996). Spinal manipulation for low back pain. An updated systematic review of randomised clinical trials. Spine, 21: 2860–2871.

Koes BW, Scholten RJPM, Mens JMA, Bouter LM (1997). Efficacy of non-steroidal anti-inflammatory drugs for low back pain: a systematic review of randomised clinical trials. Annals of the Rheumatic Diseases, 56: 214–223.

Koes BW, Scholten RJPM, Mens JMA, Bouter LM (1999). Epidural steroid injections for low back pain and sciatica: an updated systematic review of randomised clinical trials. Pain Digest, 9: 241–247.

Koes BW, van den Hoogen HMM (1994). Efficacy of bed rest and orthoses for low back pain. A review of randomised clinical trials. European Journal of Physical Medicine and Rehabilitation, 4: 86–93.

Koes BW, van Tulder M, Ostelo R, Burton AK, Waddell G (2001). Clinical guidelines for the management of low back pain in primary care: an international comparison. Spine, 26: 2504–2513.

Konrad K, Tatrai T, Hunka A, Vereckei E, Korondi (1992). Controlled trial of balneotherapy in treatment of low back pain. Annals of the Rheumatic Diseases, 51: 820–822.

Kovacs FM, Llobera J, Abraira V, Kovacs FM, Lazaro P, Pozo F, Kleinbaum D, Aguilar MD, Bauza JR, Gomez M, Mateu C, Perello L, Ripoll J, Bauza JR, Bauza K, Coll J, Duro E, Olivera A, Reyes V, Ribot S, Fernandez C, Llinas M, Pascual P, Ripoll J, Gestoso M, Mufraggi N, Gil-del-Real MT, Gili J, Gonzalez J, Nicolau C, Rodriguez E, Ibanez P, Jover A, Muriel A (2002). Effectiveness and cost-effectiveness of neuroreflexotherapy for subacute and chronic low back pain. Spine, 27: 1149–59.

Krause N, Dasinger LK, Deegan LJ, Rudolph L, Brand RJ (2001). Psychosocial job factors and return-to-work after compensated low back injury: a disability phase-specific analysis. American Journal of Industrial Medicine, 40: 374–392.

Kuch K (2001). Psychological factors and the development of chronic pain. The Clinical Journal of Pain, 17: S33–38.

Kummel B (2001). The use of nonorganic signs and symptoms as a screening tool for return to work in patients with acute low back pain (Letter to the Editor). Spine, 26: Aug 01.

Lacey PH, Dodd GD, Shannon DJ (1984). A double-blind placebo controlled study of piroxicam in the management of acute musculoskeletal disorders. European Journal of Rheumatology and Inflammation, 7: 95–104.

Larsen K, Weidich F, Leboeuf-Yde C (2002). Can custom-made biomechanic shoe orthoses prevent problems in the back and lower extremities? A randomised controlled intervention trial of 146 military conscripts. Journal of Manipulative and Physiological Therapeutics, 25: 326–331.

Larsson U, Choler U, Lidstrom A et al. (1980). Auto-traction for treatment of lumbago-sciatica. Acta Orthopedica Scandinavia, 51; 791–798.

Laws D (1994). Double blind parallel group investigation in general practice of the efficacy and tolerability of acemetacin, in comparison with diclofenac, in patients suffering with acute low back pain. British Journal of Clinical Research, 5: 55–64.

Leboeuf-Yde C, van Dijk J, Franz C, Hustad SA, Olsen D, Pihl T, Robech R, Skov Vendrup S, Bendix T, Kyvik KO (2002). Motion palpation findings and self-reported low back pain in a population-based study sample. Journal of Manipulative and Physiological Therapeutics, 25: 80–87.

Leclaire R, Esdaile JM, Suissa S, Rossignol M, Proulx R, Dupuis M (1996). Back school in a first episode of compensated acute low back pain: a clinical trial to assess efficacy and prevent relapse. Archives of Physical Medicine and Rehabilitation, 77: 673–679.

Lindequist S, Lundberg B, Wikmakr R, Bergstad B, Loof G, Ottermark AC (1984). Information and regime at low back pain. Scandinavian Journal of Rehabilitation Medicine, 16: 113–116.

Lindstrom I, Ohland C, Eek C, Wallin L, Peterson LE, Nachemson A (1992a). Mobility, strength and fitness after a graded activity program for patients with subacute low back pain. A randomised prospective clinical study with a behavioural therapy approach. Spine, 17; 6: 649–649.

Lindstrom I, Ohland C, Eek C, Wallin L, Peterson LE, Fordyce WE, Nachemson AL (1992b). The effect of graded activity on patients with subacute low back pain: a randomised prospective clinical study with an operant-conditioning behavioural approach. Physical Therapy, 72; 4: 279–290.

Linton SJ (2002). A prospective study of the effects of sexual or physical abuse on back pain. Pain, 96: 347–351.

Linton SJ (2001). Occupational psychological factors increase the risk for back pain: a systematic review. Journal of Occupational Rehabilitation, 11: 53–66.

Linton S, Andersson MA (2000) Can chronic disability be prevented? A randomized trial of a cognitive-behavioiur intervention and two forms of information for patients with spinal pain. Spine, 25: 2825–2831.

Linton SJ, Hallden K (1998). Can we screen for problematic back pain? A screening questionnaire for predicting outcome in acute and subacute back pain. The Clinical Journal of Pain, 14: 209–215.

Linton SJ, Hellsing AL, Andersson D (1993). A controlled study on the effects of an early intervention on acute musculoskeletal pain problems. Pain, 54: 353–359.

Linton SJ, Ryberg M (2001). A cognitive-behavioral group intervention as prevention for persistent neck and back pain in a non-patient population: a randomized controlled trial. Pain, Feb 1, 90: 83–90.

Linton SJ, van Tulder MW (2001). Preventive interventions for back and neck pain problems: what is the evidence? Spine, 26: 778–787.

Little P, Roberts L, Blowers H, Garwood J, Cantrell T, Langridge J, Chapman J (2001). Should we give detailed advice and information booklets to patients with back pain? A randomized controlled factorial trial of a self-management booklet and doctor advice to take exercise for back pain. Spine, 26: 2065–2072.

Loisel P, Abenheim L, Durand P, Esdaile J, Suissa S, Gosselin L, Simard R, Turcotte J, Lemaire J (1997). A population-based, randomised clinical trial on back pain management. Spine, 22: 2911–2918.

Loisel P, Gosselin L, Durand P, Lemaire J, Poitras S, Abenhaim L (2001). Implementation of a participatory ergonomics program in the rehabilitation of workers suffering from subacute back pain. Applied Ergonomics, 32: 53–60.

Loisel P, Vachon B, Lemaire J, Durand MJ, Poitras S, Stock S, Tremblay C (2002). Discriminative and predictive validity assessment of the Quebec task force classification. Spine, 27: 851–857.

Lorig KR, Laurent DD, Deyo RA, Marnell ME, Minor MA, Ritter PL (2002). Can a back pain e-mail discussion group improve health status and lower health care costs? A randomized study. Archives of Internal Medicine, 162: 792–796.

Lundin O, Hellstrom M, Nilsson I, Sward L (2001). Back pain and radiological changes in the thoraco-lumbar spine of athletes. A long term follow up. Scandinavian Journal of Medicine and Science in Sports, 11: 103–109.

MacDonald RS, Bell CMJ (1990). An open controlled assessment of osteopathic manipulation in non-specific low back pain. Spine, 15: 364–370.

Maher C, Adams R (1994). Reliability of pain and stiffness assessments in clinical manual lumbar spine examination. Physical Therapy, 74: 801–811.

Maher C, Latimer J, Refshauge K (1999). Prescription of activity for low back pain: what works? Australian Journal of Physiotherapy, 45: 122–132.

Maier-Riehle B, Harter M (2001). The effects of back schools: a meta-analysis. International Journal of Rehabilitation Research, 24: 199–206.

Main CJ, Waddell G (1998). Behavioral responses to examination: a reappraisal of the interpretation of "nonorganic signs". Spine, 23: 2367–2371.

Malmivaara A, Hakkinen U, Aro T, Heinrichs ML, Koskenniemi L, Kuosma E, Lappi S, Paloheimo R, Servo C, Vaaranen V, Hernberg S (1995). The treatment of low back pain: bed rest, exercises, or ordinary activity? New England Journal of Medicine, 332: 351–355.

Mathews JA, Mills SB, Jenkins VM, Grimes SM, Morkel MJ, Mathews W. et al. (1987). Back pain and sciatica: controlled trials of manipulation, traction, sclerosant and epidural injections. British Journal of Rheumatology, 26: 416–423.

Mayer RS, Chen IH, Lavender SA, Trafinow JH, Andersson GBJ (1995). Variance in the measurement of sagittal lumbar spine range of motion among examiners, subjects, and instruments. Spine, 20: 1489–1493.

McCombe PF, Fairbank JCT, Cockersole BC, Punsent PB (1989). Reproducibility of physical signs in lowback pain. Spine, 14: 908–918.

McGuirk B, King W, Govind J, Lowry J, Bogduk N (2001). The safety, efficacy, and cost-effectiveness of evidence-based guidelines for the management of acute low back pain in primary care. Spine, 26: 2615–2622.

McIntosh G, Frank J, Hogg-Johnson S, Bombardier C, Hall H (2000a). Prognostic factors for time receiving workers' compensation benefits in a cohort of patients with low back pain. Spine, 25: 147.

McIntosh G, Frank J, Hogg-Johnson S, Hall H, Bombardier C (2000b). Low back pain prognosis: structured review of the literature. Journal of Occupational Rehabilitation, 10: 101–115.

McNally EG, Wilson DJ, Ostlere SJ (2001). Limited magnetic resonance imaging in low back pain instead of plain radiographs: experience with first 1000 cases. Clinical Radiology, 56: 922–925.

Melzack R, Vetere P, Finch L (1983). Transcutaneous electrical stimulation for low back pain: a comparison of TENS and massage for pain and range of motion. Physical Therapy, 63: 489–493.

Merskey H, Bogduk N (eds) (1994). Classification of Chronic Pain. Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms (2nd Edition). IASP Press: Seattle.

Middleton RSW (1984). A comparison of two analgesic muscle relaxant combinations in acute back pain. British Journal of Clinical Practice, 38: 107–109.

Milgrom C, Finestone A, Lev B, Wiener M, Florman T (1993). Overexertional lumbar and thoracic back pain among recruits: a prospective study of risk factors and treatment regimens. Journal of Spinal Disorders, 6: 187–193.

Milne S, Welch V, Brosseau L, Saginur M, Shea B, Tugwell P, Wells G (2002). Transcutaneous electrical nerve stimulation (TENS) for chronic low back pain. In: The Cochrane Library, Issue 2. Oxford: Update Software.

Mitchell LV, Lawler FH, Bowen D, Mote W, Asundi P, Pursell J (1994). Effectiveness and cost-effectiveness of employer-issued back belts in areas of high risk for back injury. Journal of Occupational Medicine, 36: 90–94.

Moffett J, Torgerson D, Bell-Syer SB, Jackson D, Llewlyn-Phillips H, Farrin A, Barber J 1999). Randomised controlled trial of exercise for low back pain: clinical outcomes, costs and preferences. British Medical Journal, 319: 279–283.

Mohseni-Bandpei MA, Stephenson R, Richardson B (1998). Spinal manipulation in the treatment of low back pain: a review of the literature with particular emphasis on randomised controlled trials. Physical Therapy Rev, 3:185–194.

Mooney V, Robertson J (1976). The facet syndrome. Clinical Orthopaedics and Related Research, 115:149–156.

Morrison GEC, Chase W, Young V, Roberts WL (1988). Back pain. Treatment and prevention in a community hospital. Archives of Physical and Medical Rehabilitation, 69: 605–609.

Muncie HL Jr., King JE, DeForge B (1986). Treatment of mild to moderate pain of acute soft tissue injury: diflunisal vs acetaminophen with codeine. Journal of Family Practice, 23; 2: 125–127.

Musgrave D, Vogt M, Nevitt M, Cauley J (2001). Back problems among post-menopausal women taking estrogen replacement therapy. Spine, 26: 1606–1612.

Nadler SF, Steiner DJ, Erasala GN, Hengehold DA, Hinkle RT, Goodale MB, Abeln SB, Weingand KW (2002). Continuous low-level heat wrap therapy provides more efficacy than ibuprofen and acetaminophen for acute low back pain. Spine, 27: 1012–1017.

National Health and Medical Research Council (1999a). A Guide to the Development, Implementation and Evaluation of Clinical Practice Guidelines. AGPS: Canberra.

National Health and Medical Research Council (1999b). Acute Pain Management: Scientific Evidence. Commonwealth of Australia: Canberra.

Nelemans PJ, Bie RA de, Vet HCW de, Sturmans F (2002). Injection therapy for subacute and chronic benign low back pain. In: The Cochrane Library; Issue 2. Oxford: Update Software.

Newswanger DL, Patel AT, Ogle A (2000). Osteopathic medicine in the treatment of low back pain (Letter to the Editor). American Family Physician, 62: 2414–2415.

Newton-John T, Ashmore J, McDowell M (2001). Early intervention in acute back pain: problems with flying the yellow flag. Physiotherapy, 87: 397–401.

Nice DA, Riddle DL, Lamb RL, Mayhew TP, Ruckler K (1992). Intertester reliability of judgements of the presence of trigger points in patients with low back pain. Archives of Physical Medicine and Rehabilitation, 73: 893–898.

Njoo KH, van der Does E, Stam HJ (1995). Interobserver agreement on iliac crest pain syndrome in general practice. The Journal of Rheumatology, 22: 1532–1535.

Njoo KH, van der Does (1994). The occurrence and inter-rater reliability of myofascial trigger points in the quadratus lumborum and gluteus medius: a prospective study in non-specific low back pain patients and controls in general practice. Pain, 58: 317–323.

Nwuga VCB (1982). Relative therapeutic efficacy of vertebral manipulation and conventional treatment in back pain management. American Journal of Physical Medicine, 61: 273–278.

Nwuga G, Nwuga V (1985). Relative therapeutic efficacy of the Williams and McKenzie protocols in back pain management. Physiotherapy Practice, 1: 99–105.

Onorato A, Rosin C, Schierano S, Zampa A (2001). The soft-cast semi-rigid jacket in the treatment of low back pain. Europa Medicophysica, 37: 191–195.

O'Neill CW, Kurgansky ME, Derby R, Deaglan PR (2002). Disc stimulation and patterns of referred pain. Spine, 27: 2776–2781.

Orava S. (1986). Medical treatment of acute low back pain. Diflunisal compared with indomethacin in acute lumbago. International Journal of Clinical Research, 6: 45–51.

O'Sullivan P (2000). Twelve months after onset of acute low back pain, most are back at work but many remain in pain. The Australian Journal of Physiotherapy, 46: 237.

Pal B, Mangion P, Hossain MA, Diffey BL (1986). A controlled trial of continuous lumbar traction om the treatment of back pain and sciatica. British Journal of Rheumatology, 25: 1181–1183.

Palangio M, Morris E, Doyle RTJ, Dornseif BE, Valente TJ (2002). Combination hydrocodone and ibuprofen versus combination oxycodone and acetaminophen in the treatment of moderate or severe acute low back pain. Clinical Therapeutics, 24: 87–99.

Patel AT, Ogle AA (2000). Diagnosis and management of acute low back pain. American Family Physician, 61: 1779–1786, 1789–1790.

Pengel HM, Maher CG, Refshauge KM (2002). Systematic review of conservative interventions for subacute low back pain. Clinical Rehabilitation, 16: 811–820.

Penrose KW, Chook K, Stump JL (1991). Acute and chronic effects of pneumatic lumbar support on muscular strength, flexibility and functional impairment index. Sports Training and Medical Rehabilitation, 2: 121–129.

Pentinnen J, Nevala-Puranen N, Airaksinen O, Jaaskelainen M, Sintonen H, Takala J (2002). Randomized controlled trial of back school with and without peer support. Journal of Occupational Rehabilitation, 12: 21–29.

Philips HC, Grant L, Berkowitz J. (1991) The prevention of chronic pain and disability: a preliminary investigation. Behavior Research and Therapy, 29: 443–450.

Phillips DR, Twomey LT (1996). A comparison of manual diagnosis with a diagnosis established by a unilevel lumbar spinal block procedure. Manual Therapy, 2: 82–87.

Pincus T, Burton AK, Vogel S, Field AP (2002). A systematic review of psychological factors as predictors of chronicity/disability in prospective cohorts of low back pain. Spine, 27: E109–120.

Pohjolainen T, Jekunen A, Autio L, Vuorela H (2000). Treatment of acute low back pain with the COX-2 selective anti-inflammatory drug nimesulide. Spine, 25: 1579–1585.

Pope MH, Philips RB, Haugh LD, Hseih CY, MacDonald L, Haldeman S (1994). A prospective randomised three-week trial of spinal manipulation, transcutaneous muscle stimulation, massage and corset in the treatment of subacute low back pain. Spine, 19: 2571–2577.

Postacchini F, Facchini M, Palieri P (1988). Efficacy of various forms of conservative treatment in low back pain: a comparative study. Neuro-orthopedics, 6: 28–35.

Preyde M (2000). Effectiveness of massage therapy for subacute low back pain: a randomised controlled trial. Canadian Medical Association Journal, 162: 1815–1820.

Pullliam CB, Gatchel RJ, Gardea MA (2001). Psychosocial differences in high risk versus low risk acute low back pain patients. Journal of Occupational Rehabilitation, 11: 43–52.

Quittan M (2002). Management of back pain. Disability and Rehabilitation, 24: 423–434.

Ramey DW, Sampson W (2001). Review of the evidence for the clinical efficacy of human acupuncture. Scientific Review of Alternative Medicine, 5: 195–201.

Rasmussen GG (1979). Manipulation in treatment of low back pain: a randomised clinical trial. Manual Medicine, 1: 8–10.

Reinus WR, Strome G, Zwemer F (1998). Use of lumboscral spine radiographs in a level II emergency department. AJR, 170: 443–447.

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.

Roberts L, Little P, Chapman J, Cantrell T, Pickering R, Langridge J (2002). The back home trial: general practitioner-supported leaflets may change back pain behavior. Spine, Sep 1; 27: 1821–8.

Roland MO (1986). A critical review of the evidence for a pain-spasm-pain cycle in spinal disorders. Clinical Biomechanics, 1: 102–109.

Rollings HE, Glassman JM, Soyka JP (1983). Management of acute musculoskeletal conditions — thoracolumbar strain or sprain: a double-blind evaluation comparing the efficacy and safety of carisoprodol with cyclobenzaprine hydrochloride. Current Therapeutics Research, 34: 917– 928.

Rossignol M, Abenheim L, Seguin P, Neveu A, Collet JP, Ducruet T, Shapiro S, Cherkin D (2000). Coordination of primary health care for back pain: a randomised controlled trial. Spine, 25: 251–259.

Royal Australian College of General Practitioners (2002). Evidence-Based Primary Care Handbook on Acute Low Back Pain. RACGP: Victoria.

Rozenberg S, Delval C, Rezvani Y, Olivieri-Apicella N, Kuntz J, Legrand E, Valat J, Blotman F, Meadeb J, Rolland D, Hary S, Duplan B, Feldman J, Bourgeois P (2002). Bed rest or normal activity for patients with acute low back pain: a randomized controlled trial. Spine, 27: 1487–1493.

Rupert RL, Wagnon R, Thompson P, Ezzeldin MT (1985). Chiropractic adjustments: results of a controlled clinical trial in Egypt. ICA International Review of Chiropractic, Winter: 58–60.

Sanders GE, Reinert O, Tepe R, Maloney P (1990). Chiropractic adjustive manipulation on subjects with acute low back pain: visual analog pain scores and plasma beta-endorphin levels. Journal of Manipulative and Physiological Therapeutics, 13: 391–395.

Scavone JG, Latshaw RF, Weidner WA (1981a). AP and lateral radiographs: an adequate lumbar spine examination. AJR, 136: 715–717.

Scavone JG, Latshaw RF, Rohrer V (1981b). Use of lumbar spine films: statistical evaluation at a university teaching hospital. JAMA, 246: 1105–1108.

Schiottz-Christensen B, Nielsen GL, Hansen VK, Schodt T, Sorensen HT, Olesen F (1999). Long-term prognosis of acute low back pain

in patients seen in general practice: a 1-year prospective follow-up study. Family Practice, 16: 223–231.

Schreiber S, Vinokur S, Shavelzon V, Pick CG, Zahavi E, Shir Y (2001). A randomized trial of fluoxetine versus amitriptyline in musculoskeletal pain. The Israel Journal of Psychiatry and Related Sciences, 38: 88–94.

Seferlis T, Nemeth G, Carlsson AM, Gillstrom P (1998). Conservative treatment in patients sick-listed for acute low back pain: a prospective randomised study with 12 months' follow up. European Spine Journal, 7: 461–470.

Shannon HS, Woodward CA, Cunningham CE, McIntosh J, Lendrum B, Brown J, Rosenbloom D (2001). Changes in general health and musculoskeletal outcomes in the workforce of a hospital undergoing rapid change: a longitudinal study. Journal of Occupational Health Psychology, 6: 3–14.

Shaw WS, Pransky G, Fitzgerald TE (2001). Early prognosis for low back disability: intervention strategies for health care providers. Disability and Rehabilitation, 15: 815–828.

Shekelle PG, Adams AH, Chassin MR, Hurwitz EL, Brook RH (1992). Spinal manipulation for low back pain. Annals of Internal Medicine, 117: 590–598.

Sheppard L (2001). Group cognitive behavioural intervention lowers the risk of developing long term spinal disability. The Australian Journal of Physiotherapy, 47: 152.

Sinclair SJ, Hogg-Johnson S, Mondloch MV, Shields SA (1997). The effectiveness of an early active intervention programme for workers with soft-tissue injuries: the early claimant cohort study. Spine, 22: 2919–2931.

Skargren EI, Carlsson PG, Oberg BE (1998). One-year follow up comparison of the cost and effectiveness of chiropractic and physiotherapy as primary management for back pain: subgroup analysis, recurrence and additional health care utilisation. Spine 23: 1875–1883.

Skargren EI, Oberg BE, Carlsson PG, Gade M (1997). Cost and effectiveness analysis of chiropractic and physiotherapy treatment for low back and neck pain: six month follow up. Spine, 22: 2167–2177.

Smith D, McMurray N, Disler P (2002). Early intervention for acute back injury: can we finally develop an evidence-based approach? Clinical Rehabilitation, 16: 1–11.

Smyth M J, Wright V (1959). Sciatica and the intervertebral disc. An experimental study. The Journal of Bone and Joint Surgery, 40A: 1401–1418.

Sorby WA (1989). An evaluation of magnetic resonance imaging at The Royal North Shore Hospital of Sydney, 1986–1987. Medical Journal of Australia, 151: 8–18.

Stam C, Bonnet MS, van Haselen RA (2001). The efficacy and safety of a homeopathic gel in the treatment of acute low back pain: a multicentre, randomised, double-blind comparative clinical trial. The British Homeopathic Journal, 280: 21–28.

Stankovic R, Johnell O (1995). Conservative treatment of acute low back pain: a 5 year follow-up study of two methods of treatment. Spine, 20: 469–172.

Stevinson C, Ernst E (2002). Risks associated with spinal manipulation. American Journal of Medicine, 112: 566–571.

Stig LC, Nilsson O, Leboeuf-Yde C (2001). Recovery patterns of patients treated with chiropractic and spinal manipulative therapy for long lasting or recurrent low back pain. Journal of Manipulative and Physiological Therapeutics, 24: 288–291.

Strand LI, Ljunggren AE, Haldorsen EH, Espehaug B (2001). The impact of physical function and pain on work status at 1-year follow-up in patients with back pain. Spine, 26: 800–808.

Strender LE, Sjoblom A, Sundell K, Ludwig R, Taube A (1997). Interexaminer reliability in physical examination of patients with low back pain. Spine, 22: 814–820.

Suarez-Almazor ME, Belseck E, Russell AS, Mackel JV (1997). Use of lumbar radiographs for the early diagnosis of low back pain: proposed guidelines would increase utilization. JAMA, 277: 1782–1786.

Susman J (2001). The care of low back problems: Less is more. American Family Physician, 65: 2217–2218.

Sweetman BJ, Baig A, Parsons DL (1987). Mefenamic acid, chlormezanone-paracetamol, ethoheptazine-aspirin-meprobamate: a comparative study in acute low back pain. British Journal of Clinical Practice, 41: 619–624.

Szpalski M, Hayez JP (1994). Objective functional assessment of the efficacy of tenoxicam in thte treatment of acute low back pain treated with Tenoxicam. British Journal of Rheumatology, 33: 74–78.

Torgerson WR, Dotter WE (1976). Comparative roentgenographic study of the asymptomatic and symptomatic lumbar spine. Journal of Bone and Joint Surgery, 58A: 850–853.

Torp S, Riise T, Moen BE (2001). The impact of psychosocial work factors on musculoskeletal pain: a prospective study. Journal of Occupational and Environmental Medicine, 43: 120–126.

Trainor TJ, Wiesel SW (2002). Epidemiology of back pain in the athlete. Clinics in Sports Medicine, 21: 93–103.

Truchon M, Fillion L (2000). Biopsychosocial determinants of chronic disability and low back pain: a review. Journal of Occupational Rehabilitation, 10: 117–142.

Turner JA (1996). Educational and behavioral interventions for back pain in primary care. Spine, 21: 2851–2859.

Underwood MR, Morgan J (1998). The use of a back class teaching extension exercises in the treatment of acute low back pain in primary care. Family Practice, 15: 9–15.

Valle-Jones JC, Walsh H, O'Hara J, O'Hara H, Davey NB, Hopkin-Richards H (1992). Controlled trial of a back support in patients with non-specific low back pain. Current Medical Research Opinion, 12: 604–613.

van den Hoogen MM, Koes BW, Eijk JTM, Bouter LM (1995). On the accuracy of history, physical examination, and erythrocyte sedimentation rate in diagnosing low back pain in general practice: a criteria-based review of the literature. Spine, 20: 318–327.

van den Hoogen HJM, Koes BW, van Eijk JThM, Bouter LM, Deville W (1998). On the course of low back pain in general practice: a one-year follow up study. Annals of the Rheumatic Diseases, 57: 13–19.

van der Heijden GJMG, Beurskens AJHM, Koes BW, Assendelft WJ, de Vet HC, Bouter LM (1995). The efficacy of traction for back and neck pain: a systematic, blinded review of randomised clinical trial methods. Physical Therapy, 75: 93–104.

van Dieen JH, de Looze MP, Hermans V (2001). Effects of dynamic office chairs on trunk kinematics, trunk extensor EMG and spinal shrinkage. Ergonomics, 44: 739–750.

van Tulder MW, Assendelft WJJ, Koes BW, Bouter LM (1997a). Spinal radiographic findings and non-specific low back pain. A systematic review of observational studies. Spine, 22; 427–434.

van Tulder MW, Cherkin DC, Berman B, Lao L, Koes BW (2002a). Acupuncture for low back pain. In: The Cochrane Library; Issue 2. Oxford: Update Software.

van Tulder MW, Esmail R, Bombardier C, Koes BW (2002b). Back schools for non-specific low back pain. In: The Cochrane Library; Issue 2. Oxford: Update Software.

van Tulder MW, Jellema P, van Poppel MNM, Nachemson AL, Bouter LM (2002c). Lumbar supports for prevention and treatment of low

back pain. In: The Cochrane Library; Issue 2. Oxford: Update Software.

van Tulder MW, Koes BW, Bouter LM (1997b). Conservative treatment of acute and chronic non-specific low back pain: a systematic review of randomised controlled trials of the most common interventions. Spine, 22: 2128–2156.

van Tulder MW, Malmivaara A, Esmail R, Koes B (2000). Exercise therapy for low back pain: a systematic review within the framework of the Cochrane collaboration back review group. Spine, 25: 2784–2796.

van Tulder MW, Malmivaara A, Esmail R, Koes B (2002d). Exercise therapy for low back pain (Cochrane Review). In: The Cochrane Library; Issue 2. Oxford: Update Software.

van Tulder MW, Ostello R, Vlaeyen JWS, Linton SJ, Morley SJ, Assenfeldt WJJ (2002e). Behavioural treatment for chronic low back pain. In: The Cochrane Library; Issue 2. Oxford: Update Software.

van Tulder MW, Scholten RJPM, Koes BW, Deyo RA (2002f). Non-steroidal anti-inflammatory drugs for low back pain. In: The Cochrane Library; Issue 2. Oxford: Update Software.

Veenema KR, Leahey N, Schneider S (2000). Ketorolac versus meperidine: ED treatment of severe musculoskeletal low back pain. American Journal of Emergency Medicine, 18: 404–407.

Videman T, Osterman K (1984). Double blind parallel study of piroxicam versus indomethacin in the treatment of low back pain. Annals of Clinical Research, 16: 156–160.

Videman T, Heikkila J, Partanen (1984). Double blind parallel study of meptazinol versus diflunisal in the treatment of lumbago. Current Medical Research Opinion, 9, 246–252.

von Korff M, Moore JC (2001). Stepped care for back pain: activating approaches for primary care. Annals of Internal Medicine, 134: 911–917.

Vroomen PJAJ, Marc CTFM, Wilmink JT, Kester ADM, Knottnerus JA (1999). Lack of effectiveness of bedrest for sciatica. New England Journal of Medicine, 340: 418–423.

Waddell G (1992). Biopsychosocial analysis of low back pain. In: Nordin M, Voscher TL (eds). Bailliere's Clinical Rheumatology, Common Low Back Pain: Prevention of Chronicity. WB Saunders: London.

Waddell G, Feder G, Lewis M (1997). Systematic reviews of bed rest and advice to stay active for acute low back pain. British Journal of General Practice, 47: 647–652.

Waddell G, Feder G, McIntosh A, et al. (2001). Low Back Pain Evidence Review. Royal College of General Practitioners: London.

Waddell G, Main CJ, Morris EW, Venner RM, Rae P, Sharmy SH, Galloway H (1982). Normality and reliability in the clinical assessment of backache. British Medical Journal, 284: 1519–15223.

Waddell G, McCulloch JA, Kummel E, Venner RM (1980). Non-organic physical signs in low-back pain. Spine, 5(2): 117–125.

Waldvogel FA, Vasey H (1980). Osteomyelitis: the past decade. New England Journal of Medicine, 303: 360–370.

Waterworth RF, Hunter IA (1985). An open study of diflunisal, conservative and manipulative therapy in the management of acute mechanical low back pain. New Zealand Medical Journal, May: 372–375.

Watts RW, Silagy CA (1995). A meta-analysis on the efficacy of epidural corticosteroids in the treatment of sciatica. Anaesthesia and Intensive Care 23: 564–569.

Werneke M, Hart DL (2001). Centralisation phenomenon as a prognostic factor for chronic low back pain and disability. Spine, 26: 758–764.

Wiesel SW (1986). A study of computer-assisted tomography. 1. The incidence of positive CAT scans in an asymptomatic group of patients. Spine, 9: 549–551.

Wiesel SW, Cuckler JM, Deluca F, Jones F, Zeide MS, Rothman RH (1980). Acute low back pain: an objective analysis of conservative therapy. Spine, 5: 324–330.

Wilkinson MJ (1995). Does 48 hours' bed rest influence the outcome of acute low back pain? British Journal of General Practice, 45; 398: 481–484.

Wreje U, Nordgren B, Aberg H (1992). Treatment of pelvic joint dysfuntion in primary care: a controlled trial. Scandinavian Journal of Primary Health Care, 10: 310–315.

Yassi A, Cooper JE, Tate RB, Gerlach S, Muir M, Cohn RN, Trottier J, Doht T, Massey K (2001). A randomized controlled trial to prevent patient lift and transfer injuries of health care workers. Spine, 26: 1739–1746.

Yelland MJ (2001). Back, chest and abdominal pain: how good are spinal signs at identifying musculoskeletal causes of back, chest or abdominal pain? Australian Family Physician, 30: 908–912.

Zuberbier OA, Kozlowski AJ, Hunt DG, Berkowitz J, Schultz IZ, Crook JM, Milner RA (2001). Analysis of the convergent and discriminant validity of published lumbar flexion, extension and lateral flexion scores. Spine, 26: E472–478.

Zylbergold RS, Piper MC (1981). Lumbar disc disease: comparative analysis of physical therapy treatments. Archives of Physical Medicine and Rehabilitation, 62: 176–179.

Chapter 5

Acute Thoracic Spinal Pain

➔ *There are currently no guidelines for the management of thoracic spinal pain. This document provides an overview of the evidence in this area to raise awareness of the need for formal population studies on the diagnosis and management of thoracic spinal pain.*

Definition of Acute Thoracic Spinal Pain

In these guidelines, the term 'acute' refers to pain that has been present for less than three months; it does not refer to the severity or quality of pain. Chronic pain is defined as pain that has been present for at least three months (Merskey and Bogduk 1994).

These guidelines describe the diagnosis and treatment of acute thoracic spinal pain of unknown or uncertain origin. The following is a definition of thoracic spinal pain developed by the International Association for the Study of Pain (Merskey and Bogduk 1994):

> …pain perceived anywhere in the region bounded superiorly by a transverse line through the tip of the spinous process of T1, inferiorly by a transverse line through the tip of the spinous process of T12, and laterally by vertical lines tangential to the most lateral margins of the erector spinae muscles. This area can be divided into upper, middle and lower thirds. Pain felt lateral to this area is defined as posterior chest wall pain, and does not constitute thoracic spinal pain.

Scope

These guidelines describe the diagnosis and treatment of acute, non-specific thoracic spinal pain. The following conditions are beyond the scope of this document:

- serious conditions: infection, neoplasm, neuropathic conditions and fractures of the thoracic spine
- chronic pain

Guideline Development Process

Evaluation of Existing Guidelines

Guidelines developed by other groups were sought to determine whether an existing document could be adapted for use in the Australian context. No published guidelines currently exist for the management of thoracic spinal pain. The decision was made to update the existing draft guidelines for acute thoracic spinal pain developed for the National Musculoskeletal Medicine Initiative by Dr Michael Yelland.

Updating Existing Guidelines

The update of the existing guidelines involved a review and appraisal of the evidence on the diagnosis, prognosis and interventions for acute thoracic spinal pain, conducted by a multidisciplinary review group. Studies that were published subsequent to the most recent update of the existing guidelines were identified and appraised according to standards for guideline development (NHMRC 1999a). Those studies meeting the criteria for inclusion were used to update the existing text of the guidelines. All studies assessed for this update are included in either the Table of Included Studies or the Table of Excluded Studies for Diagnosis, Prognosis and Interventions. Studies that were described in the existing guidelines were not appraised during this update and are not present in the tables.

For details of included and excluded studies, refer to Appendix E: Tables of Included and Excluded Studies.

Relevant studies on areas related to diagnosis were identified in the literature search and used to update the sections on Aetiology and Prevalence, History, Physical Examination and Investigations where possible. These sections are largely comprised of the existing work developed using a conventional literature review. Group members had the opportunity to evaluate the literature forming the basis of the existing guidelines, review the interpretation of the literature, nominate additional articles to undergo the appraisal process or request that an article be re-appraised.

Refer to Chapter 9: Process Report for further detail.

Study Selection Criteria

The chart, 'Study Selection Criteria' is an outlines the method used to update the content of the existing thoracic spinal pain guidelines. Textbooks of Rheumatology were consulted where necessary as a supplement to the scarce literature.

Search Strategy

Sensitive searches were performed; electronic searches were limited to adults, humans and articles published in English in peer-reviewed journals. Where available, methodological filters were used. There were no hand searches conducted.

Because of the paucity of information on this topic, the decision was made by the review group to include articles in journals that are no longer in print and those in the personal collections of the review group members. Such articles underwent critical appraisal as per the established process.

The following databases were searched in August 2002:

- PubMed 1966 to 2002
- MEDLINE 1966 to 2002
- CINAHL 1982 to 2002
- EMBASE — Physical and Rehabilitation Medicine 1992 to 2002
- The Cochrane Library, 2002, Issue 2
- Australasian Medical Index 1968 to 2002

Access to CHIROLARS/MANTIS and PEDro was unavailable for this review.

During the development of the original guidelines, the Journal of Manipulative and Physiological Therapeutics (1992 to 1997) and the Journal of Manual and Manipulative Therapy (1993 to 1997) were searched by hand. In addition, electronic searches of MEDLINE (1966–1997), EMBASE, CINAHL,

Study Selection Criteria

DIAGNOSIS	
The sections on Aetiology and Prevalence, History, Examination and Investigations comprise information from the existing draft (developed by conventional literature review) combined and updated with relevant articles located and appraised according to the following inclusion and exclusion criteria:	
Inclusion criteria	Systematic reviews, cross-sectional studies, case series, case reports Adults Specific diseases and conditions including those referring pain to the thoracic spine Non-specific diseases and conditions of the thoracic spine Peer-reviewed journal
Exclusion criteria	Chronic pain
PROGNOSIS	
Information from the existing draft was combined and updated with relevant articles located and appraised independently by two reviewers according to the following inclusion and exclusion criteria:	
Inclusion criteria	Systematic reviews, cohort studies Adults Non-specific diseases and conditions of the thoracic spine Peer-reviewed journal
Exclusion criteria	Chronic pain
INTERVENTIONS	
Information from the existing draft was updated with information from relevant articles located and appraised independently by two reviewers according to the following inclusion and exclusion criteria:	
Inclusion criteria	Systematic reviews, randomised controlled trials, cohort studies, case-control studies Adults Non-specific diseases and conditions of the thoracic spine Peer reviewed journal
Exclusion criteria	Chronic pain

Current Contents, The Cochrane Library (1997) and Netscape Excite were conducted.

Search Terms

- Thoracic vertebrae .exp
- Back pain .exp
- Thoracic spine .tw
- Costovertebral .tw
- Costotransverse .tw
- Rib .tw
- Sternum .tw
- Dorsalgia .tw
- Pain .exp
- Upper back .exp
- Thoracic .exp
- Therapies .exp
- Diagnosis .exp
- Prognosis .exp
- Systematic review .tw

Research Agenda for Acute Thoracic Spinal Pain

- Well-designed studies on the prevalence and clinical features of serious conditions presenting as acute thoracic spinal pain in the primary care setting.
- Research on the risk factors (aetiological and prognostic) for thoracic spinal pain.
- Thoracic discography studies to test whether disc protrusions without neurological signs may be a source of pain.
- Carefully controlled injection studies to investigate the role of somatic structures as sources of thoracic spinal pain.
- Research into the reliability and validity of tests used in paradigms of mechanical thoracic spinal pain.
- Research into the validity and reliability of physical examination tests of the thoracic spine.
- Studies to determine the effective treatments for acute thoracic spinal pain, including manipulation, medication and exercise.
- Research into the anatomical and pathophysiological origins of acute thoracic spinal pain of mechanical or somatic origin.

Summary of Key Messages: Acute Pain Management

	EVIDENCE LEVEL
Management Plan	
It is recommended that the clinician and patient develop a management plan for acute musculoskeletal pain comprising the elements of assessment, management and review: • Assessment — Conduct a history and physical examination to assess for the presence of serious conditions; ancillary investigations are not generally indicated unless features of serious conditions are identified. • Management — Provide information, assurance and advice to resume normal activity and discuss other options for pain management as needed. • Review — Reassess the pain and revise the management plan as required.	CONSENSUS: Steering Committee
Non-Pharmacological Interventions	
Simple interventions (providing information, assurance and encouraging reasonable maintenance of activity) may be used alone or in combination with other interventions for the successful management of acute musculoskeletal pain.	CONSENSUS: Steering Committee
Pharmacological Interventions	
Specific pharmacological interventions may be required to relieve pain; such agents can be used in conjunction with non-pharmacological interventions.	CONSENSUS: Steering Committee; NHMRC 1999b
Paracetamol or other simple analgesics, administered regularly, are recommended for relief of mild to moderate acute musculoskeletal pain.	CONSENSUS: Steering Committee; NHMRC 1999b
Where paracetamol is insufficient for pain relief, a non-steroidal anti-inflammatory (NSAID) medication may be used, unless contraindicated.	CONSENSUS: Steering Committee; NHMRC 1999b
Oral opioids may be necessary to relieve severe musculoskeletal pain. It is preferable to administer a short-acting agent at regular intervals, rather than on a pain-contingent basis. Ongoing need for opioid analgesia is an indication for reassessment.	CONSENSUS: Steering Committee; NHMRC 1999b
Adjuvant agents such as anticonvulsants and antidepressants are not recommended in the management of acute musculoskeletal pain.	CONSENSUS: Steering Committee; NHMRC 1999b
Any benefits from muscle relaxants may be outweighed by their adverse effects, therefore they cannot be routinely recommended.	CONSENSUS: Steering Committee

Summary of Key Messages: Effective Communication

	EVIDENCE LEVEL
Clinicians should work with patients to develop a management plan so that patients know what to expect, and understand their role and responsibilities.	CONSENSUS: Steering Committee
Information should be conveyed in correct but neutral terms, avoiding alarming diagnostic labels; jargon should be avoided.	CONSENSUS: Steering Committee
Explanation is important to overcome inappropriate expectations, fears or mistaken beliefs that patients may have about their condition or its management.	CONSENSUS: Steering Committee
Printed materials and models may be useful for communicating concepts.	CONSENSUS: Steering Committee
Clinicians should adapt their method of communication to meet the needs and abilities of each patient.	CONSENSUS: Steering Committee
Clinicians should check that information that has been provided has been understood; barriers to understanding should be explored and addressed.	CONSENSUS: Steering Committee

Summary of Key Messages: Acute Thoracic Spinal Pain

DIAGNOSIS	EVIDENCE LEVEL
Aetiology and Prevalence	
Pain may be referred to the upper thoracic spine from visceral structures and cervical spinal structures or arise in the thoracic interspinous ligaments, paravertebral muscles and zygapophyseal joints	*LEVEL IV: Kelley 1997; Dwyer et al. 1990; Aprill et al. 1990; Fukui et al. 1996; Feinstein et al. 1954; Kellgren et al. 1939; Hockaday and Whitty 1967; Cloward 1959; Kellgren 1939; Dreyfuss et al. 1994
Men and women aged over 60 are at risk for spontaneous osteoporotic fractures of the thoracic spine; extent of vertebral deformity and multiple fractures appear linked with pain intensity.	*LEVEL IV: Ross et al. 1994; Patel et al. 1991; Huang et al. 1994
Clinicians should be alert to the potential for rare, serious conditions presenting as acute thoracic spinal pain; however most cases of thoracic spinal pain are of mechanical origin.	*LEVEL IV: Deyo and Diehl. 1988
History	
History serves to differentiate sources of acute thoracic spinal pain to identify features of potentially serious conditions; however it carries little diagnostic weight.	CONSENSUS: Flynn 1996; Kenna and Murtagh 1989; Corrigan and Maitland 1988
Physical Examination	
The reliability of palpation for tenderness of the thoracic spine is good but its validity is unknown.	*LEVEL IV: Christensen et al. 2002
The reliability of motion palpation of the thoracic spine is marginal.	*LEVEL IV: Love et al. 1987; Christensen et al. 2002
Following blunt trauma, a negative clinical examination in the presence of a clear sensorium makes a thoracic spinal fracture unlikely.	*LEVEL IV: Durham et al. 1995; Samuels and Kerstein 1993
Despite the absence of supportive, scientific data on the utility of physical examination of the thoracic spine, such examination provides an important opportunity to identify features of serious conditions.	*LEVEL IV: Deyo et al. 1988; Malawaski et al. 1991; Durham et al. 1995; Samuels and Kerstein 1993
Ancillary Investigations	
In the absence of trauma, plain radiography is of limited use in defining the cause of pain.	*LEVEL IV: Wood et al. 1995; Nathan 1962; Crawford and Singer 1995
Fractures are more likely to occur in people over age 60 with a history of blunt trauma; a lower threshold for investigation is warranted in this group.	*LEVEL IV: Frankel et al. 1994; Durham et al. 1995; Meldon and Moettus. 1995; Samuels and Kerstein 1993
In the presence of trauma, xray of the thoracolumbar spine is not indicated in those who are awake, alert and have no clinical evidence of injury; however those with equivocal or positive clinical findings or with an altered level of consciousness should undergo thoracolumbar spine evaluation.	*LEVEL IV: Samuels and Kerstein 1993; Durham et al. 1995
CT scanning is only indicated for the evaluation of the neural canal and posterior elements of the thoracic spine when fractures have been detected with plain films.	*LEVEL IV: Keene et al. 1982
There is no research to inform ancillary investigations for acute thoracic spinal pain; investigations should be selected on the basis of clinical features suggesting the presence of serious conditions.	CONSENSUS: Steering Committee
Terminology	
The appropriate labels for non-specific 'mechanical' thoracic spinal pain are 'thoracic spinal pain of unknown origin' or 'somatic thoracic spinal pain'.	CONSENSUS: Merskey and Bogduk 1994
PROGNOSIS	**EVIDENCE LEVEL**
There is a lack of published data on the natural history and influence of prognostic risk factors for acute thoracic spinal pain.	NO EVIDENCE

Acute Thoracic Spinal Pain continued

INTERVENTIONS	EVIDENCE LEVEL
Evidence of Benefit	
Spinal Manipulation — There is evidence from one small study that spinal manipulation is effective compared to placebo in thoracic spinal pain.	LEVEL II: Schiller 2001

Note: *Indicative only. A higher rating of the level of evidence might apply (refer to the note in Chapter 1: Executive Summary, Limitations of Findings).

- Research into the reliability and validity of history taking for acute thoracic spinal pain, in particular for features characteristic of serious conditions.
- Research on the reliability and validity of clinical signs in acute thoracic spinal pain used for the detection of serious conditions and for accurate diagnosis and treatment of somatic causes.
- Research into thoracic spinal pain of somatic or uncertain origin to allow more accurate labelling and targeted treatment.
- Research into the diagnostic utility and cost-effectiveness of investigations for acute thoracic spinal pain in patients without trauma.
- Research into the natural history and prognostic risk factors for acute thoracic spinal pain to inform prevention and treatment strategies.
- Research on chiropractic and other treatments with rigorous trial design.

DIAGNOSIS

>Aetiology and Prevalence

Potential causes of thoracic spinal pain may be classified as:

- painful conditions of the thoracic spine
- conditions referring pain to the thoracic spine

A classification of these causes is presented in Table 5.1. Serious conditions causing pain in the thoracic spine include those that may cause progressive pain and disability, neurological deficits and even death. These include neoplastic and inflammatory disorders, infections and fractures. Disc protrusion is another serious condition that can also cause progressive pain and disability.

Conditions referring pain to the thoracic spine have anatomical structures whose sensory afferent neural pathways converge with those of the sensory nerves of the thoracic spine in the central nervous system. They can be classified as:

- somatic conditions referring pain to the thoracic spine
- visceral conditions referring pain to the thoracic spine

Some of these conditions are serious, such as cervical disc disorders and myocardial ischaemia, and are important to include in the differential diagnosis of acute thoracic spinal pain.

Key Message

Pain may be referred to the upper thoracic spine from visceral structures and cervical spinal structures or arise in the thoracic interspinous ligaments, paravertebral muscles and zygapophyseal joints. (*LEVEL IV)

Serious Conditions

Tumours

Cancer is a rare, but important cause of thoracic spinal pain. In a study of 1,975 ambulatory patients in primary care addressing the epidemiology of low back pain, approximately 315 (16%) had thoracic spinal pain as their chief complaint (Deyo and Diehl 1988). Of these, two had cancer as the cause of pain yielding a pre-test probability of cancer of 0.63% (similar to the figure of 0.66% for low back pain in this study). The predictive power of clinical features in the diagnosis of cancer as a cause of thoracic spinal pain has not been determined.

Spinal metastases are the commonest form of cancer in the thoracic spine, being most common in the T4 and T11 regions (Simeone and Lawner 1982). The largest hospital-based series comprised 28 cases (Kleineman et al. 1978). Intractable day and night pain was common. In this series, 43% presented with interscapular or dorsal pain. Eleven percent presented with 'girdle-type' pain and stabbing intercostal pain secondary to intercostal nerve involvement. Seven percent had anterior chest pain and 39% had signs of neurological deficit. The average interval between onset of pain and treatment was four months.

There is limited information specifically addressing the presenting features of primary thoracic spinal cancer. Two hospital-based series are available. The first, a series of 29 cases included two adults with thoracic spinal malignancies presenting with one month of pain. One had a chondrosarcoma and the other had a plasmacytoma. This series had nine cases of thoracic spinal cancer, all presenting with pain of greater than three months duration and some with signs of impair-

Table 5.1

A Systematic Classification of Causes of Acute Thoracic Pain

Painful Conditions of the Thoracic Spine	
Serious conditions	Infection; fracture; neoplastic disorders; inflammatory disorders; disc protrusion
Mechanical conditions	Discogenic pain; zygapophyseal joint pain; other structures
Conditions Referring Pain to the Thoracic Spine	
Somatic conditions	Disorders of cervical zygapophyseal joints, muscles and discs
Visceral conditions	Myocardial ischaemia; dissecting thoracic aortic aneurysm; peptic ulcer; acute cholecystitis; pancreatitis; renal colic; acute pyelonephritis

ment of nerve root and/or spinal cord function (Delamarter et al. 1990).

The second was a series of 22 cases of spinal osteoid osteoma and osteoblastoma, of which six were in the thoracic spine (Ozaki et al. 2002). Of these, only two cases had pain for three months or less, one with an osteoid osteoma presenting with scoliosis and the other with osteoblastoma presenting with spinal palsy. Amongst the cases with longer durations of pain, scoliosis, spastic diplegia and leg pain were amongst the presenting symptoms. Although the sample was small, the study suggests that painful scoliosis and long tract neurological signs in the lower limbs should raise the possibility of primary thoracic spinal malignancy.

Inflammatory Arthritis

Ankylosing spondylitis can affect the discovertebral, zygapophyseal, costovertebral and costotransverse joints and paravertebral ligamentous structures of the thoracic spine (Khan 1994). Ankylosis of these structures can lead to a marked limitation of chest expansion (Stewart et al. 1976). However, it would be very unusual for an individual with ankylosing spondylitis to present with only thoracic spinal pain. The diagnosis is usually confirmed by the radiological demonstration of sacro-iliitis (Calin 1993).

Rheumatoid arthritis rarely affects the thoracic spine (Hastings 1994). It has been shown to involve the costotransverse and costovertebral joints as well as the discs in the thoracic spine (Weinberg et al. 1972; Bywaters 1974); zygapophyseal synovitis may rarely present as an epidural mass (Hastings 1994). In rheumatoid arthritis, compression fractures of the thoracic spine secondary to the associated osteoporosis are more likely causes of spinal pain.

Infection

The pre-test probability of infection as the cause of back pain in primary care is less than 0.01% (Khan 1994). These infections include osteomyelitis, discitis and epidural infections.

The largest study of pyogenic infection of the spine is a retrospective series of 442 patients from a Polish hospital (Malawski and Lukawski 1991). In this series, 349 (35%) of the 997 affected vertebrae were in the thoracic spine and of these, 23% presented with pain of less than three months duration. Some aspects of clinical presentation for the series as a whole are outlined in Table 5.2. These data indicate that spinal infection can persist for a long time without being recognised. However, fever is the major alerting feature.

In a German retrospective series of 13 patients with epidural spinal infection causing a neurologic deficit, seven had pain in the thoracic spine (Kuker et al. 1997). All had intense back pain and no one experienced a neurologic deficit without prior or simultaneous back pain.

Fractures

Traumatic Fractures

Elements of the thoracic vertebrae may be fractured as a result of blunt injuries or falls.

Osteoporotic Fractures

Although the focus in the literature on osteoporosis-related fractures has been on women, a large population study in Finland found a prevalence of 6.2 per 1,000 in men vs 3.9 per 1,000 in women (Santavirta et al. 1992). The age-adjusted odds ratio was 1.85 (95% CI 1.45, 2.36). In men the prevalence increases gradually with age whereas in women it increases abruptly after the age of 65. This study drew the conclusion that the great majority of these fractures were asymptomatic as no differences in self-assessed general health and use of analgesics were found between fracture and non-fracture groups.

In an age-stratified random sample of American women 50 years and above, a similar relationship between age and the prevalence of vertebral fractures was found (Melton et al. 1989). This ranged from 6.5% in those aged 50–59 years to 77.8% in those over 90. The peak areas for fractures, (96% of which were non-traumatic), was in at T7–8, T11 and L1. Bone mineral density in the lumbar spine did not entirely account for the age-related increase in fracture prevalence, suggesting that it is not a perfect indicator of bone fragility.

The assessment of bone density in the spine is usually done on the lumbar spine, however for technical reasons, this may underestimate the degree to which the thoracic spine is affected by osteoporosis. A hospital-based study of 96 patients with normal lumbar densitometry found thoracic spinal fractures in 11; of these, nine had pain related to these fractures (Bhambhani et al. 1992). Normal bone densitometry and radiographs of the lumbar spine do not necessarily indicate the

Table 5.2

Aspects of Clinical Presentation for a Series of 442 Patients with Pyogenic Infection of the Spine

Aspect of Presentation	Definition	Proportion of all Cases
Disease Onset		
Acute	High fever, violent pains and malaise	50%
Subacute	Fever, moderate pain and slight malaise	33%
Mild	Afebrile, local pain and otherwise well	17%
Clinical Features		
Infection	fistula, abscess, meningitis	20%
Neuromotor deficit	paralysis/paresis	5%
Interval from Onset of Symptoms to Presentation		
< 3 months		23%
3–6 months		17%
6–12 months		13%
1–8 years		47%

Note: Based on data from Malawski and Lukawski (1991).

absence of osteoporosis in the thoracic spine in those with risk factors for osteoporosis.

An association between recent fractures of the thoracolumbar spine and pain has been established in a Hawaiian study of 203 women over 50 who underwent serial radiographs approximately two years apart (Ross et al. 1994). In comparison with women with no fractures, the odds ratios for increased frequency of pain in women with prevalent (pre-existing but no new) fractures was 1.7 (95% CI 0.5, 5.6). For those with incident (pre-existing plus recent) fractures the odds ratio was 6.4 (95% CI 2.6, 15.6). Odds ratios for back pain and disability for those with incident fractures are reported in Table 5.3.

Key Message

Men and women aged over 60 are at risk for spontaneous osteoporotic fractures of the thoracic spine; extent of vertebral deformity and multiple fractures appear linked with pain intensity. (*Level IV)

In an extension of the above study (Huang et al. 1994), the adjusted odds ratio for the incidence of back pain with one incident fracture in the preceding 4.3 years was 2.79 (95% CI 1.50, 2.19) compared with 1.3 (95% CI 0.94, 2.12) for early fractures (between 4.3 and 6 years prior) and 0.73 (95% CI 0.45, 1.20) for fractures prior to this. For two incident fractures the odds ratio increased to 7.8 and for three to 21.7. Extrapolation of these results to Australian women requires some caution given the different ethnic background of women in Hawaii.

A triggering event for osteoporotic fractures is often not present. In a hospital-based case series of 30 patients with acute thoracolumbar vertebral compression fractures, 46% were classified as spontaneous, 36% associated with a trivial strain and 18% associated with moderate or severe injury (Patel et al. 1991).

The severity of the vertebral deformity has been correlated with more severe back pain and disability. In a cross sectional population-based study, 2992 Caucasian women were xrayed from T5 to L4 and classified according to their most deformed vertebra (Huang et al. 1994). These included wedge, endplate and crush deformities. Women with deformities ≥ 4 SD below the mean had a 1.9 (95% CI 1.5, 2.4) times higher risk of moderate to severe back pain and a 2.6 (95% CI 2.0, 3.2) times higher risk of disability involving the back.

Key Message

Clinicians should be alert to the potential for rare, serious conditions presenting as acute thoracic spinal pain; however, most cases of thoracic pain are of mechanical origin. (*Level IV)

Disc Protrusion

Disc protrusion is a morphological condition of the thoracic spine that is not to be confused with discogenic pain.

Thoracic disc protrusion is an uncommon condition, and accounts for only 0.15% to 1.8% of all surgically treated intervertebral disc abnormalities (Love and Schorn 1965; Otani et al. 1982; Bhole and Gilmer 1984). It affects the sexes equally and is more common between the fourth and sixth decades with a peak in the fourth decade (Otani et al. 1982; Bhole and Gilmer 1984). Although thoracic disc protrusions have been reported at every level, 75% occur below T8 with a peak at T11–12 where there is greater spinal mobility (Love and Schorn 1965; Haley and Perry 1950; Arce and Dohrmann 1985). Central protrusions are the most common (Love and Schorn 1965).

Disc protrusions in the thoracic spine with or without the contribution of facet joint hypertrophy can cause spinal stenosis and spinal cord compression with long tract signs of myelopathy (Skubic 1993). These include leg weakness, spasticity, ataxia, numbness, bowel and bladder disturbance with associated sphincter dysfunction. As such it needs to be differentiated from other causes of long tract signs including spinal cord neoplasia and multiple sclerosis.

Radicular symptoms may also occur. In a series of 67 cases, radicular symptoms, usually pain and dysaesthesia, occurred in 49% of patients; 93% had symptoms ascribable to myelopathy and 42% had both radiculopathy and myelopathy (Russell 1989). In those with myelopathy, sensory and motor symptoms were equally common. In 57% of cases the onset of symptoms was gradual. The onset was sudden in 13% and intermittent but worsening in 24%. Typically, therefore, the symptoms are myelopathic in nature and of gradual onset.

There are no data that implicate thoracic disc protrusion as a source of spinal pain in the absence of neurological features.

Mechanical Conditions

Anatomical Origins of Thoracic Spinal Pain

The evidence (or lack of evidence) for the anatomical origins of thoracic spinal pain is summarised in an extensive review of the literature by Chua (1996). Four criteria were established for structures to be labelled as a source of pain:

- Evidence of innervation of the structure.
- Evidence of pain provocation in experimental studies on normal volunteers.
- Evidence of pain relief in clinical studies on patients where the structure is selectively anaesthetised.
- The structure should be susceptible to diseases or injuries that are known to be painful.

Attempts to define a specific structure as the source of pain include the terms discogenic pain, facet (zygopophyseal) joint pain, costovertebral joint pain and costotransverse joint pain, however unless these diagnoses are supported by evidence from tests on the putative sources that relieve or reproduce the pain, they remain speculative.

Table 5.3

Odds Ratios for Incident Fractures of the Thoracolumbar Spine for Back Pain and Disability in Women Over 50

	Age Adjusted Odds Ratio	95% CI	Multiply Adjusted Odds Ratio	95% CI
Back pain since previous examination	5.5	2.5, 12.2	4.9	2.1, 11.2
Any severity of back pain > none in last 30 days	4.9	2.2, 11.0	4.2	1.8, 10.0
≥ 3 back disability responses	4.0	1.2, 13.4	3.8	1.0, 14.5

Note: Based on data from Ross et al. (1994). Odds ratio were adjusted for age and multiply adjusted for self-reported disc disease, spinal arthritis and traumatic back injury.

Discogenic Pain

According to the taxonomy produced by the International Association for the Study of Pain (Merskey et al. 1994), thoracic discogenic pain is defined as thoracic spinal pain, with or without referred pain, stemming from a thoracic intervertebral disc. The difficulty in diagnosing thoracic discogenic pain lies in the wide variety and distribution of reported symptoms (Schellhas 1994; Skubic 1993). There are no specific clinical features that differentiate pain arising from the disc from that arising from other somatic structures in the thoracic spine.

The diagnosis of discogenic thoracic spinal pain requires confirmation by an appropriate response to selective anaesthetisation or provocation discography of the painful disc. These procedures are expensive, not widely available and carry a risk of complications including pneumothorax. They are not applicable in primary care. It is not surprising, therefore, that there are no data on the prevalence of thoracic discogenic pain in primary care. Even data from tertiary care is limited. The only study on this topic simply demonstrated that thoracic discography could be performed (Schellhas et al. 1994).

In summary, on the evidence available, it is difficult to justify making a diagnosis of thoracic discogenic pain in primary care in those with acute thoracic spinal pain.

Zygapophyseal Joint Pain

The strongest evidence pertains to the zygapophyseal joints as a source of pain. They have been shown to be painful in normal volunteers and in those with thoracic spinal pain who have had their pain relieved by blocks of these joints (Dreyfuss 1994). The pain may be referred from the cervical zygapophyseal joints and felt paravertebrally or just lateral to the paravertebral region (Fukui 1996).

Other Structures

Thoracic interspinous ligaments and paravertebral muscles have been shown experimentally to be potential sources of pain (Kellgren 1939), but no controlled clinical studies have been published that indicate if these structures are sources of pain, or how commonly.

Because costovertebral joints are innervated they are potentially a source of pain, however as the techniques of blocking these joints have not been described, the prevalence of pain arising from these joints is unknown.

The thoracic spinal dura mater, longitudinal ligaments and costotransverse joints are innervated (Groen et al. 1990; Groen et al. 1988; Wyke 1975) but there have been no clinical or experimental studies to implicate them as sources of pain.

With respect to the costotransverse ligaments, there are no data on their innervation and no clinical or experimental studies to identify them as a source of pain.

Pain of Mechanical Origin

Pain of mechanical origin should include any pain that is somehow related to movement or sustained posture. In a clinical sense, however, this category specifically excludes serious conditions, even though they are often affected by movement or sustained posture. Synonyms for mechanical pain, include:

- Spondylogenic or non-radicular pain (Kenna and Murtagh 1989)
- Somatic dysfunction (Greenman 1989)
- Painful minor intervertebral dysfunction (Maigne 1996)
- Hyper- or hypo-mobility lesion of the mobile intervertebral joint complex (Corrigan and Maitland 1988)
- Intervertebral derangement (McKenzie 1981)
- Vertebral subluxation (Charlton 1991)

Proponents of these different interpretations base their diagnoses on their own defined set of historical and examination findings, with or without the exclusion of non-mechanical causes of pain. They are largely based on untested and unvalidated biomechanical theories and observations. Some ascribe their diagnosis or findings to specific anatomical structures such as zygapophyseal joints or intervertebral discs. Others claim to localise the problem to a specific segment or segments, based principally on finding altered joint or muscle function, minor changes in the anatomical relationships of bony and soft tissue landmarks, and changes in skin texture, sensation and response to stimuli. However, no paradigm has been subjected to rigorous tests of reliability and validity of diagnostic tests, or efficacy of treatment.

Readily available investigations such as xrays and blood tests serve principally to exclude non-mechanical causes of pain. However, some proponents of mechanical models of thoracic spinal pain place significant diagnostic importance on positive radiological findings such as disc bulges, which is not supported by the literature.

Conditions Referring Pain to the Thoracic Spine

The thoracic spine may be the source of referred pain, or a site to which pain may be referred. Therefore the location of pain in the thoracic region does not necessarily imply a local (thoracic) source. These structures may be somatic or visceral.

Somatic Sources of Pain

There are no clinical or epidemiological studies; however the following sources of somatic referred pain have been studied:

- Cervical zygapophyseal joints, especially those at the C5–6 and C6–7 spinal levels
- Cervical intervertebral discs and nerve roots, especially at the C5–6 and C6–7 spinal levels

Experimental studies in normal volunteers and in patients have demonstrated that pain from structures in the cervical spine can be referred into the upper thoracic spinal region. Referred pain in this region can arise from the lower cervical zygapophyseal joints (Dwyer et al. 1990; Aprill et al. 1990; Fukui et al. 1996), the cervical muscles (Feinstein et al. 1954; Kellgren 1939; Hockaday and Whitty 1967), or the cervical intervertebral discs (Cloward 1959; Schellhas and Pollei 1994). The assessment of thoracic spinal pain, therefore, requires consideration of possible cervical sources of pain.

Visceral Sources of Pain

Referral of pain from visceral structures should always be considered, especially when there are no clear mechanical features to the pain and other non-spinal symptoms are present. Visceral conditions that may refer pain to the thoracic spine are listed in Table 5.1.

Myocardial ischaemia usually presents with anterior chest pain or heaviness and sometimes nausea, but occasionally presents with pain radiating to the back (Kelley 1997).

The pain of a dissecting thoracic aortic aneurysm is usually felt in the chest, but can radiate to the back, most commonly the lower back.

The pain of a peptic ulcer on the posterior wall of the stomach or duodenum may also radiate to the back, however it usually is triggered, or in some cases relieved by eating.

Gall bladder pain may be referred to the right infrascapular region and is often accompanied by nausea and vomiting.

The pancreas is another posterior abdominal structure that may refer pain to the back, around the level of the thoracolumbar junction. The pain of acute pancreatitis may be so severe that there may be difficulty determining whether it originates in the abdomen or the back.

Renal pain caused by obstruction of the ureteropelvic junction or acute pyelonephritis is usually felt in the costovertebral area or the flank.

Conditions Referring Pain from the Thoracic Spine

Although this document is focused on pain within the anatomical limits of the thoracic spine, a brief discussion of sites to which thoracic spinal pain can refer is warranted as this is often a feature of the presentation of thoracic spinal pain.

Experimental studies in normal volunteers and patients have demonstrated that pain from thoracic spinal structures can be referred to the posterior and anterior chest wall and into the upper limb. Such patterns of referred pain have been demonstrated for the thoracic interspinous ligaments (Feinstein et al. 1954; Kellgren et al. 1939; Hockaday and Whitty 1967), and the thoracic zygapophyseal joints (Dreyfuss et al. 1994; Fukui et al. 1997). This type of referred pain is described as dull and aching; it tends to be poorly localised, not corresponding to dermatomes, and is felt deeply in the tissues. Pain from distended zygapophyseal joints of normal volunteers between T3 and T10 follows reasonably constant patterns of referral (Dreyfuss et al. 1994). Referral zones spread from one half of a segment superior to two and a half segments inferior to the joint and extend laterally to no further than the posterior axillary line. Pain from the C7–T1, T1–2 and T1–3 is referred variably to an area including the suprascapular region, the medial angle of the scapula and the midscapular region (Fukui et al. 1997). Pain from the T11–12 joints is felt paravertebrally in the lower thoracic and upper lumbar spines.

Pain outside the thoracic spine has been documented in a hospital-based case series of 30 patients with acute thoracolumbar vertebral compression fractures. Areas of radiation included the flanks and anteriorly (66%), the legs (6%), the abdomen (20%) and the chest (13%) (Patel et al. 1991).

Thoracic spinal pain, therefore, may not be restricted to the thoracic spinal region, but may spread to involve the trunk wall. The distribution of referred pain does not imply any particular source but it is a reasonable guide to the segmental location of the source structure. The higher the location of referred pain, the higher the segmental origin of the source. Pain patterns should usually indicate the source to within one segment, but this prediction has not been formally tested.

Thoracic spinal pain has also been documented as spreading to the medial aspect of the arm following noxious stimulation of the T1 interspinous tissues (Feinstein et al. 1954; Kellgren etal. 1939; Hockaday and Whitty 1967), but it has not been produced from thoracic segments below T1. Thus, pain in the arm can be expected in the case of T1 disorders, but there is no experimental data to justify belief in referral from lower thoracic segments.

The textbook literature describes a 'T4 syndrome' in which pain and paraesthesia in the upper limbs has been ascribed to 'segmental dysfunction' between T2 and T7 (McGuckin 1986). This relationship, however, was based on manual assessment using techniques of unknown reliability and validity, and on response to manipulative therapy that was poorly documented and uncontrolled. No causal relationship was established by an appropriate experimental protocol of controlled local anaesthetic blocks to these areas. Cervical causes of the arm and head symptoms were not excluded.

Some authorities have reported that in some 40% of cases of low back pain, the origin of the pain is in the thoracic spine or at the thoracolumbar junction (Maigne 1980), but the diagnostic techniques on which these claims were based are of unknown validity.

Prevalence of Conditions Causing Acute Thoracic Spinal Pain

The Australian Institute of Health and Welfare (2000) describes the prevalence and incidence of 'back pain' but does not distinguish thoracic spinal pain. Three estimates of the prevalence of thoracic spinal pain are available from the literature on spinal pain in general. A pain clinic in the Netherlands reported a relative incidence of cervical, thoracic and lumbar spinal pain in their patient cohort as 5:2:20, respectively (Stolker et al. 1993). In a primary care series of 1,975 ambulatory patients with back pain, approximately 16% had thoracic spinal pain as their chief complaint (Deyo and Diehl 1988). In a Hawaiian study of 645 postmenopausal women the prevalence in the preceding 4.3 years of pain in the neck and above the shoulder blades was reported as 7.6%. The prevalence of pain between the shoulder blades and the lowest rib level was 4.8% (Huang et al. 1994). Prevalence data for particular conditions underlying presentations are presented in Table 5.4.

>History

This chapter deals with aspects of history-taking that are specific to the problem of acute thoracic spinal pain and differ from the elements of history-taking for other pain problems. For a discussion of pain history in acute musculoskeletal pain in general, the reader is referred to Chapter 2: Acute Pain Management.

The evidence-base for the aetiology and pathology of acute thoracic spinal pain on which history taking should be based is far from comprehensive. There is no universally accepted method of eliciting a history and no research on the reliability and validity of the elements of a history in relation to acute thoracic spinal pain. Where possible, the following information derives from the evidence on the aetiology of thoracic spinal pain. As a priority, the aim is to assess for the presence of serious conditions presenting as thoracic pain. Reference has been made to texts on musculoskeletal pain and internal medicine where deficiencies exist (Flynn 1996; Kenna and Murtagh 1989; Corrigan et al. 1988).

Pain History

Site and Distribution

Although these guidelines are focused within the anatomical boundaries of the thoracic spine, it is important to obtain an accurate history of all painful areas to detect sources of pain referred to or from the thoracic spine (see Aetiology and Prevalence).

For example, pain experienced concurrently in the neck and the upper back suggests a cervical spinal origin. Anterior chest pain in association with thoracic spinal pain raises the possibility of ischaemic heart disease or dissection of the thoracic aorta. Pain may be referred from the thoracic spine to structures whose innervation arises from a similar level or levels in the spinal cord, commonly structures in the chest and abdominal walls.

Table 5.4
Relative Prevalence of Local Causes of Thoracic Pain

Frequency	Entities	Prevalence
Rare conditions	Primary and secondary neoplasia	0.63% (Deyo et al. 1988)
	Disc protrusion	0.15% of all surgically treated disc abnormalities (Love and Schorn 1965)
	Rheumatoid arthritis	Unknown
	Spinal infection	< 0.01% (Liang and Komaroff 1982)
Uncommon conditions	Traumatic fractures	Unknown
Common conditions	Somatic pain	16% of presentations with back pain in primary care (Deyo and Diehl 1988)
	Osteoporotic fractures	6.5% in 50–59 year olds and 77.8% in > 90 year olds (Melton et al. 1989)

Similarly, recent history of penetrating injury in the form of a surgical or dental procedure, catheterisation or cannulation, a wound, or self-injection constitutes an alerting feature for possible thoracic osteomyelitis, epidural abscess or discitis.

Quality

The quality of pain may be particularly important in the thoracic spine as the differentiation of somatic, radicular and visceral pain is of diagnostic importance. Somatic pain is usually deep, dull and aching. Bone pain is often described as 'boring'. Muscular pain is often called 'cramping' or 'spasm' (Kenna and Murtagh 1989).

Radicular pain is mostly sharp and 'electric' or 'shooting'. It may be difficult to differentiate this from the sharp pain of pleurisy, although the association with breathing may be helpful here. Neuropathic pain, for example in shingles, is often 'burning'. In both radicular and neuropathic pain, sensory disturbance in the associated dermatome may be present (Kenna and Murtagh 1989).

Visceral pain is dull at first and sharp when lining tissues such as the peritoneum become involved. In the case of cardiac pain, the sensation may be more of a tightness or a heaviness in the chest.

Aggravating and Relieving Factors

Biomechanically, the upper thoracic spine moves with gross movements of the neck and the lower thoracic spine moves with the trunk. Consequently, pain in the upper thoracic spine may be aggravated or relieved by certain movements and postures of the neck, and lower thoracic spinal pain affected by movement and postures of the trunk. In common with pain arising from the pleura, thoracic spinal pain may be aggravated by coughing, sneezing and deep inspiration (Kenna and Murtagh 1989). Where movement and posture has no effect on the severity of the pain, serious conditions should be considered. The exception here is in the mid thoracic spine, which, braced by ribs, may be less susceptible to movement stresses.

Other Aspects of the Pain History

Pain on general exertion may suggest ischaemic heart disease, although if the exertion involves specific thoracic spinal movements, such as twisting, a somatic cause would be more likely. Such relationships are not constant, however, and caution should be exercised in taking inferences from particular patterns of periodicity (Flynn 1996).

Conducting a broader enquiry into the general medical and psychosocial history improves knowledge of how the pain may be influenced by other biological and psychosocial factors. A history of the following clinical features alerts to the possibility of serious conditions as causes of acute thoracic spinal pain:

Chest pain raises the possibility of cardiac, vascular and pulmonary conditions particularly if associated with respiratory symptoms.

Fever accompanying the chest pain may occur in pulmonary infections. When it accompanies abdominal or flank pain, acute pyelonephritis and cholecystitis should be considered.

Unexplained weight loss and fatigue may occur with malignant causes of thoracic spinal pain.

Abdominal pain which waxes and wanes in association with thoracic spinal pain raises the possibility of biliary or renal colic.

Shortness of breath, cough and abdominal symptoms raise the possibility of cardiac and visceral disorders.

While it is acknowledged that clinical assessment lacks reliability and validity, it enables the clinician to investigate the index complaint and identify potentially serious conditions. Table 5.7 summarises features ('red flags') that may be associated with serious conditions such as malignancy, infection and fracture. While the predictive values of these alerting features have not been tested specifically in relation to thoracic spinal pain, their presence in conjunction with acute thoracic pain should prompt further investigation.

Key Message

History serves to differentiate sources of acute thoracic spinal pain to identify features of potentially serious conditions; however it carries little diagnostic weight. (Consensus)

>Physical Examination

Systems and techniques for the physical examination of the thoracic spine are based on the general principles of physical examination and on extrapolation of systems and techniques used for the lumbar spine. There are few data on the reliability of physical signs in the examination of the thoracic spine.

A physical examination of the thoracic spine may include inspection, palpation and movement.

Inspection

The purpose of inspection is to identify visible abnormalities. In the context of the thoracic spine this usually means the detection of postural abnormalities or deformities.

Table 5.5

Distribution of Agreement Amongst Four Examiners Concerning the Presence or Absence of Dysfunction in 10 Unmarked Thoracic Spinal Segments in 15 Subjects

Level of Agreement	Observed Agreement	Expected Agreement
Total agreement (4–0, 0–4)	61	20.75
Slight agreement (3–1, 1–3)	56	75
Total disagreement (1–2)	33	54.25

Note: Based on data from Johnston et al. (1983).

Posture

Spinal posture may influence the range and pattern of movement (Magarey 1994). It has been suggested that pain influences posture, and that postural abnormalities may contribute to the development of spinal pain syndromes (Enwemeka et al. 1986). However, a causal relationship in this regard has not been established.

In a small study examining the reliability of the assessment of cervicothoracic and shoulder posture, the intra-examiner reliability of three examiners using 10 subjects registered a Kappa coefficient of 0.825 (Griegel-Morris et al. 1992). The inter-examiner reliability for the same examiners using five subjects was substantial at 0.611.

An association between the incidence of interscapular pain and the more severe degrees of forward head posture, thoracic kyphosis and rounded shoulders has been shown in a convenience sample of 88 subjects aged 20 to 50 years ($\chi^2 = 6$, $df = 2$, $p < 0.05$). This association did not apply for pain severity or frequency. However, there was no clear association between cervicothoracic posture and pain in a study comparing 18 patients with pain and 18 pain-free controls (Refshauge et al. 1995).

Deformity

Because of the functional disability that it can impose, thoracic deformity is a problem in its own right; however, its relationship to pain is unclear.

Thoracic kyphosis is largely determined by the shape of the vertebral bodies and discs (Edmonston et al. 1993) particularly in the elderly. In younger people it may be increased due to Scheuermann's disease or simply to poor habitual posture. The thoracic kyphosis increases with age and has little potential for change, due to age related anatomical changes and decreased joint mobility (Singer et al. 1990). In such cases, postural correction is largely achieved through compensatory changes in the lumbar and cervical regions and the shoulder girdle.

There are no published data on the reliability of clinical assessment of kyphosis. With respect to validity, one study has shown that in older women with severe thoracic kyphosis secondary to osteoporosis, the degree of back pain and disability may be no greater than in women without such marked structural change (Ettinger et al. 1994). However, mobility and functional activities are more likely to be impaired in individuals with severe thoracic kyphosis (Cook et al. 1993).

Scoliosis is the archetypical deformity of the thoracic spine. Although scoliosis can be accurately quantified by a variety of radiographic techniques and other techniques such as Moire fringe topography, there appear to have been no publications on the reliability of inspection to detect scoliosis. Moreover, there is no established relationship between scoliosis and pain. The pursuit of scoliosis in the assessment of thoracic spinal pain is relevant in the case of idiopathic scoliosis in adolescents, which may be progressive and have other sequelae such as respiratory compromise.

Palpation

Most palpatory tests for the thoracic spine are of a qualitative nature and lack quantitative accuracy. Study results indicate limited or poor reliability and no validity.

Johnston et al. (1983) reported an inter-examiner agreement of 79–86% amongst seven osteopathically trained students distinguishing between the presence and absence of deep muscular tension as an indication of dysfunction of marked thoracic spinal segments. However, the distribution of agreement amongst four of these examiners concerning the presence or absence of dysfunction in 10 unmarked thoracic spinal segments in 15 subjects revealed only a slight level of agreement (Kappa = 0.31). See Table 5.5.

Minucci (1987) reported an inter-examiner reliability of 82% for 114 manual examination tests (requiring 162 decisions) on five subjects examined by two experienced manipulative therapists 24 hours apart. The intra-examiner reliability for five subjects was reported at 86%, but the number of subjects was too small to permit any firm conclusions and no Kappa scores were reported.

The most recent study on palpation of the thoracic spine measured the levels of intra-observer and inter-observer agreement for tenderness on palpation of T1–8 in 29 subjects with suspected or confirmed angina and 27 controls (Christensen et al. 2002). Using a clinically acceptable definition of 'expanded agreement' as agreement to within one vertebral level, good Kappa scores of 0.63–0.77 for intra-observer agreement and 0.67–0.70 for inter-observer agreement were achieved.

A variety of abnormalities are alleged to be detectable on physical examination of the thoracic spine. However, notwithstanding the questionable reliability of detecting these abnormalities by palpation, many of them are evident in asymptomatic individuals, confounding the validity of these signs (Table 5.6).

Tenderness has been shown to be more common in thoracic spinal pain. In a study of 60 students, a threshold for tenderness of 50 N of pressure was established with a dolorimeter over thoracic transverse processes, there were significant overall and individual differences ($p < 0.001$) between those with thoracic spinal pain compared to those without such pain (Bryner et al. 1989). However no studies have assessed the validity of any thoracic palpatory test against a criterion standard as a criterion standard is yet to be established.

Key Message

The reliability of palpation for tenderness of the thoracic spine is good but its validity is unknown. (*Level IV)

Table 5.6

Abnormal Palpatory Findings in Examination of Segments T1–8 in 25 Asymptomatic Subjects

Abnormality	Proportion of Vertebrae Exhibiting Abnormality	Most Common Location of Abnormality	*p* Value
Rotated vertebrae	15%	T3, T5	
Prominent or depressed vertebrae	15%	T4 and T5	< 0.05
'Thickening' of the interspinous ligaments	15%	T1–2, T1–3	< 0.01
'Thickening' in paraspinal tissues	33%	Left T4	< 0.05
Hypomobile passive accessory intervertebral movements	54%*	T4 and T5	< 0.01
Discomfort	33%	T3–5	< 0.05
Pain	4%	T3–5	
Passive physiological intervertebral extension	46%	T3–4	

Note: Based on data from Minucci (1987).

Movement

There is no literature dealing with the reliability of the assessment of gross movement restriction of the thoracic spine. Available data pertain to the excursion of the trunk as a whole during movements of the lumbar spine (McCombe et al. 1989), not to intrinsic movements of the thoracic spine.

With respect to motion palpation of individual segments, one study has reported that the T9–10 segment is the most hypomobile (Love and Brodeur 1987). However the reliability of the technique used to determine hypomobility is poor. The correlation coefficients for intra-examiner reliability of eight senior chiropractic students were greater than 0.300 ($p < 0.05$) for six students (range –0.065 to 0.648), but the correlation coefficients for inter-examiner reliability were no better than chance at 0.023 to 0.0852 (Love and Brodeur 1987).

Christensen et al. (2002) also tested the reliability of motion palpation in the sitting and prone positions. Even using the criteria for agreement within one segment, kappa scores for inter-observer agreement were only fair for sitting at 0.22 and for prone at 0.24. However for intra-observer agreement they were good at 0.59 to 0.68.

Key Message

The reliability of motion palpation of the thoracic spine is marginal. (*Level IV)

▶ *Alerting Features of Serious Conditions (see Table 5.7)*

While it is acknowledged that clinical assessment lacks reliability and validity, it enables the clinician to investigate the index complaint and identify potentially serious conditions.

Table 5.7 summarises some features described in the sections on History and Physical Examination that may be associated with serious conditions such as malignancy, infection and fracture. While the predictive values of these alerting features have not been tested specifically in relation to thoracic spinal pain, their presence in conjunction with acute thoracic pain should prompt further investigation.

In the detection of cancer in primary care patients with pain in the thoracolumbar spine, the study by Deyo and Diehl (1988) suggests that some signs are very poor predictors. Muscle spasm and spinal tenderness had positive likelihood ratios of only 0.5 and 0.4, respectively. Historical findings were much more useful in the detection of cancer.

Neuromotor deficits, when present, justify investigation in their own right, but they are uncommon in people with serious conditions who present with spinal pain. In one study, no patient with spinal cancer presented with neuromotor deficits, although two out of 13 subsequently developed paresis (Deyo and Diehl 1988). Neuromotor deficits were present in 5% of a series of 442 patients with spinal infection (Malawski and Lukawski 1991).

The likelihood ratios of a positive clinical examination indicating a fracture in the thoracolumbar spine in blunt trauma at trauma centres have been reported as 1.8 (Durham et al. 1995) and 44.6 (Samuels and Kerstein 1993). However, the definition of a positive clinical examination was not given in these studies. Nonetheless, the published figures indicate that clinical examination is highly specific but non-sensitive. Therefore, fracture is highly unlikely in those with no clinical abnormalities.

Key Message

Following blunt trauma, a negative clinical examination in the presence of a clear sensorium makes a thoracic spinal fracture unlikely. (*Level IV)

The presence of fever with or without long tract neurological signs and symptoms is an alerting feature for infection as a cause of thoracic spinal pain, even if it has been present for many years (Malawski and Lukawski 1991; Kuker et al. 1997).

Key Message

Despite the absence of supportive scientific data on the utility of physical examination of the thoracic spine, such examination provides an important opportunity to identify features of serious conditions. (*Level IV)

>Ancillary Investigations

Plain Radiography

The indications for medical imaging in people with thoracic spinal pain differ according to whether or not the onset of pain is associated with trauma.

Plain Radiography in the Absence of Trauma

Plain films play a role in detecting serious conditions associated with thoracic spinal pain when alerting features indicate such conditions. Given the increased odds of pain in those with recent osteoporotic fractures (Ross et al. 1994; Huang et al. 1994) and that these fractures often occur in the absence of trauma (Patel et al. 1991), risk factors for osteoporosis offer a relative indication for plain radiography. However there is no literature that adequately describes the sensitivity and specificity of plain films in the detection of other serious

Table 5.7

Alerting Features of Serious Conditions Associated with Acute Thoracic Spinal Pain

Feature or Risk Factor	Condition
Minor trauma (if > 50 years, history of osteoporosis and taking corticosteroids)	Fracture
Major trauma	
Fever	Infection
Night sweats	
Risk factors for infection (e.g. underlying disease process, immunosuppression, penetrating wound)	
Past history of malignancy	Tumour
Age > 50	
Failure to improve with treatment	
Unexplained weight loss	
Pain at multiple sites	
Pain at rest	
Night pain	
Chest pain or heaviness	Other serious conditions
Movement, change in posture has no effect on pain	
Abdominal pain	
Shortness of breath, cough	

conditions in the thoracic spine. In cases where clinical features suggest the possibility of cancer or infection, MRI scans and bone scans are the preferred imaging modalities as they are likely to be more sensitive in detecting these conditions.

For other conditions there is little evidence that plain film findings can be used to determine the cause of pain in the thoracic spine. Disc space narrowing at multiple levels is a common finding from the third decade of life, with an equal prevalence in symptomatic and asymptomatic individuals (Wood et al. 1995). It is associated with other age changes such as formation of osteophytes, particularly in the mid-thoracic region (Nathan 1962; Crawford and Singer 1995). In contrast, zygapophyseal joint degeneration is most common at the C7–T1 and T11–12 levels (Shore 1935). Costovertebral joint osteoarthrosis is also a common finding, affecting 50–60% of individuals by 40 years (Nathan et al. 1964).

Disc calcification has been reported in 75% of people with protruded thoracic discs, but in only 4% for unaffected individuals (McCallister and Sage 1976), giving it a positive likelihood ratio of 18.8. This makes disc calcification a useful screening sign for thoracic disc protrusion, but it has no relationship to thoracic pain.

Radiographically confirmed Scheuermann's kyphosis has been found to be associated with an increased prevalence of back pain and a decreased prevalence of lower extremity pain than in controls (Table 5.8). More specifically, pain in the thoracic spine was present in 28% of patients with Scheuermann's disease compared with 3% of controls. On the other hand, 72% of patients with Scheuermann's disease do not have back pain and causality is unclear.

Key Message

In the absence of trauma, plain radiography is of limited use in defining the cause of pain. (*Level IV)

Plain Radiography in the Presence of Trauma

In the younger population, significant trauma is usually required to cause fractures in the thoracic spine. Studies in this area are mostly retrospective and usually are based in accident and emergency departments. As such they suffer from an unknown number of 'missed cases'.

In an English series of 50 patients with acute fractures from T1–9, all were involved in road or train accidents or had fallen from a height (Grootboom et al. 1993). Twenty-three had neurological deficits indicating the high risk of neurological complications with thoracic spinal fractures. Fractures due to blunt trauma are more likely to occur in those 60 years or over (Table 5.9); this group should have a lower threshold for investigation (Samuels and Kerstein 1993).

Another study (Durham et al. 1995) identified an Injury Severity Score ≥ 15, a positive clinical examination, and a fall of ≥ 10 feet as three factors associated with thoracolumbar fractures.

Both of these studies concluded that when clinical findings are negative and there are no other complicating factors such as other injuries or an altered sensorium that the chances of finding a fracture requiring treatment is very small (Table 5.10). Where clinical findings are equivocal or there is an altered conscious state, a lower threshold for radiography should pertain.

These conclusions are supported by a retrospective review of 145 patients with thoracic or lumbar spine fractures from blunt trauma. Back pain or tenderness was present in only 81% of people at presentation (Meldon and Moettus 1995). The remaining 19% without back pain and tenderness had an altered sensorium, a concomitant major injury or a neurologic deficit.

These data suggest that patients who are awake, alert, and have no clinical evidence of injury, do not require radiologic study of the thoracolumbar spine. Those with equivocal or positive clinical findings or with altered levels of consciousness should have complete thoracolumbar spine evaluation.

Retrospective data on 1485 patients with blunt injuries admitted to a trauma centre were reviewed to define all categories of patients with fractures on thoracolumbar films (Frankel et al. 1994). These categories were:

- back pain or tenderness
- coexisting cervical spine fracture

Table 5.8

Comparison of the Location of Pain in Patients with Scheuermann's Kyphosis with Age and Sex Matched Controls

Location of Pain	No. of Patients (*n* = 67)	No. of Controls (*n* = 34)
No pain	21 (31%)	21 (62%)
Back pain*	41 (61%)	5 (15%)
Lower extremity	3 (4%)	5 (15%)
Back and lower extremity	2 (3%)	3 (9%)

Note: Chi-squared = 20.3; *df* = 3; *p* = 0.0001. *28% of patients had thoracic spinal pain compared with 3% of controls. Based on data from Murray et al. (1993).

Table 5.9

Prevalence of Thoracolumbar Fractures by Age in 99 Patients with Blunt Trauma Prompting Radiological Investigation

Age	*N*	Number with Fractures
< 60 years	82	9 (11%)
≥ 60 years	17	6 (35%)

Note: Based on data from Samuels and Kerstein 1993.

Table 5.10

Prevalence of Thoracic Spinal Fractures in Retrospective Studies of Blunt Trauma Victims in Trauma Centres on Whom Thoracolumbar Radiographs Were Performed

Clinical Status	Size of Study	Prevalence of Fractures		95% Confidence Limits
		n	%	
Clinical features suggesting injury	24	14	58	38–78%
	186	32*	17	11–24%
No clinical features of injury	55	0	0	—
	128	10#	8	3–12%
Equivocal features or altered sensorium	20	1	5	0–15%
	29	5	17	13–46%

Note: *19 required treatment, 13 were old or minor fractures. # none required treatment. Based on data from Samuels and Kerstein (1993) and Durham et al. (1995).

- fall ≥ 3 m
- neurologic deficit
- ejection from vehicle
- crash ≥ 80 km/h
- Glasgow Coma Score (GCS) ≤ 8

A total of 176 of the 233 patients who met these criteria had thoracolumbar films. Fractures were found in 50 of these and one further patient was diagnosed with a fracture on later assessment in hospital. When these criteria were applied prospectively to a series of 480 patients in the same centre, 167 were xrayed, of whom 15 had fractures. The odds ratios/relative risks of fracture for the retrospective/prospective groups are shown in Table 5.11. When the study combined retrospective and prospective data only 60% had pain and tenderness. No details of the conscious state or presence of concomitant injuries were given in this study, making it difficult to compare with other studies mentioned above. The combined data included 65 patients with thoracolumbar fractures. They had a total of 72 fractures, 26 of which were in the thoracic spine.

Key Messages

> Fractures are more likely to occur in people over age 60 with a history of blunt trauma; a lower threshold for investigation is warranted in this group. (*Level IV)

> In the presence of trauma, xray of the thoracolumbar spine is not indicated in those who are awake, alert and have no clinical evidence of injury; however those with equivocal or positive clinical findings or with an altered level of consciousness should undergo thoracolumbar spine evaluation. (*Level IV)

Computed Tomography (CT) Scanning

CT scans have virtually no role in evaluating thoracic spinal pain of unknown origin. Their role in the evaluation of thoracic spinal disc disease is also limited by the poor ability of this technique to define thecal sac or nerve root compression. This is due to the relative lack of epidural fat in the thoracic spine. In the evaluation of major trauma where fractures have been detected on plain films, CT scanning does have a role in defining the damage to the posterior elements and in demonstrating impingement on the neural canal as well as injuries to other organ systems (Keene et al. 1982).

Key Message

CT scanning is only indicated for the evaluation of the neural canal and posterior elements of the thoracic spine when fractures have been detected with plain films. (*Level IV)

Magnetic Resonance Imaging (MRI)

Care should be taken in the interpretation of investigations which define disc protrusions. With MRI a prevalence of thoracic disc herniation of 14.5% has been reported in a group of 48 cancer patients (Williams et al. 1989). A prevalence of asymptomatic thoracic disc herniations of 11.1% to 13.4% has been demonstrated with post-myelographic CT scanning

Table 5.11

Risk Factors for Thoracolumbar Fracture in Patients with Blunt Trauma Injuries Admitted to a Trauma Centre

Category	Retrospective Data (*n* = 1485) Odds Ratio (CI 90%)	Prospective Data Relative Risk (CI 90%)
Back pain or tenderness	9 (1.5–13.9)	1 (0.8–2.9)
Cervical spine fracture	0	N/A
Fall ≥ 3m	8 (2.3–16.9)	2 (1.5–4.3)
Neurologic deficit	10 9 (3.1–31.6)	N/A
Ejection from vehicle	6 (2.2–6.3)	2 (1.5–4.9)
Crash ≥ 80 km/h	2 (1.4–10.1)	2 (1.4–10.1)
GCS ≤ 8	2 (0.8–2.6)	0

Note: Odds ratios (CI 90%) are given for retrospective data and relative risk ratios (CI 90%) for prospective data. Only categories in which fractures occurred are given. Based on data from Frankel et al. (1994).

(Anwad et al. 1991). This prevalence is as high as 37% with the use of MRI (Wood et al. 1997). Follow-up of this last cohort over a mean period of 26 months showed there was a trend for small disc herniations either to remain unchanged or increase in size and for large disc herniations often to decrease in size. None of the cohort became symptomatic in this period.

The available data, therefore, indicate that thoracic disc protrusion is common in asymptomatic individuals, but when it does become symptomatic it is responsible for neurological symptoms and signs. There are no data that implicate thoracic disc protrusion as a source of spinal pain in the absence of neurological features. In the absence of alerting features of serious conditions, MRI is not indicated in the diagnosis of acute thoracic spinal pain.

Other Investigations

No research has been located that specifically deals with other tests used in the diagnosis and management of acute thoracic spinal pain. Therefore the choice of investigations is determined by the clinical features suggestive of pain other than those of somatic origin. For example, acute thoracic spinal pain with associated chest tightness and diaphoresis calls for an urgent electrocardiogram to exclude myocardial ischaemia. Associated abdominal pain and vomiting calls for tests such as serum lipase, abdominal ultrasound and CT scanning to exclude pancreatic and gall bladder pathology.

Refer to Appendix C: Ancillary Investigations.

Cost Effectiveness of Investigations

There are no data on the cost effectiveness of investigations for acute thoracic spinal pain.

Key Message

There is no research to inform ancillary investigations for acute thoracic spinal pain; investigations should be selected on the basis of clinical features suggesting the presence of serious conditions. (Consensus)

>Terminology

Recommended Terms

In the absence of any features of serious conditions, the following terms are recommended to describe non-specific thoracic spinal pain.

The taxonomy produced by the International Association for the Study of Pain (Merskey and Bogduk 1994) offers seven diagnostic categories that substitute for the umbrella term of chronic pain of mechanical origin. Most are relevant in the diagnosis of acute pain, and in the absence of a recognised taxonomy for acute pain, they offer a useful scheme for diagnostic labelling. These require rigorous criteria to be satisfied if anatomical location of the source of pain is to be specified in the diagnosis. This would often make their application in the acute setting unjustifiable.

The following categories require rigorous confirmation as summarised below:

- **Thoracic discogenic pain**: Appropriate response to selective anaesthetisation of the putatively symptomatic disc or to provocation discography.
- **Thoracic zygapophyseal joint pain**: Complete relief of pain on selective, radiologically controlled intra-articular anaesthesia of the targeted joint followed by validation procedures to exclude false positive results.
- **Costo-transverse joint pain**: Complete relief of pain on selective, radiologically controlled intra-articular anaesthesia of the targeted joint followed by validation procedures to exclude false positive results.
- **Thoracic trigger point syndrome**: Presence of a palpable, tender, firm fusiform nodule or band in a specified muscle, which reproduces the pain and/or referred pain on palpation. Elimination of the trigger point by stretching, dry needling or local anaesthesia relieves the pain.
- **Thoracic segmental dysfunction**: Aggravation of pain by selectively stressing the specified affected segment. Stressing adjacent segments does not reproduce the pain.

The investigations required to permit diagnosis in the first three categories are not widely available and are rarely pursued in clinical practice. For practical and logistic reasons, they are entities best reserved for the investigation of chronic thoracic spinal pain.

The criteria for the latter two entities require tests of known reliability and validity, but studies of these features have not been published. Therefore, although 'trigger point syndrome' and 'segmental dysfunction' can be defined in theory, they are entities that cannot yet be diagnosed in practice, without making assumptions about the reliability and validity of tests used to make the diagnosis.

Two other mechanical categories with less rigorous criteria are:

- **Thoracic muscle sprain**: Pain in a specific muscle with a history of activities consistent with strain of that muscle. Tenderness on palpation of that muscle and aggravation of the pain on selective stretching or relief on selective anaesthetisation of that muscle.

- **Thoracic spinal pain of unknown or uncertain origin**: No other cause of pain has been found or can be attributed.

The latter term acknowledges the presence of pain while recognising the limitations in formulating a patho-anatomic diagnosis.

For the purposes of this document, either of these two terms can be used to describe acute thoracic spinal pain, provided that the criteria for them are satisfied. If these are believed too ambiguous, another alternative is offered:

- **Somatic thoracic spinal pain**: Pain that may arise from the somatic tissues of the thoracic spine.

This term acknowledges the presence of pain, and indicates a belief that the pain may arise from one or other of the somatic tissues of the thoracic spine.

Key Message

The appropriate labels for non-specific 'mechanical' thoracic spinal pain are 'thoracic spinal pain of unknown origin' or 'somatic thoracic spinal pain'. (Consensus)

PROGNOSIS

Natural History

There have been no published studies on the evolution or progression of thoracic spinal pain as a complaint, with or without treatment. It is not known whether acute thoracic spinal pain behaves in the same manner as acute lumbar spinal pain or acute cervical spinal pain.

Influence of Risk Factors and Diagnostic and Therapeutic Interventions

Only one study can be found which examines the risk factors for thoracic spinal pain as a distinct or separate entity. This study prospectively examined risk factors in the development of thoracic and lumbar spinal pain in 395 male infantry recruits on a 14-week intensive training course (Milgrom et al. 1993). An increased lumbar inclination (lordosis) was the only predictive factor for thoracic spinal pain. This was 101 $^{+}$/– 3.1 degrees in the 30 recruits with pain compared to 99.2 $^{+}$/– 4.3 degrees in the 363 recruits without pain (p = 0.04; two recruits with thoracic and lumbar spinal pain were excluded). The small difference and the overlap of standard errors nullify the clinical utility of this finding. A number of other anthropometric measurements, postural deviations and muscle power tests were not found to be of significance.

There have been no published studies on the evolution or progression of thoracic spinal pain as a complaint, with or without treatment. It is not known if acute thoracic spinal pain behaves in the same manner as other acute spinal pain.

Key Message

There is a lack of published data on the natural history and influence of prognostic risk factors for acute thoracic spinal pain. (No studies located)

INTERVENTIONS

A search of the literature for Level I to Level III-3 evidence yielded little evidence for the effectiveness of interventions for thoracic spinal pain. More research is required in this area.

The evidence for therapies specifically for acute thoracic spinal pain is limited. Any evidence for the efficacy of thoracic spinal therapies is commonly buried in studies on 'back pain' and no distinction is made between cases of thoracic spinal and lumbar spinal pain. The summary of the evidence presented here is confined to studies that specifically discuss the thoracic spine. Management decisions should be based upon knowledge of the existing evidence, consideration of individual patient needs and clinical judgment.

The criteria formulated to categorise the following intervention and the definitions of the levels of evidence are described in Chapter 9: Process Report.

Evidence of Benefit

Manual Treatment

There have been no systematic reviews of therapy for thoracic spinal pain. Schiller (2001) compared the use of spinal manipulation with non-functional ultrasound placebo in a small, randomised controlled trial of 30 patients with mechanical thoracic spinal pain. This demonstrated significantly better reductions in numerical pain ratings and improvements in lateral flexion with manipulation at the end of a two to three week treatment period. These changes were maintained a month later, but were no longer better than in the placebo group. Notably there were no significant differences in McGill pain questionnaires and Oswestry Back Disability Indices between groups at any point in the trial. The small sample size was suggested as a reason for this, leaving unanswered questions about the real efficacy of manipulation.

Key Message

There is evidence from one small study that spinal manipulation is effective compared to placebo in thoracic spinal pain. (Level II)

Other Treatment

No studies can be found that address the treatment of acute thoracic spinal pain with the following therapies:

- consumer education
- reassurance and home rehabilitation
- drug therapy
- bed rest
- mobilisation
- functional restoration
- behavioural therapy
- back school
- exercises
- injection treatments
- surgery

>References

Anwad EE, Martin DS, Smith KR, Baker BK (1991). Asymptomatic versus symptomatic herniated thoracic discs: their frequency and characteristics as detected by computed tomography after myelography. Neurosurgery, 28: 180–186.

Aprill C, Dwyer A, Bogduk N (1990). Cervical zygapophyseal joint pain patterns II: a clinical evaluation. Spine, 15: 458–461.

Arce CA, Dohrmann GJ (1985). Thoracic disc herniation: improved diagnosis with computed tomographic scanning and a review of the literature. Surgical Neurology, 23: 356–361.

Australian Institute of Health and Welfare (2000). Australia's Health 2000: The Seventh Biennial Report of the Australian Institute of Health and Welfare. AIHW: Canberra.

Bhambhani M, Crisp AJ, Compston JE (1992). Differential involvement of the dorsal and lumbar spine in osteoporosis. Annals of the Rheumatic Diseases, 51: 1069–1070.

Bhole R, Gilmer RE (1984). Two-level thoracic disc herniation. Clinical Orthopaedics and Related Research, 129–131.

Bryner P, Baxter AJ, Sherwood BF (1989). Thoracic paraspinal tenderness of chronic pain sufferers. Journal of the Australian Chiropractorss Association, 19: 131–136.

Bywater EGL (1974). Rheumatoid discitis in the thoracic region due to spread from the costovertebral joints. Annals of the Rheumatic Diseases, 33: 48.

Calin A (1993). Ankylosing spondylitis. In: Maddison PJ, Isenberg DA, Woo P, Glass DN. Oxford Textbook of Rheumatology. Oxford University Press: Oxford. pp 2: 681–690.

Charlton K. Approaches to demonstration of changes of vertebral subluxation in the cervical spine. 1991 M Phil thesis, Griffith University, Nathan, Queensland

Christensen HW, Vach W, Vach K, Manniche C, Haghfelt T, Hartivgsen L, Flemming Hoilund-Carlsen P (2002). Palpation of the upper thoracic spine: an observer reliability study. Journal of Manipulative and Physiological Therapeutics, 25: 285–292.

Chua WH (1996). Thoracic spinal pain — a review. Australasian Musculoskeletal Medicine, 1: 13–22.

Cloward RB (1959). Cervical discography: a contribution to the aetiology and mechanism of neck shoulder and arm pain. Annals of Surgery, 150: 1051–1064.

Cockerill W, Ismail AA, Cooper C, Raspe H, Silman AJ, O'Neill TW (2000). Does location of vertebral deformity within the spine influence back pain and diasbility? European Vertebral Osteoporosis Study (EVOS) Group. Annals of the Rheumatic Diseases, 59: 368–371.

Comeaux Z, Eland D, Chila A, Pheley A, Tate M (2001). Measurement challenges in physical diagnosis: refining inter-rater palpation, perception and communication. Journal of Bodywork and Movement Therapies, 5: 245–253.

Cook DJ, Guyatt GH, Adachi JD, Clifton J, Griffith LE, Epstein RS, Juniper EF, (1993). Quality of life issues in women with vertebral fractures due to osteoporosis. Arthritis and Rheumatism, 36: 750–756.

Corrigan B, Maitland GD (1988). Practical Orthopaedic Medicine (5th Edition). Butterworths: Sydney. pp 223–224.

Crawford R, Singer KP (1995). Normal and degenerative anatomy of the thoracic intervertebral discs. In: Proceedings of the Manipulative Physiotherapists Association of Australia 9th Biennial Conference: Gold Coast. pp 24–29.

Delamarter RB, Sachs BL, Thompson GH, Thompson GH, Bohlman HH, Makley JT, Carter JR (1990). Primary neoplasms of the thoracic and lumber spine: an analysis of 29 consecutive cases. Clinical Orthopaedics and Related Research, 256: 87–100.

Deyo RA, Diehl AK (1988). Cancer as a cause of back pain: frequency, clinical presentation and diagnostic strategies. Journal of General Internal Medicine, 3: 230–238.

Dreyfuss P, Tibiletti C, Dreyer SJ (1994). Thoracic zygapophyseal joint pain patterns. Spine, 19: 807–811.

Durham RM, Luchtefeld WB, Wibbenmeyer L, Maxwell P, Shapiro MJ, Mazuski JE (1995). Evaluation of the thoracic and lumbar spine after blunt trauma. American Journal of Surgery, 170: 681–684.

Dwyer A, Aprill C, Bogduk N (1990). Cervical zygapophyseal joint pain patterns I: a study in normal volunteers. Spine, 15: 453–457.

Edmondston SJ, Singer KP, Price RI, Breidahl PD (1993). Accuracy of lateral dual energy Xray absorptiometry for the determination of bone mineral content in the thoracic and lumbar spine: an in vitro study. British Journal of Radiology, 66: 309–313.

Enwemeka CS, Konet IM, Prudhithumrong S, Ogbahon FE, Gbenedio NA (1986). Postural correction in persons with neck pain: A survey of neck positions recommended by physical therapists. Journal of Orthopaedic and Sports Physical Therapy, 5: 235–239.

Ettinger B, Black DM, Palermo L, Nevitt MC, Melnikoff S, Cummings SR (1994). Kyphosis in older women and its relation to back pain, disability and osteopenia: the study of osteoporotic fractures. Osteoporosis International, 4: 55–60.

Evans SF, Davie MWJ (2000). Vertebral fractures and bone mineral density in idiopathic, secondary and corticosteroid associated osteoporosis in men. Annals of the Rheumatic Diseases, 59: 269–75.

Feinstein B, Langton JNK, Jameson RM, Schiller F (1954). Experiments of pain referred from deep somatic tissues. Journal of Bone and Joint Surgery, 36A: 981–997.

Flynn TW (1996). The Thoracic Spine and Rib Cage: Musculoskeletal Evaluation and Treatment. Butterworth-Heinemann: Boston. pp 121–130.

Frankel HL, Rozycki GS, Ochsner MG, Harviel JD, Champion HR (1994). Indications for obtaining surveillance thoracic and lumbar spine radiographs. Journal of Trauma, 37: 673–676.

Fukui S, Ohseto K, Shiotani M (1997). Patterns of pain induced by distending the thoracic zygapophyseal joints. Regional-Anaesthesia, 22: 331–336.

Fukui S, Ohseto K, Shiotani M, Ohno K, Karasawa H, Naganuma Y, Yuda Y (1996). Referred pain distribution of the cervical zygapophyseal joints and cervical dorsal rami. Pain, 68: 79–83.

Giles LGF, Muller R (1999). Chronic spinal pain syndromes: a clinical pilot trial comparing acupuncture, a nonsteroidal antiinflammatory drug and spinal manipulation. Journal of Manipulative and Physiological Therapeutics, 22: 376–381.

Greenman PE (1989). Principles of manual medicine (1st Edition). Williams and Wilkins: Baltimore. pp 1–12.

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: 425–430.

Groen GJ, Baljet B, Drukker J (1990). Nerves and nerve plexuses of the human vertebral column. American Journal of Anatomy, 188: 281–296.

Groen GJ, Baljet B, Drukker J (1988). The innervation of the spinal dura mater: anatomy and clinical implications. Acta Neurochirurgica (Wien), 92: 39–46.

Grootboom MJ, Govender S (1993). Acute injuries of the upper dorsal spine. Injury, 24: 389–392.

Haldeman S, Rubinstein SM (1993). The precipitation or aggravation of musculoskeletal pain in patients receiving spinal manipulative therapy. Journal of Manipulative and Physiological Therapeutics, 16: 47–50.

Haley JC, Perry JH (1950). Protrusions of intervertebral discs: study of their distribution, characteristics and effects on the nervous system. American Journal of Surgery, 80: 394–404.

Haneline MT (2000) Chest pain in chiropractic practice. Journal of the Neuromusculoskeletal System, 8: 84–88.

Harreby M, Neergaard K, Hesselsoe G, Kjer J (1995). Are radiologic changes in the thoracic and lumbar spine of adolescents risk factors for low back pain in adults? A 25-year prospective cohort study of 640 school children. Spine, 20: 2298–2302.

Hastings DE (1994). Rheumatoid arthritis: Clinical features: Early, progressive and late disease. In: Klippel JH, Dieppe PA (eds). Rheumatology. Mosby: Chicago. pp 3: 4.1–4.14.

Hockaday JM, Whitty CWM (1967). Patterns of referred pain in the normal subject. Brain, 90: 481–495.

Huang C, Ross PD, Wasnich RW (1994). Vertebral fractures and other predictors of back pain among older women. Journal of Bone and Mineral Research, 11: 1026–32.

Hudak PL, Cole DC, Frank JW (1998). Perspectives on prognosis of soft tissue musculoskeletal disorders. International Journal of Rehabilitation Research, 21: 29–40.

Johnston WL, Allan BR, Hendra JL, Neff DR, Rosen ME, Thomas SC (1983). Interexaminer study of palpation in detecting location of spinal segmental dysfunction. Journal of the American Osteopathic Association, 82: 839–845.

Kandabarow A (1997). Injuries of the thoracolumbar spine. Topics in Emergency Medicine, 19: 65–80.

Keating L, Lubke C, Powell V, Young T, Souvlis T, Jull G (2001). Mid-thoracic tenderness: a comparison of pressure pain threshhold between spinal regions in asymptomatic subjects. Manual Therapy, 6: 34–39.

Keene JS, Goletz TH, Lilleas F, Alter AJ, Sackett JF (1982). Diagnosis of vertebral fractures. A comparison of conventional radiography, conventional tomography, and computed axial tomography. Journal of Bone and Joint Surgery, 64A: 586–594.

Kelley WN (ed) (1997). Textbook of Internal Medicine. Lippincott-Raven: Philadelphia.

Kellgren JH (1939). On the distribution of pain arising from deep somatic structures with charts of segmental pain areas. Clinical Science, 4: 35–46.

Kenna C, Murtagh J (1989). Back Pain and Spinal Manipulation — A Practical Guide. Butterworths: Sydney.

Khan MA (1994). Ankylosing spondylitis: clinical features. In: Klippel JH, Dieppe PA (eds). Rheumatology. Mosby: Chicago. pp 3: 25.1–25.10.

Kleineman WB, Kiernan HA, Michelan WJ (1978). Metastatic cancer of the spinal column. Clinical Orthopaedics and Related Research, 136: 166–172.

Kuker W, Mull M, Mayfrank L, Topper R, Thron A (1997). Epidural spinal infection. Variability of clinical and magnetic resonance imaging findings. Spine, 22: 544–550.

LaBan MM, Siegel CB, Schutz LK, Taylor RS (1994). Occult radiographic fractures of the chest wall identified by nuclear scan imaging: report of seven cases. Archives of Physical Medicine and Rehabilitation, 75: 353–354.

Liang M, Komaroff AL (1982). Roentgenograms in primary care patients with acute low back pain: a cost-effectiveness analysis. Archives of Internal Medicine, 142: 1108–1112.

Love JG, Schorn VS (1965). Thoracic-disk protrusions. Journal of the American Medical Association, 191: 627–631.

Love RM, Brodeur RR (1987). Inter- and intra-examiner reliability of motion palpation for the thoracolumbar spine. Journal of Manipulative and Physiological Therapeutics, 10: 1–4.

Magarey ME (1994). Examination of the cervical and thoracic spine. In: Grant R (ed). Physical Therapy for the Cervical and Thoracic Spine (2nd Edition). Churchill-Livingstone: Edinburgh. p 124.

Maigne R (1996). Diagnosis and treatment of pain of vertebral origin (1st Edition). Williams and Wilkins: Paris. pp 99–108.

Maigne R (1980). Low back pain of thoracolumbar origin. Archives of Physical Medicine and Rehabilitation, 61: 389–395.

Malawski SK, Lukawski S (1991). Pyogenic infection of the spine. Clinical Orthopaedics, 58–66.

McCallister VL, Sage MR (1976). The radiology of thoracic disc protrusion. Radiology, 27: 294–299.

McCombe PF, Fairbank JC, Cockersole BC, Pynsent PB (1989). 1989 Volvo Award in Clinical Sciences. Reproducibility of physical signs in low-back pain. Spine, 14: 908–918.

McGuckin N (1986). The T4 syndrome. In: Grieve GP (ed). Modern Manual Therapy of the Vertebral Column. Churchill-Livingstone: London. pp 370–376.

McKenzie RA (1981). The derangement syndrome. In: The lumbar spine: mechanical diagnosis and therapy. Spinal Publications: Waikane 11:109–12

Meldon SW, Moettus LN (1995). Thoracolumbar spine fractures: clinical presentation and the effect of altered sensorium and major injury. The Journal of Trauma, 39: 1110–1114.

Melton LJ, Kan SH, Frye MA, Wahner HW, O'Fallon WM, Riggs BL (1989). Epidemiology of vertebral fractures in women. American Journal of Epidemiology, 129: 1000–1011.

Merskey H, Bogduk N (eds) (1994). Classification of Chronic Pain: Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms (2nd Edition). IASP Press: Seattle. pp 111–120.

Milgrom C, Finestone A, Lev B, Wiener M, Floman Y (1993). Overexertional lumbar and thoracic back pain among recruits: a prospective study of risk factors and treatment regimens. Journal of Spinal Disorders, 6: 187–193.

Minucci A (1987). Palpation of the thoracic spine (T1 to T8). In: Dalziel BA, Snowsill JC (eds). Proceedings of the Manipulative Therapists Association of Australia Fifth Biennial Conference, 367–376.

Mitra SR, Gurjar SG, Mitra KR (1996). Degenerative disease of the thoracic spine in central India. Spine, 34: 333–337.

Murray PM, Weinstein SL, Spratt KF (1993). The natural history and long-term follow-up of Scheuermann kyphosis. Journal of Bone and Joint Surgery, 75A: 236–248.

Musgrave DS, Vogt MT, Nevitt MC, Cauley JA (2001). Back problems among postmenopausal women taking estrogen replacement therapy: the study of osteoporotic fractures. Spine, 26: 1606–1612.

Nathan H (1962). Osteophytes of the vertebral column. Journal of Bone and Joint Surgery, 44A: 243–268.

Nathan H, Weinberg H, Robin GC, Avaid I (1964). Anatomical-clinical observations in arthritis. Arthritis and Rheumatism, 7: 228–240.

National Health and Medical Research Council (1999a). A guide to the development, implementation and evaluation of clinical practice guidelines. NHMRC: Canberra.

National Health and Medical Research Council (1999b). Acute Pain Management: Scientific Evidence. Commonwealth od Australia: Canberra.

Norlander S, Aste-Norlander U, Nordgren B, Sahlstedt B (1996). Mobility in the cervico-thoracic motion segment: an indicative factor of musculo-skeletal neck-shoulder pain. Scandinavian Journal of Rehabilitation Medicine, 28: 183–192.

Norlander S, Gustavsson B, Lindell J, Nordgren B (1997). Reduced mobility in the cervico-thoracic motion segment, a risk factor for musculoskeletal neck-shoulder pain: a two-year prospective follow-up study. Scandinavian Journal of Rehabilitation Medicine, 29: 167–174.

O'Neill TW, McCloskey EV, Kanis JA, Bhalla AK, Reeve J, Reid DM, Todd C, Woolf AD, Silman AJ (1999). The distribution, determinants, and clinical correlates of vertebral osteophytosis: a population based survey. Journal of Rheumatology, 26: 841–848.

Otani K, Nakai S, Fujimura Y, Manzoku S, Shibasaki K (1982). Surgical treatment of thoracic disc herniation using the anterior approach. Journal of Bone and Joint Surgery, 64B: 340–343.

Ozaki T, Liljenqvist U, Hillmann A, Halm H, Lindner N, Gosheger G, Winkelman W (2002). Osteoid osteoma and osteoblastoma

of the spine: experiences with 22 patients. Clinical Orthopaedics and Related Research, 397: 394–402.

Patel U, Skingle S, Campbell GA, Crisp AJ, Boyle IT (1991). Clinical profile of acute vertebral compression fractures in osteoporosis. British Journal of Rheumatology, 30: 418–421.

Quast LM (1987). Thoracic disc disease, diagnosis and surgical treatment. Journal of Neuroscience Nursing, 19: 198–204.

Raine S, Twomey L (1994). Posture of the head, shoulders and thoracic spine in comfortable erect standing. Journal of the Australian Physiotherapy Association, 40: 25–32.

Refshauge K, Bolst L, Goodsell M (1995). The relationship between cervicothoracic posture and the presence of pain. Journal of Manipulative and Physiological Therapeutics, 3: 21–24.

Ross PD, Davis JW, Epstein RS, Wasnich R (1994). Pain and disability associated with new vertebral fractures and other spinal conditions. Journal of Clinical Epidemiology, 47: 231–239.

Russell T (1989). Thoracic intervertebral disc protrusion: experience of 67 cases and review of the literature. British Journal of Neurosurgery, 3: 153–160.

Samuels LE, Kerstein MD (1993). 'Routine' radiologic evaluation of the thoracolumbar spine in blunt trauma patients: a reappraisal. Journal of Trauma, 34: 85–89.

Santavirta S, Konttinen YT, Heliovaara M, Knekt P, Luthje P, Aromaa A (1992). Determinants of osteoporotic thoracic vertebral fracture. Screening of 57,000 Finnish women and men. Acta Orthopaedica Scandinavica, 63: 198–202.

Schellhas KP, Pollei SR (1994). Thoracic discography: a safe and reliable technique. Spine, 19: 2103–2109.

Schiller L (2001). Effectiveness of spinal manipulative therapy in the treatment of mechanical thoracic spinal pain. Journal of Manipulative and Physiological Therapeutics, 24: 394–401.

Shore LR (1935). On osteo-arthritis in the dorsal intervertebral joints. The British Journal of Surgery, 22: 833–839.

Simeone FA, Lawner PM (1982). Intraspinal neoplasms. In: Rothman RH, Simeone FA (eds). The Spine (2nd Edition). W B Saunders: Philadelphia. p 1041.

Singer KP, Jones TJ, Breidahl PD (1990). A comparison of radiographic and computer-assisted measurements of thoracic and thoracolumbar sagittal curvature. Skeletal Radiology, 19(1): 21–26.

Skubic JW (1993). Thoracic disc disease. Current Opinion in Orthopaedics, 4: 96–103.

Stewart R, Ridyard J, Pearson J (1976). Regional lung function in ankylosing spondylitis. Thorax, 31: 433–477.

Stolker RJ, Vervest AC, Groen GJ (1993). Percutaneous facet denervation in chronic thoracic spinal pain. Acta Neurochirurgica Wien, 122: 82–90.

Sward L, Hellstrom M, Jacobsson B, Nyman R, Peterson L (1991). Disc degeneration and associated abnormalities of the spine in elite gymnasts. A magnetic resonance imaging study. Spine, 16: 437–443.

Triano JJ, Erwin M, Hansen DT (1999). Costovertebral and costotransverse joint pain: a commonly overlooked pain generator. Topics in Clinical Chiropractic, 6: 79–92.

Vanichkachorn JS, Vaccaro AR (2000) Thoracic disk disease: diagnosis and treatment. The Journal of the American Academy of Orthopedic Surgeons, 8: 159–169.

Wedderkopp N, Leboeuf-Yde C, Andersen LB, Froberg K, Hansen HS (2001). Back pain reporting pattern in a Danish population-based sample of children and adolescents. Spine, 26: 1879–1883.

Weinberg H, Nathan H, Magora F (1972). Arthritis of the first costovertebral joint as a cause of thoracic outlet syndrome. Clinical Orthopaedics and Related Research, 86: 159–163.

Williams MP, Cherryman GR, Husband JE (1989). Significance of thoracic disc herniation demonstrated by MR imaging. Journal of Computer Assisted Tomography, 13: 211–214.

Wise CM, Semble EL, Dalton CB (1992). Musculoskeletal chest wall syndromes in patients with noncardiac chest pain: a study of 100 patients. Archives of Physical Medicine and Rehabilitation, 73: 147–149.

Wood KB, Blair JM, Aepple DM, Schendel MJ, Garvey TA, Gundry CR, Heithoff KB (1997) The natural history of asymptomatic thoracic disc herniations. Spine, 22: 525–529.

Wood KB, Garvey TA, Gundry C, Heithoff KB (1995). Magnetic resonance imaging of the thoracic spine. Evaluation of asymptomatic individuals. Journal of Bone and Joint Surgery, 77A: 1631–1638.

Wood KB, Schellhas KP, Garvey TA, Aeppli D (1999). Thoracic discography in healthy individuals: a controlled prospective study of magnetic resonance imaging and discography in asymptomatic and symptomatic individuals. Spine, 24: 1548–1555.

Wyke B (1975). Morphological and functional features of the innervation of the costovertebral joints. Folia Morphologica Praha, 23: 286–305.

Yelland MJ, Glasziou PP, Purdie J. (2002). The interobserver reliability of thoracic spinal examination. Australasian Musculoskeletal Medicine, May: 16–22.

Chapter 6

Acute Neck Pain

➔ *Neck pain is one of several regional pain problems affecting the musculoskeletal system. Neck pain is rivalled only by low back pain and osteoarthritis in general, among disorders of the musculoskeletal system.*

International figures indicate that at any point in time approximately 10–15% of the population will be suffering an episode of neck pain, and 40% will suffer neck pain during a twelve-month period (Ariens et al. 1999). Figures for the Australian population are lacking, although one survey reported that 18% of individuals woke with cervical pain and 4% suffered from it all day (Gordon et al. 2002).

Textbooks of medicine provide different and limited advice as to the causes and treatment of acute neck pain; the information they provide is inconsistent with current scientific information on the management of such pain.

These guidelines were developed to provide an educational resource for the management of acute neck pain, enabling clinicians and patients to make informed treatment decisions.

Definition of Acute Neck Pain

In these guidelines, the term 'acute' refers to pain that has been present for less than three months (Merskey 1979); it does not refer to the severity or quality of pain. Chronic pain is pain that has been present for at least three months (Merskey and Bogduk 1994).

Although no organisation has explicitly defined neck pain, it is taken to mean cervical spinal pain, which the International Association for the Study of Pain (IASP) defines as:

> …pain perceived as arising from anywhere within the region bounded superiorly by the superior nuchal line, inferiorly by an imaginary transverse line through the tip of the first thoracic spinous process and laterally by sagittal planes tangential to the lateral borders of the neck (Merskey and Bogduk 1994).

This definition is based exclusively on where the individual indicates they perceive pain.

Scope

These guidelines outline the evidence for the management of acute idiopathic neck pain and acute whiplash-associated neck pain. The following conditions are beyond the scope of these guidelines:

- serious conditions: neurological conditions, infection, neoplasm, fracture of the cervical spine
- neuropathic pain
- cervicogenic headache
- pain in the throat
- headache
- cervical radicular pain (pain perceived in the upper limb)
- thoracic spinal pain
- chronic pain

Guideline Development Process

Evaluation of Other Guidelines

Guidelines developed by other groups were obtained and reviewed to compare guideline development processes and to assess whether existing guidelines could be adapted for use in the Australian context. The following guidelines were located:

- The Philadelphia Panel Evidence-Based Clinical Practice Guidelines on Selected Rehabilitation Interventions for Neck Pain (2001). These guidelines focus on interventions for neck pain in general rather than acute neck pain.
- Guidelines for the Management of Whiplash-Associated Disorders, prepared by the Motor Accident Authority of New South Wales (2001). These guidelines focus on acute neck pain associated with whiplash.

The decision was made to update and disseminate the existing draft guidelines for acute neck pain developed for the National Musculoskeletal Medicine Initiative by Professor Nikolai Bogduk.

Updating Existing Guidelines

The update of the existing work involved a review of the evidence on acute neck pain conducted by a multi-disciplinary group. Group members had the opportunity to evaluate the literature forming the basis of the existing guidelines, review the interpretation of the literature, nominate additional articles to undergo the appraisal process or request that an article be re-appraised.

A systematic process was used to identify new studies on the diagnosis, prognosis and interventions for acute neck pain in line with current standards for guideline development (NHMRC 1999a).

Studies were appraised against selection criteria and those meeting the criteria for inclusion were used to update the existing text of the guidelines.

Relevant studies on areas related to diagnosis were identified in the literature search and used to update the sections on

Aetiology and Prevalence, History, Physical Examination and Ancillary Investigations where possible. These sections are largely comprised of the existing work developed using a conventional literature review.

The most recent Clinical Evidence text (2002) was used as the basis for updating the section on interventions. Studies cited in Clinical Evidence were checked against the selection criteria established for this update. In cases where there were no studies involving purely acute populations with neck pain, studies involving mixed acute and chronic populations were considered in this update. Additional studies published subsequent to the search date in Clinical Evidence were sought to determine whether new evidence existed.

All studies appraised for this update are included in either the Table of Included Studies or the Table of Excluded Studies (refer to Appendix E). Studies that were included in the existing guidelines or described in Clinical Evidence (2002) are not described in these tables.

Refer to Chapter 9: Process Report for further detail.

Study Selection Criteria

The chart below is an outline of the criteria used to identify, select and appraise new studies on acute neck pain.

Search Strategy

Sensitive searches were performed. Electronic searches were limited to adults, humans and articles published in English in peer-reviewed journals. Where available, methodological filters were used. There were no hand searches conducted.

Searches for information on the diagnosis and prognosis of acute neck pain covered the period from 1992 to 2002. Searches for articles on interventions covered the period from 2001 to 2002, taking into account the search date (September 2001) used in the Clinical Evidence text (2002) which provided a review of the evidence on interventions for neck pain.

The following databases were searched in August 2002:

- PubMed (Clinical Queries)

Study Selection Criteria

DIAGNOSIS	
The sections on Aetiology and Prevalence, History, Examination and Investigations comprise information from the existing draft (developed by conventional literature review) combined and updated with relevant articles located and appraised according to the following inclusion and exclusion criteria:	
Inclusion criteria	Systematic reviews, cross-sectional studies, case series, case reports Adults Specific diseases and conditions (to identify serious conditions) Acute idiopathic neck pain Acute whiplash-associated neck pain
Exclusion criteria	Chronic pain
PROGNOSIS	
Information from the existing draft was combined and updated with relevant articles located and appraised independently by two reviewers according to the following inclusion and exclusion criteria:	
Inclusion criteria	Systematic reviews, cohort studies Adults Acute idiopathic neck pain Acute whiplash-associated neck pain
Exclusion criteria	Chronic pain Specific diseases Serious conditions (cervical fracture, infection, neurological conditions, tumour) Thoracic spinal pain, throat pain, cervicogenic headache, cervicoradicular pain
INTERVENTIONS	
Information from the existing draft was updated with information obtained from Clinical Evidence (2002) together with relevant articles located and appraised according to the following criteria. In cases where no evidence was available on interventions specifically for acute neck pain, studies containing mixed populations (acute and chronic neck pain) were considered in the review:	
Inclusion criteria	Systematic reviews, randomised controlled trials (RCTs) Acute idiopathic neck pain Acute whiplash-associated neck pain Adults
Exclusion criteria	Chronic pain Specific diseases Serious conditions (cervical fracture, infection, neurological conditions, tumour) Thoracic spinal pain, throat pain, cervicogenic headache, cervicoradicular pain

- CINAHL
- EMBASE — Physical and Rehabilitation Medicine
- The Cochrane Library, 2002, Issue 2

Access to CHIROLARS/MANTIS and PEDro was unavailable for this review.

Search Terms

- Neck pain .exp
- Evidence-based practice .tw
- Pathology .exp
- Interventions .exp
- Mortality .exp
- Cervical pain .exp
- Pain .exp
- Acute .exp
- Diagnosis .exp
- Morbidity .exp
- Pain management .exp
- Prognosis .exp
- Whiplash .exp
- Randomised controlled trial .exp

Summary of Key Messages: Acute Pain Management

	EVIDENCE LEVEL
Management Plan	
It is recommended that the clinician and patient develop a management plan for acute musculoskeletal pain comprising the elements of assessment, management and review: • Assessment — Conduct a history and physical examination to assess for the presence of serious conditions; ancillary investigations are not generally indicated unless features of serious conditions are identified. • Management — Provide information, assurance and advice to resume normal activity and discuss other options for pain management as needed. • Review — Reassess the pain and revise the management plan as required.	CONSENSUS: Steering Committee
Non-Pharmacological Interventions	
Simple interventions (providing information, assurance and encouraging reasonable maintenance of activity) may be used alone or in combination with other interventions for the successful management of acute musculoskeletal pain.	CONSENSUS: Steering Committee
Pharmacological Interventions	
Specific pharmacological interventions may be required to relieve pain; such agents can be used in conjunction with non-pharmacological interventions.	CONSENSUS: Steering Committee; NHMRC 1999b
Paracetamol or other simple analgesics, administered regularly, are recommended for relief of mild to moderate acute musculoskeletal pain.	CONSENSUS: Steering Committee; NHMRC 1999b
Where paracetamol is insufficient for pain relief, a non-steroidal anti-inflammatory (NSAID) medication may be used, unless contraindicated.	CONSENSUS: Steering Committee; NHMRC 1999b
Oral opioids may be necessary to relieve severe musculoskeletal pain. It is preferable to administer a short-acting agent at regular intervals, rather than on a pain-contingent basis. Ongoing need for opioid analgesia is an indication for reassessment.	CONSENSUS: Steering Committee; NHMRC 1999b
Adjuvant agents such as anticonvulsants and antidepressants are not recommended in the management of acute musculoskeletal pain.	CONSENSUS: Steering Committee; NHMRC 1999b
Any benefits from muscle relaxants may be outweighed by their adverse effects, therefore they cannot be routinely recommended.	CONSENSUS: Steering Committee

Summary of Key Messages: Effective Communication

	EVIDENCE LEVEL
Clinicians should work with patients to develop a management plan so that patients know what to expect, and understand their role and responsibilities.	CONSENSUS: Steering Committee
Information should be conveyed in correct but neutral terms, avoiding alarming diagnostic labels; jargon should be avoided.	CONSENSUS: Steering Committee
Explanation is important to overcome inappropriate expectations, fears or mistaken beliefs that patients may have about their condition or its management.	CONSENSUS: Steering Committee
Printed materials and models may be useful for communicating concepts.	CONSENSUS: Steering Committee
Clinicians should adapt their method of communication to meet the needs and abilities of each patient.	CONSENSUS: Steering Committee
Clinicians should check that information that has been provided has been understood; barriers to understanding should be explored and addressed.	CONSENSUS: Steering Committee

Summary of Key Messages: Acute Neck Pain

DIAGNOSIS	EVIDENCE LEVEL
Aetiology and Prevalence	
Acute neck pain is most commonly idiopathic or attributed to a whiplash accident; serious causes of acute neck pain are rare (< 1%).	*LEVEL III-3: Based on cross-sectional and prospective radiological surveys (Heller et al. 1983; Johnson and Lucas 1997)
Degenerative changes, osteoarthrosis or spondylosis of the neck are neither causes nor risk factors for idiopathic neck pain.	*LEVEL III: Based on epidemiological and radiological surveys (van der Donk et al. 1991; Fridenberg and Miller 1963)
The most consistent determinant of idiopathic neck pain is the social nature of the work environment; occupation and stress at work are weakly associated risk factors.	*LEVEL III: Based on multiple epidemiological surveys (Makela et al. 1991; Kamwendo et al. 1991a; Linton and Kamwendo 1989; Vasseljen et al. 1995; Fredriksson et al. 2002; Ariens et al. 2001)
Involvement in a motor vehicle accident is not a risk factor for developing neck pain; however individuals who experience neck pain soon after such an event are more likely to develop chronic neck pain.	*LEVEL III: Based on a prospective epidemiological study (Berglund et al. 2000)
History	
Attention should be paid to the intensity of pain because regardless of its cause, severe pain is a prognostic risk factor for chronicity and patients with severe pain may require special or more concerted interventions.	CONSENSUS: Review Group and Steering Committee
The hallmarks of serious causes of acute neck pain are to be found in the nature and mode of pain onset, its intensity and alerting features.	CONSENSUS: Review Group and Steering Committee
Eliciting a history aids the identification of potentially threatening and serious causes of acute neck pain and distinguishes them from non-threatening causes.	CONSENSUS: Review Group and Steering Committee
Physical Examination	
Physical examination does not provide a patho-anatomic diagnosis of acute idiopathic or whiplash-associated neck pain as clinical tests have poor reliability and lack validity.	*LEVEL III: Gross et al. 1996; Fjellner et al. 1999; Smedmark et al. 2000; Nansel et al. 1989; De Boer et al. 1985; Mior et al. 1985; Youdas et al. 1991; Viikari-Juntura 1987

Acute Neck Pain continued

Despite limitations, physical examination is an opportunity to identify features of potentially serious conditions.	CONSENSUS: Review Group and Steering Committee
Tenderness and restricted cervical range of movement correlate well with the presence of neck pain, confirming a local cause for the pain.	*LEVEL III: Sandmark and Nisell 1995
Ancillary Investigations	
Plain radiography is not indicated for the investigation of acute neck pain in the absence of a history of trauma, or in the absence of clinical features of a possible serious disorder.	*LEVEL III: Based on radiological surveys (Heller et al. 1983; Johnson and Lucas 1997; Hoffman et al. 2000)
In symptomatic patients with a history of trauma, radiography is indicated according the Canadian C-Spine Rule.	*LEVEL III: Based on a large epidemiological survey (Stiell et al. 2001)
CT is indicated only when: plain films are positive, suspicious or inadequate; plain films are normal but neurological signs or symptoms are present; screening films suggest injury at the occiput to C2 levels; there is severe head injury; there is severe injury with signs of lower cranial nerve injury, or pain and tenderness in the sub-occipital region.	CONSENSUS: Based on published consensus views (El Khoury et al. 1995; Kathol 1997)
Acute neck pain in conjunction with features alerting to the possibility of a serious underlying condition is an indication for MRI.	CONSENSUS: Consensus view (El Khoury et al. 1995)
Terminology	
Except for serious conditions, precise identification of the cause of neck pain is unnecessary.	CONSENSUS: Review Group and Steering Committee
Once serious causes have been recognised or excluded, terms to describe acute neck pain can be either ‘acute idiopathic neck pain’ or ‘acute whiplash-associated neck pain’.	CONSENSUS: Review Group and Steering Committee
PROGNOSIS	**EVIDENCE LEVEL**
Approximately 40% of patients recover fully from acute idiopathic neck pain, approximately 30% continue to have mild symptoms and 30% of patients continue to have moderate or severe symptoms.	*LEVEL III: Based on retrospective surveys (Gore et al. 1987; Lees and Turner 1963)
Approximately 56% of patients fully recover within three months from onset of acute whiplash-associated neck pain, 80% recover fully within one or two years; 15–40% continue to have symptoms and 5% are severely affected.	*LEVEL III, LEVEL IV: Based on prospective studies (Radanov et al. 1995; Kasch et al. 2001) and other studies with limitations (Brison et al. 2000)
Psychosocial factors are not determinants of chronicity in whiplash-associated neck pain.	*LEVEL III: Radanov et al. 1991; Borchgrevink et al. 1997
Risk factors for chronicity of following whiplash-associated neck pain are older age at time of injury, severity of initial symptoms, past history of headache or head injury.	*LEVEL III: Based on prospective studies (Radanov and Sturzenegger 1996; Suissa et al. 2001)
INTERVENTIONS	**EVIDENCE LEVEL**
Evidence of Benefit	
Advice to Stay Active (Activation) — Encouraging resumption of normal activities and movement of the neck is more effective compared to a collar and rest for acute neck pain.	LEVEL I, II: Based on systematic reviews (Spitzer et al. 1995; Verhagen et al. 2002) and a controlled trial (Borchgrevink et al. 1998)
Exercises — Gentle neck exercises commenced early post-injury are more effective compared to rest and analgesia or information and a collar in acute neck pain. Exercises performed at home are as effective for neck pain as tailored outpatient treatments at two months and appear to be more effective at two years after treatment.	LEVEL II: Based on controlled trials for short-term data (McKinney et al. 1989; Rosenfeld et al. 2000) and a blinded prospective randomised trial for long-term data, with limitations (McKinney 1989)

Acute Neck Pain continued

Multi-Modal Therapy — Multi-modal (combined) treatments inclusive of cervical passive mobilisation in combination with specific exercise alone or specific exercise with other modalities are more effective for acute neck pain in the short term compared to rest, collar use and single modality approaches.	LEVEL I, II: Based on a systematic review (Gross et al. 2002c) and two randomised controlled trials (Bonk et al. 2000; Hoving et al. 2002)
Pulsed Electromagnetic Therapy (PEMT) — Pulsed electromagnetic therapy reduces pain intensity compared to placebo in the short term but is no different to placebo at 12 weeks for acute neck pain.	LEVEL I: Based on systematic reviews (Gross et al. 2002b; Kjellman et al. 1999) of two controlled trials (Foley-Nolan et al. 1990, 1992)
Insufficient Evidence	
Acupuncture — There are no randomised controlled studies on the effect of acupuncture or infrared acupuncture in the treatment of acute neck pain. There is conflicting evidence that acupuncture is more effective compared to placebo and other treatments for neck pain in mixed populations.	No Level I or II evidence LEVEL I: Based on systematic reviews (White and Ernst 1999; Harms-Ringdahl and Nachemson 2000; Gross et al. 2002b; Smith et al. 2000)
Analgesics, Opioid — Opioids may be used, however there are no randomised controlled studies of its effectiveness for acute neck pain. In general, opioid and compound analgesics have a substantially increased risk of side effects compared with paracetamol alone.	No Level I or II evidence LEVEL I: Based on a systematic review not specific to neck pain (de Craen et al. 1996)
Analgesics, Simple — Simple analgesics may be used to treat mild to moderate pain however there is insufficient evidence that paracetamol is more effective than placebo, natural history or other measures for relieving acute neck pain.	No Level I or II evidence
Cervical Manipulation — There are no randomised controlled trials investigating the effect of cervical manipulation in the treatment of acute neck pain. Adverse effects of cervical manipulation are rare but potentially serious.	No Level I or II evidence LEVEL I: Based on systematic reviews (Hurwitz et al. 1996; Gross et al. 2002c)
Cervical Passive Mobilisation — There are no randomised controlled studies on the effect of cervical passive mobilisation compared to natural history or placebo in the treatment of acute neck pain.	No Level I or II evidence
Electrotherapy — There is insufficient evidence that electrotherapy is effective compared to no treatment in acute neck pain.	LEVEL I: Based on a systematic review (Verhagen et al. 2002) that identified two controlled trials with limitations (Fialka et al. 1989; Hendriks and Horgan 1996)
Gymnastics — There are no randomised controlled trials on the effect of gymnastics for acute neck pain. Gymnastics may be no more effective than natural history in mixed populations.	No Level I or II evidence LEVEL I: Based on a systematic review (Kjellman et al. 1999) that identified one controlled trial involving mixed populations (Takala et al. 1994)
Microbreaks — There is insufficient evidence that taking regular breaks from computer work is more effective compared to irregular breaks for preventing acute neck pain.	LEVEL II: Based on one controlled study with limitations (McLean et al. 2001)
Multi-Disciplinary Biopsychosocial Rehabilitation — There are no randomised controlled studies investigating the effect of multi-disciplinary treatment in acute neck pain. There is insufficient evidence that multi-disciplinary treatment is effective compared to other interventions for reducing neck pain in mixed populations.	No Level I or II evidence LEVEL I, II: Based on a systematic review (Karjalainen et al. 2002) that identified two controlled trials and two subsequent trials that all involved mixed populations

Acute Neck Pain continued

Muscle Relaxants — There are no randomised controlled trials investigating the efficacy of muscle relaxants for the treatment of acute neck pain.	No Level I or II evidence
Muscle relaxants are no more effective than placebo for neck pain in mixed populations.	LEVEL I, II: Based on a systematic review (Aker et al. 1996) of two studies plus one additional study, all involving mixed populations
Drowsiness, dizziness and dependency are common adverse effects of muscle relaxants.	LEVEL I: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997)
Neck School — There are no randomised controlled trials on the effect of neck school for acute neck pain.	No Level I or II evidence
Neck school appears no more effective than no treatment for neck pain in mixed populations.	LEVEL II: Based on one controlled trial (Kamwendo and Linton 1991) involving a mixed population
Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) — There are no randomised controlled trials on the effectiveness of NSAIDs for acute neck pain.	No Level I or II evidence
There is evidence that NSAIDs are no more effective than placebo ultrasound for neck pain in mixed populations.	LEVEL I: Based on a systematic review (Aker et al. 1996) that located two studies involving mixed populations
Serious adverse effects of NSAIDs include gastrointestinal complications. (e.g. bleeding, perforation)	LEVEL I: Based on systematic reviews (Bigos et al. 1994; Henry et al. 1996)
Patient Education — There are no randomised controlled trials investigating the effect of patient education as a single strategy in the treatment of acute neck pain.	No Level I or II evidence
Spray and Stretch Therapy — There are no randomised controlled trials investigating the effect of spray and stretch therapy in acute neck pain.	No Level I or II evidence
Spray and stretch therapy appears no more effective than placebo for neck pain in mixed populations.	LEVEL I: Based on one study reported in abstract form (Snow et al. 1992) cited in three systematic reviews (Aker et al. 1996; Harms-Ringdahl and Nachemson 2000; Gross et al. 2002b)
Traction — There are no randomised controlled trials investigating the effectiveness of traction for acute neck pain.	No Level I or II evidence
In mixed populations, there is evidence that traction is of no benefit compared to a range of other interventions for neck pain.	LEVEL I: Based on systematic reviews (Aker et al. 1996; Harms-Ringdahl and Nachemson 2000; Verhagen et al. 2002; van der Heijden et al. 1995; Gross et al. 2002b) of five studies with limitations involving mixed populations
Transcutaneous Electrical Nerve Stimulation (TENS) — There is insufficient evidence of benefit from TENS compared to a collar or manual therapy in acute neck pain.	LEVEL I: Based on a systematic review (Gross et al. 2002b) that identified one controlled trial (Nordemar and Thorner 1981) with equivocal results
Evidence of No Benefit	
Collars — Soft collars are not effective for acute neck pain compared to advice to resume normal activity and other interventions.	LEVEL I, II: Based on a systematic review (Harms-Ringdahl and Nachemson 2000) and multiple controlled trials

Note: * Indicative only. A higher rating of the level of evidence might apply (refer to the note in Chapter 1: Executive Summary, Limitations of Findings).

Research Agenda for Acute Neck Pain

- Observational studies to determine the sources of pain in patients whose recovery from acute neck pain is slow in order to implement diagnostic blocks before the pain becomes chronic.
- Research into prognostic indicators for idiopathic neck pain and neck pain following whiplash from mechanistic hypotheses of pain, sensory motor function and psychosocial factors.
- Randomised controlled trials to evaluate the effectiveness of specific and multi-modal interventions for acute neck pain, using the minimalist treatment of assurance, advice to stay active as the control intervention versus exercise programs. Include cost benefit analysis.
- Studies to determine if concerted and specific management of patients with risk factors for chronicity is effective at reducing progression to chronicity.

DIAGNOSIS

>Aetiology and Prevalence

In principle, neck pain may result from various disorders that affect the bones, joints, ligaments, muscles and vessels of the cervical spine. In practice, however, the specific source of neck pain is difficult to establish. This is particularly so in the case of acute neck pain. Conventional tests such as medical imaging are rarely contributory and diagnostic. Consequently, there is little information on what constitutes the differential diagnosis of acute neck pain.

Textbooks of Rheumatology (Nakano 2001; Hardin and Halla 2001; Binder 1993) provide lists of the possible causes of neck pain, however, many of the entities listed either do not pertain to the differential diagnosis of acute neck pain or there is evidence that questions their validity.

Potential sources of neck pain may be considered in the following contexts:

- Pain may be referred to the neck from another region. The classical example is angina pectoris.
- Neck pain may be one feature of a neurological disorder affecting the cervical spinal cord or nerve roots.
- The neck may be involved as one of several foci of a more widespread or systemic disease, such as rheumatoid arthritis, spondylarthropathy or polymyalgia rheumatica.
- Neck pain may be the sole presenting feature, with no indication of any visceral, neurological or systemic disorder. When this is the case, possible causes can be categorised into threatening and non-threatening disorders (Table 6.1). Threatening disorders (those that threaten to compromise the cervical spinal cord or general health) are regarded as serious conditions that should be recognised as rapidly as possible. Non-threatening disorders do not pose an immediate health threat.

Rare Causes of Acute Neck Pain

Threatening Causes (Serious Conditions)

The serious causes of acute neck pain are rare, with a prevalence in primary care of less than 1%. They include tumours and infections of the cervical spine or spinal cord, epidural haematomas and aneurysms of the vertebral artery, internal carotid artery or aorta.

Tumours and infections of the cervical vertebral column may be regarded as serious causes of neck pain because they threaten the integrity of the column and the spinal cord. They are rare causes of neck pain in general and acute neck pain in particular.

Explicit studies of the incidence and prevalence of these disorders have not been published, but inferential data are available. Two studies of plain radiography of the cervical spine, each involving over 1,000 patients, both reported not detecting any serious disorder that was not otherwise suspected on clinical grounds (Heller et al. 1983; Johnson and Lucas 1997). This zero prevalence has an upper 95% confidence limit of 0.4%, from which it can be deduced that the prevalence of serious causes of neck pain is less than 0.4%.

The literature on spinal osteomyelitis and epidural abscess is generic and does not provide explicit information on the prevalence of this condition in the cervical spine (Goodman 1988; Auten et al. 1991; Darouiche et al. 1992; Danner and Hartman 1987; Hlavin et al. 1990; Verner and Musher 1985; Nolla et al. 2002). There are no data on cervical discitis. Septic arthritis of the neck is a rare condition, described only in case reports (Muffoletto et al. 2001). A cervical epidural abscess can present with neck pain, prior to producing neurological signs, but is rare (Auten et al. 1991; Elias 1994; Scully et al. 1992; Waldman 1991; Lasker and Harter 1987; Del Curling et al. 1990).

Meningitis produces neck pain but in the context of a patient who is also very ill. A positive Kernig's sign is the hallmark of this condition.

Early in its evolution, an epidural haematoma may present with neck pain (Williams and Allegra 1994; Lobitz and Grate 1995). However, motor and sensory deficits usually develop within hours of the onset of pain (Williams and Allegra 1994; Beatty and Winston 1984; Matsumae et al. 1987). The presence of such deficits converts the presentation from one of neck pain to that of a neurological emergency.

Table 6.1

Acute Neck Pain as the Principal Presenting Feature: Possible Causes

Prevalence	Threatening	Non-Threatening
Rare (< 1%)	Spinal tumours	Retropharyngeal tendonitis
	Spinal infection	Rheumatoid arthritis
	Epidural haematoma	Spondylarthropathies
	Aneurysms	
Uncommon (< 5%)	Fractures	Fractures
		Torticollis
Common		Idiopathic
		Whiplash

Vascular disorders constitute an important differential diagnosis of acute neck pain that is often overlooked. Although headache is the usual presenting feature of aneurysms of either the internal carotid artery or the vertebral artery, they can present initially with neck pain alone. Neck pain has been the sole presenting feature in approximately 6% of cases of internal carotid aneurysm (Silbert et al. 1995; Biousse et al. 1994). It has been the initial feature of 50% to 90% of cases of vertebral aneurysm, although usually combined with headache (Silbert et al. 1995; Sturzenegger 1994). Aortic aneurysms typically present with chest pain and cardiac distress, but neck pain has been the presenting feature in approximately 6% of cases (Garrard and Barnes 1996; Hirst et al. 1958).

Key Message

Acute neck pain is most commonly idiopathic or attributed to a whiplash accident; serious causes of acute neck pain are rare (< 1%). (*Level III-3)

Non-Threatening Causes

Inflammatory arthropathies can involve the cervical spine to produce neck pain. However, they do so in the context of other features of the primary disorder. It is rare for these conditions to present with cervical involvement alone.

Only rarely does rheumatoid arthritis present with neck pain with no peripheral manifestations (Sharp et al. 1958). Approximately 10% of patients with ankylosing spondylitis may present with neck pain (Hochberg et al. 1978).

Other disorders such as Reiter's syndrome and psoriatic arthritis can affect the cervical spine, but are rare causes of neck pain (Hardin and Halla 2001) especially in the absence of peripheral features of these disorders. Polymyalgia rheumatica can involve the neck, but is a systemic disorder that does not affect the neck in isolation (Bird et al. 1979).

Patients with chondrocalcinosis of peripheral joints may develop calcification of the transverse ligament of the atlas (Constantin et al. 1996). Most often this is asymptomatic but occasionally it has been associated with an episode of acute neck pain with stiffness, fever and an erythrocyte sedimentation rate greater than 50mm/hr (Constantin et al. 1996).

Retropharyngeal tendonitis is a condition of unknown cause that is characterised by inflammation and oedema of the upper portions of longus colli. One estimate places its incidence at 1 per 400 000 population per year (Fahlgren 1986). The inflammation is often associated with calcification opposite the C2 vertebra (Fahlgren 1986; Sarkozi and Fam 1984; Ekbom et al. 1994; Karasick and Karasick 1981; Hartley 1964; Bernstein 1975; Newmark et al. 1978; Ring et al. 1994; Mihmanli et al. 2001; Guss and Jacobi 2002), but this calcification appears to be unrelated to pain, for it can be painless (Newmark et al. 1981). The condition presents with acute neck pain, but is self-limiting. Symptoms abate within one or two weeks (Fahlgren 1986; Ekbom et al. 1994; Bernstein 1975; Ring et al. 1994; Mihmanli et al. 2001).

Cervical spondylosis, cervical osteoarthrosis, degenerative disc disease and degenerative joint disease all constitute normal age changes of the cervical spine (Gore et al. 1986; Elias 1958). Some studies report that cervical spondylosis occurs slightly more frequently in symptomatic than asymptomatic individuals (Heller et al. 1983; van der Donk et al. 1991), but the odds ratios for disc degeneration or osteoarthrosis as predictors of neck pain are only 1.1 and 0.97 respectively for women and 1.7 and 1.8 for men (van der Donk et al. 1991). In other studies, the prevalence of disc degeneration has been found not to differ in symptomatic and asymptomatic individuals (Fridenberg et al. 1963). Moreover, uncovertebral osteophytes and osteoarthrosis of the synovial joints of the neck were found to be less prevalent in symptomatic individuals (Fridenberg et al. 1963).

The lack of correlation between age changes and pain means that finding spondylosis, osteoarthrosis or degenerative joint disease on a radiograph does not constitute finding the cause of the neck pain.

Key Message

Degenerative changes, osteoarthrosis or spondylosis of the neck are neither causes nor risk factors for idiopathic neck pain. (*Level III)

Uncommon Causes of Acute Neck Pain

Threatening Causes

Fractures of the cervical spine are an uncommon cause of acute neck pain (< 5%), even in patients with suspected trauma who present to accident and emergency departments. Unsuspected fractures have had a zero prevalence in radiological surveys of neck pain (Heller et al. 1983; Johnson and Lucas 1997), placing their prevalence at less than 0.4%. Even amongst patients presenting to emergency rooms with suspected cervical trauma, fractures are evident in only about 3.5% of cases (± 0.5%) (Fischer 1984; Jacobs and Schwartz 1986; Mace 1985; Roberge et al. 1988; McNamara 1988; Kreipke et al. 1989; Hoffman et al. 1992; Gerrelts et al. 1991; Bachulis et al. 1987).

Non-Threatening Causes

Torticollis is not a cause of neck pain but a condition in its own right characterised by a distinctive rotatory deformity of the head and neck. Often idiopathic, this condition can be caused by atlanto-axial subluxation (Wortzman and Dewar 1968; Jayakrishnan and Teasdale 2000; Wise et al. 1997; Fielding and Hawkins 1977; van Holsbeeck and Mackay 1989), or vertebral osteomyelitis (McKnight and Friedman 1992). Neurological causes include basal ganglion disorders and phenothiazine toxicity. A putative mechanical cause is extrapment of a meniscoid in a cervical zygapophyseal joint (Mercer and Bogduk 1993).

Common Causes of Acute Neck Pain

The common causes of acute neck pain are unknown. Two entities may be identified:

- idiopathic neck pain, which is pain for which no cause is evident or apparent
- whiplash-associated neck pain, which is pain attributed to a motor vehicle accident.

Idiopathic Neck Pain

The majority of cases of acute neck pain are idiopathic in nature as there is no identifiable or discernable source.

Whiplash-Associated Neck Pain

Whiplash is a mechanism of injury to the neck. It is not, in itself, a diagnosis. The cardinal complaint of whiplash injury is neck pain and that invites a consideration of its causes.

Although biomechanical studies have demonstrated plausible mechanisms of injury due to whiplash, these mechanisms and the injuries that they cause pertain to only a minority of cases (Bogduk and Yoganandan 2001). In the majority of cases, people recover spontaneously or with minimal intervention. Nevertheless, some can suffer serious injuries similar to those that pertain to idiopathic acute neck pain.

Fractures are uncommon in those with whiplash injuries. One study of 283 patients with neck pain after whiplash found none with fractures (Hoffman et al. 1992). Another study of 2788 patients with a history or rear-end motor-vehicle collision found only two to have a fracture (Stiell et al. 2001), yielding a prevalence of 0.07%.

Fractures attributed to whiplash have been described only in case studies or small descriptive series. The majority involve fractures of the odontoid process (Seletz 1958; Signoret et al. 1986), the laminae and articular processes of C2 (Seletz 1958; Signoret et al. 1986; Craig and Hodgson 1991) and the occipital condyles (Stroobants et al. 1994).

Vascular injuries can affect either the internal carotid or vertebral artery. Either vessel can sustain an aneurysm as a result of whiplash (Hinse et al. 1991; Janjua et al. 1996). The vertebral artery can be injured by an adjacent fracture (Tulyapronchote et al. 1994). The internal carotid artery can be strangulated by the hypoglossal nerve (Wosazek and Balzer 1990).

Other causes of neck pain, such as cervical zygapophyseal joint pain and cervical discogenic pain, may be pertinent for the differential diagnosis of chronic neck pain after whiplash, however their prevalence in those with acute neck pain after whiplash has not been investigated.

Other Issues

Referred Pain

Depending on its source and cause, neck pain may be referred to the head, to the upper limb girdle and upper limb or to the anterior chest wall. Reciprocally, pain from other sources may be referred to the neck, usually in disorders of viscera that receive a cervical innervation. Examples include angina pectoris, myocardial infarction, aortic aneurysm and disorders of the respiratory tract or oesophagus (Binder 1993). In these conditions, the clinical picture will usually indicate, or suggest, a non-cervical source of pain. Either the pain will principally be perceived as arising elsewhere than in the neck or associated features of distress or visceral dysfunction will implicate a visceral disorder.

Neurological Disorders

Neurological symptoms and signs indicate the presence of a neurological disorder. While acute neck pain may be an associated complaint, the neurological features rather than the neck pain determine the investigation and management of the condition. Reciprocally, investigations appropriate for neurological conditions are not indicated when neurological features are absent and neck pain is the only presenting feature.

Although spinal cord tumours may be associated with neck pain, their defining feature is myelopathy or radiculopathy. Similarly, conditions such as thoracic outlet syndrome, disc herniation, foraminal stenosis and synovial cysts of the cervical spine are characterised by the neurological symptoms and signs that they cause in the upper limb.

The investigation of these conditions is aimed at determining the cause of neurological impairment and should follow conventional neurological practice. Neck pain is essentially immaterial to the investigation and management of these conditions and the present guidelines do not apply. Neurological conditions should be identified early and constitute grounds for the patient to exit the management algorithm for acute neck pain.

Only rarely has neck pain been reported as the sole feature in a patient with a neurological disorder. In one case the cause was an intracranial lesion (Schattner 1996). In the other it was irritation of the dorsal root entry zone of the spinal accessory nerve by an aberrant vertebral artery that caused neuralgic pain across the trapezius (Yano et al. 1993).

Spurious Diagnoses

Neck pain has in some instances been ascribed to certain conditions to provide a diagnosis; however, they lack defining criteria or objective evidence of their existence. Examples of these are as follows:

- 'Soft-tissue injury' is a descriptor but does not serve as a diagnosis. Neither the nature of the presumed injury nor its location is specified. In effect the label means no more than neck pain in the absence of a fracture or other radiologically demonstrable lesion (Bogduk and Yoganandan 2001).
- 'Cervical strain' is an inference concerning the presumed mechanism of injury, but does not specify the nature of the lesion or its location.
- 'Psychogenic pain' lacks diagnostic criteria and is not recognised as an entity by the DSM-IV (American Psychiatric Association 1994).
- 'Fibrositis' and 'myofascial pain' are conditions whose diagnosis relies on physical examination, which has been shown to be unreliable and to lack validity in the context of neck pain (see Physical Examination).
- 'Fibromyalgia' is not a differential diagnosis for neck pain. By definition, this condition must affect multiple regions of the body (Wolfe et al. 1990). Although it can involve the neck, the patient must have pain in other regions remote from the neck.

Disputed Causes

Certain conditions have been listed in textbooks as causes of neck pain (Hardin and Halla 2001; Binder 1993), but pursuit of the literature reveals no evidence that this is the case. Rather, they may be asymptomatic or present with myelopathy or radiculopathy.

Diffuse idiopathic skeletal hyperostosis is often asymptomatic, but when symptomatic typically causes stiffness and dysphagia rather than neck pain (Hardin and Halla 2001; Binder 1993).

Ossification of the posterior longitudinal ligament can be asymptomatic. When symptomatic, it is more likely to present with myelopathy rather than neck pain (Hardin and Halla 2001; Binder 1993).

Paget's disease is regarded as a possible cause of pain when it affects other regions of the skeleton, but one large survey has reported that Paget's disease is often painless and that patients with cervical involvement had no pain referable to that region (Harinck et al. 1986).

Synovial cysts of the cervical spine are not known to cause neck pain. All case reports of this condition indicate that they cause myelopathy or radiculopathy (Takano et al. 1992; Lunardi et al. 1999; Shuma et al. 2002).

Aetiological Risk Factors (Idiopathic neck pain)

In an effort to gain insight into what might be the cause of idiopathic neck pain, epidemiologists and others have assessed possible risk factors for the development of such pain. However, these studies have refuted more factors than they have implicated.

Medical, Social and Occupational Factors

Medical, social and occupational factors refuted as risk factors for the development of idiopathic neck pain are presented in

Table 6.2

Medical, Social and Occupational Risk Factors Shown Not To Be Aetiological Risk Factors for Neck Pain

Medical	
Zygapophyseal osteoarthrosis	van der Donk et al. 1991
Degenerative disc disease	van der Donk et al. 1991
Previous pain symptoms	Westgard and Jansen 1992
Social	
Marital status	Westgard and Jansen 1992
Children	Westgard and Jansen 1992,
Economic status	Westgard and Jansen 1992; Andersen and Gaardboe 1993b
Living conditions	Westgard and Jansen 1992
Exercise	Westgard and Jansen 1992
Workload at home	Westgard and Jansen 1992
Activities outside work	Westgard and Jansen 1992
Smoking	Makela et al. 1991
Occupational	
Prolonged sitting at a work station	Kamwendo et al. 1991a (Part I)
Ergonomic variables	Kamwendo et al. 1991b (Part II)

Table 6.3

Medical, Social and Occupational Risk Factors Weakly Associated with Neck Pain

Medical	
Previous injury	Makela et al. 1991
Social	
Female gender	van der Donk et al. 1991
Education level < 8 years	Makela et al. 1991
Education level 8–12 years	Makela et al. 1991
Occupation	
Clerical	Makela et al. 1991
Industry	Makela et al. 1991
Agriculture	Makela et al. 1991
Occupational	
Physical stress at work	Makela et al. 1991
Mental stress at work	Makela et al. 1991
Working with machines	Kamwendo et al. 1991a (Part I)

Table 6.2. The odds ratios for these factors are barely greater than 1.0, with 95% confidence intervals that overlap 1.0.

A systematic review (Ariens et al. 1999; Borghouts et al. 1998) that investigated physical risk factors for neck pain found that few studies were of high quality. There was some evidence that neck pain was related to the duration of prolonged sitting at work, and to bending and twisting of the trunk at work. Factors such as neck flexion, arm-force, arm posture, hand-arm vibration and workplace design emerged as factors only if less stringent standards of evidence were accepted.

Factors that are significantly, but weakly, associated with idiopathic neck pain are listed in Table 6.3. These have odds ratios ranging between 1.0 and 2.5, with 95% confidence intervals between 1.0 and 3.0. Comorbid illnesses such as back pain, headache, and cardiovascular and digestive disorders are risk factors for idiopathic neck pain, but only amongst patients who are moderately or severely affected by these other conditions (Cote et al. 2000).

Psychosocial Risk Factors

The results of cross-sectional studies demonstrate that psychosocial factors are not significantly related to neck pain. Those factors appearing significant on univariate analysis disappear on multivariate analysis (Westgard and Jansen 1992; Linton and Kamwendo 1989). Psychological state accounted for only 2% of the variance in symptoms (Westgard and Jansen 1992). Specific psychosocial factors that are not associated risk factors for neck pain are listed in Table 6.4.

Only two psychosocial factors have been shown to be significantly associated with neck pain. One is a sense of inadequacy (van der Donk et al. 1991). The other is general tension (Vasseljen et al. 1995). The latter feature was not defined

Table 6.4

Psychosocial Risk Factors Shown Not To Be Related to Neck Pain

- Social support
- Self-confidence
- Sense of humour
- Impulsiveness
- Anxiety
- Extroversion
- Neuroticism
- Depression
- Coping ability
- Ability to solve problems
- Irritability
- Psychosis
- Lying

Note: Based on data from Vasseljen et al. 1995.

prospectively in the study. Rather it reflected what the subjects perceived as a general state of tension.

The one prospective study of risk factors for neck pain studied 2222 men over three years. It found no consistent relationships between neck pain and psychosocial factors as measured by the Middlesex Hospital Questionnaire and the Maudsley Personality Inventory (Pietri-Taleb et al. 1994). Such relationships that were found were not consistent across all occupations and had odds ratios barely greater than 1.0, with 95% confidence intervals that overlapped 1.0.

Work Environment Factors

The risk factors that have consistently emerged across multiple studies as strongly related to neck pain pertain to the psychosocial work environment (Kamwendo et al. 1991a; Linton and Kamwendo 1989; Vasseljen et al. 1995; Fredriksson et al. 2002; Ariens et al. 2001).

The critical components of this factor are:

- lack of co-operation between workers (Linton and Kamwendo 1989; Ariens et al. 2001)
- lack of camaraderie (Linton and Kamwendo 1989)
- lack of possibility to influence or vary ones workload (Linton and Kamwendo 1989; Vasseljen et al. 1995)
- high work demands (Linton and Kamwendo 1989; Fredriksson et al. 2002; Ariens et al. 2001)
- reduced opportunity to acquire or use new knowledge (Fredriksson et al. 2002)
- lack of opportunity to participate in planning of work (Fredriksson et al. 2002)

The strongest determinants of neck pain, therefore, are not physical or ergonomic factors or personal psychosocial factors. They lie in the social nature of the work environment, ostensibly in whether an individual feels that they work in a cooperative environment or an oppressive one. The nature of these factors is such that they may be amenable to change and could be targeted in the management of neck pain.

Key Message

The most consistent determinant of idiopathic neck pain is the social nature of the work environment; occupation and stress at work are weakly associated risk factors. (*Level III)

Aetiological Risk Factors (Whiplash-Associated Neck Pain)

Involvement in a motor vehicle accident does not mean that an individual will develop neck pain. Many passengers and drivers do not develop neck pain after a motor vehicle accident and the subsequent prevalence of chronic neck pain amongst such individuals is not greater than that in the general community who have never been involved in an accident (Berglund et al. 2000). However, people who develop neck pain soon after a motor vehicle accident have a relative risk of 2.7 (95%CI 2.1, 3.5) of developing chronic neck pain (Berglund et al. 2000). These data indicate that some victims of a motor vehicle accident sustain an injury that renders them symptomatic. The risk factor for chronicity is not being involved in a motor vehicle accident, per se, but developing acute neck pain soon after the accident.

Key Message

Involvement in a motor vehicle accident is not a risk factor for developing neck pain; however individuals who experience neck pain soon after such an event are more likely to develop chronic neck pain. (*Level III)

>History

No particular method of assessing the history of acute neck pain is universally accepted, nor has the validity of particular elements of history been formally assessed in the context of neck pain. However, eliciting a history can be critical in the assessment of neck pain. Its cardinal role is to identify alerting features of a serious underlying cause for the pain.

Pain History

Site

Determining the site of pain establishes that an individual is in fact, experiencing neck pain. Clinicians should note if the pain appears to arise in the neck as opposed to being referred to the neck from another site.

Distribution

Neck pain can be referred to the head, upper limb girdle or chest wall. The extent or pattern of referral is not diagnostic of the cause of pain, but it can provide prima facie information as to the possible location of the source of pain. Experiments in normal volunteers (Dwyer et al. 1990; Aprill et al. 1990) and observations in those undergoing invasive diagnostic procedures (Fukui et al. 1996; Bogduk and Marsland 1988; Barnsley et al. 1995; Lord et al. 1996; Grubb and Kelly 2000; Schellhas et al. 1996) have shown that pain stemming from the zygapophyseal joints and cervical intervertebral discs follows segmental patterns. It is emphasised, however, that these patterns do not implicate a particular structure or disorder as the cause of pain. They only indicate its likely segmental location.

Somatic referred pain should be distinguished from cervical radicular pain. To some extent, the pattern of radiation of pain serves to make this distinction. Although somatic pain has been reported to extend into the forearm and hand in some studies of normal volunteers (Kellgren 1939; Feinstein et al. 1954), no clinical studies have reported relief of such a distal referral of pain following anaesthetisation or successful treatment of a somatic source of neck pain. Accordingly, somatic referred pain tends to concentrate around the upper limb girdle or proximal arm. A more distal radiation of pain implies radicular pain, but the distinction between radicular pain and somatic referred pain is better made on the basis of the quality of pain and its associated features (see below), which are far more important than the distribution of pain.

Duration of Illness

Establishing the duration of illness is relevant because duration of illness predicates investigations and treatment. Measures that may be appropriate for chronic neck pain may not be appropriate for acute neck pain. Therefore, for the management of acute neck pain, it should be established that the patient has pain that has not lasted longer than three months.

Onset (Precipitating Event)

Pursuing the circumstances of onset may provide clues to the possible aetiology of acute neck pain. In most instances, no valid information will be obtained, but some circumstances may be relevant.

A history of injury alerts clinicians to the possibility of a fracture being the source of pain. A diagnosis of fracture will be established by imaging. Fractures are less likely to be the cause of pain if the injury has been inertial but more likely if an external force has been applied to the neck or head.

Therefore, it is worthwhile to obtain a description of the nature of injury and an estimate of the magnitude of forces involved. Information on imaging following trauma is provided in the section 'Ancillary Investigations'.

A recent viral illness prior to the onset of pain may be a clue to the possibility of retropharyngeal tendonitis.

Similarly, recent history of penetrating injury in the form of a surgical or dental procedure, catheterisation or cannulation, a wound, or self-injection constitutes an alerting feature for possible cervical osteomyelitis, epidural abscess or discitis.

Mode of Onset

A history of sudden onset of pain, particularly if the pain is also severe, should be taken as an alerting feature for a possible serious cause of pain. However, the validity of this feature has not been measured in the context of neck pain.

Quality

The quality of somatic neck pain is usually and typically dull, aching or pressure-like. Deviations from this description constitute prima facie evidence of an unusual cause of pain. Lancinating or stabbing pain, particularly if it is 'electrical' in quality, is suggestive of a neuropathic cause. Lancinating pain travelling from the neck into the upper limb is strongly suggestive of radicular pain, particular if it extends into the forearm and hand.

Intensity

The intensity of pain should be recorded on a measurement device (refer to Chapter 2: Acute Pain Management) to provide a baseline from which to evaluate progress.

Pain intensity, however, has not been proven to predict the cause of pain and description of severity will vary from person to person. Serious causes of neck pain should be suspected primarily on other grounds, such as sudden onset and alerting features.

When severe pain is the only available clinical feature, it may be difficult to distinguish whether it is an amplification or exaggeration of intensity and, therefore, a sign of distress or a serious cause.

Given that severe pain at onset is a prognostic risk factor for chronicity, those with high pain intensity should be identified at the outset and earmarked for more concerted or special management.

Key Message

Attention should be paid to the intensity of pain because regardless of its cause severe pain is a prognostic risk factor for chronicity and patients with severe pain may require special or more concerted interventions. (Consensus)

Frequency

Most causes of neck pain do not exhibit any characteristic frequency or periodicity. Paroxysmal pain is virtually diagnostic of neuropathic pain, but neck pain is rarely neuropathic in origin.

Duration

Pain duration is not diagnostic of any particular cause of neck pain. The pain may be constant or of variable duration, irrespective of cause.

Time of Onset

Onset of neck pain during waking hours is not diagnostic of any cause. However, pain that affects or prevents sleep should alert clinicians to a possible serious cause.

Precipitating and Aggravating Factors

Various neck movements may precipitate virtually any form of neck pain and are not diagnostic of any particular cause. However, people who consciously avoid rotation of the head for fear of precipitating their pain should be taken seriously, as this behaviour can be a feature of atlanto-axial instability due to odontoid fractures or tears of the alar ligaments (Dvorak et al. 1987).

Most patients will report that neck movements aggravate their neck pain, but aggravating factors are not diagnostic of any particular source or cause of pain. Of greater significance is the absence of any aggravating factors. This may suggest a vascular lesion or a lesion within a vertebral body that is not affected by movement; or a cause of pain that is not located in the cervical spine.

Although postural abnormalities may accompany neck pain, the sensitivity of the sign is poor (Greigel-Morris et al. 1992).

Relieving Factors

In most cases neck pain will be relieved to some extent by lying down or otherwise resting the neck. Of note is the absence of relieving factors. Pain that is not relieved to any extent by simple physical measures or by simple analgesics may be indicative of a serious cause, particularly if it is of sudden, recent onset.

Key Message

The hallmarks of serious causes of acute neck pain are to be found in the nature and mode of pain onset, its intensity and alerting features. (Consensus)

Alerting Features of Serious Conditions (see Table 6.5)

Features that alert to the presence of specific and serious conditions can be identified through a comprehensive review of past history of illness and general health status. The presence of the following features in conjunction with acute neck pain should prompt further investigation. The following list is a guide only; it is not exhaustive.

Tumour

- A past history of cancer and unexplained weight loss are features alerting to the possibility of metastatic disease.
- Dysphagia may be a sign of a prevertebral lesion that causes neck pain.
- Headache and vomiting in the presence of neck pain are alerting features of an intracranial lesion (Schattner 1996).

Infection

- Immunosuppression, diabetes mellitus, cirrhosis, HIV/AIDS, use of steroids, recent or concurrent infection and recent penetrating injury are considered risk factors for infection (Vilke and Honingford 1996). Fever or night sweats may be indicative of infection or neoplasm; fever is a feature of spinal osteomyelitis in about 42% of cases (Goodman 1988).
- Exposure to infectious organisms should be considered (e.g. *Neisseria meningitidis, Mycobacterium tuberculosis*).

Fracture

The use of corticosteroids constitutes a risk factor for pathological fracture due to osteoporosis, but pathological fractures of the cervical spine are rare.

Neurological Conditions

Neurological symptoms in the upper limb or lower limb indicate the possibility of a neurological condition.

Inflammatory Arthropathies

- Pain in other regions of the musculoskeletal system is a cue to consider systemic arthropathy or a systemic inflammatory disorder.
- Psoriasis and related skin lesions may be indicative of spondylarthropathy.

Features of Other Conditions

- Transient ischaemic attacks are the cardinal features of aneurysms of the vertebral or internal carotid arteries (Silbert et al. 1995; Biousse et al. 1994; Sturzenegger 1994). The onset of such attacks after the onset of neck pain may indicate the presence of an aneurysm.
- Anticoagulant use is a risk factor for cerebral or spinal haemorrhage (Schattner 1996; Hurst et al. 1989; Mustafa and Gallino 1988; Krolick and Cintrom 1991; Rose et al. 1990).
- Amongst endocrine disorders, hyperparathyroidism can cause osteitis fibrosa, which can be a cause of spinal pain with no other clinical features.

Table 6.5 is a summary of some features described in the sections on History and Physical Examination that may be associated with serious conditions such as malignancy, infection and fracture. Although these features have only face validity in the context of acute neck pain, a similar device has proved effective in screening for serious causes of low back pain (McGuirk et al. 2001).

While the predictive values of these alerting features have not been tested specifically in relation to acute neck pain, their presence in conjunction with such pain should prompt further investigation.

Key Message

Eliciting a pain history aids the identification of potentially threatening and serious causes of acute neck pain and distinguishes them from non-threatening causes. (Consensus)

>Physical Examination

Physical examination of the neck can be divided into three main categories:

- general examination
- neurological examination
- musculoskeletal examination

General Examination

A general physical examination is relevant for the assessment of medical conditions that are not musculoskeletal in nature but can result in neck pain. These tests have not been formally assessed for reliability and validity. They include:

- Kernig's sign for meningitis
- palpation of the viscera of the throat in cases of anterior neck pain
- palpation of cervical lymph nodes to assess for lymphadenopathy
- detecting ptosis and miosis to assess for Horner's syndrome
- recognising pigmentation in neurofibromatosis

Conducting routine observations such as obtaining a temperature is an essential step in screening for spinal infection. However, although fever is highly specific for infection, it has a low sensitivity. Only some 42% of patients with spinal infection exhibit a fever (Goodman 1988).

Neurological Examination

The presence of neurological symptoms warrants a full neurological examination.

Cervical Radicular Pain

Neck pain should not be confused with cervical radicular pain (pain in the upper limb). While radicular pain warrants a careful neurological examination, such pain does not constitute neck pain. Therefore, indications for a neurological examination that apply for radicular pain do not apply for neck pain.

One exception to this rule is the presence of neck pain and headache without neurological symptoms. It is possible for an intracranial lesion to present with neck pain. For such conditions, fundoscopy should be undertaken to search for signs of elevated intracranial pressure.

Screening for Neurological Sources

A screening examination may be undertaken to determine if there is gross sensory loss or weakness in the upper and lower

Table 6.5

Alerting Features of Serious Conditions Associated with Acute Neck Pain

Feature or Risk Factor	Condition
Symptoms and signs of infection (e.g. fever, night sweats)	Infection
Risk factors for infection (e.g. underlying disease process, immunosuppression, penetrating wound, exposure to infectious diseases)	
History of trauma	Fracture
Use of corticosteroids	
Past history of malignancy	Tumour
Age > 50 years	
Failure to improve with treatment	
Unexplained weight loss	
Dysphagia, headache, vomiting	
Neurological symptoms in the limbs	Neurological condition
Cerebrovascular symptoms or signs, anticoagulant use	Cerebral or spinal hemorrhage
Cardiovascular risk factors, transient ischaemic attack	Vertebral or carotid aneurysm

limbs. If these symptoms are absent and the individual is ambulatory, they are unlikely to have a neurological cause for their neck pain. If non-ambulatory, a careful neurological examination is warranted followed by the next stage of assessment of neck pain following trauma (An 1998).

Musculoskeletal Examination

There are no signs that might be elicited that allow identification of a patho-anatomic source of idiopathic pain. Conventional clinical tests lack reliability or validity or both.

Key Message

Physical examination does not provide a patho-anatomic diagnosis of acute idiopathic or whiplash-associated neck pain as clinical tests have poor reliability and lack validity. (*Level III)

Despite these limitations, musculoskeletal examination of the neck informs the examiner whether or not the neck, or another structure, is the site of pain. The presence of tenderness and limited range of motion correlates well with the presence of neck pain (Sandmark and Nisell 1995). Finding such features implies the presence of a local abnormality in the neck.

Key Message

Despite limitations, physical examination is an opportunity to identify features of potentially serious conditions. (Consensus)

Perhaps more significant is finding no physical signs. The absence of musculoskeletal signs invites a consideration of pain referred to the neck, rather than a local cause of pain or a deep source of pain that is not palpable and which is not affected by movements of the neck, such as a vascular disorder or vertebral tumour.

Palpation

For the examination of the cervical spine, the reliability is poor or only fair for detecting intersegmental movements or 'fixations' (Gross et al. 1996; Fjellner et al. 1999; Smedmark et al. 2000; Nansel et al. 1989; De Boer et al. 1985; Mior et al. 1985).

For the detection of tenderness in the posterior neck muscles, reliability is fair to good (Viikari-Juntura 1987; Levoska et al. 1991; Andersen and Gaardboe 1993a). Reliability is quite good for detecting tenderness over the zygapophyseal joints (Hubka and Phelan 1994). Tenderness, however, is also a non-specific sign not indicative of any particular disorder.

So-called trigger points in the neck lack operational criteria. The source reference on this topic specifically excuses trigger points in the neck from satisfying the conventional diagnostic criteria (Travell and Simons 1993). A review of this problem revealed that cervical trigger points cannot be distinguished clinically from tenderness overlying a painful zygapophyseal joint (Bogduk and Simons 1993).

Movement

There is poor reliability for determining range of motion by visual inspection (Youdas et al. 1991), but reasonable reliability for assessing whether movements are limited or markedly limited (Viikari-Juntura 1987). Restricted movement, however, is a non-specific sign not indicative of any particular disorder.

Key Message

Tenderness and restricted cervical range of movement correlate well with the presence of neck pain, confirming a local cause for the pain. (*Level III)

>Ancillary Investigations

Most causes of neck pain will not be evident on any form of medical imaging. If undertaken in the pursuit of a diagnosis, medical imaging will, therefore, most often yield normal results. Conversely, medical imaging may yield false-positive results or show spurious findings that may be misconstrued as the cause of pain.

Serious causes of neck pain are rare. The potential yield of imaging undertaken as a screening test will be very small. If a comprehensive history has been taken and there is no evidence of a serious disorder clinically and there are no risk factors for a serious disorder, the possibility of an occult cause of pain is remote. Furthermore, the sensitivity of tests such as plain radiography is low. Therefore, a normal plain film does not guarantee that a serious disorder has been excluded.

Refer to Appendix C: Ancillary Investigations for information on other ancillary investigations.

Plain Radiography

Plain radiography demonstrates the structure of bones and, to a limited extent, the structure of joints. It will not demonstrate lesions that do not affect bones and has a limited sensitivity even for lesions that do affect bones. Consequently, plain radiography serves poorly either as a diagnostic test to detect causes of neck pain or as a screening test to exclude occult lesions.

Infection

Early in the course of osteomyelitis or discitis, plain films may be normal. They are diagnostic only once there has been substantial destruction of bone, which may be three to six weeks after onset of pain (Goodman 1988). Furthermore, not all spinal infections involve the vertebrae; bone is involved in 44% of epidural abscesses (Darouiche et al. 1992).

Bone scan and magnetic resonance imaging (MRI) are both very sensitive for infection (Goodman 1988; Bassett 1987; Berquist et al. 1985; Modic et al. 1985) and more useful to diagnose spinal infection than plain radiography. MRI offers better resolution of the intervertebral discs and paravertebral soft-tissues (Bassett 1987). These properties, however, do not justify the wholesale application of bone scan or MRI as screening tests. Their use is justified only when risk factors and clinical signs of an infection are present or if a blood count reveals leucocytosis or an elevated erythrocyte sedimentation rate.

Tumour

Tumours are rare causes of neck pain, according to two studies. A British study (Heller et al. 1983) of 1263 patients at one hospital over 12 months found that, 'There were no unexpected findings of malignancy or infection in any of the films', and, 'The request for xray films of the cervical spine 'just in case' such a finding is present is probably unjustified.'

A similar study in the United States examined 848 outpatients and found that, 'In no patient was a serious diagnosis detected, including fractures, dislocations, or tumours.' Furthermore, on follow-up for as long as five years, the study found that, '… no medically dangerous diagnoses would have been missed if the cervical spine series had not been done.' (Johnson and Lucas 1997).

Given that neither of these studies found any malignancies and that each comprised approximately 1,000 people, the 95% confidence limit of a zero sets the upper limit at 0.38% for the possible prevalence of tumours as a cause of neck pain. This figure does not justify the use of plain films to screen for possible tumours.

The pursuit of tumours is justified only in cases where alerting features for malignancy have been identified. In that event, MRI is the investigation of choice because of its combined high sensitivity and specificity for spinal tumours.

Fracture

Fear of missing a fracture is a strong motivation for ordering plain films of the cervical spine. According to one survey, 33% of cervical spine studies are undertaken for medicolegal purposes (Miller et al. 1994). Another study found that 'medicolegal purposes' is the most common reason for cervical spine radiography (Eliastam et al. 1980). Yet, that study found that although 236 of 304 cervical spine investigations were undertaken for medicolegal purposes, only one fracture was detected (Roberge et al. 1988).

The fear of missing a fracture is not justified on epidemiological grounds. Assessment for the presence of alerting features can be used to determine the need for radiography.

Trauma

No Symptoms Following Trauma

People without neck pain who are alert and otherwise competent and who have no neurological signs, have zero chance of having a fracture (Roberge et al. 1988; Fischer 1984; Kreipke et al. 1989; Velmahos et al. 1996; Vandemark 1990; Roth et al. 1994; Wales et al. 1980; Saddison et al. 1991). In such cases, the American College of Radiology resolved that radiographs were unnecessary (Kathol 1997). When tested in 34,509 patients, the criteria for avoiding cervical spine radiography listed in Figure 6.1 had a sensitivity of 99.6% and a negative predictive value of 99.9%, with confidence intervals of 99.8% to 100% (Hoffman et al. 2000). Only two patients were identified with clinically significant fractures that would not have been detected had the criteria been applied. One had an avulsion fracture of a vertebral endplate that was aysmptomatic; the other had a fracture of the lamina of C6.

Occult fractures in asymptomatic patients are rare and are reported in case reports (Thambyrajah 1972; Maull and Sachatello 1977; Bresler and Rich 1982; Walter et al. 1984; Haines 1986; Ogden and Dunn 1986; McKee et al. 1990; Mace 1991; Mace 1992). Close scrutiny of this literature, however, reveals that either insufficient clinical data were presented in these reports or the patients had tenderness with no pain, had been intoxicated or in fact did have pain (Roberge et al. 1988; Velmahos et al. 1996; Mirvis et al. 1989; Roberge 1993). Accordingly, provided the operational criteria are strictly followed, occult fracture is unlikely in the absence of symptoms.

Criteria for Not Undertaking Radiography in Patients with a History of Cervical Spine Trauma

- Absence of posterior midline tenderness
- Absence of neurological deficit
- Normal level of alertness
- No evidence of intoxication
- Absence of clinically apparent pain that might distract the patient from the pain of a cervical spine injury

Figure 6.1

Criteria for not undertaking radiography in patients with a history of cervical spine trauma. Based on Kathol 1997.

Symptomatic Following Trauma

In cases where there is a history of trauma with symptoms, no clinical signs have been shown to be predictive of fractures of the cervical spine. Whereas various features have high sensitivity, they lack specificity. Neurological signs have high specificity but low sensitivity (McNamara 1988; Roberge and Wears 1992). This pattern arises because pain, tenderness and reduced range of motion are common in people with and without fractures. Neurological signs are often absent irrespective of whether a fracture is present or not. In essence, there are no characteristic clinical features of a cervical spine fracture.

Even if clinical features are combined, formal studies have shown that clinical impression has a specificity of 0.92 and a sensitivity of only 0.50 for the diagnosis of a fracture (Jacobs and Schwarz 1986). For predicting the result of the radiograph the specificity was 0.94 and the sensitivity only 0.46.

Key Message

Plain radiography is not indicated for the investigation of acute neck pain in the absence of a history of trauma, or in the absence of clinical features of a possible serious disorder. (*Level III)

Protocols

Plain radiography is an imperfect tool for the detection of cervical spinal fractures. Single, cross-table lateral views miss fractures of the odontoid process, the lateral masses, laminae, transverse processes and vertebral endplates (Streitweiser et al. 1983; MacDonald et al. 1990; Cohn et al. 1991; Lee and Woodring 1991). For this reason, the recommended protocol for the assessment of fractures requires at least a lateral view, an antero-posterior view and an open-mouth view (Kathol 1997; Dreyzin and Esses 1993; Johnson 1996). Some have questioned the utility of the antero-posterior view on the grounds that it reveals nothing that is not otherwise evident on the open-mouth view (Holliman et al. 1991).

Canadian C-Spine Rule

Canadian physicians developed a rule to apply to trauma patients who are stable and alert. The rule operates as an algorithm (Figure 6.2). When tested prospectively in 8,924 patients, the C-Spine Rule (Stiell et al. 2001) achieved a sensitivity of 100% and a specificity of 42.5%. The lack of specificity meant that radiographs were taken in 57.5% of patients who did not have fractures, but following the rule nevertheless resulted in an estimated 15.5% decrease in the use of radiographs. However, the high sensitivity meant that all significant fractures were detected.

With respect to the fear of missing a fracture, the Canadian C-Spine Rule provides reassurance backed by statistics. Physicians who follow the rule can be assured that a fracture will not be missed, with a 95% confidence range of 98% to 100%.

Key Message

In symptomatic patients with a history of trauma, radiography is indicated according the Canadian C-Spine Rule. (*Level III)

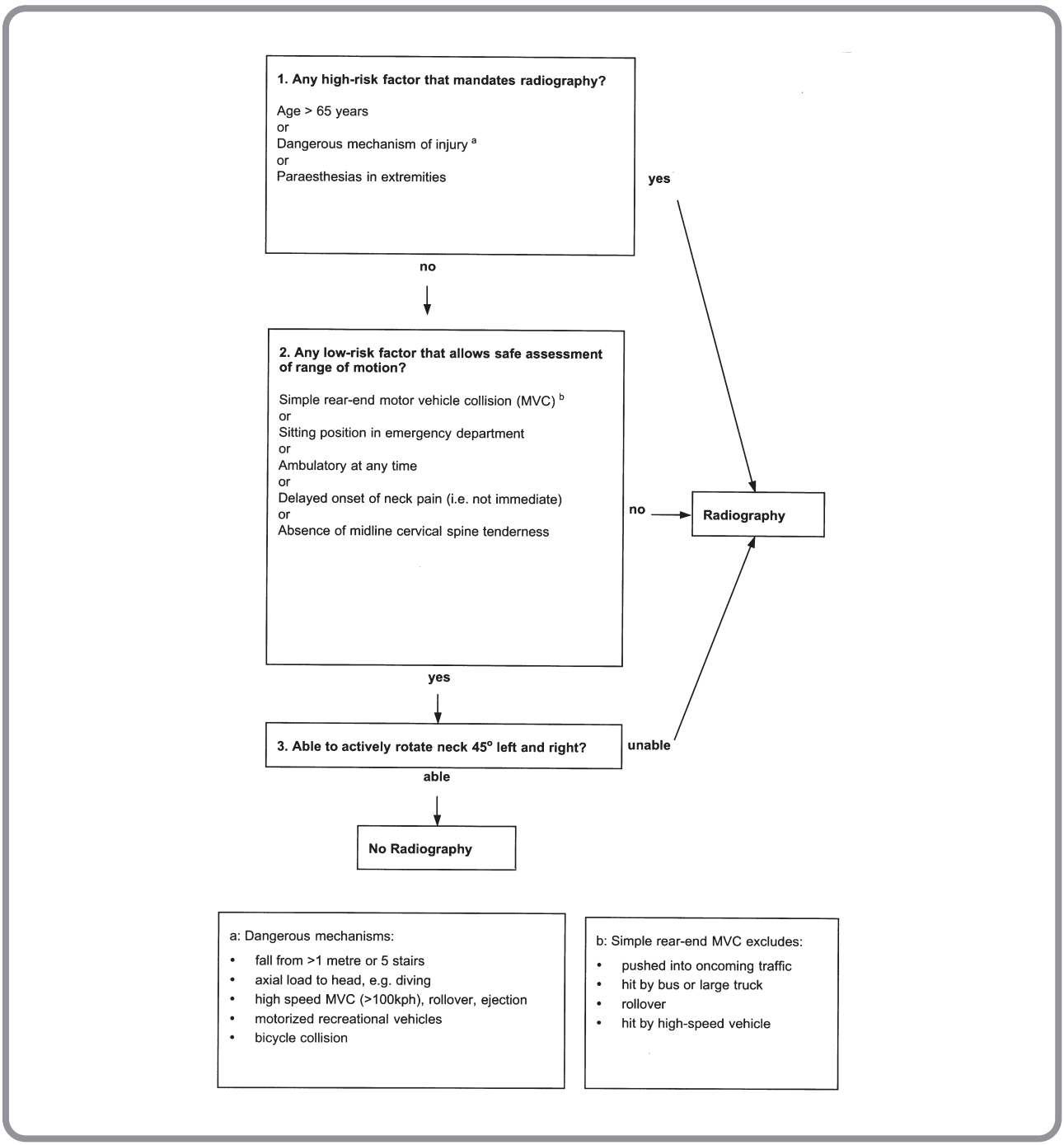

Figure 6.2
The Canadian C-Spine Rule. Based on Stiell et al. (2001).

Spurious Conditions

Plain radiography often reveals features of the cervical spine identified as abnormalities. These features may be mistakenly used as a diagnosis or an explanation for the pain.

Cervical spondylosis is the most common radiological finding in those with neck pain (Heller et al. 1983; Johnson and Lucas 1997), but it does not constitute a diagnosis. The radiological changes of cervical spondylosis are normal changes that occur increasingly frequently with age in asymptomatic individuals (Gore et al. 1986; Elias 1958). Most commonly, they affect the C5–6 and C6–7 segments, followed by C4–5 and C3–4 (Fridenburg and Miller 1963).

In some studies, the changes of cervical spondylosis were weakly associated with neck pain (Heller et al. 1983; van der Donk et al. 1991), but the odds ratios were only 1.1 and 0.97 respectively for women and 1.7 and 1.8 for men (van der Donk et al. 1991). In other studies, the prevalence of disc degeneration was not significantly different between symptomatic and asymptomatic individuals (Fridenberg and Miller 1963). Indeed, uncovertebral osteophytes and zygapophyseal osteoarthrosis were less prevalent in symptomatic individuals (Fridenberg and Miller 1963). The lack of significant correlation precludes cervical spondylosis from being a legitimate diagnosis of neck pain.

Loss of lordosis is a normal variant of the cervical spine. It is equally prevalent in the presence of acute neck pain, chronic neck pain and no symptoms. It is independent of age and symptoms, but is more common in females (Helliwell et al. 1994).

Flexion-Extension Views

The role of flexion-extension views of the cervical spine has been difficult to define precisely. However, authorities agree that they are indicated for patients with neck pain following trauma, but not for determining the source of neck pain. They are a test for ligament damage and instability (Fazl et al. 1990; Lewis et al. 1991; Wilberger and Maroon 1990).

The American College of Radiology recommends that flexion-extension views be used for symptomatic patients in whom ligamentous injury is suspected and whose plain films are normal (Kathol 1997). So-called 'fingerprints' of ligamentous injury include, kyphosis, subluxation, wedging of a disc space, facet displacement and fanning of the spinous processes (Fazl et al. 1990). Other authorities reserve this investigation for 'high-risk' cases in consultation with a spine specialist, only if the patient can perform movements under physician monitoring, which may be 10–14 days after injury (Vandemark 1990).

A retrospective study, however, found that the yield of flexion-extension radiographs was very low (Wang et al. 1999). In 290 patients, flexion-extension radiographs revealed instability in only one patient who had no symptoms at one month and required no additional treatment. This study calculated that the 95% confidence interval for a positive finding of instability that required treatment was 0% to 1.3%. It also found that lack of movement, ostensibly because of pain, confounded flexion-extension radiography in some 34% of cases. The study recommended that flexion-extension radiographs not be used routinely. Instead, patients should be assessed clinically for the amplitude of movement possible. Patients without adequate movement could be evaluated at a later time, if indicated, when they are better able to flex and extend their cervical spine in order to achieve an adequate study.

Computed Tomography Scanning

Although computed tomography (CT) scanning may be useful in the investigation of radiculopathy and myelopathy (Ellenberg et al. 1994; Bernhardt et al. 1993; Bell and Ross 1992), it is not indicated for the primary investigation of neck pain. Indeed, two recent textbooks have advised against the use of CT for the investigation of neck pain (Poletti and Handal 1995; Barnsley 1998).

Descriptive studies (Mirvis et al. 1989; Gerrelts et al. 1991; Borock et al. 1991; Tehranzadeh et al. 1994) and review articles (Acheson et al. 1987; Daffner 1992) have consistently reported that CT should be reserved for cases where fracture is suspected and plain films are positive, suspicious or inadequate. If plain films are adequate, the chances of finding a fracture are remote (Schleehauf et al. 1989; Borock et al. 1991, Hoffman et al. 1992). Moreover, such fractures usually affect the spinous processes, laminae or transverse processes, which do not constitute a threat to the integrity of the cervical spine.

In severely injured patients, suboccipital injuries and rotatory atlanto-axial dislocations can escape detection on plain films (El Khoury et al. 1995; Kathol 1997). In one study, 8% of severely injured patients had fractures of the odontoid process or of C1 or C2 (Blacksin and Lee 1995). Accordingly, CT of the suboccipital region is indicated when views of the odontoid process are inadequate on plain films, in cases of severe head injury and in patients with signs of lower cranial nerve injury or pain and tenderness at the base of the occiput (El Khoury et al. 1995).

The American College of Radiology (Kathol 1997) resolved that CT of the cervical spine is indicated in patients:

- with neurological signs or symptoms whose plain films were normal
- with screening films suggesting injury at the occiput to C2 levels.

CT for Small Fractures

CT has the ability to detect small fractures of the articular pillars or the facets of the cervical zygapophyseal joints (Lee and Woodring 1991; Clark et al. 1988; Woodring and Goldstein 1982; Binet et al. 1977; Yetkin et al. 1985). In cervical spine injury, such fractures constitute about 20% (Clark et al. 1988; Woodring and Goldstein 1982; Binet et al. 1977) of all fractures detected. However, approximately 87% of these small fractures are not detected on plain films (Woodring and Goldstein 1982). About one-third present with neurological signs; the remainder present only with neck pain (Clark et al. 1988; Woodring and Goldstein 1982).

Small articular fractures may constitute occult sources of acute neck pain, but that does not justify the use of CT as a primary investigation in the pursuit of these lesions. Although articular fractures constitute 20% of all fractures, the prevalence of fractures in general is less than 4% in patients with a history of injury and less than 0.4% in those with no injury (See Aetiology and Prevalence). There is a low chance of CT being diagnostic.

Indications for the use of CT or conventional tomography for the pursuit of small fractures include:

- patients with positive or suspicious findings on antero-posterior radiographs (Lee and Woodring 1991)
- patients who develop radiculopathy (Woodring and Goldstein 1982)
- patients with persistent pain (Binet et al. 1977).

The pursuit of small fractures involves considerable radiation exposure in order to obtain high-resolution images across the entire cervical spine. It may be more efficient and involve less radiation exposure if a suspected painful joint were to be identified initially with diagnostic blocks.

Key Message

CT is indicated only when: plain films are positive, suspicious or inadequate; plain films are normal but neurological signs or symptoms are present; screening films suggest injury at the occiput to C2 levels; there is severe head injury; there is severe injury with signs of lower cranial nerve injury or pain and tenderness in the sub-occipital region. (Consensus)

Magnetic Resonance Imaging

There is no literature on the diagnostic utility of magnetic resonance imaging (MRI) for idiopathic neck pain. The only available literature addresses neck pain after whiplash. Otherwise, the literature describes findings in the cervical spines of asymptomatic individuals.

In asymptomatic individuals, studies disagree on the prevalence of particular abnormalities, but agree that abnormalities such as disc degeneration, spondylosis, disc herniation, bulging disc and foraminal stenosis are common in individuals with no neck pain (Boden et al. 1990; Teresi et al. 1987). This observation is consonant with the literature on plain radiography,

which reports that degenerative changes and spondylosis are asymptomatic age-related changes.

In those with whiplash-associated neck pain, MRI demonstrates abnormalities that are evident in asymptomatic individuals, with approximately the same prevalence (Ellertsson et al. 1978; Pettersson et al. 1994; Fagerlund et al. 1995; Borchgrevink et al. 1995; Ronnen et al. 1996; Karlsborg et al. 1997; Voyvodic et al. 1997).

One study reported that MRI revealed disrupted discs or ligaments in 36% (n = 174) of patients with 'potent instability' of the cervical spine, but this term was not defined (Benzel et al. 1996).

Reviews of imaging for cervical spine injuries restrict the utility of MRI to patients with spinal cord injuries, vertebral artery lesions (El Khoury et al. 1995) and neurological deficits (Daffner 1992; Bell and Ross 1992; Kathol 1997). Uncomplicated neck pain is not an indication for MRI.

Key Message

Acute neck pain in conjunction with features alerting to the possibility of a serious underlying condition is an indication for MRI. (Consensus)

Single Photon Emission Computed Tomography

One study of single photon emission computed tomography (SPECT) in a highly selected small sample of patients with whiplash-associated neck pain suggested that SPECT may be useful in the early detection of small fractures in such individuals (Seitz et al. 1995). These findings have not been confirmed, or elaborated in a large and representative sample of patients.

Other Ancillary Investigations

Refer to Appendix C: Ancillary Investigations.

>Terminology

The common causes of acute neck pain are unknown. Generically only two entities might be identified:

- *idiopathic neck pain*, being neck pain with no obvious aetiological basis, and
- *whiplash associated neck pain*, defined solely by the association of onset of pain with a motor vehicle accident.

If the patient does not have a neurological disorder, a vascular disorder, a tumour or an infection and if they have no history of trauma, further pursuit of a diagnosis is unnecessary. Effectively, the patient will have either idiopathic neck pain, or whiplash-associated neck pain if the pain is related to a traumatic incident.

Recommended terms to describe acute, non-specific neck pain are outlined below.

Specific and Serious Causes of Acute Neck Pain

- Torticollis will be evident by the characteristic posture of the neck.
- Neurological conditions will be identified by the presence of neurological signs and symptoms.
- Patients with rheumatic disorders will exhibit peripheral features of their disease. Of the rheumatic disorders only gout might, rarely, present with neck pain alone.
- Vascular disorders are an important consideration in any patient with a new onset of neck pain. However, it is the subsequent onset of cerebrovascular features that establishes the diagnosis. Vigilance for these features is what is required in the first instance. Only upon emergence of cerebrovascular features are investigations indicated.
- Neurological conditions that may present as neck pain alone without manifesting neurological signs are rare conditions (e.g. irritation of the dorsal root entry zone of the spinal accessory nerve and intracranial lesions). Of the rheumatic disorders, only gout might present solely with neck pain and not produce other features.
- Tumours and infections are rare cause of acute neck pain and should be suspected if the history reveals features or risk factors for these conditions. In the absence of alerting features, however, tumours and infections are extremely unlikely to be the cause of neck pain. Investigations are indicated only if the patient fails to recover or if they develop new signs of the disorder.
- Fractures are an uncommon cause of neck pain, even amongst patients with a history of trauma. They cannot be diagnosed clinically. They require radiography. However, guidelines apply for the investigation of patients with suspected fractures of the cervical spine (see Ancillary Investigations).

Other entities are not causes of neck pain, or are known not to be associated with neck pain in an epidemiological sense. These include diffuse idiopathic skeletal hyperostosis, ossification of the posterior longitudinal ligament, Paget's disease, and cervical spondylosis.

Spurious conditions are ones that lack defining diagnostic criteria. These include soft-tissue lesion, cervical strain, psychogenic pain, postural abnormalities, and myofascial pain.

Key Message

Except for serious conditions, precise identification of the cause of neck pain is unnecessary. (Consensus)

Terms to Describe Acute Neck Pain

For the nomenclature of neck pain whose cause cannot be established, the IASP recommends the term cervical spinal pain of unknown origin (Merskey and Bogduk 1994). Although this term serves adequately for the purposes of an honest and disciplined, formal taxonomy, it is nonetheless unwieldy for conventional or everyday practice. For those purposes the terms 'idiopathic neck pain' and 'whiplash-associated neck pain' may be less than optimal, but no other terms of better quality are available.

Key Message

Once serious causes have been recognised or excluded, terms to describe acute neck pain can be either 'acute idiopathic neck pain' or 'acute whiplash-associated neck pain'. (Consensus)

PROGNOSIS

Awareness of the prognosis of acute neck pain is seminal to its management. The fundamental determinants of prognosis are:

- the natural history of acute neck pain
- the presence of risk factors.

Natural History

Idiopathic Neck Pain

There are very few data on the natural history of acute idiopathic neck pain. Such data are limited to a review based on surrogate data and two retrospective studies.

A systematic review published in two sources (Ariens et al. 1999; Borghouts et al. 1998), attempted to describe the

natural history of acute neck pain using surrogate data, that is, the outcomes of control groups in randomised controlled trials. Those data proved less than satisfactory, for many studies enrolled people with both acute and chronic pain and most had periods of follow-up of less than three months. Those data indicated that the proportion of people who improve ranges from 10% to 100%, with an average of 30% to 50%, depending on the study.

A retrospective study of 250 people with neck pain seen ten years previously, found that 43% had no symptoms, 25% had mild symptoms, 25% had moderate levels of pain and 7% were severely affected (Gore et al. 1987). A smaller retrospective study (N = 51), found that after two to 19 years following the onset of pain, 44% had no symptoms, 29% had mild or intermittent symptoms and 28% had troublesome symptoms or moderate disability (Lees and Turner 1963).

Collectively, these data paint a mixed picture of the natural history or prognosis of neck pain. Approximately 40% of patients can expect to recover fully with the passage of time, 25–30% can expect persistence of mild or intermittent symptoms and 30% can expect moderate to severe symptoms.

Key Message

Approximately 40% of patients recover fully from acute idiopathic neck pain, approximately 30% continue to have mild symptoms and 30% continue to have moderate or severe symptoms. (*Level III)

Whiplash-Associated Neck Pain

Several studies have provided prospectively acquired data on the natural history of neck pain following whiplash. One series of studies of acute neck pain after whiplash provides some indication of the natural history of this condition, but suffers from an initially small sample and dwindling numbers at follow-up over two, 10, and 15 years (Gargan and Bannister 1990; Gargan and Bannister 1994; Squires et al. 1996; Norris and Watt 1983). The confidence intervals of the proportions of patients remaining in the study render the data difficult to interpret with any sense of certainty (Barnsley et al. 1998).

Data on the natural history of whiplash-associated neck pain suggest that 97% of patients recover fully within 12 months (Spitzer et al. 1995). However, as these data are based on closure of insurance claims and certified 'work-readiness', they do not necessarily reflect clinical status.

Three studies provide clinical data. A Swiss study followed 164 patients recruited from primary care practices within two weeks of onset of neck pain (Radanov et al. 1995). By three months, 56% of these patients were fully recovered; at six months, one year and two years, this proportion had risen to 70%, 76% and 82%, respectively. Over the same periods, the proportion of patients still having mild or moderate symptoms fell from 38%, to 26%, 20% and 14%. Around 5% of patients had severe pain at each point of follow-up.

A Danish study of 141 patients found that 7.8% had not returned to their usual level of activity or work (Kasch et al. 2001). A Canadian study found that the proportion of patients still experiencing symptoms at three months was 37%; this figure remained stable at 34–36% at six months through 24 months after injury (Brison et al. 2000). The study did not indicate the severity of persisting symptoms, but did comment that of the 8% of patients who sought compensation, 2% were successful.

These outcomes have been corroborated by data from a randomised controlled trial of treatment for acute whiplash-associated neck pain (Borchgrevink et al. 1998). The study involved an index intervention that required patients to act as usual without any other treatment. At six months, 48% (95%CI 38%, 58%) no longer had pain, 41% had mild to moderate pain and 11% (95%CI 5%, 17%) had severe symptoms.

These various figures indicate that the prognosis of whiplash-associated neck pain is somewhat better than that of idiopathic neck pain. Many people recover fully after whiplash, with one in seven (14%) to two in five (40%) having mild to moderate persisting symptoms and one in 20 (5%) having severe symptoms.

Key Message

Approximately 56% of patients fully recover within three months from onset of acute whiplash-associated neck pain, 80% recover fully within one or two years, 15–40% continue to have symptoms and 5% are severely affected. (*Level III, IV)

Prognostic Risk Factors

Idiopathic Neck Pain

The literature is devoid of any data on prognostic risk factors for idiopathic neck pain. A systematic review on this matter (Ariens et al. 1999; Borghouts et al. 1998) found only six studies that addressed prognostic factors, but none provided a statistical analysis that yielded either the relative risk or odds ratios for any association.

Whiplash-Associated Neck Pain

Data based on insurance claims reveal certain demographic and clinical determinants of chronicity of whiplash-associated neck pain (Table 6.6). This source (Suissa et al. 2001; Harder et al. 1998), however, did not include psychosocial variables.

Prospective studies have shown that personality and psychosocial stress were not determinants of chronicity of neck pain after whiplash (Radanov et al. 1991; Borchgrevink et al. 1997). The cardinal determinants are listed in Table 6.7.

Table 6.6

Factors Associated with Chronic Neck Pain After Whiplash: Insurance Data

Demographic	Older Age
	Female gender
	Having dependents
	Not employed full-time
Clinical	Neck pain on palpation
	Muscle pain
	Headache
	Pain or numbness radiating to the upper limb

Note: Based on insurance claims data from Suissa et al. 2001 and Harder et al. 1998.

Table 6.7

Demographic and Clinical Factors Associated with Chronic Neck Pain After Whiplash

Demographic	Age
Past History	Of headache
	Of head injury
Clinical	Impaired neck movements
	Initial pain intensity
	Initial headache intensity
Psychometric	Nervousness score
	Neuroticism score
	Test score on focused attention

Note: Based on data from Radanov et al. 1991.

A small study (Karlsborg et al. 1997) has warned that distress over concurrent life events unrelated to the accident may also hinder recovery.

Key Message

Psychosocial factors are not determinants of chronicity in whiplash-associated neck pain. (*Level III)

Collectively, the insurance data and the clinical data agree that older age and the severity of initial symptoms are the leading determinants of chronicity of neck pain after whiplash.

Although chronicity is often ascribed to litigation neurosis, reviews have found no evidence to support this notion (Shapiro and Roth 1993; Teasell and Shapiro 1998; Mendelson 1982, 1984; Norris and Watt 1983). Competent follow-up studies have shown chronicity to be independent of litigation (Norris and Watt 1983; Maimaris et al. 1988; Pennie and Agambar 1991; Parmar and Raymakers 1993; Swartzman et al. 1996).

Key Message

Risk factors for chronicity following whiplash-associated neck pain are older age at time of injury, severity of initial symptoms, past history of headache or head injury. (*Level III)

INTERVENTIONS

Acute idiopathic neck pain and acute whiplash-associated neck pain differ only with respect to aetiology. Therefore, in practice the same interventions may be of use in treating both entities. There is no evidence to demonstrate that a particular intervention offers greater benefit for either idiopathic or whiplash-associated neck pain. Accordingly, no distinction is made in the following evidence-based information on interventions for acute neck pain.

It is important to note that a lack of evidence (i.e. insufficient evidence) does not mean that a particular intervention has no place in the management of acute neck pain, however, it is preferable to employ interventions for which there is evidence of benefit, where appropriate. Management decisions should be based upon knowledge of the existing evidence, consideration of individual patient needs and clinical judgment.

The criteria formulated to categorise the following interventions and definitions of the levels of evidence are described in Chapter 9: Process Report.

Adverse effects have not specifically been investigated during this review, however information has been included in the text where adverse effects have been described in the cited material.

Evidence of Benefit

Advice to Stay Active (Activation)

Activation is an intervention in which the practitioner deliberately and conscientiously encourages the patient to resume normal activities of daily living. The intervention is implemented in the context of having assessed the patient and found no evidence of a serious cause of pain, having explained this to the patient and having explained the natural history and prognosis of acute neck pain (see Chapter 2: Acute Pain Management and Chapter 3: Effective Communication).

Activation is not simply the difference between prescribing active treatments, to which the patient contributes some sort of therapeutic activity, and prescribing passive treatments that the patient simply receives. Activation does not entail any specific artificial activity that the practitioner imposes on the patient. It requires only the resumption of activities that the patient would normally perform. Nor should activation be misrepresented as a dismissive announcement that nothing is wrong with the patient and that, therefore, they should get back to work or summarily resume a normal life (as if nothing had happened). Conscientious encouragement involves recognition that the patient has suffered an episode of pain, considered application of the epidemiological evidence on natural history and recovery and securing the patient's understanding and confidence that it is not only safe but appropriate to resume activities. Securing the patient's confidence to do this implies that their fears about their condition have been allayed. In this way, activation differs from concluding that the patient's complaints and disability are trivial.

Systematic reviews (Spitzer et al. 1995; Peeters et al. 2001; Verhagen et al. 2002) have emphasised a preference for people with neck pain to become and remain active rather than undertake passive treatments. This emphasis, however, is largely based on the success of activation in the treatment of acute low back pain.

In the context of neck pain, some systematic reviews (Sptizer et al. 1995) showed that active treatments were more effective than passive treatments. Other reviews (Peeters et al. 2001; Verhagen et al. 2002) showed that active treatments were superior to rest. None of these reviews, however, explicitly address activation as a sole intervention. Rather, they extolled the virtues of interventions involving exercises (see Exercises,

below). Nevertheless, they established the importance of having the patient keep the neck active.

One study (Borchgrevink et al. 1998) described in one systematic review (Peeters et al. 2001; Verhagen et al. 2002) has explicitly assessed the efficacy of advice to stay active as a sole intervention. In that study, 201 patients with acute neck pain received instructions for self-training on the first day of treatment and a five-day prescription for non-steroidal anti-inflammatory drugs (NSAIDs). The patients were subsequently randomised either to receive 14 days of sick leave and a soft collar, or to act as usual, with no collar and no sick leave. There was a reduction in symptoms in both groups at six weeks and six months after treatment. Pain outcomes were significantly better in the group resuming usual activities, with 48% (95%CI 37%, 59%) no longer bothered by their pain and 11% still suffering with severe symptoms. The corresponding figures for the comparison group were 34% and 15%, respectively.

The cardinal role of activation could best be described as providing a foundation upon which other effective interventions might be added in order to optimise the rate of recovery.

Key Message

Encouraging resumption of normal activities and movement of the neck is more effective compared to a collar and rest for acute neck pain. (Level I, II)

Exercises

When assessing the efficacy of exercises for acute neck pain, systematic reviews have differed in the literature that they have identified and accepted. Reviews by Harms-Ringdahl and Nachemson (2002), Gross et al. (2002b; last updated 1998), Kjellman et al. (1999) and Verhagen et al. (2002) identified seven studies (Goldie and Landquist 1970, Levoska and Keinanen-Kiukaanniemie 1993; Takala et al. 1994; Mealy et al. 1986; McKinney et al. 1989, Provinciali et al. 1996; Karlberg et al. 1996). Four of these studies (Goldie and Landquist 1970; Levoska and Keinanen-Kiukaanniemie 1993; Karlberg et al. 1996; Takala et al. (1994) involved mixed populations. Two studies meeting the criteria for this review (Provinciali et al. 1996; Mealy et al. 1986) involved multi-modal interventions and the efficacy of exercises alone could not be specifically distinguished. These studies are considered under the heading, 'Multi-modal Therapy', below. Of the studies identified in the systematic reviews, only that of McKinney et al. (1989) provides evidence on the efficacy of exercises for acute neck pain. It is complemented by a recent study (Rosenfeld et al. 2000) not yet included in a systematic review.

McKinney et al. (1989) showed that mobilising exercises performed at home, plus postural advice, were significantly more effective at two months than rest and analgesia and no less effective than outpatient treatments tailored to individual patient needs (comprising thermal modalities, short wave diathermy, hydrotherapy, active and passive movements, traction, advice on posture and home exercises). Moreover, at two years, a significantly greater proportion (72%) of those treated with home exercises were pain-free compared with those treated either by rest (54%) or tailored outpatient treatments (56%) (McKinney 1989). The drop-out rate in this latter study (27%) compromises the validity of the conclusions, but both best-case and worst-case analysis of the missing data still favour home exercises.

Rosenfeld et al. (2000) treated one group of patients with an active program of gentle, active, small-range and small amplitude rotation movements (consistent with McKenzie principles) performed at home 10 times every waking hour. If pain persisted more than 20 days, individualised exercises were added. A comparison group was provided with a leaflet providing information about injury mechanisms, advice on suitable activities, instructions on postural correction and a collar. The study also tested the effects of early (within 96 hours) versus delayed (after two weeks) treatment. At six months, those patients seen within 96 hours and treated with exercises showed an 80% reduction in pain, while the comparison group showed no reduction ($p < 0.001$). Approximately 38% of the neck exercise group were pain-free and a further 52% had low levels of pain. The corresponding figures in the comparison group were 17% and 30%. The odds ratio for achieving complete relief of pain was 2.9, with 95% confidence intervals of 1.1 to 7.8, but for achieving low or no pain, the odds ratio was 10.4, with confidence intervals of 2.4 to 41.3. Such differences were not evident if exercises were commenced late (two weeks) after onset of pain.

Key Messages

> Gentle neck exercises commenced early post-injury are more effective compared to rest and analgesia or information and a collar in acute neck pain. (Level II)

> Exercises performed at home are as effective for neck pain as tailored outpatient treatments at two months and appear to be more effective at two years after treatment. (Level II)

Multi-Modal Therapy

Multi-modal therapy is a program of treatment in which two or more interventions are provided in combination. The combination may be designed to obtain an additive therapeutic effect for the same symptom or symptoms or each intervention may target a different aspect of symptoms, impairments or disabilities.

A recent systematic review of the efficacy of multi-modal manual therapy by Gross et al. (2002c) located 20 studies on manual therapy for mechanical neck disorders. Of these, Provinciali et al. (1996), Giebel et al. (1997), Mealy et al. (1986) and McKinney et al. (1989) met the criteria for this update, although Giebel et al. (1997) was not published in English. Gross et al. (2002c) concluded that while there are themes emerging in the area of multi-modal therapy, results remain inconclusive because of the small sizes and methodological limitations of the studies.

Provinciali et al. (1996) rated as 'good' quality in a systematic review by Verhagen et al. (2002) but rated low (2/5 on the Jadad scale) in the Gross et al. (2002c) review, assessed a combination of treatments in patients with both cervical and encephalic symptoms. The multi-modal package consisted of relaxation training based on diaphragmatic breathing, postural re-education, psychological support, proprioceptive exercise and cervical passive mobilisation. The comparison treatment was application of transcutaneous electrical nerve stimulation (TENS), pulsed electromagnetic therapy (PEMT), ultrasound and calcic iontophoresis. At one and six months after treatment, pain scores were significantly less in the multi-modal group. Although the authors did not provide any data on the variance of their outcomes, a systematic review derived an effect size of –0.79 (95%CI –1.32, –0.26) (Gross et al. 2002c). At six months, 12 out of 30 people in the multi-modal group reported marked improvement but only seven were totally improved. This proportion was significantly greater than the control group, where the corresponding figures were two and one, respectively, in a group of 30 people.

The poor outcome in the control group is conspicuous in this study, amplifying the attributable effect. Nevertheless, multi-modal therapy of the nature provided in this study appears to be effective for achieving subjective improvement at six months. What is not evident from the study is whether the attributable effect depends on providing all of the components of this combination of therapy. A further limitation is that the results cannot be extended to all patients with acute neck pain. The study explicitly excluded patients with 'symptom exaggeration with the intention of enhancing financial rewards'.

Giebel et al. (1997) evaluated the efficacy of a combined package of cervical passive mobilisation, traction and strengthening and proprioceptive exercises versus treatment using a collar. The study found that a greater proportion of those who received multi-modal therapy had recovered at two weeks; however by 12 weeks there were no significant differences between the two treatment groups. A statistical analysis by Gross et al. (2002c) calculated a treatment advantage of 5.5% in favour of multi-modal therapy. The methodological limitations of the study were noted in another systematic review (Verhagen et al. 2002).

Mealy et al. (1986) compared cervical passive mobilisation therapy coupled with a home exercise program versus wearing a collar and resting for two weeks. At four weeks and eight weeks after treatment, the mobilisation and exercise group exhibited a significantly greater reduction of pain, from a mean score at baseline of 5.7 to one of 1.7 at eight weeks. For the control group the corresponding figures were 6.4 and 3.9, amounting to an effect size of 0.7. Even so, there was considerable variance in the outcomes of the index treatment group. The standard deviation of the 1.7 score was 2.3, indicating that many people still had high pain scores.

McKinney et al. (1989) compared the effects of a combination of outpatient treatments (thermal modalities, short wave diathermy, hydrotherapy, active and passive movements, traction, advice on posture and home exercises) tailored to individual patient needs with both the effects of rest and analgesia and the effects of instruction to perform mobilisation exercises at home and postural education. Tailored multi-modal therapy was not more effective than home exercises, but both interventions were significantly more effective in reducing neck pain than rest and analgesia.

This study is the only one that has provided long-term follow-up (McKinney 1989). At two years, 77% of the home exercise group were pain-free compared with 56% in the outpatient group and 54% in the rest and analgesia group. Thus, with respect to expecting eventual complete relief of pain, the odds ratio for home exercise (2.9; 95%CI 1.7, 4.9) was substantially greater than that of the tailored package of outpatient treatments (1.3; 95%CI 0.79, 2.0) when compared to rest and analgesia. There was a loss to follow-up of 21–27% in the various groups that compromised the results of the study. Nevertheless, both worst-case and best-case analysis of the missing data favours home exercises.

Two more recent studies (Bonk et al. 2000; Hoving et al. 2002) have been published subsequent to the systematic review of Gross et al. (2002c). Both provide additional supporting data on the efficacy of multi-modal therapy.

Bonk et al. (2000) compared the effectiveness of active therapy (three weeks of active and passive cervical mobilisation, postural exercises and advice) with that of rest in a collar for three weeks. At three weeks, those patients treated with active therapy had significantly less pain than those treated with a collar.

The second additional study was published in an article reporting results at seven weeks (Hoving et al. 2002) and a thesis that reported longer-term outcomes (Hoving 2001). Hoving et al. (2002) compared 60 people treated with manual therapy (mobilisation and stabilisation techniques), 59 with physical therapy (exercise therapies, manual traction, massage and heat) and 64 who received usual care consisting of advice on prognosis and home exercises, encouragement to await spontaneous recovery and prescription of analgesics. Approximately 30% of those treated by manual therapy or physical therapy had chronic neck pain, as did 20% of those treated by their general practitioner. The remainder had acute neck pain.

Both publications (Hoving et al. 2002; Hoving 2001) reported a range of standard outcome measures, such as visual analogue scores for pain, disability scores and quality of life measures. With respect to those measures, the study found that those treated with manual therapy exhibited a 56% reduction in pain at seven weeks compared with 39% for those treated with physical therapy and 30% for those under usual care. The reduction in pain in the manual therapy group was significantly greater than that in the usual care group but was not significantly greater than that of the physical therapy group. Reductions in disability amounted to 30% and were not significantly different between groups. Improvements in quality of life measures were significantly better for the manual therapy group and amounted to 22% for manual therapy, 12% for physical therapy and 10% for usual care.

For relief of pain, the effect size for manual therapy was small (0.3) when compared with physical therapy and medium (0.7) when compared with usual care. Similarly, for reduction of disability the effect size for manual therapy was small (0.3) compared with physical therapy and medium (0.6), when compared with usual care. For improvement in quality of life, the effect size for manual therapy was not much higher than that of physical therapy (0.01) and was medium (0.5) when compared with usual care. Overall, these results indicated that manual therapy was moderately more effective than usual care and marginally more effective than physical therapy (Hoving et al. 2002; Hoving 2001).

The investigators used another measure that provided greater differences in favour of manual therapy. They reported that 68% of their patients treated with manual therapy had recovered at seven weeks compared with 51% of patients treated by physical therapy and 36% of patients under usual care. The odds ratio for recovery under manual care is 3.8 (95%CI 2.8, 5.0) compared with usual care and 1.33 (95%CI 1.1, 1.6) compared to physical therapy. In these terms, therefore, manual therapy is substantially more favourable than usual care but not more than physical therapy (Hoving et al. 2002; Hoving 2001).

The investigators defined success as the proportion of people who felt that they had either 'completely recovered' or were 'much improved'. However, in reporting their results, the investigators did not stratify the outcomes according to these two categories. Instead, the success rate reported was the combined total of both categories. Thus it is not evident from either publication (Hoving et al. 2002; Hoving 2001) the extent to which 'recovered' means 'completely recovered' or 'much improved'.

An editorial that accompanied the paper raised concerns about the subjective nature of 'perceived recovery' as an outcome measure and questioned if manual therapy appeared more successful because of the intensity of the patient-therapist

interactions associated with manual therapy (Posner and Glew 2002). This could be an important factor in light of the fact that those treated with manual therapy averaged six visits, whereas those under usual care averaged only two visits.

Nevertheless, the results of this study at seven weeks indicate that the outcomes of manual therapy are substantially better than those of usual care and only marginally better than those of physical therapy. The thesis (Hoving 2001), however, reveals that any difference in outcome diminishes with time. At 13 weeks, a significantly higher proportion (72%) of people who had manual therapy felt they had recovered compared with 42% in the usual group. Neither of these proportions was different from that of the physical therapy group (59%). However, pain scores were not significantly different between any of the groups. By 52 weeks, no statistically significant differences in any of the outcome measures persisted between the groups.

Key Message

Multi-modal (cominbed) treatments inclusive of cervical passive mobilisation in combination with specific exercise alone or specific exercise with other modalities are more effective for acute neck pain in the short term compared to rest, collar use and single modality approaches. (Level I, II)

Pulsed Electromagnetic Therapy (PEMT)

Pulsed electromagnetic therapy (PEMT) involves wearing a collar embedded with a device that delivers a pulsed electromagnetic stimulus for eight hours a day. Two studies from the same group have advocated PEMT for the treatment of acute neck pain. Each compared active therapy with wearing a collar embedded with a placebo device.

Gross et al. (2002b) located two randomised controlled trials (RCTs) on PEMT (Foley-Nolan et al. 1990; Foley-Nolan et al. 1992). The first study (Foley-Nolan et al. 1990) reported that PEMT was superior to control treatment in that it achieved a statistically significant, greater reduction of pain. However, as everyone subsequently undertook active treatment, any lasting differences in the effect were obscured. The second study (Foley-Nolan et al. 1992) involved wearing either the active collar or the placebo collar for 12 weeks. Those treated with the active device exhibited significantly greater reduction in pain scores at two and four weeks during treatment, but not at 12 weeks. At four weeks, a significantly greater proportion ($p < 0.05$) of patients treated with the active device reported feeling moderately better and fewer were worse; but at 12 weeks there were no differences in these proportions. The second study (1992) involved patients with acute whiplash-associated neck pain whereas the first study (1990) involved people with mixed durations of neck pain. Gross et al. (2002b) noted that neither study provided sufficient data to calculate an effect size.

Another systematic review recognised that PEMT provided some reduction of pain during treatment, but concluded that 'there is limited evidence that this treatment does not influence perceived pain intensity' (Harms-Ringdahl and Nachemson 2000). The review also questioned the propriety of requiring people to wear a collar for 12 weeks, when other measures, including activation, might be at least as effective (Harms-Ringdahl and Nachemson 2000).

Key Message

Pulsed electromagnetic therapy reduces pain intensity compared to placebo in the short term but is no different to placebo at 12 weeks for acute neck pain. (Level I)

Insufficient Evidence of Benefit

Acupuncture

The literature on acupuncture for neck pain is limited to studies involving chronic pain, mixed acute and chronic pain or specific conditions causing pain. It provides insufficient evidence concerning the management of acute neck pain.

Exploring the literature on mixed populations does not provide any evidence that might be extrapolated to acute neck pain. Clinical Evidence (2002) cited two systematic reviews (White and Ernst 1999; Smith et al. 2000). White and Ernst (1999) identified 14 RCTs and Smith et al. (2000) included three. Both identified the study of Coan et al. (1981) that showed that acupuncture was significantly better for pain-relief than being on a waiting list. The other studies did not yield statistically significant results. Both reviews concluded that there is insufficient evidence that acupuncture is effective compared with placebo or other interventions in the treatment of neck pain.

A Cochrane Review by Gross et al. (2002b, last updated 1998) identified two studies on acupuncture. That of Petrie and Langley (1983) reported that acupuncture was significantly superior to sham transcutaneous electrical nerve stimulation (TENS) ($p < 0.01$). Loy (1983) reported that acupuncture was more effective than shortwave diathermy and traction for treating neck pain, although no details of the analysis were provided.

A review by Harms-Ringdahl and Nachemson (2000) identified one additional RCT comparing acupuncture versus sham TENS. The difference in pain outcomes was not statistically significant between groups (Petrie and Hazleman 1986).

An alternative to needle acupuncture is the application of infrared heat to acupuncture points. Gross et al. (2002b) described a study (Lewith and Machin 1981) that compared this form of therapy to sham TENS and reported no significant difference between the therapies.

Key Messages

- There are no randomised controlled studies on the effect of acupuncture or infrared acupuncture in the treatment of acute neck pain. (No Level I or II studies)
- There is conflicting evidence that acupuncture is more effective compared to placebo and other treatments for neck pain in mixed populations. (Level I)

Analgesics (Opioid)

No studies have described or investigated the efficacy of opioids for treatment of acute neck pain. For the treatment of acute spinal pain, the guidelines on acute musculoskeletal pain management published by the National Health and Medical Research Council of Australia (1999b) state that 'Opioids (oral) may be required in the acute stage, with regular rather than pain-contingent dosing with a short-acting agent such as oxycodone or codeine'. Deyo (1996) draws a similar conclusion in a review of drug therapy for back pain. This appears to be a consensus view taking into account the possible need for stronger analgesia than that afforded by paracetamol or non-steroidal anti-inflammatory drugs (NSAIDs) in patients with acute spinal pain. Whereas opioids may be considered a

humane, temporising measure, people with neck pain severe enough to warrant use of opioid medication should be carefully assessed and re-assessed lest they have an unrecognised serious cause for their pain.

Harms have been associated with the use of opioids. A systematic review (not specific to acute neck pain) of 29 RCTs (de Craen et al. 1996) reported a pooled 5% reduction in pain with compound analgesia (opioid plus paracetamol) compared with paracetamol alone. However there was a substantial increase in side effects with multiple doses of compound analgesics compared with multi-dose paracetamol alone (OR = 2.5, 95%CI 1.5, 4.2). The most commonly reported adverse effects were nausea, dizziness, vomiting, constipation and drowsiness.

Key Messages

> Opioids may be used, however there are no randomised controlled studies of its effectiveness for acute neck pain. (No Level I or II studies)

> In general, opioid and compound analgesics have a substantially increased risk of side effects compared with paracetamol alone. (Level I)

Analgesics (Simple)

Clinical Evidence (2002) reports that although it is widely used as first line therapy, there is insufficient evidence on the efficacy of paracetamol in the treatment of acute neck pain. Reviews by Spitzer et al. (1995), Aker et al. (1996) and Bogduk (2000) were cited, however no RCTs were located.

A review by Kjellman et al. (1999) located one study on the efficacy of analgesics in the treatment of acute whiplash-associated neck pain. McKinney et al. (1989) compared the use of a compound analgesic (paracetamol plus codeine) plus rest versus a regime of active treatment versus mobilisation advice. Both the active treatment and the advice groups fared better than the rest and analgesia group at one and two months ($p = 0.01$).

Key Message

Simple analgesics may be used to treat mild to moderate pain however there is insufficient evidence that paracetamol is more effective than placebo, natural history or other measures for relieving acute neck pain. (No Level I or II studies)

Cervical Manipulation

Cervical manipulation is movement performed to move a joint beyond its immediately available range of movement.

Clinical Evidence (2002) located three systematic reviews (Aker et al. 1996; Hurwitz et al. 1996; Kjellman et al. 1999) describing studies on cervical manipulation. No studies on the efficacy of cervical manipulation in acute neck pain were located. Five RCTs involving mixed populations (Cassidy et al. 1992; Vernon et al. 1990; Howe et al. 1983; Sloop et al. 1982; Koes et al. 1992a,b) were identified, however they provided no conclusive evidence on the effectiveness of cervical manipulation.

The study of Cassidy et al. (1992), rated as strong in methodological quality by Kjellman et al. (1999), assessed the immediate effects of cervical manipulation versus muscle-energy techniques. Kjellman et al. (1999) provided data on the results of the study, which showed no significant difference between the groups ($p = 0.16$; effect size –0.01; 95% CI –0.4, 0.4). However, the effect disappeared when the data were adjusted for pre-treatment differences. Vernon et al. (1990) reported a significant difference in pain in the spinal manipulation group ($n = 5$) ($p < 0.0001$) compared to passive mobilisation, however this effect was measured after only five minutes. Howe et al. (1983) compared cervical manipulation with a non-steroidal anti-inflammatory drug (NSAID), azapropazone. Although differences in favour of cervical manipulation were apparent immediately after treatment, there were no differences at one week and three weeks after treatment. Sloop et al. (1982) compared the effect of manipulation plus an amnesic dose of intravenous diazepam versus diazepam alone. They reported no significant difference ($p = 0.20$) between the groups ($N = 39$). Koes et al. (1992a,b) compared manual therapy (manipulation and mobilisation), usual care (analgesic, postural advice, home exercises and other treatments), physical therapy (exercises, massage, physical therapy modalities) and placebo treatment (detuned short wave diathermy and ultrasound) in a study of patients back and neck pain and found no significant difference in pain outcomes between groups at three, six and 12 weeks.

No further studies were identified in additional systematic reviews by Gross et al. (2002c) and Harms-Ringdahl and Nachemson (2000).

Hurwitz et al. (1996) and Gross et al. (2002c) report rare but serious adverse events associated with cervical manipulation. They estimated the risk of all serious effects is 5–10 per 10,000,000 manipulations (Hurwitz et al. 1996).

Key Messages

> There are no randomised controlled trials investigating the effect of cervical manipulation in the treatment of acute neck pain. (No Level I or II studies)

> Adverse effects of cervical manipulation are rare but potentially serious. (Level I)

Cervical Passive Mobilisation

Cervical passive mobilisation is the application of forces to the neck in a slow, rhythmic fashion in order to increase the available range of motion in a joint. Systematic reviews have differed in their interpretations and treatment of the studies available on mobilisation therapy.

Clinical Evidence (2002) located four systematic reviews (Koes et al. 1991; Aker et al. 1996; Hurwitz et al. 1996; Kjellman et al. 1999) describing studies on cervical mobilisation. These reviews identified three studies involving patients with acute neck pain (Nordemar and Thorner 1981; Mealy et al. 1986; McKinney et al. 1989). Only the study by Nordemar and Thorner (1981) involved mobilisation as a sole intervention. Those of Mealy et al. (1986) and McKinney et al. (1989) compared the effects of combinations of treatments involving mobilisation as only one component. These studies are discussed in the section on 'Multi-modal Therapy'.

Nordemar and Thorner (1981) compared use of a collar versus collar plus TENS versus collar plus traction and mobilisation (all patients took analgesics). After one week, the group receiving traction and mobilisation had a greater but not statistically significant reduction in pain compared to the other two groups. At six weeks and three months, there were no differences between the groups.

An additional review by Harms-Ringdahl and Nachemson (2000) concluded that there was insufficient evidence about the effect of manual therapy when used alone and compared with other treatments in mixed populations. However, when manual therapy was used in combination with active treatments, the review found that 'there is moderate evidence that some patient groups may benefit from mobilising techniques

as part of an activating program'. A more recent review (Gross et al. 2002c) offered similar conclusions.

Four studies identified in the reviews involved patients with a mixture of acute and chronic pain (Cassidy et al. 1992; Vernon et al. 1990; Brodin 1985; Koes 1992). The results were conflicting and none of the studies compared cervical passive mobilisation to natural history or placebo.

Any benefit of cervical passive mobilisation appears restricted to its use in combination with other interventions. This is discussed in the section on 'Multi-modal Therapy'.

Clinical Evidence (2002) identified no reports of serious adverse effects from cervical mobilisation.

Key Message

There are no randomized controlled studies on the effect of cervical passive mobilisation compared to natural history or placebo in the treatment of acute neck pain. (No Level I or Level II studies)

Electrotherapy

Certain forms of electrotherapy for acute neck pain have been tested. Systematic Reviews (Peeters et al. 2001; Verhagen et al. 2002) identified two studies (Fialka et al. 1989; Hendricks and Horgan 1996) of patients with acute, idiopathic neck pain. Fialka et al. (1989) compared the efficacy of middle frequency electrotherapy with no treatment, treatment with iontophoresis gel, and a combined treatment involving traction, exercises and massage. Although the authors did not formally compare differences between groups, their data show no significant differences in outcome. Hendriks and Horgan (1996) compared ultra-reiz current with no treatment and found that electrotherapy was more effective at 15 minutes after treatment, but not at six weeks.

Key Message

There is insufficient evidence that electrotherapy is effective compared to no treatment in acute neck pain. (Level I)

Gymnastics

One study (Takala et al. 1994) on the effect of group gymnastics versus control in a population with a mix of acute and chronic neck pain was located. Gymnastics reduced neck pain no more than natural history and seasonal variations (Takala et al. 1994).

Key Messages

- There are no randomised controlled trials on the effect of gymnastics for acute neck pain. (No Level I or II studies)
- Gymnastics may be no more effective than natural history in mixed populations. (Level I)

Microbreaks

McLean et al. (2001) investigated the effects of taking short work breaks (microbreaks) on the development of acute neck pain in subjects working at computer terminals. The subjects were pain-free at inception and undertook a three-hour task, during which they took breaks at their own discretion or at scheduled 20-minute or 40-minute intervals. The development of neck discomfort was compared with that incurred when the same task was performed without breaks. Taking breaks, of any sort, was found to reduce neck discomfort by about 30% at the end of the three-hour task. Microbreaks at 20-minute intervals were found to reduce subjective discomfort in the neck ($p < 0.05$).

Key Message

There is insufficient evidence that taking regular breaks from computer work is more effective compared to irregular breaks for preventing acute neck pain. (Level II)

Multi-Disciplinary Treatment

Multi-disciplinary treatment comprises a combination of treatment modalities, including physical treatments for musculoskeletal pain and psychological, behavioural and educational interventions.

Clinical Evidence (2002) cited one Cochrane Review (Karjalainen et al. 2002, last updated 1999) on multi-disciplinary biopsychosocial rehabilitation of neck and shoulder pain with two RCTs (Ekberg et al. 1994; Jensen et al. 1995) as well as two subsequent RCTs (Taimela et al. 2000; Linton and Andersson 2000) on multi-disciplinary treatment. All of the studies involved mixed acute and chronic populations.

Ekberg et al. (1994) found no difference in the effects of active, multi-disciplinary rehabilitation versus usual care at 12 and 24 months follow up. Jensen et al. (1995) reported no statistically significant differences in pain outcomes between groups receiving cognitive behavioural therapy (CBT) provided by a clinical psychologist versus a program delivered by other members of the rehabilitation team. Taimela et al. (2000) compared home exercises and education versus proprioceptive exercises versus a lecture recommending exercise. At three months, there was significantly less pain ($p = 0.018$) in the home exercise and proprioceptive exercise groups compared to the advice only group, but no difference after 12 months. Linton and Andersson (2000) included subjects with acute and subacute spinal pain, including neck pain, in their study on prevention of chronic spinal pain. After one year, there were no statistically significant differences in pain between the CBT group and two groups receiving information.

Key Messages

- There are no randomised controlled studies investigating the effect of multi-disciplinary treatment in acute neck pain. (No Level I or II studies)
- There is insufficient evidence that multi-disciplinary treatment is effective compared to other interventions for reducing neck pain in mixed populations. (Level I, II)

Muscle Relaxants

Studies investigating the effect of muscle relaxants on neck pain are limited to those involving populations with specific conditions or mixed acute and chronic pain. Clinical Evidence (2002) located one systematic review (Aker et al. 1996) that included two RCTs on the use of muscle relaxants for neck pain (Basmajian 1978; Bercel 1977). Both studies reported significant improvement in pain from use of oral cyclobenzaprine compared to diazepam and placebo but neither provided follow up data. An additional study (Basmajian 1983) compared the effect of diazepam, phenobarbital and placebo for the treatment of neck pain related to trauma, arthritis or congenital defects. They reported there was no evidence that diazepam had improved neck pain on palpation or on movement.

Adverse effects of muscle relaxants are common, including drowsiness, dizziness and dyspepsia. Dependency has been reported after one week of use (Bigos et al. 1994; van Tulder et al. 1997).

Key Messages

> There are no randomised controlled trials investigating the efficacy of muscle relaxants for the treatment of acute neck pain. (No Level I or II studies)

> Muscle relaxants are no more effective than placebo for neck pain in mixed populations. (Level I, II)

> Drowsiness, dizziness and dependency are common adverse effects of muscle relaxants. (Level I)

Neck School

The only study of neck school for neck pain involves patients with a mix of acute and chronic pain (Kamwendo and Linton 1991). The study compared neck school (exercise, self-care and relaxation) to no treatment, with and without individual advice, and found no significant reduction in pain in the intervention groups compared to no treatment.

Key Messages

> There are no randomised controlled trials on the effect of neck school for acute neck pain. (No Level I or II studies)

> Neck school appears no more effective than no treatment for neck pain in mixed populations. (Level II)

Non-Steroidal Anti-inflammatory Drugs (NSAIDs)

Studies investigating the effect of non-steroidal anti-inflammatory drugs (NSAIDs) are limited to those involving populations with specific conditions or mixed populations. A systematic review by Aker et al. (1996) located two studies (Coletta et al. 1988; Koes et al. 1992a,b) investigating the efficacy of NSAIDs for neck pain. Coletta et al. (1988) compared a topical anti-inflammatory drug (etofenamate) plus transcutaneous electrical nerve stimulation (TENS) versus TENS alone and reported significantly better pain relief ($p < 0.02$) in the group receiving combination therapy. However, the study had methodological limitations and Aker et al. (1996) reported they were unable to calculate an effect size from the data. Koes et al. (1992a,b) found no difference in outcome between those treated with analgesics, NSAIDs and education and those treated with placebo ultrasound in a study involving patients with neck and back pain.

Adverse effects can occur to varying degrees with the use of NSAIDs and appear to be dose-related. They include gastrointestinal bleeding, tiredeness and dizziness (Bigos et al. 1994; Henry et al. 1996).

Key Messages

> There are no randomised controlled trials on the effect of NSAIDs for acute neck pain. (No Level I or II studies)

> There is evidence that NSAIDs are no more effective than placebo ultrasound for neck pain in mixed populations. (Level I)

> Serious adverse effects of NSAIDs include gastrointestinal complications, (e.g. bleeding, perforation). (Level I)

Patient Education

There is no evidence on the efficacy of patient education for the treatment of acute neck pain. The only study identified by systematic reviews (Gross et al. 2002a; Linton and van Tulder 2001) that involved patients with acute neck pain was that of McKinney et al. (1989), but in this study patient education was only one of several components of a multi-modal intervention. Consequently, it is not possible to determine the effect of education from this study.

Key Message

There are no randomised controlled trials investigating the effect of patient education as a single strategy in the treatment of acute neck pain. (No Level I or II studies)

Spray and Stretch

Spray and stretch therapy involves the application of vapocoolant spray followed by passive stretching (Gross et al. 2002b).

A Cochrane Review by Gross et al. (2002b, last updated 1998) identified one study published in abstract form by Snow et al. (1992), assessing the efficacy of spray and stretch therapy for chronic myofascial neck and back pain. The study compared spray and stretch therapy versus placebo versus control (heat, exercise and education). The authors concluded that vapocoolant spray was no more effective than placebo and when combined with other interventions did not improve treatment efficacy.

A systematic review (Aker et al. 1996) accepted the Snow et al. (1992) study as providing evidence that spray and stretch was not effective for neck pain. Another systematic review (Harms-Ringdahl and Nachemson 2000) noted the negative result, but commented that the result should be interpreted with caution because of the small size of the study.

Key Messages

> There are no randomised controlled trials investigating the effect of spray and stretch therapy in acute neck pain. (No Level I or II studies)

> Spray and stretch therapy appears no more effective than placebo for neck pain in mixed populations. (Level I)

Traction

Clinical Evidence (2002) cites a systematic review on traction for back and neck pain by van der Heijden et al. (1995) and a Cochrane Review by Gross et al. (2002b, last updated 1998). Five studies (Goldie and Landquist 1970; Zylbergold and Piper 1985; British Association of Physical Medicine 1966; Loy 1983; Pennie and Agambar 1990) were located in the reviews; none of these studies involved patients with acute neck pain.

Goldie and Landquist (1970) compared traction versus exercise versus control (analgesic and muscle relaxant plus posture advice) and reported no significant difference between groups. Aker et al. (1996) noted that it was not possible to calculate an effect size from the results. Zylbergold and Piper (1985) found no differences in outcome when static traction, intermittent traction, manual traction and no traction were added to a regimen of instruction, moist heat and a program of exercises. The study by the British Association of Physical Medicine (1966) involved patients with pain in the arm. Loy (1983) reported that traction was less effective compared to a combination of electroacupuncture and shortwave diathermy for treating neck pain. No details of the analysis were provided. In a non-randomised study, Pennie and Agambar (1990) found no added benefit from intermittent traction and exercise instruction compared to two weeks of rest in a standard or moulded collar.

Other systematic reviews (Aker et al. 1996; Harms-Ringdahl and Nachemson 2000; Verhagen et al. 2002) did not locate additional studies.

Key Messages

> There are no randomised controlled trials investigating the effectiveness of traction for acute neck pain. (No Level I or II studies)

> In mixed populations, there is evidence that traction is of no benefit compared to a range of other interventions for neck pain. (Level I)

Transcutaneous Electrical Nerve Stimulation (TENS)

The Cochrane Review by Gross et al. (2002b, last updated 1998) included one study assessing the efficacy of transcutaneous electrical nerve stimulation (TENS) for the treatment of acute, non-radiating neck pain (Nordemar and Thorner 1981). Over a one-week period, collar use, rest and analgesics were compared to manual therapy (massage, gentle traction and cervical mobilisation) versus TENS three times a week. The TENS and manual therapy groups also wore a collar and received analgesics. At six weeks and three months there were no significant differences in pain levels between the groups. However, all people in all groups recovered fully within six weeks.

Key Message

There is insufficient evidence of benefit from TENS compared to a collar or manual therapy in acute neck pain. (Level I)

Evidence of No Benefit

Collars

In a Cochrane Review, Verhagen et al. (2002, last updated 2000) identified one non-randomised study (Gennis et al. 1996) that compared the effect of a soft collar for two weeks versus no treatment (both groups received rest and analgesics) in subjects with acute neck pain. Collars were found to be no more effective than rest and analgesics.

In their systematic review, Harms-Ringdahl and Nachemson (2000) concluded that no evidence exists that collars have a positive effect on neck pain. They cited a number of studies that met the criteria for this update (Mealy et al. 1986; McKinney et al. 1989; Borchgrevink et al. 1998; Foley-Nolan et al. 1992; Nordemar and Thorner 1981). An additional study (Rosenfeld et al. 2000) comparing the effect of active mobilisation versus passive treatment was located.

In many of these studies, collars were used as the control treatment, or as part of the index treatment. In that regard, collars were found to be less effective than manual therapy (Mealy et al. 1986), active outpatient treatment (McKinney et al. 1989), therapist-directed home exercises (McKinney et al. 1989; Rosenfeld et al. 2000) and no more effective than TENS (Nordemar and Throner 1981) and instructions to resume normal activities (Borchgrevink et al. 1998). The Foley-Nolan et al. (1992) study is described in 'Pulsed Electromagnetic Therapy (PEMT)'.

Key Message

Soft collars are not effective for acute neck pain compared to advice to resume normal activity and other interventions. (Level I, II)

>References

Acheson MB, Livingston RR, Richardson ML, Stimac GK (1987). High-resolution CT scanning in the evaluation of cervical spine fractures: comparison with plain film examinations. American Journal of Roentgenology, 148: 1179–1185.

Aker PD, Gross AR, Goldsmith CH, Peloso P (1996). Conservative management of mechanical neck pain: systematic overview and meta-analysis. British Medical Journal, 313: 1291–1296.

American Psychiatric Association (1994). Diagnostic and Statistical Manual of Mental Disorders (4th Edition) (DSM-IV). American Psychiatric Association: Washington DC. p 683.

An HS (1998). Cervical spine trauma. Spine, 23: 2713–2729.

Andersen JH, Gaardboe O (1993a). Musculoskeletal disorders of the neck and upper limb among sewing machine operators: a clinical investigation. American Journal of Industrial Medicine, 24: 689–700.

Andersen JH, Gaardboe O (1993b). Prevalence of persistent neck and upper limb pain in a historical cohort of sewing machine operators. American Journal of Industrial Medicine, 24: 677–687.

Aprill C, Dwyer A, Bogduk N (1990). Cervical zygapophyseal joint pain patterns II: a clinical evaluation. Spine, 15: 458–461.

Ariens GAM, Bongers PM, Hoogendoorn WE, Houtman ILD, van dar Wal G, van Mechelen W (2001). Risk quantitative job demands and low coworker support as risk factors for neck pain. Spine, 26: 1896–1903.

Ariens GAM, Borghouts AJ, Koes BW (1999). Neck pain. In: Crombie IK (ed). Epidemiology of Pain. IASP Press: Seattle. pp 235–255.

Auten GM, Levy CS, Smoth MA (1991). Haemophilus parainfluenzae as a rare cause of epidural abscess. Case report and review. Reviews of Infectious Diseases, 13: 609–612.

Bachulis BL, Long WB, Hynes GD, Johnson MC (1987). Clinical indications for cervical spine radiographs in the traumatised patient. American Journal of Surgery, 153: 473–477.

Barnsley L (1998). Neck pain. In: Klippel JH Dieppe PA (eds). Rheumatology (2nd Edition), Volume 1. Mosby, London. pp 4.1–4.12.

Barnsley L, Lord S, Bogduk N (1998). The pathophysiology of whiplash. In: Malanga GA (ed). Cervical Flexion-Extension/Whiplash Injuries. Spine: State of the Art Reviews. Hanley and Belfus: Philadelphia. pp 12: 209–242.

Barnsley L, Lord SM, Wallis BJ, Bogduk N (1995). The prevalence of chronic cervical zygapophyseal joint pain after whiplash. Spine, 20: 20–26.

Basmajian JV (1978). Cyclobenzaprine hydrochloride effect on skeletal muscle spasm in the lumbar region and neck: two double-blind controlled clinical and laboratory studies. Archives of Physical Medicine and Rehabilitation, 59: 58–63.

Basmajian JV (1983). Reflex cervical muscle spasm: treatment by diazepam, phenobarbital or placebo. Archives of Physical Medicine and Rehabilitation, 64: 121–124.

Bassett LW (1987). Magnetic resonance imaging in musculoskeletal disorders. Bulletin on the Rheumatic Diseases, 37: 1–6.

Beatty RM, Winston KR (1984). Spontaneous cervical epidural haematoma: a consideration of etiology. Journal of Neurosurgery, 61: 143–148.

Bell GR, Ross JS (1992). Diagnosis of nerve root compression: myelography, computed tomography, and MRI. Orthopedic Clinics of North America, 23: 405–419.

Benzel EC, Hart BL, Ball PA, Baldwin NG, Orrison WW, Espinosa MC (1996). Magentic resonance imaging for the evaluation of patients with occult cervical spine injury. Journal of Neurosurgery, 85: 824–829.

Bercel NA (1977). Cyclobenzaprine in the treatment of skeletal muscle spasm in osteoarthritis of the cervical and lumbar spine. Current Therapeutic Research, 22: 462–468.

Berglund A, Alfredsson L, Cassidy JD, Jensen I, Nygren A (2000). The association between exposure to a rear-end collision and future neck or shoulder pain: a cohort study. Journal of Clinical Epidemiology, 35: 1089–1094.

Bernhardt M, Hynes RA, Blume HW, White AA (1993). Current concepts review: cervical spondylotic myelopathy. Journal of Bone and Joint Surgery, 75A: 119–128.

Bernstein SA (1975). Acute cervical pain associated with soft-tissue calcium deposition anterior to the interspace of the first and

second cervical vertebrae. Journal of Bone and Joint Surgery, 57A: 426–428.

Berquist TH, Brown ML, Fitzgerald RH, May GR (1985). Magnetic resonance imaging: application in musculoskeletal infection. Magnetic Resonance Imaging, 3: 219–230.

Binder A (1993). Cervical pain syndromes. In: Maddison PJ, Isenberg DA, Woo P, Glass DN (eds). Oxford Textbook of Rheumatology. Oxford University Press: Oxford. pp 1060–1070.

Binet EF, Moro JJ, Marangola JP, Hodge CJ (1977). Cervical spine tomography in trauma. Spine, 2: 163–172.

Bigos S, Bowyer O, Braen G. et al. (1994). Acute low back problems in adults. Clinical Practice Guidelines No. 14. AHCPR Publication No. 95-0642. Agency for Health Care Policy and Research, Public Health Service. US Department of Health: Rockville MD.

Biousse V, D'Anglejan-Chatillon J, Massiou H, Bousser MG (1994). Head pain in non-traumatic carotid artery dissection: a series of 65 patients. Cephalalgia, 14: 33–36.

Bird HA, Esselinkcz W, Dixon AStJ, Mowat AG, Wood PHN (1979). An evaluation of criteria for polymyalgia rheumatica. Annals of the Rheumatic Diseases, 38: 434–439.

Blacksin MF, Lee HJ (1995). Frequency and significance of fractures of the upper cervical spine detected by CT in patients with severe neck trauma. American Journal of Roentgenology, 165: 1201–1204.

Boden SD, McCowin PR, Davis DG, Dina TS, Mark AS, Wiesel S (1990). Abnormal magnetic-resonance scans of the cervical spine in asymptomatic subjects: a prospective investigation. Journal of Bone and Joint Surgery, 72A: 1178–1184.

Bogduk N (2000). Whiplash: why pay for what does not work? Journal of Musculoskeletal Pain, 8: 29–53.

Bogduk N, Marsland A (1988). The cervical zygapophysial joints as a source of neck pain. Spine, 13: 610–617.

Bogduk N, Simons DG (1993). Neck pain: joint pain or trigger points. In: Vaeroy H, Merskey H (eds). Progress in Fibromyalgia and Myofascial Pain. Elsevier: Amsterdam. pp 267–273.

Bogduk N, Yoganandan N (2001). Biomechanics of the cervical spine Part 3: minor injuries. Clinical Biomechanics, 16: 267–275.

Bonk AD, Ferrari R, Giebel GD, Edelmann M, Huser R (2000). Prospective randomized controlled study of activity vs collar and the natural history of whiplash injury in Germany. Journal of Musculoskeletal Pain, 8: 123–132.

Bono G, Antonaci F, Ghirmai S, D'Angelo F, Berger M, Nappi G (2000). Whiplash injuries: clinical picture and diagnostic work-up. Clinical and Experimental Rheumatology, 18: 523–528.

Borchgrevink GE, Kaasa A, McDonagh D, Stiles TC, Haraldseth O, Lereim I (1998). Acute treatment of whiplash neck sprain injuries. A randomised trial of treatment during the first 14 days after a car accident. Spine, 23: 25–31.

Borchgrevink GE, Smevik O, Nordby A, Rinck PA, Stiules TC, Lereim I (1995). MR imaging and radiography of patients with cervical hyperextension-flexion injuries after car accidents. Acta Radiologica, 36: 425–428.

Borchgrevink GE, Stiles TC, Borchgrevink PC, Lereim J (1997). Personality profile among symptomatic and recovered patients neck sprain injury, measured by MCM-I acutely and 6 months after car accidents. Journal of Psychosomatic Research, 42: 357–376.

Borghouts JAS, Koes BW, Bouter LM (1998). The clinical course and prognostic factors of non-specific neck pain: a systematic review. Pain, 77: 1–13.

Borock EC, Gabram SGA, Jacobs LM, Murphy A (1991). A prospective analysis of a two-year experience using computed tomography as an adjunct for cervical spine clearance. Journal of Trauma, 31: 1001–1006.

Bresler MJ, Rich GH (1982). Occult cervical spine fracture in an ambulatory patient. Annals of Emergency Medicine, 11: 440–442.

Brison RJ, Hartling L, Pickett W (2000). A prospective study of acceleration-extension injuries following rear-end motor vehicle collisions. Journal of Musculoskeletal Pain, 8: 97–113.

British Association of Physical Medicine (1966). Pain in the neck and arm: a multicentre trial of the effects of physiotherapy. British Medical Journal, January 29; 5482: 253–258.

Brockow T, Dillner A, Franke A, Resch KL (2001). Analgesic effectiveness of subcutaneous carbon-dioxide insufflations as an adjunct treatment in patients with non-specific neck or low back pain. Complementary Therapies in Medicine, 9: 68–76.

Brodin H (1985). Cervical pain and mobilization. Manipulative Medicine, 2: 18–22.

Cassidy JD, Lopes AA, Yong-Hing K (1992). The immediate effect of manipulation versus mobilization on pain and range of motion in the cervical spine: a randomized controlled trial. Journal of Manipulative and Physiological Therapeutics, 15: 570–575.

Clark CR, Igram CM, El-Khoury GY, Ehara S (1988). Radiographic evaluation of cervical spine injuries. Spine, 13: 742–747.

Clinical Evidence (June 2002). BMJ: London. pp 7:1049–1062.

Coan RM, Wong G, Coan PL (1981). The acupuncture treatment of neck pain: a randomised controlled study. American Journal of Chinese Medicine, 9: 326–332.

Cohn SM, Lyle G, Linden CH, Lancey RA (1991). Exclusion of cervical spine injury: a prospective study. Journal of Trauma, 31: 570–574.

Coletta R, Maggiolo F, di Tizio S (1988). Etofenamate and transcutaneous electrical nerve stimulation treatment of painful spinal syndromes. International Journal of Clinical Pharmacology Research, 8: 295–298.

Constantin A, Marin F, Bon E, Fedele M, Lagarrigue B, Bouteiller G (1996). Calcification of the transverse ligament of the atlas in chondrocalcinosis: computed tomography study. Annals of the Rheumatic Diseases, 55: 137–139.

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.

Craig JB, Hodgson BF (1991). Superior facet fractures of the axis vertebra. Spine, 16: 875–877.

Dabbs V, Lauretti J (1995). A risk assessment of cervical manipulation vs NSAIDs for the treatment of neck pain. Journal of Manipulative and Physiological Therapeutics, 18: 530–536.

Daffner RH (1992). Evaluation of cervical vertebral injuries. Seminars in Roentgenology, 27: 239–253.

Danner RL, Hartman RJ (1987). Update of spinal epidural abscess: 35 cases and review of literature. Reviews of Infectious Diseases, 9: 265–274.

Darouiche RO, Hamill RJ, Greenberg SB, Weathers SW, Musher DM (1992). Bacterial spinal epidural abscess. Review of 43 cases and literature survey. Medicine, 71: 369–385.

De Boer KF, Harman R, Tuttle CD, Wallace H (1985). Reliability study of detection of somatic dysfunctions in the cervical spine. Journal of Manipulative and Physiological Therapeutics, 8: 9–16.

de Craen AJ, Di Giulio G, Lampe-Schoenmaeckers JE, Kessels AG, Kleijnen J (1996) Analgesic efficacy and safety of paracetamol-codeine combinations versus paracetamol alone: a systematic review. British Medical Journal, 313: 321–325.

Del Curling O, Gower DJ, McWhorter JM (1990). Changing concepts in spinal epidural abscess. A report of 29 cases. Neurosurgery, 27: 185–192.

Demure B, Luippold RS, Bigalow C, Ali D, Mundt KA, Liese B (2000). Video display terminal workstation improvement program: I. Baseline associations between musculoskeletal

discomfort and ergonomic features of workstations. Journal of Occupational and Environmental Medicine, 42: 783–791.

Deyo RA (1996). Drug therapy for back pain: which drugs help which patients? Spine, 21: 2840–2850.

Dreyzin V, Esses SI (1993). Trauma of the cervical spine. Current Opinion in Orthopaedics, 4: 78–88.

Dvorak J, Hayek J, Zehnder R (1987). CT-functional diagnostics of the rotatory instability of the upper cervical spine. Part 2: an evaluation on healthy adults and patients with suspected instability. Spine, 12: 726–731.

Dwyer A, Aprill C, Bogduk N (1990). Cervical zygapophysial joint pain patterns I: a study in normal volunteers. Spine, 15: 453–457.

Ekberg K, Bjorkqvist B, Malm P, Bjerre-Kielly B, Axelson O (1994). Controlled two year follow up of rehabilitation for disorders in the neck and shoulders. Occupational and Environmental Medicine, 51: 833–838.

Ekbom K, Torhall J, Annell K, Traff J (1994). Magnetic resonance image in retropharyngeal tendonitis. Cephalalgia, 14: 266–269.

Elias F (1958). Roentgen findings in the asymptomatic cervical spine. New York State Journal of Medicine, 58: 3300–3303.

Elias M (1994). Cervical epidural abscess following trigger point injection. Journal of Pain and Symptom Management, 9: 71–72.

Eliastam M, Rose E, Jone H, Kaplan E, Kaplan R, Seiver A (1980). Utilization of diagnostic radiologic examinations in the emergency department of a teaching hospital. Journal of Trauma, 20: 61–66.

El-Khoury GY, Kathol MH, Daniel WW (1995). Imaging of acute injuries of the cervical spine: value of plain radiography, CT and MR imaging. American Journal of Roentgenology, 164: 43–50.

Ellenberg MR, Honet JC, Treanor WJ (1994). Cervical radiculopathy. Archives of Physical Medicine and Rehabilitation, 75: 342–352.

Ellertsson AB, Sigurjonsson K, Thorsteinsson T (1978). Clinical and radiographic study of 100 cases of whiplash injury. Acta Neurologica Scandinavica, 5 Supp 67: 269.

Ernst E (2002). Spinal manipulation: Its safety is uncertain. Canadian Medical Association Journal, 166: 40–41.

Fagerlund M, Bjornebrink J, Pettersson K, Hildingsson C (1995). MRI in acute phase of whiplash injury. European Radiology, 5: 297–301.

Fahlgren H (1986). Retropharyngeal tendonitis. Cephalalgia, 6: 169–174.

Fazl M, La Febvre J, Willinsky RA, Gertzbein S (1990). Past traumatic ligamentous disruption of the cervical spine an easily overlooked diagnosis: presentation of three cases. Neurosurgery, 26: 647–648.

Feinstein B, Langton JBK, Jameson RM, Schiller F (1954). Experiments on referred pain from deep somatic tissues. The Journal of Bone and Joint Surgery, 36A: 981–997.

Fialka V, Preisnger E, Bohler A (1989). Zur physikalischen diagnostik und physikalischer therapie der distorsio columnae cervicalis. Z Phys Med Baln Med Klim, 18: 390–397.

Fielding JW, Hawkins RJ (1977). Atlanto-axial rotatory fixation (fixed rotatory subluxation of the atlanto-axial joint). Journal of Bone and Joint Surgery, 59A: 37–44.

Fischer RP (1984). Cervical radiographic evaluation of alert patients following blunt trauma. Annals of Emergency Medicine, 13: 905–907.

Fjellner A, Bexander C, Faleij R, Strender LE (1999). Interexaminer reliability in physical examination of the cervical spine. Journal of Manipulative and Physiological Therapeutics, 22: 511–516.

Foley-Nolan D, Barry C, Coughlan RJ, O'Connor P, Roden D (1990). Pulsed high frequency (27mhz) electromagnetic therapy for persistent pain: a double blind, placebo-controlled study of 20 patients. Orthopaedics, 13: 445–451.

Foley-Nolan D, Moore K, Codd M, Barry C, O'Connor P, Coughlan RJ (1992). Low energy high frequency pulsed electromagnetic therapy for acute whiplash injuries. Scandinavian Journal of Rehabilitation Medicine, 24: 51–59.

Fredriksson K, Alfredsson L, Ahlberg G, Josephson M, Kilbom A, Wigaeus Hjelm E, Wiktorin C, Vingard E (2002). Work environment and neck and shoulder pain: the influence of exposure time. Results from a population based case-control study. Occupational and Environmental Medicine, 59: 182–55.

Fridenberg ZB, Miller WT (1963). Degenerative disc disease of the cervical spine. A comparative study of asymptomatic and symptomatic patients. Journal of Bone and Joint Surgery 45A: 1171–1178.

Fukui S, Ohseto K, Shiotani M, Ohno K, Karasawa H, Nagaauma Y, Yuda Y (1996). Referred pain distribution of the cervical zygapophyseal joints and cervical dorsal rami. Pain, 68: 79–83.

Gargan MF, Bannister GC (1990). Long-term prognosis of soft-tissue injuries of the neck. Journal of Bone and Joint Surgery, 72B: 901–903.

Gargan MF, Bannister GC (1994). The rate of recovery following whiplash injury. European Spine Journal, 3: 162–164.

Garrard P, Barnes D (1996). Aortic dissection presenting as a neurological emergency. Journal of the Royal Society of Medicine, 89: 271–272.

Gennis P, Miller L, Gallagher EJ, Giglio J, Carter W, Nathanson N (1996). The effect of soft collars on persistent neck pain in patients with whiplash injury. Academic Emergency Medicine, 3: 568–573.

Gerrelts BD, Petersen EU, Mabry J, Petersen SR (1991). Delayed diagnosis of cervical spine injuries. Journal of Trauma, 31: 1622–1626.

Giebel GD, Edelmann M, Huser R (1997). Die Distorsion der Halswirbelsaule: fruhfunktionelle vs ruhigstellende Behandlung. Zentralblatt fur Chirurgie, 51: 377–384.

Goldie I, Landquist A (1970). Evaluation of the effects of different forms of physiotherapy in cervical pain. Scandinavian Journal of Rehabilitation Medicine, 2–3: 117–121.

Goodman BW (1988). Neck pain. Primary Care, 15: 689–708.

Gordin V, Stowe C (2001). Diagnostic and therapeutic injections for the nonoperative treatment of axial neck pain and cervical radiculopathy. Current Opinion in Orthopaedics, 12: 238–244.

Gordon SJ, Trott P, Grimmer KA (2002). Waking cervical pain and stiffness, headache, scapular or arm pain: gender and age effects. Australian Journal of Physiotherapy, 48: 9–15.

Gore DR, Sepic SB, Gardner GM (1986). Roentgenographic findings of the cervical spine in asymptomatic people. Spine, 1: 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.

Greigel-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.

Gross AR, Aker PD, Goldsmith CH, Peloso P (2002a). Patient education for mechanical neck disorders. In: The Cochrane Library; Issue 2. Oxford: Update Software.

Gross AR, Aker PD, Goldsmith CH, Peloso P (2002b). Physical medicine modalities for mechanical neck disorders. In: The Cochrane Library; Issue 2. Oxford: Update Software.

Gross AR, Aker PD, Quartly C (1996). Manual therapy in the treatment of neck pain. Rheumatic Diseases Clinics of North America, 22: 579–598.

Gross AR, Kay T, Hondras M, Goldsmith C, Haines T, Peloso P, Kennedy C, Hoving J (2002c). Manual therapy for mechanical neck disorders: a systematic review. Manual Therapy, 7: 131–149.

Grubb JA, Kelly CK (2000). Cervical discography: clinical implications from twelve years' experience. Spine, 25: 1382–1389.

Guss DA, Jacoby IJ (2002). Longus colli tendonitis causing acute neck pain. Journal of Emergency Medicine, 22: 211–212.

Haines JD (1986). Occult cervical spine fractures. Postgraduate Medicine, 80: 73–74, 77.

Harder S, Veilleux M, Suissa S (1998). The effect of socio-demographic and crash-related factors on the prognosis of whiplash. Journal of Clinical Epidemiology, 51: 377–384.

Hardin JG, Halla JT (2001). Cervical spine syndromes. In: Koopman WJ (ed). Arthritis and Allied Conditions. A Textbook of Rheumatology (14th Edition). Lippincott Williams and Wilkins: Philadelphia. pp 2009–2018.

Harinck HI, Buvoet OL, Vellenga CJ, Blanksma HJ, Frijlink WB (1986). Relation between signs and symptoms in Paget's disease of bone. Quarterly Journal of Medicine, 58: 133–151.

Harms-Ringdahl K, Nachemson A (2000). Acute and subacute neck pain: nonsurgical treatment. In: Nachemson A, Jonsson E (eds). Neck and Back Pain: The Scientific Evidence of Causes, Diagnosis, and Treatment. Lippincott Williams and Wilkins: Philadelphia. pp 327–338.

Hartley J (1964). Acute cervical pain associated with retropharyngeal calcium deposit. Journal of Bone and Joint Surgery, 46A: 1753–1754.

Heller CA, Stanley P, Lewis-Jones B, Heller RF (1983). Value of x-ray examinations of the cervical spine. British Medical Journal, 287: 1276–1278.

Helliwell PS, Evans PF, Wright V (1994). The straight cervical spine: does it indicate muscle spasm? Journal of Bone and Joint Surgery, 76B: 103–106.

Hendriks O, Horgan A (1996). Ultra-reiz current as an adjunct to standard physiotherapy treatment of the acute whiplash patient. Physiotherapy Ireland, 17: 3–7.

Henry D, Lim LLY, Rodriguez LAG, Gutthann SP, Carson JL et al. (1996). Variability in the risk of gastrointestinal complications with individual non-steroidal anti-inflammatory drugs: results of a collaborative meta-analysis. British Medical Journal, 312: 1563–1566.

Hinse P, Thie A, Lachenmayer L (1991). Dissection of the extracranial vertebral artery: report of four cases and review of the literature. Journal of Neurology, Neurosurgery, and Psychiatry, 54: 863–869.

Hirst AE, Johns VJ, Kime FW (1958). Dissecting aneurysm of the aorta: a review of 505 cases. Medicine, 37: 217–275.

Hlavin ML, Kaminski HJ, Ross JS, Ganz E (1990). Spinal epidural abscess: a ten year perspective. Neurosurgery, 27: 177–184.

Hochberg M, Borenstein D, Arnett F (1978). The absence of back pain in classic ankylosing spondylitis. The Johns Hopkins Medical Journal, 143: 181–183.

Hoffman JR, Mower WR, Wolfson AB, Todd KH, Zucker MI, for the National Emergency X-radiography Utilization Study Group (2000). Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. New England Journal of Medicine, 343: 94–99.

Hoffman JR, Schriger DL, Mower W, Luo JS, Zucker M (1992). Low-risk criteria for cervical-spine radiography in blunt trauma: a prospective study. Annals of Emergency Medicine, 21: 1454–1460.

Holliman CJ, Mayer JS, Cook RT, Smith JS (1991). Is the anteroposterior cervical spine radiograph necessary in initial trauma screening? American Journal of Emergency Medicine, 9: 421–425.

Horneij E, Hemborg B, Jensen I, Ekdahl C (2001). No significant differences between intervention programmes on neck, shoulder and low back pain: a prospective randomized study among home-care personnel. Journal of Rehabilitation Medicine, 33: 170–176.

Hoving JL (2001). Neck Pain in Primary Care (PhD Thesis). Vrie Universitet, Amsterdam.

Hoving JL, Gross AR, Gasner D, Kay T, Kennedy C, Hondras MA, Haines AT, Bouter LM (2001). A critical appraisal of review articles on the effectiveness of conservative treatment for neck pain. Spine, 26: 196–205.

Hoving JL, Koes BW, de Vet HC, van der Windt DA, Assendelft WJ, van Mameren H, Deville WL, Pool JJ, Scholten RJ, Bouter LM (2002). Manual therapy, physical therapy, or continued care by a general practitioner for patients with neck pain. A randomized, controlled trial. Annals of Internal Medicine, 136: 713–722.

Howe DH, Newcombe RG, Wade MT (1983). Manipulation of the cervical spine — pilot study. Journal of the Royal College of General Practitioners, 33: 574–579.

Hubka MJ, Phelan SP (1994). Interexaminer reliability of palpation for cervical spine tenderness. Journal of Manipulative and Physiological Therapeutics, 17: 591–595.

Hudak PL, Cole DC, Frank JW (1998). Perspectives on prognosis of soft tissue musculoskeletal disorders. International Journal of Rehabilitation Research, 21: 29–40.

Hurst PG, Seeger J, Carter P, Marus F I (1989). Value of magnetic resonance imaging for diagnosis of cervical epidural hematoma associated with anticoagulation after cardiac valve replacement. American Journal of Cardiology, 63: 1016–1017.

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.

Jacobs LM, Schwartz R (1986). Prospective analysis of acute cervical spine injury: a methodology to predict injury. Annals of Emergency Medicine, 15: 44–49.

Janjua KJ, Goswami V, Sagar G (1996). Whiplash injury associated with acute bilateral internal carotid arterial dissection. Journal of Trauma, 40: 456–458.

Jayakrishnan VK, Teasdale E (2000). Torticollis due to atlanto-axial rotatory fixation following general anaesthesia. British Journal of Neurosurgery, 14: 583–585.

Jensen I, Nygren A, Gamberale F, Goldie I, Westerholm P, Jonsson E (1995). The role of the psychologist in multidisciplinary treatments for chronic neck and shoulder pain. A controlled cost-effectiveness study. Scandinavian Journal of Rehabilitation Medicine, 27: 19–26.

Johnson DW (1996). Imaging of the traumatized cervical spine. Current Opinion in Orthopaedics, 7: 61–68.

Johnson MJ, Lucas GL (1997). Value of cervical spine radiographs as a screening tool. Clinical Orthopaedics and Related Research, 340: 102–108.

Jull G, Bogduk N, Marsland A (1988). The accuracy of manual diagnosis for cervical zygapophysial joint pain syndromes. Medical Journal of Australia, 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. Australian Journal of Physiotherapy, 43: 125–129.

Kamwendo K, Linton SJ (1991). A controlled study of the effect of neck school in medical secretaries. Scandinavian Journal of Rehabilitation Medicine, 23: 143–152.

Kamwendo K, Linton SJ, Moritz U (1991a). Neck and shoulder disorders in medical secretaries. Part I. Pain prevalence and risk factors. Scandinavian Journal of Rehabilitation Medicine, 23: 127–133.

Kamwendo K, Linton SJ, Moritz U (1991b). Neck and shoulder disorders in medical secretaries. Part II. Ergonomical work

environment and symptom profile. Scandinavian Journal of Rehabilitation Medicine, 23: 135–142.

Karasick D, Karasick S (1981). Calcific retropharyngeal tendonitis. Skeletal Radiology, 7: 203–205.

Karjalainen K, Malmivaara A, van Tulder M, Roine R, Jauhiainen M, Hurri H, Koes B (2002). Multi-disciplinary biopsychosocial rehabilitation for neck and shoulder pain among working age adults. In: The Cochrane Library; Issue 2. Oxford: Update Software.

Karlberg M, Magnusson M, Malmstrom EM et al. (1996). Postural and symptomatic improvement after physiotherapy in patients with dizziness of suspected cervical origin. Archives of Physical and Medical Rehabilitation, 77: 874–882.

Karlsborg M, Smed A, Jespersen H, Stephensen S, Cortsen M, Jennum P, Herning M, Korfitsen E, Werdelin L (1997). A prospective study of 39 patients with whiplash injury. Acta Neurologica Scandinavica, 95: 65–72.

Kasch H, Bach FW, Jensen TS (2001). Handicap after acute whiplash injury: a 1-year prospective study of risk factors. Neurology, 56: 1637–1643.

Kathol MH (1997). Cervical spine trauma: what is new? Radiology Clinics of North America, 35: 507–532.

Kellgren JH (1939). On the distribution of pain arising from deep somatic structures with charts of segmental pain areas. Clinical Science, 4: 35–46.

Ketola R, Toivonen R, Hakkanen M, Luukkonen R, Takala EP, Viikari-Juntura E (2002). Effects of ergonomic intervention in work with video display units. Scandinavian Journal of Work, Environment and Health, 1: 18–24.

Kjellman GV, Skargren EI, Oberg BE (1999). A critical analysis of randomized clinical trials on neck pain and treatment efficacy: a review of the literature. Scandinavian Journal of Rehabilitation Medicine, 31: 139–152.

Kligman M, Rabichev M, Roffman M (2000). Acute neck pain due to calcification anterior to the odontoid process. Bulletin (Hospital for Joint Diseases, New York, N.Y.), 59: 111–112.

Koes BW (1992). Efficacy of manual therapy for back and neck complaints. (Thesis) Cip-Gegevens Koninklijke Bibliotheek: den Haag.

Koes BW, Assendelft WJ, Van der Heijden GJ, Bouter LM, Knipschild PG (1991). Spinal manipulation and mobilization for back and neck pain a blinded review. British Medical Journal, 303: 1298–1303.

Koes BW, Bouter LM, van Mameren H, Essers AH, Verstegen GM, Hofhuizen DM, Houben JP, Knipschild PG (1993). A randomised clinical trial of manual therapy and physiotherapy for persistent back and neck complaints: subgroup analysis and relationship between outcome measures. Journal of Manipulative and Physiological Therapeutics, 16: 211–219.

Koes BW, Bouter LM, van Mameren H, Essers AH, Verstegen GM, Hofhuizen DM, Houben JP, Knipschild PG (1992a). Randomised clinical trial of manipulative therapy and physiotherapy for persistent back and neck complaints: results of one year follow up. British Medical Journal, 304: 601–605.

Koes BW, Bouter LM, Van Mameren H, Essers AHM, Verstegen GMJR, Hofhuizen DM, Houben JP, Knipschild PG (1992b). The effectiveness of manual therapy, physiotherapy, and treatment by the general practitioner for nonspecific back and neck complaints: a randomized clinical trial. Spine, 17: 28–35.

Kreipke DL, Gillespie KR, McCarthy MC, Mail JT, Lappas JC, Broadie TA (1989). Reliability of indications for cervical spine films in trauma patients. Journal of Trauma, 29: 1438–1439.

Krolick MA, Cintrom GB (1991). Spinal epidural hematoma causing cord compression after tissue plasminogen activator and heparin therapy. Southern Medical Journal, 84: 670–671.

Lasker RB, Harter DH (1987). Cervical epidural abscess. Neurology, 37: 1747–1753.

Lee C, Woodring JH (1991). Sagittally oriented fractures of the lateral masses of the cervical vertebrae. Journal of Trauma, 31: 1638–1643.

Lees F, Turner JWA (1963). Natural history and prognosis of cervical spondylosis. British Medical Journal, 2: 1607–1610.

Levoska S, Keinanen-Kiukaanniemi S, Bloigu R (1991). Repeatability of measurement of tenderness in the neck-shoulder region by a dolorimeter and manual palpation. Clinical Journal of Pain, 9: 229–235.

Lewis LM, Docherty W, Ruoff BE, Fortney JP, Keltner RA, Britton P (1991). Flexion extension views in the evaluation of cervical-spine injuries. Annals of Emergency Medicine, 20: 117–121.

Lewith GT, Machin D (1981). A randomised trial to evaluate the effect of infra-red stimulation of local trigger points, versus placebo on the pain caused by cervical osteoarthrosis. Acupuncture and Electro-Therapeutics Research, 6: 277–284.

Linton SJ, Andersson MA (2000). Can chronic disability be prevented? A randomized trial of a cognitive-behaviour intervention and two forums of information for patients with spinal pain. Spine, 21: 2825–2831.

Linton SJ, Kamwendo K (1989). Risk factors in the psychosocial work environment for neck and shoulder pain in secretaries. Journal of Occupational Medicine, 31: 609–613.

Linton SJ, van Tulder M (2001). Preventive interventions for back and neck pain problems. What is the evidence? Spine, 26: 778–787.

Lobitz B, Grate I (1995). Acute epidural hematoma of the cervical spine: an unusual cause of neck pain. Southern Medical Journal, 88: 580–582.

Lord S, Barnsley L, Wallis BJ, Bogduk N (1996). Chronic cervical zygapophysial joint pain after whiplash: a placebo-controlled prevalence study. Spine, 21: 1737–1745.

Loy TT (1983). Treatment of cervical spondylosis: electroacupuncture versus physiotherapy. Medical Journal of Australia, 1983; 2: 32–34.

Lunardi P, Acqui M, Ricci G, Agrillo A, Ferrante L (1999). Cervical synovial cysts: case report and review of the literature. European Spine Journal, 8: 232–237.

MacDonald RL, Schwartz ML, Mirich D, Sharkey PW, Nelson WR (1990). Diagnosis of cervical spine injury in motor vehicle crash victims: how many x-rays are enough? Journal of Trauma, 30: 392–397.

Mace SE (1985). Emergency evaluation of cervical spine injuries: CT versus plain radiographs. Annals of Emergency Medicine, 14: 973–975.

Mace SE (1992). The unstable occult cervical spine fracture: a review. American Journal of Emergency Medicine, 10: 136–142.

Mace SE (1991). Unstable occult spine fracture. Annals of Emergency Medicine, 20: 1373–1375.

Maimaris C, Barnes MR, Allen MJ (1988). 'Whiplash injuries' of the neck: a retrospective study. Injury, 19: 393–396.

Makela M, Heliovaara M, Sievers K, Impivaara O, Knekt P, Aromaa A (1991). Prevalence, determinants and consequences of chronic neck pain in Finland. American Journal of Epidemiology, 124: 1356–1367.

Matsumae M, Shimoda M, Shibuya N, Veda M, Yamamoto I, Sato O (1987). Spontaneous cervical epidural hematoma. Surgical Neurology, 28: 381–384.

Maull KI, Sachatello CR (1977). Avoiding a pitfall in resuscitation: the painless cervical fracture. Southern Medical Journal, 70: 477–478.

McGuirk B, King W, Govind J, Lowry J, Bogduk N (2001). The Safety, Efficacy, and Cost-Effectiveness of Evidence-Based

Guidelines for the Management of Acute Low Back Pain in Primary Care. Spine, 26: 2615–2622.

McKee TR, Tinkoff G, Rhodes M (1990). Asymptomatic occult cervical spine fracture: case report and review of the literature. Journal of Trauma, 30: 623–626.

McKinney LA (1989). Early mobilisation and outcomes in acute sprains of the neck. British Medical Journal, 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. Archives of Emergency Medicine, 6: 27–33.

McKnight P, Friedman J (1992). Torticollis due to cervical epidural abscess and osteomyelitis. Neurology, 42: 696–697.

McLean L, Tingley M, Scott RN, Rickards J (2001). Computer terminal work and the benefit of microbreaks. Applied Ergonomics, 32: 225–237.

McNamara RM (1988). Post-traumatic neck pain: a prospective and follow-up study. Annals of Emergency Medicine, 17: 906–911.

Mealy K, Brennan H, Fenelon GCC (1986). Early mobilisation of acute whiplash injuries. British Medical Journal, 292: 656–657.

Mendelson G (1984). Follow-up studies of personal injury litigants. International Journal of Law and Psychiatry, 7: 179–188.

Mendelson G (1982). Not 'cured by a verdict'. Effect of legal settlement on compensation claimants. Medical Journal of Australia, 2: 132–134.

Mercer S, Bogduk N (1993). Intra-articular inclusions of the cervical synovial joints. British Journal of Rheumatology, 32: 705–710.

Merskey H (1979). Pain terms: a list with definitions and notes on usage recommended by the IASP Subcommittee on Taxonomy. Pain, 6: 249–252.

Merskey H, Bogduk N (eds) (1994). Classification of Chronic Pain. Descriptions of Chronic pain Syndromes and Definitions of Pain Terms (2nd Edition). IASP Press: Seattle.

Mihmanli I, Karaarslan E, Kanberoglu K (2001). Inflammation of vertebral bone associated with acute calcific tendonitis of the longus colli muscle. Neuroradiology, 43: 1098–1101.

Miller RL, White S, McConnell C, Mueller C (1994). Influence of medicolegal factors in the use of cervical spine and head computed tomographic examinations in an emergency setting. Emergency Radiology, 1: 279–282.

Mior SA, King RS, McGregor M, Bernard M (1985). Intra and interexaminer reliability of motion palpation in the cervical spine. Journal of the Canadian Chiropractic Association, 29: 195–198.

Mirvis SE, Diaconis JN, Chirico PA, Reiner BI, Joslyn JN, Militello P (1989). Protocol-driven radiologic evaluation of suspected cervical spine injury: efficacy study. Radiology, 170: 831–834.

Modic MT, Feiglin DH, Piraino DW, Boumphrey F, Weinstein MA, Duchesneau PM, Rehm S (1985). Vertebral osteomyelitis: assessment using MR. Radiology, 157: 157–166.

Motor Accidents Authority: Sydney NSW (2001). Guidelines for the Management of Whiplash-Associated Disorders.

Muffoletto AJ, Nader R, Westmark RM, Nauta HJ, Garges KJ, Hadjipavlou AG (2001). Hematogenous pyogenic facet joint infection of the subaxial cervical spine. A report of two cases and review of the literature. Journal of Neurosurgery, 95: 135–138.

Munchau A, Palmer JD, Dressler D, O'Sullivan JD, Tsang KL, Jahanshahi M, Quinn NP, Lees AJ, Bhatia KP (2001). Prospective study of selective peripheral denervation for botulinum-toxin resistant patients with cervical dystonia. Brain, 124: 769–783.

Mustafa MH, Gallino R (1988). Spontaneous spinal epidural hematoma causing cord compression after streptokinase and heparin therapy for acute coronary artery occlusion. Southern Medical Journal, 81: 1202–1203.

Nakano KK (2001). Neck pain. In: Ruddy S, Harris ED, Sledge CB (eds). Kelley's Texbook of Rheumatology, (6th Edition). WB Saunders: Philadelphia. pp 457–474.

Nansel DD, Peneff AL, Jansen RD, Cooperstein R (1989). Interexaminer concordance in detecting joint-play asymmetries in the cervical spines of otherwise asymptomatic subjects. Journal of Manipulative and Physiological Therapeutics, 12: 428–433.

National Health and Medical Research Council (1999a). A Guide to the Development, Implementation and Evaluation of Clinical Practice Guidelines. Commonwealth of Australia: Canberra.

National Health and Medical Research Council (1999b). Acute Pain Management: Scientific Evidence. Commonwealth of Australia: Canberra. p 109.

Newmark H, Forrester DM, Brown JC, Robinson A, Olken SM, Bledsoe R (1978). Calcific tendonitis of the neck. Radiology, 128: 355–358.

Newmark H, Zee CS, Frankel P, Robinson A, Blau L, Gans DC (1981). Chronic calcific tendonitis of the neck. Skeletal Radiology, 7: 207–208.

Nolla JM, Ariza J, Gomez-Vaquero C, Fiter J, Bermejo J, Valverde J, Escofet DR, Gudiol F (2002). Spontaneous pyogenic vertebral osteomyelitis in nondrug users. Seminars in Arthritis and Rheumatism, 31: 271–278.

Nordemar R, Thorner C (1981). Treatment of acute cervical pain — a comparative group study. Pain, 10: 93–101.

Norris SH, Watt I (1983). The prognosis of neck injuries resulting from rear-end vehicle collisions. Journal of Bone and Joint Surgery, 65B: 608–611.

Ogden W, Dunn JD (1986). Cervical radiographic evaluation following blunt trauma. Annals of Emergency Medicine, 15: 604–605.

Palmer KT, Walker-Bone K, Griffin MJ, Syddall H, Pannett B, Coggon D, Cooper C (2001). Prevalence and occupational associations of neck pain in the British population. Scandinavian Journal of Work, Environment and Health, 27: 49–56.

Parmar HV, Raymakers R (1993). Neck injuries from rear impact road traffic accidents: prognosis in persons seeking compensation. Injury, 24: 75–78.

Peeters GGM, Verhagen AP, de Bie RA, Oostendorp RAB (2001). The efficacy of conservative treatment in patients with whiplash injury. A systematic review of clinical trials. Spine, 26: E64-E73.

Pennie B, Agambar L (1991). Patterns of injury and recovery in whiplash. Injury, 22: 57–59.

Pennie BH, Agambar LJ (1990). Whiplash injuries: a trial of early management. Journal of Bone and Joint Surgery, 72B: 277–279.

Petrie JP, Hazleman BL (1986). A controlled study of acupuncture in neck pain. British Journal of Rheumatology, 25: 271–275.

Petrie JP, Langley GB (1983). Acupuncture in the treatment of chronic cervical pain: a pilot study. Clinical and Experimental Rheumatology, 1: 333–335.

Pettersson K, Hildingsson C, Toolanen G, Fagerlund M, Bjornebrink J (1994). MRI and neurology in acute whiplash trauma. Acta Orthopaedica Scandinavica, 65: 525–528.

Philadelphia Panel Evidence-based clinical practice guidelines on selected rehabilitation interventions for neck pain (2001). Physical Therapy, 81: 1701–1717.

Pietri-Taleb F, Riihimaki H, Viikari-Juntura E, Lindstrom K (1994). Longitudinal study on the role of personality characteristics and psychological distress in neck trouble among working men. Pain, 58: 261–267.

Poletti SC, Handal JA (1995). Degenerative disc disease of the cervical spine: degenerative cascade and the anterior approach. In: White AH (ed). Spine Care, Volume Two. Mosby: St Louis. pp 1351–1357.

Posner J, Glew C (2002). Editorial. Neck pain. Annals of Internal Medicine, 136: 758–759.

Provinciali L, Baroni M, Illuminati L, Ceravolo MG (1996). Multimodal treatment to prevent the late whiplash syndrome. Scandinavian Journal of Rehabilitation Medicine, 28: 105–111.

Radanov BP, Di Stefano G, Schnidrig A, Ballinari P (1991). Role of psychosocial stress in recovery from common whiplash. Lancet, 338: 712–715.

Radanov BP, Sturzenegger M (1996). Predicting recovery from common whiplash. European Neurology, 36: 48–51.

Radanov BP, Sturzenegger M, Di Stefano G (1995). Long-term outcome after whiplash injury: a 2-year follow-up considering features of injury mechanism and somatic, radiologic, and psychosocial findings. Medicine, 74: 281–297.

Ring D, Yaccaro AR, Scudori G, Pathria MN, Garfin SE (1994). Acute calcific retropharyngeal tendonitis. Clinical presentation and pathological characterization. Journal of Bone and Joint Surgery, 76: 1636–1642.

Roberge RJ (1993). Unstable occult cervical-spine fracture. Annals of Emergency Medicine, 22: 868.

Roberge RJ, Wears RC (1992). Evaluation of neck discomfort, neck tenderness, and neurologic deficits as indicators for radiography in blunt trauma victims. Journal of Emergency Medicine, 10: 539–544.

Roberge RJ, Wears RC, Kelly M, Evans TC, Kenny MA, Daffner RD, Kremenr, Murray K, Cottington EC (1988). Selective application of cervical spine radiology in alert victims of blunt trauma: a prospective study. Journal of Trauma, 28: 784–788.

Ronnen HR, de Korte PJ, Brink PRG, van der Bijl HJ, Tonino AJ, Franke CL (1996). Acute whiplash injury: is there a role for MR imaging? A prospective study of 100 patients. Radiology, 201: 93–96.

Rose KD, Croissant PD, Parliament CF, Levin MB (1990). Spontaneous spinal epidural hematoma with associated platelet dysfunction from excessive garlic consumption. Neurosurgery, 26: 880–882.

Rosenfeld M, Gunnarsson R, Borenstein P (2000). Early intervention in whiplash-associated disorders. A comparison of two treatment protocols. Spine, 25: 1782–1787.

Roth BJ, Martin RR, Foley K, Barcia PJ, Kennedy P (1994). Roentgenographic evaluation of the cervical spine: a selective approach. Archives of Surgery, 129: 643–645.

Saddison D, Vanek VW, Racanelli JL (1991). Clinical indications for cervical spine radiographs in alert trauma patients. American Surgeon, 57: 366–369.

Sandmark H, Nisell R (1995). Validity of five common manual neck pain provoking tests. Scandinavian Journal of Rehabilitation Medicine, 27: 131–136.

Sarkozi J, Fam AG (1984). Acute calcific retropharyngeal tendonitis: an unusual cause of neck pain. Arthritis and Rheumatism, 27: 708–710.

Schattner A (1996). Pain in the neck. Lancet, 348: 411–412.

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.

Schleehauf K, Ross SE, Civil AD, Schwab CW (1989). Computed tomography in the initial evaluation of the cervical spine. Annals of Emergency Medicine, 18: 815–817.

Schrader H, Obelieniene D, Bovim G, Surkiene D, Mickeviciene D, Miseviciene I, Sand T (1996). Natural evolution of late whiplash syndrome outside the medicolegal context. Lancet, 347: 1207–1211.

Schreiber S, Vinokur S, Shavelzon V, Pick CG, Zahavi E, Shir Y (2001). A randomized trial of fluoxetine versus amitriptyline in musculo-skeletal pain. Israel Journal of Psychiatry and Related Science, 38: 88–94.

Scully RE, Mark EJ, McNeely WF, Ebeling SH, Phillips LD (1992). Case records of the Massachusetts General Hospital. New England Journal of Medicine, 326: 1070–1076.

Seitz JP, Unguez CE, Corbus HF, Wooten WW (1995). SPECT of the cervical spine in the evaluation of neck pain after trauma. Clinical Nuclear Medicine, 20: 667–673.

Seletz E (1958). Whiplash injuries: neurophysiological basis for pain and methods used for rehabilitation. Journal of the American Medical Association, 168: 1750–1755.

Shapiro AP, Roth RS (1993). The effect of litigation on recovery from whiplash. In: Teasell R, Shapiro A (eds). Spine: State of the Art Reviews. Cervical Flexion-Extension/Whiplash Injuries. Hanley and Belfus: Philadelphia. pp 7: 551–556.

Sharp J, Purser DW, Lawrence JS (1958). Rheumatoid arthritis of the cervical spine in the adult. Annals of the Rheumatic Diseases, 17: 303–313.

Shuma Y, Rothman SLG, Yasura K, Takahashi S (2002). Degenerative intraspinal cyst of the cervical spine. Case report and literature review. Spine, E18-E22.

Signoret F, Feron JM, Bonfait H, Patel A (1986). Fractured odontoid with fractured superior articular process of the axis. Journal of Bone and Joint Surgery. 68B: 182–184.

Silbert PL, Makri B, Schievink WI (1995). Headache and neck pain in spontaneous internal carotid and vertebral artery dissections. Neurology, 45: 1517–1522.

Skargren EI, Oberg BE, Carlsson PG, Gade M (1997). Cost and effectiveness analysis of chiropractic and physiotherapy treatment for low back and neck pain. Spine, 22: 2167–2177.

Sloop PR, Smith DS, Goldenberg E, Dore C (1982). Manipulation for chronic neck pain: a double-blind controlled study. Spine, 7: 532–535.

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 LA, Oldman AD, McQuay HJ, Moore RA (2000). Teasing apart quality and validity in systematic reviews: an example from acupuncture trials in chronic neck and back pain. Pain, 86: 119–132.

Snow CJ, Aves Wood R, Dowhopouuk H, Howdle C, Decker G, Elfenbaum E, Wong C, Miles-Tapping (1992). Randomized controlled clinical trial of spray and stretch for relief of back and neck myofascial pain. (Abstract). Physiotherapy Canada, 44: 2 S8.

Spitzer WO, Skovron ML, Salmi LR, Cassidy JD, Duranceau J, Suissa S, Zeiss E (1995). Scientific monograph of the Quebec Task Force on whiplash-associated disorders: redefining “whiplash” and its management. Spine, 20(suppl. 8): 1–73.

Squires B, Gargan MF, Bannister GC (1996). Soft tissue injuries of the cervical spine: 15-year follow-up. Journal of Bone and Joint Surgery, 78B: 955–957.

Sterling M, Jull G, Wright A (2001). Cervical mobilisation: concurrent effects on pain, sympathetic nervous system activity and motor activity. Manual Therapy, 6: 72–81.

Sterner Y, Lofgren M, Nyberg V, Karlsson AK, Bergstrom M, Gerdle B (2001). Early interdiscipilinary rehabilitation program for whiplash associated disorders. Disability and Rehabilitation, 23: 422–429.

Stiell IG, Wells GA, Vandemheen KL, Clement CM, Lesiuk H, De Maio VJ, Laupacis A, Schull M, McKnight RD, Verbeek R, Brison R, Cass D, Dreyer J, Eisenhauer MA, Greenberg GH, MacPahil I, Morrison L, Readon M, Worthington JW (2001). The Canadian C-spine rule for radiography in alert and stable

trauma patients. Journal of the American Medical Association, 286: 1841–1848.

Stolk-Hornsveld F, Gijsberts TJ, Duquet W, Stoekart R, Oostendorp R (1999). Pain-provocation tests for C0-C4 as a tool in the diagnosis of cervicogenic headache. Cephalalgia, 19: 436.

Streitweiser DR, Knopp R, Wales LR, Williams JL, Tonnemacher K (1983). Accuracy of standard radiographic views in detecting cervical spine fractures. Annals of Emergency Medicine, 12: 538–542.

Stroobants J, Fidler L, Storms JL, Klaes R, Dua G, van Hoye M (1994). High cervical pain and impairment of skull mobility as the only symptoms of an occipital condyle fracture. Journal of Neurosurgery, 81: 137–138.

Sturzenegger M (1994). Headache and neck pain: the warning symptoms of vertebral artery dissection. Headache, 34: 187–193.

Suissa S, Harder S, Veilleux M (2001). The relation between initial symptoms and signs and the prognosis of whiplash. European Spine Journal, 10: 44–49.

Suttner NJ, Chandy KS, Kellerman AJ (2002). Osteoid osteomas of the body of the cervical spine. Case report and review of the literature. British Journal of Neurosurgery, 16: 69–71.

Swartzman LC, Teasell RW, Shapiro AP, McDermid AJ (1996). The effect of litigation status on adjustment to whiplash injury. Spine, 21: 53–58.

Taimela S, Takala EP, Asklof T, Seppala K, Parvianen S (2000). Active treatment of chronic neck pain: a prospective randomized intervention. Spine, 25: 1021–1027.

Takala EP, Viikari-Juntura E, Tynklynen EM (1994). Does group gymnastics at the workplace help in neck pain? Scandinavian Journal of Rehabilitation Medicine, 26: 17–20.

Takano Y, Homma T, Okumura H, Takahashi HE (1992). Ganglion cyst occurring in the ligamentum flavum of the cervical spine. Case report. Spine, 17: 1531–1533.

Teasell RW, Shapiro AP (1998). Whiplash injuries: an update. Pain Research and Management, 3: 81–90.

Tehranzadeh J, Bonk T, Ansari A, Mesgarzadeh M (1994). Efficacy of limited CT for nonvisualized lower cervical spine in patients with blunt trauma. Skeletal Radiology, 23: 349–352.

Teresi LM, Lufkin RB, Reicher MA, Moffit BJ, Vinuela FV, Wilson GM, Bentson JR, Hanafee WN (1987). Asymptomatic degenerative disk disease and spondylosis of the cervical spine: MR imaging. Radiology, 164: 83–88.

Thambyrajah K (1972). Fracture of the cervical spine with minimal or no symptoms. Medical Journal of Malaya, 26: 244–249.

Travell JG, Simons DG (1993). Myofascial Pain and Dysfunction. The Trigger Point Manual. Williams and Wilkins: Baltimore. pp 312.

Trinkoff AM, Lipscomb JA, Geiger-Brown J, Brady B (2002). Musculoskeletal problems of the neck, shoulder, and back and functional consequences in nurses. American Journal of Industrial Medicine, 41: 170–178.

Tulyapronchote R, Selhorst JB, Malkoff MD, Gomez CR (1994). Delayed sequelae of vertebral artery dissection and occult cervical fractures. Neurology, 44: 1397–1399.

van der Donk J, Schouten JSAG, Passchier J, van Romunde LKJ, Valkenburg HA (1991). The associations of neck pain with radiological abnormalities of the cervical spine and personality traits in a general population. Journal of Rheumatology, 18: 1884–1889.

van der Heijden GJ, Beurskens AJ, Koes BW, Assendelft WJ, De Vet HC, Bouter LM (1995). The efficacy of traction for back and neck pain: a systematic, blinded review of randomized clinical trial methods. Physical Therapy, 75: 93–104.

Vandemark RM (1990). Radiology of the cervical spine in trauma patients: practice pitfalls and recommendations for improving efficiency and communication. American Journal of Roentgenology, 155: 465–472.

van Holsbeek EMA, Mackay NNS (1989). Diagnosis of acute atlanto-axial rotary fixation. Journal of Bone and Joint Surgery, 71B: 90–91.

van Tulder MW, Koes BW, Bouter LM (1997). Conservative treatment of acute and chronic non-specific low back pain: a systematic review of randomised controlled trials of the most common interventions. Spine, 22: 2128–2156.

Vasseljen O, Westgaard RH, Larsen S (1995). A case-control study of psychological and psychosocial risk factors for shoulder and neck pain at the workplace. International Archives of Occupational and Environmental Health, 66: 375–382.

Velmahos GC, Theodorou D, Tatevossian R, Belzberg H, Cornwell EE, Berne TV, Asensio JA, Demetriades D (1996). Radiographic cervical spine evaluation in the alert asymptomatic blunt trauma victim: much ado about nothing? Journal of Trauma, 40: 768–774.

Verhagen AP, Peeters GGM, de Bie RA, Oostendorp RAB (2002). Conservative treatment for whiplash. In: The Cochrane Library; Issue 2. Oxford: Update Software.

Verner EF, Musher DM (1985). Spinal epidural abscess. Medical Clinics of North America, 69: 375–384.

Vernon HT, Aker P, Burns S, Viljakaanen S, Short L (1990). Pressure pain threshold evaluation of the effect of spinal manipulation in the treatment of chronic neck pain: a pilot study. Journal of Manipulative and Physiological Therapeutics, 13: 13–6.

Viikari-Juntura E (1987). Interexaminer reliability of observations in physical examinations of the neck. Physical Therapy, 67: 1526–1532.

Vilke GM, Honingford EA (1996). Cervical spine epidural abscess in a patient with no predisposing risk factors. Annals of Emergency Medicine, 27: 777–780.

Virani SN, Ferrari R, Russell AS (2001). Physician resistance to the late whiplash syndrome. Journal of Rheumatology, 28: 2096–2099.

Voyvodic F, Dolinis J, Moore VM, Ryan GA, Slavotinek JP, Whyte AM, Hoile RD, Taylor GW (1997). MRI of car occupants with whiplash injury. Neuroradiology, 39: 25–40.

Waldman SD (1991). Cervical epidural abscess after cervical epidural nerve block with steroids. Anesthesia and Analgesia, 72: 717–718.

Wales LR, Knopp RK, Morishima MS (1980). Recommendations for evaluation of the acutely injured cervical spine: a clinical radiologic algorithm. Annals of Emergency Medicine, 9: 422–428.

Walsh MJ (2000). Evaluation of clinical findings for uncomplicated mechanical neck pain. Chiropractors Journal of Australia, 30: 122–126.

Walter J, Doris PE, Shaffer MA (1984). Clinical presentation of patients with acute cervical spine injury. Annals of Emergency Medicine, 13: 512–515.

Wang JC, Hatch JD, Sandhu HS, Delamarter RB (1999). Cervical flexion and extension radiographs of acutely injured patients. Clinical Orthopaedics and Related Research, 364: 111–116.

Werneke M, Hart DL, Cook D (1999). A descriptive study of the centralisation phenomenon: a prospective analysis. Spine, 24: 676–683.

Westgard RH, Jansen T (1992). Individual and work related factors associated with symptoms of musculoskeletal complaints. II Different risk factors among sewing machine operators. British Journal of Industrial Medicine, 49: 154–162.

Wheeler AH, Goolkasian P, Gretz SS (1998). A randomized, double-blind, prospective pilot study of botulinum toxin injection for refractory, unilateral, cervicothoracic, paraspina, myofascial pain syndrome. Spine, 23: 1662–1667.

White AR, Ernst E (1999). A systematic review of randomized controlled trials of acupuncture for neck pain. Rheumatology, 38: 143–147.

Wilberger JE, Maroon JC (1990). Occult posttraumatic cervical ligamentous instability. Journal of Spinal Disorders, 3: 156–161.

Williams JM, Allegra JR (1994). Spontaneous cervical epidural haematoma. Annals of Emergency Medicine, 23: 1368–1370.

Wise JJ, Cheney R, Fischgrund J (1997). Traumatic bilateral rotatory dislocation of the atlanto-axial joints: a case report and review of the literature. Journal of Spinal Disorders, 10: 451–453.

Wolfe F, Smythe HA, Yunus MB, Bennett RM, Bombardie C, Goldberg DL, Tugwell P, Campbell SM, Abeles M, Clark P, et al. (1990). The American College of Rheumatology 1990 criteria for the classification of fibromyalgia: report of the Multicenter Criteria Committee. Arthritis and Rheumatism, 33: 160–172.

Wood TG, Colloca CJ, Matthews R (2001). A pilot randomized clinical trial on the relative effect of instrumental (MFMA) versus manual (HVLA) manipulation in the treatment of cervical spine dysfunction. Journal of Manipulative and Physiological Therapeutics, 24: 260–271.

Woodring JH, Goldstein SJ (1982). Fractures of the articular processes of the cervical spine. American Journal of Roentgenology, 139: 341–344.

Wortzman G, Dewar FP (1968). Rotatory fixation of the atlantoaxial joint: rotational atlantoaxial subluxation. Radiology, 90: 479–487.

Wosazek GE, Balzer K (1990). Strangulation of the internal carotid artery by the hypoglossal nerve. Journal of Trauma, 30: 332–335.

Yano K, Murase S, Kuroda T, Noguchi K, Tanabe Y, Yamada (1993). Cervical cord compression by the vertebral artery causing a severe cervical pain: case report. Surgical Neurology, 40: 43–46.

Yetkin Z, Osborn AG, Giles DS, Haughton VM (1985). Incovertebral and facet joint dislocations in cervical articular pillar fractures: CT evaluation. American Journal of Neuroradiology, 6: 633–637.

Youdas JW, Carey JR, Garrett TR (1991). Reliability of measurements of cervical spine range of motion — comparison of three methods. Physical Therapy, 71: 98–106.

Zylbergold RS, Piper MC (1985). Cervical spine disorders: a comparison of three types of traction. Spine, 10: 867–871.

Chapter 7

Acute Shoulder Pain

➔ *Approximately 10% of the general adult population will experience an episode of shoulder pain in their lifetime (van der Heijden et al. 1996). Pain in the shoulder is the third most commonly experienced musculoskeletal pain, exceeded only by low back and neck pain (Cailliet 1981). Shoulder pain is a common reason for care seeking as it impacts upon on a range of activities of daily living, including sleep. It is estimated that around 95% of people with shoulder pain are treated in primary care settings (van der Heijden 1999).*

Many people presenting with acute shoulder pain are likely to have conditions that will resolve spontaneously regardless of treatment. Indeed, there are reports that 50% of people with shoulder pain do not seek care at all. van der Windt et al. (1996) report that 23% of all new episodes of shoulder pain resolve fully within one month and 44% resolve within three months of onset. However, the results of studies on the natural history of shoulder pain vary considerably because of the range of definitions used to describe shoulder disorders (van der Heijden 1999).

The risk that uncomplicated shoulder pain will persist beyond the acute phase appears to be related to personality traits, coping style and occupational factors (van der Heijden 1999). van der Windt et al. (1996) note that 41% had persistent symptoms after one year. It is important to take prognostic risk factors into consideration and to intervene early to prevent progression to chronic pain.

Definition of Acute Shoulder Pain

In these guidelines, the term 'acute' is defined as pain that has been present for less than three months; it does not refer to the severity or quality of pain. Chronic pain is pain that has persisted for longer than three months (Merskey and Bogduk 1994).

There is no universal definition of shoulder pain. For the purposes of these guidelines, 'shoulder' refers to the articulations of the scapula, clavicle and humerus together with the ligaments, tendons, muscles and other soft tissues with a functional relationship to these structures.

Scope

These guidelines describe the diagnosis and treatment of acute shoulder pain of unknown or uncertain origin. The following are beyond the scope of this document but are mentioned to place conditions in perspective:

- serious conditions: infection, neoplasm, inflammatory arthropathies and fracture, rupture, instability or joint dislocation related to trauma
- neurological conditions
- hemiplegic shoulder pain (post-cerebrovascular accident)
- conditions characterised by pain referred to the shoulder
- chronic pain (e.g. due to 'frozen shoulder' or 'adhesive capsulitis')

Guideline Development Process

Evaluation of Existing Guidelines

Guidelines developed by other groups were obtained to determine whether an existing guideline could be adapted for use in the Australian context. The Philadelphia Panel Evidence-Based Clinical Practice Guidelines on Selected Rehabilitation Interventions for Shoulder Pain (Albright et al. 2001) were viewed. As they did not specifically look at acute shoulder pain, the decision was made to update and disseminate existing draft guidelines for shoulder pain developed for the National Musculoskeletal Medicine Initiative by Dr Wade King.

Updating Existing Guidelines

The update of the existing work involved a review of the evidence on acute shoulder pain conducted by a multi-disciplinary group. Group members had the opportunity to evaluate the literature forming the basis of the existing guidelines, review the interpretation of the literature, nominate additional articles to undergo the appraisal process or request that an article be re-appraised.

A systematic process was used to identify new studies on the diagnosis, prognosis and interventions for acute shoulder pain in line with current standards developed by the National Health and Medical Research Council for guideline development (NHMRC 1999a). Studies were appraised against selection criteria and those meeting the criteria for inclusion were used to update the existing text of the guidelines.

All studies assessed for this update are included in either the Table of Included Studies or the Table of Excluded Studies for Diagnosis, Prognosis and Interventions. Studies that were previously included in the guidelines are not described in these tables. Refer to Appendix E: Tables of Included and Excluded Studies.

For further detail, refer to Chapter 1: Executive Summary and Chapter 9: Process Report.

Study Selection Criteria

The chart below outlines the criteria used to identify, select and appraise new studies on acute shoulder pain.

Search Strategy

Sensitive searches were performed; electronic searches were limited to adults, humans and articles published in English in peer-reviewed journals. Where available, methodological filters were used. There were no hand searches conducted.

Literature describing the diagnosis and prognosis of acute shoulder pain was sought through an electronic database search. The search located literature published since the initial guidelines were developed (1998–2002).

The evidence for interventions for acute shoulder pain was sourced from the Cochrane Review on interventions for shoulder pain (Green et al. 2002). This material was reviewed to locate articles specifically describing the effectiveness of interventions for shoulder pain of less than three months duration. In addition, an electronic literature search was conducted spanning the time elapsed since the last update of the Cochrane Review (2001–2002).

Articles that group members felt were important to the topic that did not appear in the search results were submitted to the review process.

The following databases were searched in August 2002:

- (PubMed) Clinical Queries
- CINAHL
- EMBASE — Physical and Rehabilitation Medicine
- The Cochrane Library, 2002, Issue 2

Access to CHIROLARS and PEDro was unavailable for this update.

Study Selection Criteria

DIAGNOSIS	
The sections on Aetiology and Prevalence, History, Examination and Investigations comprise information from the existing draft (developed by conventional literature review) combined and updated with relevant articles located and appraised according to the following inclusion and exclusion criteria:	
Inclusion criteria	Systematic reviews, cross-sectional studies All ages
Exclusion criteria	Chronic pain Specific diseases and conditions, fracture/disclocation, neoplasm, infection, inflammatory arthropathies, pain referred from/to the shoulder, intrinsic neurological conditions, hemiplegic shoulder pain
PROGNOSIS	
Information from the existing draft was combined with relevant articles located and appraised according to the following inclusion and exclusion criteria:	
Inclusion criteria	Systematic reviews, cohort studies No age specification
Exclusion criteria	Chronic pain Specific diseases and conditions, fracture/disclocation, neoplasm, infection, inflammatory arthropathies, pain referred from/to the shoulder, intrinsic neurological conditions, hemiplegic shoulder pain
INTERVENTIONS	
A systematic review of the literature was undertaken according to the following inclusion and exclusion criteria. In cases where no evidence was available on interventions specifically for acute shoulder pain, studies containing mixed populations (acute and chronic shoulder pain) were considered in the review:	
Inclusion criteria	Systematic reviews, randomised controlled trials (RCTs) No age specification
Exclusion criteria	Chronic pain (mixed acute and chronic populations were included if there were no data specifically on interventions for acute shoulder pain) Specific diseases and conditions, fracture/disclocation, neoplasm, infection, inflammatory arthropathies, pain referred from/to the shoulder, intrinsic neurological conditions, hemiplegic shoulder pain

Search Terms

- Shoulder pain .exp
- Arm injury .exp
- Analgesics
- Anti-inflammatory drugs
- NSAID syndrome .exp
- Shoulder girdle .exp
- Shoulder radiography .exp
- Shock wave therapy .tw
- Orthopaedic surgery .tw
- Physiotherapy .exp
- Mobilisat*
- Manual .tw
- LLLT .tw
- Shortwave .tw
- Acute .tw
- Acupuncture
- Cervicobrachial neuralgia .exp
- N.S.A.I.D.S.
- Shoulder impingement
- Shoulder dislocation .exp
- Extra corporeal shock wave therapy .tw
- Non steroidal anti-inflammatory .exp
- Surgery .exp
- Exercise .exp
- Manipulat*
- Ultrasound .tw
- Laser .tw
- TENS .tw
- Injection .exp
- Tendinitis .exp
- Tendonitis .tw
- Glenohumeral .tw
- Diagnosis .exp
- Systematic review .tw
- Controlled trial .tw
- Clinical trial .tw
- Etiology .exp
- Adhesive capsulitis .tw
- Rotator cuff .exp
- Frozen shoulder .exp
- Therapies .exp
- Prognosis .exp
- Pain assessment .tw
- Randomised .tw
- Drug therapy .exp

Research Agenda for Acute Shoulder Pain

- Consistent terminology needs to be established.
- A prognostic model for shoulder disorders needs to be developed.
- Standard outcome measures need to be developed.
- Well-designed studies are required to research the effectiveness of interventions (e.g. analgesics).
- Studies are needed on the cost-effectiveness of interventions and other aspects of care.

Summary of Key Messages: Acute Pain Management

	EVIDENCE LEVEL
Management Plan	
It is recommended that the clinician and patient develop a management plan for acute musculoskeletal pain comprising the elements of assessment, management and review: • Assessment — Conduct a history and physical examination to assess for the presence of serious conditions; ancillary investigations are not generally indicated unless features of serious conditions are identified. • Management — Provide information, assurance and advice to resume normal activity and discuss other options for pain management as needed. • Review — Reassess the pain and revise the management plan as required.	CONSENSUS: Steering Committee
Non-Pharmacological Interventions	
Simple interventions (providing information, assurance and encouraging reasonable maintenance of activity) may be used alone or in combination with other interventions for the successful management of acute musculoskeletal pain.	CONSENSUS: Steering Committee
Pharmacological Interventions	
Specific pharmacological interventions may be required to relieve pain; such agents can be used in conjunction with non-pharmacological interventions.	CONSENSUS: Steering Committee; NHMRC 1999b
Paracetamol or other simple analgesics, administered regularly, are recommended for relief of mild to moderate acute musculoskeletal pain.	CONSENSUS: Steering Committee; NHMRC 1999b
Where paracetamol is insufficient for pain relief, a non-steroidal anti-inflammatory (NSAID) medication may be used, unless contraindicated.	CONSENSUS: Steering Committee; NHMRC 1999b
Oral opioids may be necessary to relieve severe musculoskeletal pain. It is preferable to administer a short-acting agent at regular intervals, rather than on a pain-contingent basis. Ongoing need for opioid analgesia is an indication for reassessment.	CONSENSUS: Steering Committee; NHMRC 1999b
Adjuvant agents such as anticonvulsants and antidepressants are not recommended in the management of acute musculoskeletal pain.	CONSENSUS: Steering Committee; NHMRC 1999b
Any benefits from muscle relaxants may be outweighed by their adverse effects, therefore they cannot be routinely recommended.	CONSENSUS: Steering Committee

Summary of Key Messages: Effective Communication

	EVIDENCE LEVEL
Clinicians should work with patients to develop a management plan so that patients know what to expect, and understand their role and responsibilities.	CONSENSUS: Steering Committee
Information should be conveyed in correct but neutral terms, avoiding alarming diagnostic labels; jargon should be avoided.	CONSENSUS: Steering Committee
Explanation is important to overcome inappropriate expectations, fears or mistaken beliefs that patients may have about their condition or its management.	CONSENSUS: Steering Committee
Printed materials and models may be useful for communicating concepts.	CONSENSUS: Steering Committee
Clinicians should adapt their method of communication to meet the needs and abilities of each patient.	CONSENSUS: Steering Committee
Clinicians should check that information that has been provided has been understood; barriers to understanding should be explored and addressed.	CONSENSUS: Steering Committee

Summary of Key Messages: Acute Shoulder Pain

DIAGNOSIS	EVIDENCE LEVEL
Aetiology and Prevalence	
Clinicians should be alert to the potential for rare, serious conditions (e.g. fracture/dislocation, tumour, infection, inflammatory arthropathies) presenting as acute shoulder pain.	*LEVEL IV: numerous case studies (Jones et al. 1994; Kaempffe 1995; Barlow and Newman 1994; Welch 1994; Linos et al. 1980)
Most cases of acute shoulder pain are of 'mechanical' origin and can be managed as acute regional pain.	*LEVEL III-2, III-3: Torstensen and Hollinshead 1999; Chandnani et al. 1992; Milgrom et al. 1995; Sher et al. 1995
Biological factors such as age, female gender, past history and response to repetitive physical tasks may contribute to the development of acute shoulder pain.	*LEVEL III-3: Jones et al. 1994; Cummings et al. 1995; Sambrook 1996; Ekberg et al. 1995; Skov et al. 1996
Psychosocial factors such as job dissatisfaction and work demands may contribute to the onset of acute shoulder pain.	*LEVEL III-2: Bergenudd et al. 1994; Ekberg et al. 1995; Marcus et al. 1996; Skov et al. 1996
History	
Information obtained from the history may alert to the presence of a serious condition as the underlying cause of acute shoulder pain.	CONSENSUS: Steering Committee
The reliability and validity of individual features in histories have low diagnostic significance; the history is to be interpreted with caution when choosing a course of action.	*LEVEL III-2: Nørregaard et al. 2002; Litaker et al. 2000
Physical Examination	
Findings of shoulder examination must be interpreted cautiously in light of the evidence of limited utility; no clinical test is both reliable and valid for any specific diagnostic entity.	*LEVEL III-2: Calis et al. 2000; MacDonald et al. 2000; Naredo et al. 2002; Itoi et al. 1999; Bennett 1998
Causes of acute shoulder pain cannot be diagnosed by clinical assessment; however, with the exception of serious conditions, satisfactory outcomes do not depend on precise identification of cause.	*LEVEL III-2: Bamji et al. 1996; Liesdeck et al. 1997; de Winter et al. 1999; Pal et al. 2000; Nørregaard et al. 2002
Despite limitations, physical examination is an opportunity to identify features of potentially serious conditions.	CONSENSUS: Steering Committee

Acute Shoulder Pain continued

Ancillary Investigations	
Imaging is not necessary unless there are alerting features of serious conditions; in the absence of alerting features, the diagnostic utility of imaging is minimal and the results are unlikely to improve management.	*LEVEL III: Numerous studies (Torstensen and Hollinshead 1999; Teefey et al. 2000a,b; Tempelhof et al. 1999; Milgrom et al. 1995; Chandnani et al. 1992; Sher et al. 1995; Sher et al. 1998; Blanchard et al. 1999a)
There is a need to educate consumers about the limitations of imaging and the risks of radiation exposure.	*LEVEL IV: Roebuck 1995
Terminology	
Terms to describe acute shoulder pain should summarise the discernible features of the condition to form the basis for a management plan.	CONSENSUS: World Health Organisation 1986; Merskey and Bogduk 1994
PROGNOSIS	**EVIDENCE LEVEL**
Approximately 50% of people with acute shoulder pain (treated conservatively) recover within six months; approximately 60% recover within 12 months.	*LEVEL III-2: Van der Windt et al. 1996; Winters et al. 1997b
Shoulder pain may recur even in those who appear to fully recover in the short term.	*LEVEL III-2: Croft et al. 1996
INTERVENTIONS	**EVIDENCE LEVEL**
Evidence of Benefit	
Corticosteroid Injection — Subacromical corticosteroid injection for acute shoulder pain may improve pain at four weeks compared to placebo but this benefit is not maintained at 12 weeks.	LEVEL I: Systematic review of RCTs of adults with acute shoulder pain (Adebajo et al. 1990, Vecchio et al. 1993); systematic review of steroid injections for shoulder pain (Buchbinder et al. 2002)
Exercises — Exercises may improve shoulder pain compared to placebo in people with rotator cuff disease in both the short and longer term.	LEVEL I: Systematic review of two RCTs (Ginn et al. 1997; Brox et al. 1997)
Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) — Topical and oral NSAIDs improve acute shoulder pain by a small to moderate degree for up to four weeks compared to placebo. Serious adverse effects of NSAIDs include gastrointestinal complications (e.g. bleeding, perforation)	LEVEL I: Systematic review of three RCTs of adults with acute shoulder pain (Ginsberg and Famaey 1991; Mena et al. 1986; Adebajo et al. 1990) LEVEL I: Based on a systematic review (Bigos et al. 1994)
Ultrasound — Therapeutic ultrasound may provide short-term pain relief in calcific tendonitis compared to placebo.	LEVEL I: Systematic review of one RCT in acute shoulder pain (Ebenbichler et al. 1999)
Conflicting Evidence	
Acupuncture — There is conflicting evidence of the effectiveness of acupuncture compared to placebo ultrasound for shoulder pain and function.	LEVEL I: Systematic review (Green et al. 2003) of two RCTs (Kleinhenz et al. 1999; Berry et al. 1980)
Insufficient Evidence	
Analgesics — There are no randomised controlled trials investigating the use of analgesics (paracetamol or compound analgesics) for acute or chronic shoulder pain.	No Level I or II evidence
Extracorporeal Shock Wave Treatment (ESWT) — There are no randomised controlled trials of Extracorporeal Shock Wave Treatment for acute shoulder pain. Trials conducted in populations with chronic shoulder pain show conflicting results for ESWT compared with placebo.	No Level I or II evidence LEVEL I: Buchbinder et al. 2003a (systematic review of four RCTs)

Acute Shoulder Pain continued

Manual Therapy — Shoulder joint mobilisation with combined treatments (hot packs, active exercise, stretching, soft tissue mobilisation and education) may improve acute shoulder pain in the short term compared to the combined treatments alone.	LEVEL I: Systematic review located one RCT of 14 patients (Conroy and Hayes 1998)
Oral Corticosteroids — There are no randomised controlled trials investigating the use of oral corticosteroids for acute shoulder pain. Studies of mixed populations do not report significant benefit from oral corticosteroids compared with placebo or no treatment for adhesive capsulitis.	No Level I or II evidence LEVEL I: Green et al. 1998 (systematic review of two RCTs with methodological limitations)
Suprascapular Nerve Blocks — There are no published studies investigating the value of suprascapular nerve blocks for acute shoulder pain. There is some evidence of short-term effect from suprascapular nerve blocks for chronic adhesive capsulitis and rotator cuff disease.	No Level I or II evidence LEVEL I: Buchbinder et al. 2003b (systematic review of three RCTs)
Surgery — There are no published randomised controlled trials investigating the effectiveness of surgery for acute shoulder pain although studies exist for chronic populations.	No Level I or II evidence
Transcutaneous Electrical Nerve Stimulation (TENS) — There is insufficient evidence for the use of TENS for acute shoulder pain.	LEVEL I: Systematic review of on RCT (Shehab and Adham 2000)

Note: * Indicative only. A higher rating of the level of evidence might apply (refer to the note in Chapter 1: Executive Summary, Limitations of Findings).

DIAGNOSIS

>Aetiology and Prevalence

Acute shoulder pain has many possible sources, including all diseases, injuries and other impairments that invoke nociceptive mechanisms in the region. The following information is provided as a means to familiarise clinicians with some of the possible causes of acute shoulder pain; it is not intended as a checklist of conditions. Attempts to diagnose the cause of acute shoulder pain by systematically eliminating the possible causes are likely to be confounded by the unreliability of clinical methods and the variability in the understanding and description of clinical entities (see Table 7.1).

With the exception of conditions posing a serious threat to health, identification of a specific cause is not a precondition for effective management of acute pain. Potential causes of acute shoulder pain may be classified as:

- painful conditions of the shoulder
- conditions referring pain to the shoulder

Painful Conditions of the Shoulder

Local impairments of anatomical structures of the shoulder comprise the vast majority of causes of acute shoulder pain. They in turn may be classified, broadly, as:

- serious conditions
- intrinsic neurological conditions
- mechanical conditions

The first two types are uncommon but the conditions they encompass must not be overlooked in the assessment process as a missed diagnosis may have serious consequences.

Serious Conditions

Serious conditions manifesting as shoulder pain pose more serious health risks than common 'mechanical' disorders of local structures. The best response to the danger of serious conditions is vigilance. Appropriate vigilance depends on knowledge of the conditions and the potential for their existence, the extent of the threat they pose to health and the features that provide clues to their presence. When necessary, ancillary investigations can be used astutely. Alerting features of serious conditions are summarised in Table 7.11. Management of serious conditions is outside the scope of these guidelines.

Fractures and Dislocations

Major trauma is the common cause of fracture in otherwise healthy people. Healthy bones resist large forces and break only if subjected to severe, deforming stresses. Resultant injuries include disruption of the shaft, avulsion of the greater tuberosity and more subtle lesions such as Hill-Sachs compression fracture of the humeral head. Dislocation involves major forces with vectors that damage the soft tissue restraints of a joint rather than the bones, causing injuries such as anterior detachment of the glenoid labrum (known as the Bankart lesion) or superior labrum anterior and posterior (SLAP) lesion (Andrews et al. 1985).

Minor trauma does not cause fracture unless there is a predisposing condition of bone. Osteoporosis is the most common such condition. It affects most elderly women and many elderly men in Australia. A large study (Jones et al. 1994) showed that 56% of women and 29% of men over 60 years of age suffer osteoporotic fractures; 11% involve the humerus.

Osteomalacia is another disorder of bone metabolism leading to poor bone mineralisation, osteopaenia and tendency to fracture. It results from inadequacy of Vitamin D activity as a result of dietary deficiency, inadequate exposure to sunshine, intestinal malabsorption, renal tubular disorders, anticonvulsant medication or inherited metabolic disorders.

Paget's disease of bone (or osteitis deformans) is an uncommon condition in which increases of osteoclastic and osteoblastic activity cause thickening, weakening and deformity of affected bones. The shoulder is seldom involved. Paget's disease is usually painless but may cause low-grade pain. Occasionally it is associated with pathological fractures.

Other medical conditions in which bones are prone to fracture after minor trauma are rare. One is osteogenesis imperfecta, a hereditary disorder of collagen synthesis causing

Table 7.1

A Guide to Described Causes of Acute Shoulder Pain

Painful Conditions of the Shoulder	
Serious conditions	Fracture, dislocation, rupture and instability; tumours; infection (septic arthritis, penetrating injury); inflammatory arthropathies
Intrinsic neurological conditions	Peripheral neuropathies (suprascapular, axillary and musculocutaneous nerve impairment) (Bonnici and Welsh 1993; Biundo et al. 1995);
	Brachial plexus injuries (Travlos et al. 1990);
	Complex regional pain syndromes (types I and II) (Veldman and Goris 1995).
Mechanical conditions involving patho-anatomical entities	Sprain, subluxation or dislocation of articulations (glenohumeral joint acromioclavicular joint, sternoclavicular joint)
	Tear, contracture of joint capsules (glenohumeral joint, acromioclavicular joint, sternoclavicular joint)
	Effusion of bursae (subacromial bursa, others)
	Sprain, tear of ligaments (glenohumeral ligaments, acromioclavicular ligaments, sternoclavicular ligaments)
	Sprain, tear of muscles and tendons (supraspinatus, infraspinatus, teres minor, subscapularis, deltoid, others)
Conditions Referring Pain to the Shoulder	
Extrinsic neurological conditions	Central pain syndromes; nerve root syndromes; peripheral nerve irritation
Somatic conditions	Cervical zygapophyseal joint impairment (especially at the C5–6 and C6–7 spinal levels); cervical intervertebral disc impairment (especially at the C5–6 and C6–7 spinal levels); cervical muscle impairment
Visceral conditions	Pericardial irritation; pleural irritation; diaphragmatic peritoneal irritation; liver and gall bladder disease; vascular conditions (myocardial ischaemic pain, variant angina pectoris, aortic aneurysm, thoracic outlet syndrome) (Brown 1983)

brittle bones and lax ligaments; about two-thirds of those affected have blue sclerae and about half have crumbling teeth (dentinogenesis imperfecta).

Pathological fractures associated with neoplasia, Pagetic bone disease etc. may occur after minimal trauma or even without any trauma at all.

Tumours

Tumours are rare in the shoulder but they do occur. The shoulder is second only to the knee in the ranking of peripheral sites of neoplasia. The proximal humerus is the third most common long bone site of tumour formation, after the distal femur and the proximal tibia (Kaempffe 1995).

Primary bone tumours in the proximal humerus include osteoclastoma (giant cell tumour), osteogenic sarcoma, chondroblastoma and chondrosarcoma, amongst others (Barlow and Newman 1994).

Secondary malignancies in the bones of the shoulder mainly affect the proximal humerus. Their primary sites include lung, breast, prostate, kidney and thyroid (Welch 1994).

Soft tissue tumours in the shoulder include primaries such as malignant fibrous histiocytoma (in those aged 50 to 70 years), synovial chondromas (Buess and Friedrich 2001) and sarcomas (in younger people) and a variety of secondaries including local extension of an apical carcinoma of lung or 'Pancoast tumour' (Pancoast 1932).

Infections

Infection may be related to septic arthritis (Lossos et al. 1998) or a history of penetrating injury, including medical procedures.

Inflammatory Arthropathies

Inflammatory arthropathies are difficult to identify in the early stages. The inflammatory diseases that affect the shoulder include, amongst others:

- rheumatoid arthritis
- crystal arthropathies (gout, pseudo-gout)
- polymyalgia rheumatica
- psoriatic arthropathy
- reactive arthropathy associated with inflammatory bowel disease
- amyloid arthropathy

Key Message

Clinicians should be alert to the potential for rare, serious conditions (e.g. fracture/dislocation, tumour, infection, inflammatory arthropathies) presenting as acute shoulder pain. (*Level IV)

Intrinsic Neurological Conditions

Intrinsic neurological conditions are those primarily involving local neural structures of the shoulder (Bateman 1983).

Mechanical Conditions

'Mechanical' musculoskeletal disorders are characterised by altered biomechanical function. In the broadest sense, most conditions have biomechanical implications. Disorders termed 'mechanical' are those in which changes of function are the principal features. They are due to mechanical impairment either directly by injury or indirectly by internal change.

Stress applied to tissue produces a strain. If the strain force exceeds the tissue's load-bearing capacity, mechanical injury (sprain or tear) results. Less unaffected tissue is then available for load bearing and it has greater stresses imposed on it by subsequent applications of force. Mechanical transduction occurs when the force applied to a particular Aδ or C nerve fibre reaches its threshold for stimulation. This is the main mechanism of the pain associated with musculoskeletal injuries.

Identifying precise causes of mechanical pain is difficult. Management plans based on mistaken assumptions of cause can lead to treatment errors and iatrogenic prolongation and complication of simple conditions.

Loose terminology applied inconsistently to describe mechanical shoulder disorders further complicates the picture. The literature describes several more-or-less distinct syndromes considered 'mechanical' but the terms used to name them are unclear. The wide usage of diagnostic labels implies they have specific meanings, but traditional entities are not defined in exclusive terms. There is overlap between 'frozen shoulder', 'periarthritis' and 'capsulitis', and between 'rotator cuff lesion', 'supraspinatus tendonitis', 'subacromial bursitis' and 'impingement syndrome'. There is potential for confusion between all these supposedly distinct conditions. The difficulties of identifying and naming conditions associated with acute shoulder pain are acknowledged, and a rational taxonomy is suggested in 'Terminology'.

It may be useful to consider the array of terms and concepts by considering mechanical entities from two perspectives:

- conditions recognised by tradition
- patho-anatomical entities

Key Message

Most cases of acute shoulder pain are of 'mechanical' origin and can be managed as acute regional pain. (*Level III-2, III-3)

Mechanical Conditions Recognised by Tradition

Minor Sprains

Sprains of ligaments, tendons and muscles account for the vast majority of acute shoulder pain. Sprain of a muscle and its tendon usually affects the myotendonous junction, which is the weakest part of the structure when loaded to cause longitudinal stretch. Minor sprains usually heal spontaneously over a period of days unless perpetuating factors are at work. The evidence shows that a defect in the collagenous structure of a sprained tendon will be filled with fibroblasts producing new collagen within three days of injury and will regain its normal strength within a matter of weeks (Lundborg and Rank 1978; Manske et al. 1984).

Impingement Syndrome

The impingement syndrome, as it was described originally by Neer (1972) and corroborated later by Hawkins and Kennedy (1980), is defined as pain on active shoulder flexion (forward elevation of the arm) above horizontal that is relieved by injection of local anaesthetic into the subacromial space. The rationale is that as the greater tuberosity of the humeral head and the acromion move closer together in flexion they impinge on tissues in the subacromial space.

The term 'impingement syndrome' is also applied loosely to other conditions in which there is pain on movement, such as pain on combined active internal rotation and abduction beyond the horizontal.

Impingement syndrome is usually attributed to subacromial bursitis or rotator cuff lesions (Neer 1983; Limb and Collier 2000).

Subacromial Bursitis

Inflammation of the subacromial bursa is associated with the development of an effusion that causes the bursa to swell (Neer 1983a; Gotoh et al. 2001; Szomor et al. 2001). The swollen structure tends to become entrapped and compressed between the humeral head and the acromion as they move closer together in shoulder flexion, internal rotation and abduction. Such impingement on an already tense structure may precipitate or aggravate the pain. It may be relieved by movement to a position in which the humeral head and the acromion are further apart, such as in external rotation.

Rotator Cuff Lesions

The rotator cuff tendons may be torn by sudden overloading in a traumatic event or frayed by rubbing against the acromion over time. The injury invokes an inflammatory response that causes the tendon to swell and become painful (Neer et al. 1983b; Ozaki et al. 1988; Ogata and Uhthoff 1990; Hijioka et al. 1993). The swollen structure may also be trapped between the humeral head and the acromion, causing the impingement syndrome.

Supraspinatus Tendonosis

The supraspinatus tendon, in particular, is thought to become torn or frayed in the manner outlined above (Codman and Akerson 1931). The more specific term implies that the structure primarily involved in the mechanism of the painful condition can be identified specifically. If indeed that tendon is the primary site of pathology, the term 'supraspinatus tendonosis' is more appropriate than the traditional 'tendonitis', as it carries less presumption of the pathogenesis.

Instability

The glenohumeral joint is stabilised by the glenoid labrum, the joint capsule and the ligaments and tendons that insert into it. If one of these structures is impaired (e.g. by dislocation) and the damage does not resolve, the joint will be unstable in the direction in which its restraints are inadequate (Protzman et al. 1980; Rowe and Zarins 1981; Matsen et al. 1990).

Clinical instability of the shoulder is manifest as recurrent pain and 'giving way' or 'locking' after particular movements such as reaching upwards and outwards or overhead throwing. Episodes are sometimes accompanied by numbness, tingling and weakness, the so-called 'dead arm syndrome'. Active movements are restricted because of a reluctance to move into positions that precipitate symptoms. Abduction and external rotation are most commonly affected, especially in combination, but instability can occur in any direction.

Frozen Shoulder

The term 'frozen shoulder' (and 'adhesive capsulitis') is commonly employed to describe a condition characterised by pain and stiffness. As this condition is by nature chronic, it is not specifically addressed in these guidelines. The following notes place the condition into perspective.

Classic frozen shoulder manifests as pain and stiffness of gradual onset over weeks or months. The condition is usually unilateral and more often affects the non-dominant side. Active and passive movements are restricted progressively in the onset or 'freezing' phase. Often the range most affected is external rotation, with abduction next most restricted, then internal rotation. The pain and stiffness tend to persist for a period of months (the so-called 'frozen phase') before gradually wearing off in the 'thawing phase' (Lundberg 1969; Baslund et al. 1990). The whole process usually takes from one to two years (Reeves 1975; Grey 1978) and recovery is generally substantial (Binder et al. 1984a), although many people have persistent problems (Shaffer et al. 1992) of a relatively minor nature.

The problem was described by Duplay (1872) as 'péri-arthritie scapulo-humerale' or in English 'scapulo-humeral periarthritis'. Codman used the name 'frozen shoulder' in his authoritative textbook published in 1934. Over the years others have used the term loosely to describe combinations of pain and stiffness that do not match the classic syndrome at all (Nevasier and Nevasier 1987), and some have applied it to any shoulder condition involving both symptoms. Thus in general usage the label 'frozen shoulder' is nebulous. Quigley (1963) described the term 'frozen shoulder' as having only the 'dubious respectability of long usage, and … no greater precision than 'surgical belly' or 'back strain'.

Patho-Anatomical Entities

Reference to an anatomical matrix provides a means to classify mechanical problems of the shoulder.

Articulations

Impairments of the joints of the shoulder include:

- glenohumeral joint sprain, subluxation and dislocation (including Bankart, Hill-Sachs and superior labrum anterior and posterior (SLAP) lesions)
- acromioclavicular joint sprain, subluxation and dislocation
- sternoclavicular joint sprain, subluxation and dislocation

Joint Capsules

The capsules of the same joints may be partially torn or completely disrupted. The glenohumeral joint in particular may become contracted; thus:

- glenohumeral capsular tear, disruption and contracture
- acromioclavicular capsular tear and disruption
- sternoclavicular capsular tear and disruption

Bursae

Any of the bursae of the shoulder may become injured, resulting in effusion:

- subacromial bursal effusion is the most common
- other bursal effusions should also be considered

Ligaments

Any ligament of the shoulder may be partially torn or completely disrupted:

- glenohumeral ligamentous tears and disruptions
- acromioclavicular ligamentous tears and disruptions
- sternoclavicular ligamentous tears and disruptions

Muscles and Tendons

Any muscle attached to the shoulder may become compromised mechanically by a single large force or series of repeated insults. The most common injuries are simple sprains and tears, which typically occur at myotendonous junctions. Taking the rotator cuff group of muscles as examples, the classification of entities would be:

- supraspinatus sprains and tears
- infraspinatus sprains and tears
- teres minor sprains and tears
- subscapularis sprains and tears
- other muscle and tendon sprains and tears

Conditions Referring Pain to the Shoulder

Three groups of conditions refer pain to the shoulder:

- extrinsic neurological conditions
- somatic conditions
- visceral conditions

The mechanism of pain referral to the shoulder is convergence in the nervous system. Thus sources of shoulder pain include neural structures of both peripheral and central nervous systems that receive sensory fibres from the shoulder and any somatic or visceral structure with sensory innervation converging with that of the shoulder in the afferent sensory pathways. Patterns of shoulder pain that arise in this way vary from localised to diffuse.

Extrinsic Neurological Conditions

Neurological disorders are classified as intrinsic and extrinsic. Extrinsic conditions are those that arise at sites outside the shoulder but refer pain to it (Bateman 1983; Campbell and Koris 1995).

Conditions that irritate any of the peripheral nerves supplying the shoulder are also capable of causing shoulder pain (Brown 1983; Biundo et al. 1995), for example cervical lymphadenopathy and Pancoast tumour (Pancoast 1932).

Intrinsic neurological conditions are considered later in this chapter.

Somatic Conditions

Pain is referred to the shoulder from other somatic structures. The sources of such somatic referred pain include anatomical structures whose sensory afferent neural pathways converge with those of the sensory nerves of the shoulder in the central nervous system.

Patterns of pain referral are described in detail for the cervical zygapophyseal joints (Dwyer et al. 1990; Aprill et al. 1990; Fukui et al. 1996) but less precisely for the cervical intervertebral discs (Friedenberg and Miller 1963) and the muscles of the neck (Bogduk and Simons 1993).

Visceral Conditions

Pain may be referred to the shoulder by visceral disease processes. In particular, diseases of tissues innervated by the phrenic nerve (which forms part of the fourth and to lesser extents the third and fifth, cervical nerves) are associated with shoulder pain (Cousins 1987).

Prevalence of Conditions Causing Acute Shoulder Pain

The prevalence of some conditions causing acute shoulder pain has been established; serious (i.e. threatening) conditions are rare (see Table 7.2).

Sprains

Sprains of tendons and muscles are probably the most common reason for acute shoulder pain. Their prevalence is unknown because many are so minor as not to require professional care and are not recorded.

Rotator Cuff Tears

Tears of the rotator cuff tendons have been shown by post mortem pathological studies (Welfling et al. 1964; Rothman and Parke 1965) to occur in adults beyond the third decade of life in direct proportion to age. The finding has been borne out by imaging and arthroscopic studies that have shown rotator cuffs tears to be common in both symptomatic (Torstensen and Hollinshead 1999) and asymptomatic individuals (Chandnani et al. 1992; Milgrom et al. 1995; Sher et al. 1995). Tendon tears can occur at any age but are so common

especially in older age groups as to raise serious doubts about the clinical significance of finding a cuff tear.

Impingement Syndrome

Impingement syndrome proved to have prevalences of 4.3% of men and 9.3% of women in a large survey in Sweden (Jacobsson et al. 1989).

Fractures

Osteoporotic fractures of the humerus are uncommon overall but do occur in older people. More than half (56%) of women and one third (29%) of men over 60 years of age in Australia have osteoporotic fractures. The humerus is the site of 11% of these fractures. The prevalence of osteoporotic fracture in women over 60 years is 6% and the prevalence of osteoporotic fracture in men over age 60 is 3% (Jones et al. 1994).

Tumours

Shoulder pain caused by cancer is comparatively rare. Precise figures for prevalence have not been determined but the pre-test probability of a patient presenting with shoulder pain and having cancer as the cause is thought to be substantially less than 1%.

Primary bone tumours involved the shoulder in 7% of one reported series of 2039 cases of primary bone neoplasm; 145 tumours occurred in the shoulder, with about equal prevalence of benign and malignant lesions. Malignant tumours tended to occur in an older age group (mean age 43 years) and benign tumours in younger people (mean age 17 years). In this series, 75% of the primary lesions of the shoulder were in the proximal humerus, 20% in the scapula and 5% in the outer clavicle (Barlow and Newman 1994).

Secondary malignancies in the bones of the shoulder affect the proximal humerus most often, with about 5% to 7% of osseous metastases occuring there. Primary sites are mainly the lung, breast, prostate, kidney and thyroid (Welch 1994).

Inflammatory Arthropathies

Inflammatory arthropathies are uncommon with prevalences of less than 5% and some much less, depending on the specific condition and the age group considered. Rheumatoid arthritis is the most common with a prevalence of up to 4.7% of elderly females and 2.5% of elderly males (Linos et al. 1980).

Aetiological Risk Factors

Risk factors are features associated with the causation or perpetuation of a health problem. Their presence is correlated statistically with the chance of developing that problem or going on to suffer from it over a long period.

These correlations do not prove direct involvement in aetiology; it is likely that the factors outlined below reflect characteristics of lifestyles:

- Advanced age is a factor relevant to osteoporosis and neoplasia. Osteoporosis is uncommon below the age of 50 and its incidence increases with age after that (Jones et al. 1994).
- Female gender is associated with increased risk of osteoporosis (Cummings et al. 1995; Sambrook 1996) and with shoulder pain in general as found in two large European epidemiological studies (Ekberg et al. 1995; Skov et al. 1996).
- Past health is also relevant to both osteoporosis and neoplasia. Early menopause and endocrine disturbances are other risk factors for osteoporosis (Cummings et al. 1995; Sambrook 1996). A past history of cancer is a risk factor for developing metastatic disease.
- Sleep disturbances, smoking and caffeine consumption have all been associated with shoulder pain (in general) in large European and American epidemiological studies (Bergenudd and Nilsson 1994; Marcus and Gerr 1996; Skov et al. 1996).
- Repetitive physical tasks, whether at work or elsewhere, have been repeatedly associated with shoulder pain (Ekberg et al. 1995; English et al. 1995). Repetitive work tasks are implicated in many occupational conditions. The undertaking of an overhead task such as painting ceilings may bring on a subacromial disorder in a person unused to such activity.
- Other physical work stresses specifically associated with onset of shoulder pain in studies include work pace (Ekberg et al. 1995), long periods of driving (Skov et al. 1996) and prolonged exposure to vibration (Futatsaka et al. 1985).
- Psychosocial work stresses such as job dissatisfaction, work demands, uncertainty about performance, decreased social support in the workplace and uncertain employment prospects have all been correlated with shoulder pain in studies (Bergenudd and Nilsson 1994; Ekberg et al. 1995; Marcus and Gerr 1996; Skov et al. 1996).
- Immigrant status is another factor associated with shoulder pain (in general) in a European epidemiological survey (Ekberg et al. 1995).

Table 7.2

Prevalence Rates of Some Conditions Causing Acute Shoulder Pain

Frequency	Threatening Conditions	Non-threatening Conditions
Rare causes (< 1%)	Neoplasia (< 1%) Septic arthritis (< 0.01%)	
Uncommon causes (< 5%)	Inflammatory arthropathies (< 5% by conditions and age)	Impingement syndrome (males 4%) Frozen shoulder (2%) Osteoporotic fractures (3–6% over age 60)
Common causes		Rotator cuff tears (> 50% over age 50) Impingement syndrome (females 9%)
Unknown		Minor sprains Fractures of healthy bones

Key Messages

> Biological factors such as age, female gender, past history and response to repetitive physical tasks may contribute to the development of acute shoulder pain. (*Level III-3)

> Psychosocial factors such as job dissatisfaction and work demands may contribute to the onset of acute shoulder pain. (*Level III-2)

>History

The aim in taking a history is to assess for the presence of serious conditions that may present as acute shoulder pain. The following is a framework for collecting relevant information and identifying features ('red flags') that may alert to the presence of serious conditions. However, as there is no evidence to demonstrate that such features are reliable, valid indicators of serious conditions causing acute shoulder pain, ongoing vigilance is vital.

Pain History

Site

The site where pain is felt tends to be the anatomical reference by which the index condition is designated but it may not be the site of origin. The clinician should ask which part of the shoulder hurts most and whether the pain started there or occurred somewhere else first. If there has been pain at multiple sites, the original site should be noted and an extrinsic cause or a serious condition considered.

Distribution

Distribution provides a clue to the source of pain. For example, shoulder pain associated with abdominal pain may be visceral referred pain. A specific pattern from the sternoclavicular region up into the side of the neck has been described for sternoclavicular joint pain on the basis of provocation studies (Hassett and Barnsley 2001). Distribution of pain from other parts of the shoulder girdle can be deduced from studies of the sensory supply of shoulder components (Gardner 1948).

Quality

Somatic impairment usually causes dull, aching pain. Such pain distributed from the neck to the shoulder suggests somatic referred pain of cervical origin. Sharp, stabbing pain shooting from the neck to the shoulder and arm is likely to be radicular. Burning pain is often neuropathic. Sharp pain in the shoulder and abdomen may be visceral referred pain.

Duration

Duration may reflect type and degree of impairment. Minor sprains and tears generally heal spontaneously; they are usually of short duration. Longer-term pain may be due to more severe impairment or the effects of perpetuating factors.

Periodicity

Constant pain may be associated with conditions involving joint distension or diffuse inflammation. Intermittent pain, especially pain on movement, may be associated with injury or focal inflammation. Such relationships are not constant; caution should be exercised in drawing conclusions from particular patterns of periodicity.

Intensity

The intensity of pain should be assessed (refer to Chapter 2: Acute Pain Management). Intensity of pain is often related to shoulder movement if there is somatic impairment or other local pathology, and unrelated to activities when the pain is of extrinsic origin.

Precipitating and Aggravating Factors

Aggravating factors include biomechanical stresses that load structures beyond their physiological capacities. A study of people with shoulder pain identified lifting above shoulder height, attempting to throw overhand and sleeping on the affected side as aggravating factors common to over 85% of them (Smith et al. 2000). If pain is of extrinsic origin, precipitating and aggravating factors may be unrelated to shoulder movement or loading. Pain at rest should alert to the possibility of fracture.

Relieving Factors

If pain is due to injury or other somatic impairment, relieving factors usually reduce biomechanical stresses, e.g. avoiding particular movements and activities, or performing them in different ways. When acute shoulder pain is of extrinsic origin, any relieving factors are often unrelated to shoulder movement or loading.

Effect of Pain on Activities of Daily Living

Assessing the effect of pain on activities of daily living (ADL) allows the clinician to determine the impact of pain on the individual's lifestyle. Ongoing assessment of the impact on ADL provides a practical measure of the progress of the condition and associated disabilities.

Associated Symptoms

Symptoms associated with mechanical shoulder pain may include stiffness or limitation of shoulder movement. Unexpected weight loss, fever, night sweats or other unexplained symptoms should alert the clinician to the possibility of a serious condition.

Onset (Precipitating Event)

A history of trauma is the main feature alerting to possible fracture or dislocation. The usual history is sudden onset of shoulder pain after substantial force was applied to the region, or a history of a fall. Further alerting features are pain at rest and swelling (Fraenkel et al. 2000).

In cases of mechanical shoulder pain, the onset is usually due to an incident of trauma or to repeated biomechanical stress of the affected part. Appraisal of the onset may suggest the vectors of applied force(s), however multiple structures are involved.

If there is no history of trauma or repeated stress the clinician should consider the possibility of a serious condition. Conversely, a history of trauma may have aggravated a pre-existing condition.

Previous Similar Symptoms

History of previous similar symptoms casts doubt on the acute nature of a pain and suggests an acute manifestation of a chronic condition. If there have been previous similar episodes that apparently resolved the possible effects of risk factors should be considered (see Prognosis).

Previous Treatment for the Index Condition

If multiple interventions have all failed to provide relief, the possibility of a serious condition should be considered.

Current Treatment for the Index Condition

All forms of treatment in current use should be noted together with information on the helpfulness of each. Alleviation, even temporarily, by particular measures may provide clues to the nature of the condition. Pain that responds to physical interventions often has a mechanical basis, or at least a mechanical contribution to its pathogenesis.

General History

- Note should be taken of any current treatment (for other conditions) that may have a bearing on the index condition or its treatment.
- Reviewing past and present symptoms from each system of the body may reveal conditions that influence the index condition.
- Involvement in activities that entail shoulder use, the likely impacts of disabilities and handicaps associated with the index condition and the presence of supportive relationships and other social resources should be noted.
- Lateral dominance is relevant as a possible aetiological factor, e.g. as a determinant of the way a person engages in particular activities and as a factor in the impact of the condition on activities of daily living.
- Occupation is relevant as a guide to ways the shoulder has been used in the past and to tasks the consumer may have to undertake, or try to undertake, in the future.
- Past history of other musculoskeletal conditions or of significant trauma suggests the possibility of an acute manifestation of a chronic condition. Past history of fracture due to minor trauma, recurrent infection, immunological compromise or neoplasm suggests the possibility of a serious condition.
- Age is relevant to acute shoulder pain as a risk factor. Osteoporosis is uncommon below the age of 50 so advanced age is an alerting feature. Age over 50 is also associated with an increased risk of cancer.
- Fever is an indication of systemic infection; this may be an alerting feature for septic arthritis (Lossos et al. 1998). A history of penetrating injury is another alerting feature. Infective organisms must have a portal of entry either directly into the joint or into other parts of the body. Events providing such portals include penetrating injuries, surgery, medical procedures using needles, catheters or other instruments, acupuncture, body piercing, tattooing and injecting drug use.
- Previous malignancy, age over 50, weight loss and failure to improve with treatment are alerting features.

Note: The predictive values of these features have not been tested formally in relation to shoulder pain.

Key Message

Information obtained from the history may alert to the presence of a serious condition as the underlying cause of acute shoulder pain. (Consensus)

Psychosocial History

An assessment of whether the individual's affect, cognitions and beliefs are likely to influence the course of the condition can identify whether there are psychosocial factors that warrant additional management.

In all cases, appreciation of the psychosocial response to the condition assists clinicians to empathise with and care for the individual in the manner advocated by Cochrane (1977).

Psychosocial history should include:

- affect generally (e.g. whether anxious or depressed)
- understanding of and reaction to the index condition and any associated fears
- relevant cognitions and beliefs, both personal and socio-cultural
- coping strategies used in relation to the index condition, or lack of them

Evidence of Reliability

There are few data on the diagnostic utility of history taking. The value of the history in clinical assessment is often taken for granted. There are no reports in the literature of formal studies of histories of people with acute shoulder pain but one study exists on the reliability of histories taken from those with chronic shoulder pain.

Nørregaard et al. (2002) studied histories obtained by an orthopaedic surgeon and a rheumatologist who each assessed 86 patients in a teaching hospital shoulder clinic, in random order. The inter-observer agreement on symptoms was low; the results are presented in Table 7.3.

Evidence of Validity

There are no data on the validity of history taking only, without physical examination, pertaining solely to those with acute shoulder pain but there are data for histories of those with shoulder pain of mixed (acute and chronic) durations.

Litaker et al. (2000) studied the histories of 448 people who had double contrast arthrography for investigation of shoulder problems. The features in the histories were correlated with arthrographic evidence of rotator cuff tendon tears (Table 7.4).

These data of reliability and validity suggest a need for caution in the interpretation of clinical histories obtained from people with shoulder pain.

Key Message

The reliability and validity of individual features in histories have low diagnostic significance; the history is to be interpreted with caution when choosing a course of action. (*Level III-2)

>Physical Examination

A physical examination of the shoulder may include inspection, palpation and movement testing.

Inspection

Observations on visual inspection of the shoulder may include peculiarities of posture, of bodily contours or of bony landmarks that suggest structural abnormality. Swelling should alert to the possibility of fracture.

Inflammatory arthropathies are characterised by effusion and should be considered if an individual presents with joint swelling.

Palpation

Tenderness is the main physical sign elicited by palpation. It may be focal or diffuse. Focal tenderness is usually regarded as more significant, especially if it reproduces the individual's typical pain. On finding focal tenderness, the conventional approach is to try to determine its anatomical reference.

Other signs elicited by palpation include apparent alterations of skin sensitivity such as hypoaesthesia, suggesting neurological deficit, and hyperaesthesia, suggesting allodynia,

Table 7.3

Reliability of Symptoms Elicited by Two Experienced Clinicians

Symptom	Kappa	Standard Error
Pain deep in the shoulder	0.15	0.08
Pain in the upper shoulder	0.09	0.07
Pain in the front of the shoulder	0.15	0.08
Pain in the back of the shoulder	0.49	0.10
Pain on lifting or throwing	0.26	0.16
Pain at rest	0.54	0.08

Note: Based on data from Nørregaard et al. (2002).

Table 7.4

Validity of Histories of Rotator Cuff Lesions

Features in History	Sensitivity	Specificity	Likelihood Ratio
History of trauma	0.36	0.73	1.33
Pain on shoulder movement	0.98	0.10	1.10
Night pain	0.88	0.20	1.10

Note: Based on data from Litaker et al. (2000).

and apparent alteration of bony landmarks, soft tissue conformation and muscle tone.

Palpable deformities of bones and other tissues alert to the possibility of neoplasm.

Movement Testing

Movements of the shoulder are tested by assessing the active, passive and accessory ranges of movement and challenging the restraints to movement.

Ranges of Movement

Ranges of active movement are assessed based on the ability to extend, flex, abduct, adduct, externally rotate and internally rotate the shoulder from a neutral position. Conventions have been set (Russe et al. 1976; Green and Christensen 1994) for performing these tests and recording their results. The ranges may be assessed visually or by use of a measuring instrument, a goniometer or an inclinometer. The examiner should note any limitation of range and any movement associated with pain.

Ranges of passive and accessory movement are tested similarly, with the examiner supplying the effort to move the shoulder through each range in turn.

Challenging Restraints

The restraints to the various movements are bony contours, capsules, ligaments, tendons and muscles that limit movement in each direction. They are tested actively by asking the individual to move the shoulder as far as possible and to describe what seems to be limiting further movement, whether pain, tethering, a bony stop or otherwise. By resisting active movements the examiner can gain an impression of the strength of muscles involved and any association with pain.

Restraints are challenged passively by the examiner moving a joint through its physiological ranges and testing its accessory movements, the translations and rotations possible along and around each of the biomechanical axes. Restraints may be deemed to be intact or impaired.

- 'End-feel' is described as what is felt by the examiner when a joint is taken to the limit of its movement. It is deemed to be 'hard' or 'soft' (Frisch 1994).
- A 'painful arc' is another sign described in relation to movement testing. It is part of a range through which movement is associated with pain (Kessel and Watson 1977).
- The original 'impingement sign' is said to be present when shoulder flexion (forward elevation of the arm) is limited by pain as the humeral head and the acromion move closer together, apparently impinging on tissues in the subacromial space (Neer 1972). It should be noted that in the original description, Neer included abolition of the positive response after subacromial injection of lignocaine as a second stage of the test (1972).
- Another clinical sign described as denoting impingement is a positive 'Hawkins test' (Hawkins and Kennedy 1980), pain on passive internal rotation of the shoulder at 90° flexion (forward elevation of the arm).
- Many other clinical tests have been developed for the assessment of suspected subacromial impingement. One of many examples is the 'Yocum test' (Yocum 1983), which is described as positive when pain is provoked by raising the individual's elbow when their hand is on the opposite shoulder. Clinicians should note that tests are sometimes called by eponymous names even though they are not done as originally described, and what is described as a positive clinical test may not be the same in the hands of different examiners.
- The 'drop arm test' for a torn rotator cuff tendon is described as positive if there is a sudden drop on active adduction of the arm from 90° abduction.
- The 'apprehension sign' is described when guarding and apprehension are exhibited as the examiner starts to test restraints to a particular movement. It is said to signify instability (Blazina and Satzman 1969).

Other tests are described for assessment of the biceps tendon:

- Provocation of pain by active shoulder flexion (forward elevation) against resistance is called a positive 'Speed test' (Speed 1966). It is said to denote a disorder of the tendon of the long head of the biceps.

- Another test of the long head of the biceps is the 'Yergason test' (Yergason 1931), which is described as positive when anterior shoulder pain is provoked by resisted active supination of the forearm from pronation.
- Tests are also described for challenging the restraints of the acromioclavicular joint (American Academy of Orthopaedic Surgeons 1962) and the sternoclavicular joint (Burrows 1951).

Key Message

Findings of shoulder examination must be interpreted cautiously in light of the evidence of limited utility; no clinical test is both reliable and valid for any specific diagnostic entity. (*Level III-2)

Evidence of Reliability

Inspection

In the absence of data yielding kappa scores or other indices of agreement, the reliability of inspection of the shoulder is unknown.

Palpation

Palmer et al. (2000) showed a high degree of reliability for elicitation of tenderness somewhere around the shoulder (kappa 0.80, with a standard error of 0.11). The diagnostic utility of such non-specific tenderness is unknown.

The reliability of focal tenderness or other palpatory signs is unknown; no data exist.

Movement Testing

Ranges of Movement

There are no data on movement testing specifically related to acute shoulder pain. Data have been published for normal subjects, people with shoulder pain of mixed duration from one to 48 months and some with shoulder pain of unstated duration.

Visual estimations of ranges of shoulder movement seem of inconsistent reliability. Croft et al. (1994) reported good agreement between six trained observers for visual estimation of abduction, with an intra-class correlation coefficient (ICC) of 0.84, but poor agreement for external rotation (ICC 0.43). Other ranges were not studied.

Goniometry (using an instrument like a protractor with a scale marked in degrees and arms) might be expected to confer advantage. Williams and Callaghan (1990) studied 22 observers using visual estimation and three different types of goniometers to assess ranges of abduction. They showed visual estimation was the most reliable method. Other studies of goniometry have also showed only moderate inter-observer reliability (Boone et al. 1978; Riddle et al. 1987; Bostrom et al. 1991).

Inclinometry (using a device with gravitational reference and a dial displaying degrees) can produce reliable measurements if performed by trained clinicians but it is not uniformly reliable. Two inclinometric studies by Green et al. (1998a) and Hoving et al. (2002) showed inter-rater reliability varies for different ranges of movement and groups of observers, as in Table 7.5.

Challenging Restraints

There are no data on the reliability of challenging restraints pertaining solely to those with acute shoulder pain but data have been published for physical examination of people with shoulder pain of unstated durations, and for those without shoulder conditions (to act as controls).

Palmer et al. (2000) studied the inter-observer reliability of physical signs elicited by challenging restraints to shoulder movement. The tests were performed on 43 subjects by two trained examiners (a research nurse and a rheumatologist). The results are presented in Table 7.6, showing kappa scores and their standard errors.

Calis et al. (2000) studied seven physical tests of shoulder restraints. The tests were performed by two experienced physicians and their inter-observer reliability values were reported as 'above 98%'.

Table 7.5

Inter-Rater Reliability of Shoulder Range Inclinometry by Physiotherapists and Rheumatologists

Ranges	ICCs (6 physiotherapists)	ICCs (6 rheumatologists)
Total shoulder flexion	0.82	0.73
Total shoulder abduction	0.88	0.56
External rotation in neutral	0.95	0.30
External rotation in abduction	0.73	0.19
Internal rotation in abduction	0.48	0.02
Hand behind back	0.71	0.80

Note: ICC = intra-class correlation coefficient. Based on data from Green et al. (1998a) and Hoving et al. (2002).

Table 7.6

Reliability of Physical Signs Elicited by Challenging Restraints

Physical Signs	Kappa	Standard Error
Painful arc	0.93	0.11
Painful resisted external rotation	0.90	0.11
Painful resisted internal rotation	0.54	0.11
Painful resisted abduction	0.81	0.11
Acromioclavicular joint 'stress'	0.80	0.11

Note: Based on data from Palmer et al. (2000).

Evidence of Validity

Inspection

In the absence of data yielding indices of sensitivity and specificity and likelihood ratios, the validity of inspection of the shoulder girdle is unknown.

Palpation

There are no data on the validity of tenderness (either focal or diffuse) or of other palpatory signs associated with shoulder disorders. The diagnostic utility of palpation for such signs is unknown.

Movement Testing

Ranges of Movement

There are no data on the validity of testing ranges of movement of the shoulder girdle so the diagnostic utility of such tests is also unknown.

Challenging Restraints

There are no data on the validity of challenging restraints pertaining solely to acute shoulder pain. Data have been published for physical examination of people with shoulder pain of mixed duration (range one to 48 months) and of unstated duration.

Calis et al. (2000) studied physical examination of the shoulder for the impingement syndrome. Physical signs were compared with a criterion standard of combined radiography, magnetic resonance imaging and relief of pain after subacromial injection of local anaesthetic. The sensitivity, specificity and likelihood ratio of each sign are presented in Table 7.7.

Other investigators have studied the validity of impingement signs. MacDonald et al. (2000) investigated the Neer and Hawkins clinical tests using arthroscopy as the criterion standard. They compared specific arthroscopic findings of subacromial bursitis with the clinical findings recorded pre-operatively by the treating orthopaedic surgeon. Naredo et al. (2002) investigated physical examination using ultrasonographic findings as their criterion standard. They studied a combination of ten clinical tests, including the Neer, Hawkins and Yocum tests to elicit signs of impingement. Results of both studies are presented in Table 7.8.

MacDonald et al. (2000) also compared positive Neer and Hawkins tests with arthroscopic findings of rotator cuff tendon lesions. Naredo et al. (2002) did a similar study using ultrasonographic findings as the criterion standard. Itoi et al. (1999) studied two clinical tests, called the 'full can test' (Jobe and Moynes 1982) and 'the empty can test' (Kelly et al. 1996) for rotator cuff tears using magnetic resonance imaging as a criterion standard. Their results are presented in Table 7.9.

Table 7.7

Validity of Physical Signs Elicited by Tests to Challenge Restraints to Shoulder Movement

Physical Sign(s)	Sensitivity	Specificity	Likelihood Ratio
Pain on passive forward elevation (Neer test)	89%	31%	1.29
Pain on passive internal rotation at 90° flexion (Hawkins test)	92%	25%	1.23
Pain on passive horisontal adduction with elbow flexed	82%	28%	1.14
Painful arc between 60° and 120° of active shoulder abduction	33%	81%	1.74
Sudden drop on active adduction from horizontal (drop arm test)	8%	97%	2.60
Shoulder pain on resisted forearm supination (Yergason test)	37%	86%	2.64
Pain on resisted shoulder flexion (Speed test)	69%	56%	1.57
All 7 of the above 'impingement' tests positive	4%	97%	1.57

Note: Based on data from Calis et al. (2000).

Table 7.8

Validity of Physical Signs of Impingement

Physical Sign(s)	Sensitivity	Specificity	Likelihood Ratio
Pain on passive forward elevation (Neer test) (M)	75%	48%	1.44
Pain on internal rotation at 90° flexion (Hawkins test) (M)	92%	44%	1.64
Both Neer and Hawkins tests positive (M)	71%	51%	1.45
Neer, Hawkins, Yocum and other tests all positive (N)	43%	88%	3.6

Note: Based on data from MacDonald et al. 2000 (M) and Naredo et al. 2002 (N).

Table 7.9

Validity of Physical Signs for Rotator Cuff Lesions

Physical Sign(s)	Sensitivity	Specificity	Likelihood Ratio
Pain on passive forward elevation ('Neer test') (M)	83%	51%	1.69
Pain on internal rotation at 90° flexion ('Hawkins test') (M)	88%	43%	1.54
Both 'Neer' and 'Hawkins' tests positive (M)	83%	56%	1.89
'Neer', 'Hawkins', 'Yocum' and other tests all positive (N)	79%	50%	1.58
Pain on external rotation in elevation ('the full can test') (I)	66%	64%	1.83
Pain on internal rotation in elevation ('the empty can test') (I)	63%	55%	1.40

Note: Based on data from MacDonald et al. 2000 (M), Naredo et al. 2002 (N) and Itoi et al. 1999 (I).

Naredo et al. (2002) also investigated physical examination for biceps tendon lesions using ultrasonographic findings as the criterion standard. Bennett (1998) studied the Speed test for testing the biceps tendon at the level of the bicipital groove and compared its results with those of arthroscopy. The results are displayed in Table 7.10.

Readers will note that the tables show many of the same clinical tests being used to detect apparently distinct disorders.

Summary

The evidence on the diagnostic utility of tests used in physical examination of the shoulder girdle is summed up by Calis et al. (2000) who stated 'the highly sensitive tests seem to have low specificity values and the highly specific ones to have low sensitivity values'. This is reflected in the low likelihood ratios of all individual tests and most combinations that have been studied.

Key Messages

> Causes of acute shoulder pain cannot be diagnosed by clinical assessment; however, with the exception of serious conditions, satisfactory outcomes do not depend on precise identification of cause. (*Level III-2)

> Despite limitations, physical examination is an opportunity to identify features of potentially serious conditions. (Consensus)

Alerting Features of Serious Conditions (see Table 7.11)

Table 7.11 summarises some of the features generally associated with serious conditions such as malignancy, infection and fracture/dislocation that may be noted during clinical assessment. While the predictive values of these alerting features have not been tested specifically in relation to shoulder pain, their presence in conjunction with acute shoulder pain should prompt further investigation.

>Ancillary Investigations

Medical Imaging

Medical imaging enables indirect visualisation of internal structures of the body that otherwise can only be assessed by palpation. Imaging technology provides numerous modalities with different capacities, applications and indications.

The limitations of imaging require consideration. The evidence shows that visualisation of internal structures is compromised by limitations of reliability and validity. Imaging results may actually confuse the diagnostic process. Additionally, there are safety and cost issues to consider.

Indications for Medical Imaging

Imaging is indicated when there are clinical features of a potentially serious condition (i.e. fracture/dislocation, tumour, infection, inflammatory arthropathies). In the absence of alerting features, the diagnostic utility of imaging is minimal and imaging is not indicated.

Imaging has a much greater role to play in chronic shoulder pain. Whenever imaging is used, care must be exercised in the interpretation of the findings.

Plain Radiography

In plain radiography, the xray beam is impeded by tissue in its path to produce an image on a radiosensitive plate. Radiographic images depend on the relative radiolucencies of tissues. They show the outlines and contours of bones and joints clearly, but are less useful for assessing soft tissues. 'Stress views', in which a joint is imaged under biomechanical stress, show the relationships of the bones and provide some idea of whether anatomical restraints to joint movement are intact.

Safety

The ionising radiation used in plain radiography is teratogenic and carcinogenic. Those who are (or might be) pregnant should not be exposed to it. All others should only be exposed

Table 7.10

Validity of Physical Signs of Biceps Tendon Lesions

Physical Sign(s)	Sensitivity	Specificity	Likelihood Ratio
Neer, Hawkins, Yocum and other tests all positive (N)	74%	58%	1.76
Pain on resisted shoulder flexion (Speed test) (B)	90%	14%	1.05

Note: Based on data from Naredo et al. 2002 (N) and Bennett 1998 (B).

Table 7.11

Alerting Features of Serious Conditions Associated with Acute Shoulder Pain

Feature or Risk Factor	Condition
Symptoms and signs of infection (e.g. fever)	Infection
Risk factors for infection (e.g. underlying disease process, immunosuppression, penetrating wound)	
History of trauma	Fracture/dislocation
Sudden onset of pain	
Past history of malignancy	Tumour
Age > 50 years	
Failure to improve with treatment	
Unexplained weight loss	
Pain at multiple sites	
Pain at rest	

when necessary and then only to the minimum dose required for satisfactory images. The potentially serious consequences of radiation should be considered and the consumer warned of them to allow informed consent before radiography is undertaken (Roebuck 1995).

Reliability

There are no formal studies of the reliability of plain radiography in the investigation of acute shoulder pain. Studies of plain radiography of other joints suggest variation between radiographers in the methods used to produce images, and between radiologists in the detection, interpretation and designation of changes. Those studies show the limited reliability of plain radiography generally; it is unknown whether this finding can be extrapolated to radiography of the shoulder.

Validity

There are no formal studies of the validity of plain radiography in the diagnosis of acute shoulder pain. Plain films have been described as having the ability to show relationships between the segments of the proximal humerus and the glenohumeral joint, alterations of them due to trauma (Neer 1970), and signs of neoplasia (Stiles and Otte 1993; Tyson 1995). No studies have quantified those abilities. One paper reports sensitivity of 78% and specificity of 98% for plain xray diagnosis of instability after massive rotator cuff tears (Kaneko et al. 1995).

Cost Effectiveness

In the absence of dependable data on reliability and validity, the cost effectiveness of plain radiography in the diagnosis of acute shoulder pain is unknown.

Diagnostic Utility

Plain radiography seems useful in the diagnosis of fractures, dislocations, tumours and advanced arthritides. In acute cases it should be reserved for those with features of serious conditions. One study showed plain radiography is often uninformative in the assessment of acute shoulder pain (Fraenkel et al. 2000).

Key Message

Imaging is not necessary unless there are alerting features of serious conditions; in the absence of alerting features, the diagnostic utility of imaging is minimal and the results are unlikely to improve management. (*Level III)

Ultrasonography

In ultrasonography ('ultrasound' or 'sonography'), images are produced when an ultrasound beam is reflected by tissue in its path. Reflection occurs at surfaces and interfaces. Ultrasonic images show the surfaces and contours of soft tissues such as tendons and ligaments but do not show the internal structure of solid tissue such as bone.

Ultrasonography does not involve ionising radiation. There is no evidence that ultrasound has any harmful effects on human tissues and the method is considered non-invasive.

There are no data on the intra-observer or inter-observer reliability of ultrasonography explicitly related to the diagnosis of acute shoulder pain. Factors likely to threaten the reliability of the technique are similar to those described for other imaging modalities.

Safety

Equipment used in ultrasonography includes an ultrasonic transducer and a scanner. Current standards of shoulder ultrasonography (Middleton 1992; Teefey et al. 2000b) require use of a variable high-frequency linear-array transducer (7.5–10 megahertz). Sector transducers produce images of insufficient resolution and are best avoided.

Reliability

Scanning technique includes the position of the patient, the operator and the monitor screen and the orientation of the transducer relative to anatomical structures imaged. Ultrasonography of the shoulder is usually performed with the patient seated and the operator standing behind so both face the monitor screen and the ultrasonographer can orientate the transducer under the guidance of the image.

Skill of the operator is a major factor in inter-observer reliability. Ultrasonography is said to be highly dependent on the operator's training and experience (Tyson 1995; van Moppes et al. 1995).

Diagnostic criteria determine the changes identified and their interpretation. It is useful to understand the criteria applied in the judgment of ultrasonic findings.

Interpretation is particularly important in ultrasonography as changes in reflection of the ultrasound beam must be observed as they occur for proper appreciation. Ultrasonography cannot be interpreted effectively by subsequent viewing of the films.

Validity

There are no data on the validity of ultrasonography explicitly related to the diagnosis of acute shoulder pain, but data are available from studies of subjects with shoulder pain of mixed and unstated durations, very likely including some acute cases.

In these studies, ultrasonography has been compared with diagnostic interventions including both single and double contrast arthrography, computed tomography (CT), magnetic resonance imaging (MRI) and surgical findings (both open and arthroscopic) in investigation of the rotator cuff tendons and the subacromial bursa. In one study, ultrasonography has also been compared with clinical examination of the shoulder.

There have been several studies of the validity of ultrasonography in the investigation of rotator cuff tendon lesions using double contrast arthrography as a criterion standard. The results of seven such studies are set out in Table 7.12.

Many investigators have studied the validity of ultrasonography in diagnosis of rotator cuff lesions using surgical findings (in recent years, this mainly involved arthroscopy) as criterion standard. The results of ten such studies are set out in Table 7.13.

The results in Tables 7.12 and 7.13 relate to diagnosis of rotator cuff tears of all extents. Some authors have reported separate results for identifying full and partial-thickness tears; generally the diagnostic utility of ultrasonography was greater for full-thickness tears.

Milgrom et al. (1995) considered the clinical significance of ultrasonographic findings of rotator cuff tears in a study of 90 asymptomatic adults aged from 30 to 99 years. They set diagnostic criteria for a high-frequency ultrasonic scanner by imaging fresh human cadaver shoulders and then scanned living volunteers with no current or past shoulder symptoms. Their results are shown in Table 7.14.

Tempelhof et al. (1999) performed a similar study of 411 asymptomatic volunteers. They reported only ultrasonic findings of full-thickness (complete) tears. Their results are also shown in Table 7.14.

Milgrom et al. (1995) reported partial-thickness as well as full-thickness tears, which is why their prevalence figures are higher than those of Tempelhof et al. (1999). Both sets of figures

Table 7.12

Validity of Ultrasonography Versus Arthrography in the Diagnosis of Rotator Cuff Tears as Reported by Several Authors

Authors	*N*	Sensitivity	Specificity	Likelihood Ratio
D'Erme et al. (1993)	15	83%	0%*	0.83*
Brandt et al. (1989)	58	75%	43%	1.30
Miller et al. (1989)	56	58%	93%	8.30
Middleton et al. (1986)	100	91%	91%	10.00
Farin et al. (1996)	86	89%	95%	18.00
Mack et al. (1988)	99	88%	96%	22.00
Mack et al. (1985)	72	93%	97%	31.00

Note: * The specificity of 0% and low likelihood ratio were due to a lack of true negative scores in the results.

Table 7.13

Validity of Ultrasonography Versus Surgical Findings in the Diagnosis of Rotator Cuff Tears as Reported by Several Authors

Authors	*N*	Sensitivity	Specificity	Likelihood Ratio
D'Erme et al. (1993)	9	86%	0%*	0.86*
Brandt et al. (1989)	38	71%	29%	1.00
Kurol et al. (1991)	58	42%	88%	3.50
Brenneke and Morgan (1992)	120	78%	82%	4.30
Teefey et al. (2000b)	120	94%	85%	6.30
Crass et al. (1988)	108	90%	92%	11.00
Mack et al. (1985)	47	100%	91%	11.00
Wiener and Seitz (1993)	225	95%	94%	16.00
Farin et al. (1996)	86	87%	98%	44.00
Mack et al. (1988)	90	91%	98%	46.00

Note: * The specificity of 0% and low likelihood ratio were due to a lack of true negative scores in the results.

Table 7.14

Ultrasonographic Findings of Rotator Cuff Tears in People Without Symptoms as Found in Two Studies

Age Groups	All Tears (Milgrom et al. 1995) *N* = 90	Complete Tears (Tempelhof et al. 1999) *N* = 411
30–39	6%	—
40–49	10%	—
50–59	33%	13%
60–69	53%	20%
70–79	70%	31%
> 80	80%	51%

Note: Based on data from Milgrom et al. (1995) and Tempelhof et al. (1999).

are comparable with those of Chandnani et al. (1992), Sher et al. (1995), Miniaci et al. (1995) and Needell et al. (1996), who all reported findings of rotator cuff tears in significant proportions of people without symptoms investigated by MRI.

All of the studies of ultrasonography in the diagnosis of rotator cuff lesions are affected by selection bias; they involve only those who also underwent other investigations and/or surgery for rotator cuff problems. Extrapolating the findings to the wider population with shoulder pain is not possible.

Clinical significance is another issue raised in the diagnosis of rotator cuff tears. The finding of a tear by ultrasonography (or by other methods) does not prove the cause of the symptoms, as the presence of a tear does not correlate closely with pain. There are data showing that rotator cuff tears also occur in asymptomatic people.

Cost Effectiveness

In the absence of dependable data, the cost effectiveness of ultrasonography in the diagnosis of acute shoulder pain is unknown.

Diagnostic Utility

The diagnostic utility of ultrasonography for the investigation of acute shoulder pain is not simply a reflection of its ability to detect rotator cuff tears or other lesions. There are issues of selection bias and clinical significance to be considered in the interpretation of the validity data.

Ultrasonography seems useful for investigation of the rotator cuff and biceps tendons. It is very sensitive and specific for identifying full-thickness tears of the rotator cuff according to some reports (Mack et al. 1988; Wiener and Seitz 1993; Farin et al. 1996), although not all (Brandt et al. 1989; Miller et al. 1989; Kurol et al. 1991). It is not so useful for detecting partial thickness tears, with sensitivity of about 70% and specificity ranging from 29% to 96% in different reports (Norris and Green 1993).

If ultrasonography detects a rotator cuff tear, the decision must be made whether the finding is of clinical significance in the circumstances (Milgrom et al. 1995; Tempelhof et al. 1999).

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is based on the motion in bodily tissues of hydrogen and other atoms with odd numbers of protons. The procedure involves use of a radiofrequency pulse to deflect the atoms from their usual axes and a powerful magnetic field to realign them. Images are generated by associated electromagnetic changes (Harms et al. 1984; Seeger 1989a).

The high-resolution images produced by MRI show soft tissues clearly and bones reasonably clearly. They are used for assessing the rotator cuff muscles and tendons, the subdeltoid and subacromial spaces, the glenohumeral joint capsule and ligaments, the glenoid labrum, the biceps tendon and its groove and the architecture of the shoulder girdle bones (Seeger 1989b; Tsai and Zlatkin 1990).

Safety

A major advantage is that MRI does not involve ionising radiation. A consideration peculiar to it is the risk of metallic foreign bodies, especially intraocular ones, being drawn through tissues by the magnetic field. Another is the potential for claustrophobia from the apparatus.

Reliability

The reliability of MRI of the shoulder has been assessed in relation to the diagnosis of rotator cuff tendon tears in a study of five experienced musculoskeletal radiologists who each read 222 MR images (Balich et al. 1997). The results are described in Table 7.15.

Validity

There are no data on the validity of MRI explicitly related to the investigation of acute shoulder pain, but data are available from studies of patients with shoulder pain of mixed and unstated durations, very likely including at least some acute cases.

The literature is very varied because MRI is the only imaging modality that seems to show all the soft tissues of the shoulder well while also demonstrating the bones quite reasonably. Authors of formal scientific reports and topical reviews give credence to the ability of MRI to identify a wide range of conditions including fractures, labral injuries, osteopenic conditions, tumours in the bones and adjacent soft tissues, joint effusions and bursal swellings, cysts, muscle atrophy, tendon tears of grades I, II and III and biceps tendonosis (Tsai and Zlatkin 1990; Blanchard et al. 1999a).

The quality of evidence varies too, from purely descriptive reports to formal studies of different designs and sizes yielding indices of sensitivity and specificity and likelihood ratios. That evidence is considered according to the structures investigated and the lesions detected.

- Fractures are usually demonstrated by MRI (Reinus and Hatem 1998), although some subtle and complex fractures are not shown as well by MRI as they are by plain radiography or CT.
- Other bone conditions are shown distinctly. MRI is the most sensitive and specific technique for detecting osteonecrosis (Tsai and Zlatkin 1990). It is also more sensitive than both plain radiography and arthroscopy for demonstrating Hill-Sachs lesions (Workman et al. 1992).
- Tumours of bone and soft tissues are usually shown clearly by MRI. It often reveals rare tumours such as lipomas, haemangiomas and neuromas (Tyson 1995).
- Joint effusions and cysts including ganglia image starkly on MRI making them readily identifiable (Tsai and Zlatkin 1990; Fritts and Craig 1994).
- Biceps tendon pathology is said to be demonstrated well by MRI and several distinct appearances are described (Fritts and Craig 1994).
- Impingement syndromes and tendonosis are identified by MRI with a sensitivity of 93% and specificity of 76%, yielding a likelihood ratio of 3.9 (Iannotti et al. 1991).

Data are available for labral injuries and rotator cuff tendon tears, conditions that MRI is believed to be especially useful for investigating.

Labral injuries are often demonstrated well by MRI. Its validity in the diagnosis has been variously reported as only moderate in some publications but high in others, with sensitivity in the range 33–95% and specificity 69–100% (Green and Christensen 1994). Some representative results are shown in Table 7.16.

Rotator cuff tears are said to be imaged distinctly by MRI. Many investigators have studied the validity of MRI in the investigation of the rotator cuff tendons using surgical findings (mostly those of arthroscopy) as the criterion standard. The results of eight such studies are set out in Table 7.17.

Table 7.15

Inter-Observer Reliability of MRI in Diagnosis of Rotator Cuff Tears: κ Score Ranges Between Five Experienced Radiologists

Partial Tears	Complete Tears	All Tears
κ = 0.17–0.44	κ = 0.73–0.88	κ = 0.63–0.80

Note: Based on data from Balich et al. (1997).

Table 7.16

Validity of MRI versus Surgical Findings in the Diagnosis of Labral Injuries as Reported by Several Authors *

Authors	*N*	Sensitivity	Specificity	Likelihood Ratio
Torstensen and Hollinshead (1999)	15	73%	58%	1.7
Gross et al. (1990)	22	91%	69%	2.9
Ianotti et al. (1991)	39	88%	93%	13.0
Green and Christensen (1994)	33	75%	100%	*

Note: * The specificity of 100% due to a lack of false negative scores does not allow conventional calculation of the likelihood ratio.

Table 7.17

Validity of MRI versus Surgical Findings in the Diagnosis of Rotator Cuff Tears as Reported by Several Authors

Authors	N	Sensitivity	Specificity	Likelihood Ratio
Torstensen and Hollinshead (1999)	24	96%	49%	1.9
Tuite et al. (1998)	110	67%	77%	2.9
Blanchard et al. (1999b)	54	81%	78%	3.7
Maurer et al. (1997)	14	79%	88%	6.6
Zlatkin et al. (1989)	32	91%	88%	7.6
Evancho et al. (1988)	31	69%	94%	12.0
Balich et al. (1997)	222	84%	94%	14.0
Ianotti et al. (1991)	88	100%	95%	20.0

Table 7.18

MRI Findings of Rotator Cuff Tears in 96 People Without Symptoms

Age Groups	Partial Tears	Complete Tears	All Tears
19–39	4%	0%	4%
40–60	24%	4%	28%
> 60	26%	28%	54%
ALL	20%	15%	34%

Note: Based on data from Sher et al. (1995).

Table 7.19

MRI Findings of Rotator Cuff Tears in 100 People Without Symptoms

Age Groups	Partial Tears	Complete Tears	All Tears
19–39	8%	0%	8%
40–60	27%	4%	31%
> 60	27%	27%	54%
ALL	22%	14%	36%

Note: Based on data from Needell et al. (1996).

There are liabilities to be considered in interpretation of these data as with the evidence on validity of ultrasonography.

The evidence of the validity of MRI varies markedly from study to study. One reason for this is that the studies differ in the criteria they use for diagnosis of rotator cuff tears. In particular, those showing higher specificities and likelihood ratios are based on diagnosis of full-thickness tears, whereas those with lower specificities and likelihood ratios include partial-thickness tears. It seems MRI is extremely sensitive and specific for detection of complete rotator cuff tears, but much less specific for partial tears.

Selection bias is a problem in these data too, with most if not all study groups biased towards people destined to undergo surgery.

Clinical significance is the most challenging issue for clinicians. If MRI can detect rotator cuff tears with reasonable accuracy, the treating clinician has to decide how to interpret the imaging findings in relation to the clinical situation. Ultrasonographic data have demonstrated that rotator cuff tears occur in many asymptomatic people. Studies based on MRI have produced similar data (Sher et al. 1995; Miniaci et al. 1995; Needell et al. 1996). The results of two of these studies are strikingly similar, and are described in Tables 7.18 and 7.19.

Miniaci et al. (1995) studied a younger group of 20 asymptomatic people who had undergone shoulder MRI. Thirty-nine of the 40 subjects were under 40 years of age. They reported MRI signs of partial-thickness tears in 23% of the subjects' supraspinatus tendons and in 13% of their infraspinatus tendons.

These data raise the issue of how to interpret common findings in imaging studies. No imaging modality can show pain per se, but they can demonstrate morphological appearances that may be associated with pain. The prevalence of radiological 'abnormalities' in asymptomatic individuals brings the significance of those radiological findings into question and casts serious doubt on the validity of diagnostic imaging as a guide to management.

Chandnani et al. (1992) pursued this issue in another study involving two matched groups of 20 patients and 20 asymptomatic volunteers between ages 25 to 55 years. The results show the relative prevalence of various features seen on MRI in those with and those without symptoms (Table 7.20).

These data also cast doubt on the clinical significance of many MRI findings including acromioclavicular osteophytes, abnormal labral signal, joint fluid, absent subacromial or subdeltoid fat, abnormal tendon signal and 'tendonitis', as well as partial tears of the rotator cuff tendons. The clinician should

Table 7.20

Prevalence of MRI 'Abnormalities' in People With and Without Symptoms

MRI Findings	Symptomatic People	Asymptomatic People
Acromioclavicular osteophytes	11	7
Anterior instability	4	0
Posterior instability	1	0
Abnormal labral morphology	4	0
Abnormal labral signal	11	10
Bony glenoid defect	3	0
Joint fluid	7	10
Absent subacromial fat	4	1
Absent subdeltoid fat	2	1
Supraspinatus depression	13	6
Abnormal tendon morphology	7	1
Abnormal tendon signal	13	6
Impingement	0	0
Tendonitis	3	4
Partial rotator cuff tear	3	1
Tendon discontinuity	7	0
Complete rotator cuff tear	6	0

Note: Based on data from Chandnani et al. (1992).

be careful to interpret MRI reports accordingly and not to simply take them at face value.

Cost Effectiveness

MRI is more expensive than other imaging modalities. There are no explicit data on its cost effectiveness in the investigation of acute shoulder pain. The clinician must decide whether the diagnostic advantages of MRI in particular circumstances outweigh the cost disadvantage.

Diagnostic Utility

MRI is a useful modality for imaging the shoulder, with the ability to demonstrate all the soft tissues clearly and bone quite well. It can be used to assess the rotator cuff muscles and tendons, the subdeltoid and subacromial spaces, the glenohumeral joint capsule and ligaments, the glenoid labrum, the biceps tendon and its groove and the bones of the shoulder girdle. It may not demonstrate fractures and tumours as well as plain radiographs or CT, but is unlikely to miss such lesions. As with other imaging modalities, the findings of MRI have to be interpreted carefully, particularly with regard to clinical significance.

Computerised Tomography

Computerised tomography (CT) involves the recording of two series of tomographs along sagittal and transverse axes. The images are processed by a computer that arranges the slices for systematic scanning and three-dimensional reconstruction. This provides images with greater definition than other radiographic modalities.

Like conventional radiography, CT images bones better than it does soft tissues but the higher resolution of CT allows some assessment of soft tissue structures.

- The danger of ionising radiation is much higher with CT than with other radiological imaging modalities.
- There are no data on the reliability of CT for investigation of acute shoulder pain.
- No formal studies have been published of the validity of CT in the investigation of acute shoulder pain. No sensitivity or specificity indices are available but reports in the literature describe the utility of CT for assessing subtle and complex fractures of the proximal humerus and the scapula (Castagno et al. 1987; Kuhlman et al. 1988).
- There are no data on the cost effectiveness of CT for imaging the shoulder.
- Other modalities have supplanted CT in many of its former applications. Its main use in investigation of the shoulder is for delineation of subtle and complex fractures.

MR Arthrography

The paramagnetic agent gadolinium, injected either intravenously or intra-articularly before MRI, enhances the images and improves their capacity to show partial rotator cuff tears (Flannigan et al. 1990) and subtle changes such as inflammation of the biceps tendon sheath (Gückel and Nidecker 1998).

- The safety considerations are the same as for unenhanced MRI with the additional risks involved in joint injection and use of contrast medium.
- There are no data on the reliability of MR-arthrography in the investigation of acute shoulder pain.
- There are no data on the validity of MR-arthrography for acute shoulder pain.
- The arthrographic technique is more expensive than unenhanced MRI but probably more discriminatory of subtle lesions.
- MR-arthrography offers a means of investigating conditions that are not shown well on unenhanced MRI. The same cautions apply in interpretation of findings.

Radionuclide Bone Scanning (Scintigraphy)

An isotopic bone scan entails injection of a radioactive isotope such as technetium-99 into the blood and subsequent imaging

of isotope distribution through the body. Concentrations of the isotope show up as darker spots on the images and indicate 'pooling', or regions in which blood is collected.

Scintigraphy is used for detecting occult fractures (Matin 1979), tumours (McNeil 1984), infections (Merkel et al. 1984) and inflammatory arthropathies (Weissberg et al. 1978). Mechanical conditions can also be imaged using this modality (Clunie et al. 1997).

- There are no data on the reliability of isotopic scans for acute shoulder pain.
- There are no data on the validity of isotopic scans for acute shoulder pain.
- There are no data on the cost effectiveness of isotopic scans for acute shoulder pain.
- Isotopic scans are best reserved for investigating suspected serious conditions.

Other Ancillary Investigations

Other special investigations such as serological tests, nerve conduction studies, electromyography and bone density estimations have specific roles in the investigation of suspected serious conditions but there are no other indications for their use in the assessment of acute shoulder pain. Their applications are beyond the scope of these guidelines. Refer to Appendix C: Ancillary Investigations.

Key Message

There is a need to educate consumers about the limitations of imaging and the risks of radiation exposure. (*Level IV)

Conclusion

The evidence shows that symptoms and physical signs do not correlate sufficiently for definitive diagnosis of shoulder pain. Despite traditional teaching and the best efforts of expert clinicians, structure-specific clinical diagnosis cannot be reliably achieved. Five studies of clinical diagnosis involving different clinicians have concluded that it is of limited reliability. The results are presented in Table 7.21.

As the cause of acute shoulder pain cannot, in most cases, be identified at the initial consultation (Phillips and Polisson 1997; Solomon et al. 2000), clinicians may be inclined to proceed to ancillary investigations. While such investigations are warranted in the presence of features alerting to a serious condition, they lack utility in acute mechanical conditions as the results will not alter management or outcome.

>Terminology

The evidence on treatment of common mechanical disorders shows that satisfactory outcomes do not depend on precise identification of causes (Solomon et al. 2001). However, management must still be guided by some concept of the index condition. The clinician can formulate a working diagnosis that summarises the discernible features of the condition accurately even if it is not definitive. A descriptive label can be applied to the working diagnosis describing what is known of the condition.

Diagnostic labelling has two main purposes:

- to enable the formulation of a management plan
- to facilitate effective communication between clinician and consumer

A diagnostic label must be as specific as possible and scientifically valid. Inaccurate description or use of inappropriate terms obscures the diagnosis, hinders communication and understanding and increases the risk of treatment errors. The use of appropriate terms is essential to minimise such problems.

To promote consistency, the terms recommended by the International Association for the Study of Pain (IASP) in the latest edition of its taxonomy (Merskey and Boguk 1994) are preferred. However, the IASP taxonomy lists chronic pain terms; additional terms are needed for acute pain. Suggested terms for common mechanical conditions giving rise to acute shoulder pain on the basis of clinical assessment findings are presented in Figure 7.1.

These terms are not intended as definitive diagnoses. They express what is known about the presenting condition after clinical assessment. Clinicians should note that it is not neces-

When the origin of pain is unclear but unlikely to be related to local tissue damage:

- acute shoulder pain of uncertain origin

When the pain appears to be of local somatic origin but nothing else can be specified:

- acute somatic shoulder impairment

When the pain appears to arise from a particular region of the shoulder:

- acute anterior shoulder impairment
- acute posterior shoulder impairment
- acute lateral shoulder impairment
- acute superior shoulder impairment
- acute inferior shoulder impairment

Figure 7.1
Suggested terms to describe acute shoulder pain.

Table 7.21
Reliability of Clinical Diagnosis of Shoulder Pain as Shown by Five Studies

Authors	Clinicians	Reliability
Bamji et al. (1996)	Rheumatologists	'only 46%'
Liesdeck et al. (1997)	GPs and physiotherapists	'low' ($\kappa = 0.31$)
de Winter et al. (1999)	Physiotherapists	'moderate' ($\kappa = 0.45$)
Pal et al. (2000)	Emergency room doctors	'low'
Nørregaard et al. (2002)	Orthopaedic surgeon and rheumatologist	'poor' ($\kappa < 0.4$)

sary to identify an underlying condition at the outset unless a serious condition is suspected.

The suggested taxonomy aims to reduce the confusion arising from the inappropriate use of terms to describe acute shoulder pain. For example, 'subacromial bursitis', 'supraspinatus tendonitis', 'rotator cuff tear' and 'impingement syndrome' are terms used more or less interchangeably to describe similar clinical presentations (Buchbinder et al. 1996a,b). They create false impressions of disparate diagnostic entities that are readily distinguishable clinically. Substituting the term for all of them of 'acute superior shoulder impairment' avoids ambiguity and facilitates comparison between conditions that are similar.

These terms are deliberately not tissue-specific. The concept of impairment is central to their understanding.

'Impairment' is defined, in the World Health Organisation (1986) list of terms related to disability, as 'loss or abnormality of anatomical structure, or physiological or psychological function'. It is a general term implying damage and/or loss of function without attributing cause. It is more than a description of a symptom but not a presumption of specific pathology and it allows for both the psychosocial and physical dimensions of the condition.

Acute Shoulder Pain of Uncertain Origin

Acute shoulder pain of uncertain origin refers to pain in the shoulder where the source of pain is unclear after clinical assessment. Its use is best confined to cases in which the pain is likely to be mediated by factors other than local tissue damage, such as pain arising outside the shoulder, and then it should be supplemented by explanation. Consideration of serious conditions should be an urgent priority in such cases.

Acute Somatic Shoulder Impairment

Acute somatic shoulder impairment means the pain is due to impairment of somatic structure(s) of the shoulder. The word 'somatic' denotes that the condition is physical. While not specifying the tissue(s) affected, the descriptor implies the pain is arising locally rather than from outside the shoulder, is not of neurological origin and is not due to a serious condition.

Acute Regional Shoulder Pain

Acute anterior shoulder impairment means the pain is due to impairment of one or more of the structures at the front of the shoulder, without specifying the particular tissue(s) involved. Acute posterior, lateral, superior or inferior shoulder impairment implies impairment of one or more of the structures at the back, outer part, top or underpart of the shoulder, respectively, without specifying the particular tissue(s) involved.

Key Message

Terms to describe acute shoulder pain should summarise the discernible features of the condition to form the basis for a management plan. (Consensus)

PROGNOSIS

Prognosis is determined by:

- natural history
- the influence of risk factors
- the effects of interventions

Natural History

The natural history of a condition is the course it is likely to follow under natural circumstances (i.e. if no interventions are applied).

By the original definition, 'acute' shoulder pain is 'that due to a condition which is likely to resolve spontaneously by natural healing' (Bonica 1953). To that definition could be added 'so long as it is not compounded by iatrogenic complications'. Accordingly, acute shoulder pain can be expected to resolve within a short time (a period of less than three months) if the causative condition is simply left alone.

By the current definition of 'acute' shoulder pain, 'that of less than three months duration' (Merskey 1979), some cases will be due to conditions characterised by more severe damage or pathology that are unlikely to resolve spontaneously.

Evidence

There are few data on the natural history of acute shoulder pain and existing data are compromised by methodological constraints.

There are obvious ethical restraints to studying people with painful conditions and deliberately leaving them untreated. Most published reports document the course of shoulder pain in patients in tertiary settings. Information about natural history can be deduced from data related to those treated symptomatically only, or in other ways unlikely to have altered the natural course of the condition.

Uncertainty of diagnosis creates problems in epidemiological research and in practice. Classifying patients into diagnostic groups on the basis of clinical assessment is unreliable and all studies based on such classification are inherently internally invalid (and thus also externally invalid). Their results and conclusions must be interpreted carefully in the light of diagnostic uncertainty. Apparent differences between cohorts should be discounted if selection criteria were imprecise.

Three reports in the literature provide data on outcomes of acute shoulder pain when treated conservatively by general practitioners. These data are presented in Tables 7.22 and 7.23.

Winters et al. (1997b) studied the course of acute shoulder pain at weekly intervals until the pain resolved or 25 weeks had elapsed. Nine percent had recovered at two weeks, 48% after 6 weeks, 76% after 12 weeks and 91% after the 25 weeks, as shown in Table 7.22. Their results for recovery of range of movement followed a similar trend.

Further progress was reported in a later publication (Winters et al. 1999b) demonstrating a substantial rate of recurrence of shoulder pain in the study cohort after the initial period. The

Table 7.22

Short Term Recovery of Acute Shoulder Pain

2 weeks	6 weeks	12 weeks	25 weeks
9%	48%	76%	91%

Note: Based on data from Winters et al. (1997b).

Table 7.23

Longer Term Recovery of Acute Shoulder Pain

1 month	3 months	6 months	12 months
23%	44%	51%	59%
		49%	59%

Note: Based on data from van der Windt et al. (1996) (upper figures) and Winters et al. (1997b) (lower figures).

later data suggest more moderate recovery rates when the recurrences are taken into account. The figures are very similar to the results of a study by van der Windt et al. (1996).

Analysis of associated factors suggested recovery was more likely to be rapid when onset was related to minor trauma or an episode of overuse and in those who presented soon after onset (possibly those with no major problems).

Croft et al. (1996) reported a prospective study of disabilities associated with acute shoulder pain treated conservatively by general practitioners in England. Their results are presented in Table 7.24.

The natural history of acute shoulder pain in general, based on these studies, is for recovery in the majority of cases within 12 weeks, but with substantial risk of recurrence of pain leading to chronic problems.

This information provides the treating clinician with a sound basis for treating acute shoulder pain conservatively in the early stages, so long as there are no alerting features of serious conditions. The data also suggest the clinician should be wary of the risk of recurrence even in those who seem to have recovered and consider the possible role of prognostic risk factors.

Key Messages

> Approximately 50% of people with acute shoulder pain (treated conservatively) recover within six months; approximately 60% recover within 12 months. (*Level III-2)

> Shoulder pain may recur even in those who appear to fully recover in the short term. (*Level III-2)

Prognostic Risk Factors

Clinical Relevance

Recognising risk factors enables clinicians to counteract their influence (potential or actual) on the onset of acute shoulder pain or the progression to chronic problems. Risk factors may be immutable or potentially remediable. Biological and psychosocial factors may be involved:

- biological risk factors as both aetiological and prognostic determinants
- psychosocial risk factors as aggravating and perpetuating influences

Biological Risk Factors

Biological or physical risk factors include physique, demographic status, clinical features and physical influences on them (see Table 7.25). They may be intrinsic or extrinsic.

- Intrinsic biological factors include gender, age, bodily habitus and health status; the physical attributes that determine susceptibility to pathogenetic mechanisms.
- Extrinsic biological factors include external physical influences such as forces sustained during activities. Of special relevance are the ways in which a person goes about activities of daily living, work and leisure pursuits.

Both intrinsic and extrinsic biological risk factors may be involved in causation (aetiological risk factors) and in the progression of an acute condition to chronicity (prognostic risk factors). Because of their potential to act in both ways, biological risk factors should be considered at the initial assessment and reconsidered at each review of progress.

Table 7.24

Recovery of Disability Associated with Acute Shoulder Pain

6 months	18 months
21%	49%

Note: Based on data from Croft et al. (1996).

Psychosocial Risk Factors

Psychosocial risk factors include intrapsychic factors, interpersonal factors and sociocultural factors (see Table 7.26). Psychosocial risk factors are prognostic; they predict chronicity. The term 'yellow flags' may be used to describe psychosocial risk factors.

INTERVENTIONS

Although there are many forms of conservative therapy for acute shoulder pain, evidence of their efficacy is not well established. Furthermore, as outlined in the preceding chapters, the interpretation of the results of trials in shoulder disorders is often hampered by the fact that these disorders are labelled and defined in diverse and often conflicting ways (Green et al. 1998b).

It is important to note that a lack of evidence (i.e. insufficient evidence) does not mean that a particular intervention has no place in the management of acute shoulder pain, however, it is preferable to employ interventions for which there is evidence of benefit, where appropriate. Management decisions should be based upon knowledge of the existing evidence, consideration of individual patient needs and clinical judgment.

The criteria formulated to categorise the following interventions and definitions of the levels of evidence are described in Chapter 9: Process Report.

Evidence of Benefit

Corticosteroid Injection

There were two trials of subacromial injection of corticosteroid and local anaesthetic compared to local anaesthetic injection alone for acute shoulder pain (Adebajo et al. 1990; Vecchio et al. 1993). Adebajo et al. (1990) compared 3ml of 0.5% lignocaine and 1ml of 80mg/ml triamcinolone hexacetonide to lignocaine alone for rotator cuff disease of less than three months duration. Results favoured the steroid injection group at four weeks; mean difference between groups in pain at four weeks was 3.6 (95%CI 1.55, 5.65) and mean difference between groups in range of abduction at four weeks was 45˚ (95%CI 19.12, 70.88). Vecchio et al. (1993) compared 40mg methylprednisolone and 1% lignocaine (1ml) to lignocaine alone in 57 trial participants with rotator cuff tendonitis (defined as shoulder pain exacerbated by resistance in at least one of: abduction, external or internal rotation and normal passive motion) of less than three months duration. At three months there were no reported differences between treatment groups for pain or passive range of motion however only median changes were reported and only completers were analysed.

Systematic review of trials of mixed duration of symptoms of shoulder pain (including the two trials described above) concluded that there is some evidence to support the use of subacromial corticosteroid injection for rotator cuff disease although its effect may be small and not well maintained and it may be no better than non-steroidal anti-inflammatory drugs (Buchbinder et al. 2002). There is also a suggestion that intra-articular steroid injection may be beneficial in the short-term for adhesive capsulitis but again the effect may be small and not well maintained (Buchbinder et al. 2002). While this updated systematic review found 26 randomised controlled

Table 7.25

Biological Risk Factors for Shoulder Pain as Shown in Various Reports

Factors	Subjects	Authors
Work above shoulder height	Industrial workers	Bjelle et al. (1979)
	Car assembly workers	Punnett et al. (2000)
	Forestry workers	Miranda et al. (2001)
	Urban manual workers	Pope (2001)
Low frequency vibration	Industrial workers	Futatsaka et al. (1985)
Repetitive work tasks	Semi-rural community	Ekberg et al. (1995)
	Shoulder patients	English et al. (1995)
	Urban manual workers	Pope (2001)
Heavy workload	Forestry workers	Miranda et al. (2001)
Work pace	Semi-rural community	Ekberg et al. (1995)
Driving for long periods	Commercial travellers	Skov et al. (1996)
Shift work	Male workers	Fredriksson et al. (1999)
Sleep disturbance	Male and female workers	Bergenudd and Nilsson (1994)
Smoking	Male workers	Bergenudd and Nilsson (1994)
	Commercial travellers	Skov et al. (1996)
	Video display unit users	Marcus and Gerr (1996)
	Female sewing machinists	Kaergaard and Andersen (2000)
Caffeine consumption	Video display unit users	Marcus and Gerr (1996)
Female gender	Semi-rural community	Ekberg et al. (1995)
	Commercial travellers	Skov et al. (1996)
	Female sewing machinists	Kaergaard and Andersen (2000)

Table 7.26

Psychosocial Risk Factors for Shoulder Pain as Shown in Various Reports

Factors ('yellow flags')	Subjects	Authors
Job dissatisfaction	Male and female workers	Bergenudd and Nilsson (1994)
	Video display unit users	Marcus and Gerr (1996)
Uncertain work demands	Semi-rural community	Ekberg et al. (1995)
	Commercial travellers	Skov et al. (1996)
	Newly employed workers	Nahit et al. (2001)
Poor support at work	Video display unit users	Marcus and Gerr (1996)
	Female sewing machinists	Kaergaard and Andersen (2000)
High mental workload	Male workers	Fredriksson et al. (1999)
	Urban manual workers	Pope (2001)
Psychological distress	Urban workers	Macfarlane et al. (1998)
	Forestry workers	Miranda et al. (2001)
	Semi-rural community	van der Windt et al. (2002)
	Semi-rural community	Badcock et al. (2002)
Immigrant status	Semi-rural community	Ekberg et al. (1995)

trials of corticosteroid injections for shoulder pain,there was little overall evidence to guide yteatment due to: limitations such as small sample sizes, variable methodological quality and heterogeneity in terms of population studied, injection modality employed and choice of comparator. While most studies (22/26; 84.6%) did not confirm the accurate placement of the injection, two reviewed studies used ultrasound to confirm needle placement (Gam et al. 1998; Plafki et al. 2000). Two other studies checked the accuracy of injecion following the procedure (Richardson 1975; White and Tuite 1996). Richardson (1975) performed an arthrogram following steroid injection and reported that the injection was intra-articular 'only inconstantly' when intra-articular injection was performed using the posterior approach, but 'readily obtained' when subacromial injection was performed. White and Tuite (1996) mixed urograffin with the corticosteroid preparation and took post-injection plain films. They reported that 10/20 (50%) of intra-articular injections using the posterior approach were correctly placed, compared to 19/20 (95%) using the anterior approach. Eustace et al. (1997) also assessed the accuracy of steroid injection and found that 10/24 (42%) of intra-articular injections using the anterior approach were correctly

placed and 4/14 (29%) of subacromial injections were correctly placed. It remains to be clarified whether the accuracy of needle placement, anatomical site, frequency, dose and type of corticosteroid influences efficacy.

Two trials compared corticosteroid injection to non-steroidal anti-inflammtory drugs (NSAIDs) for acute shoulder pain (labeled 'rotator cuff tendonitis' in both trials) (Adebajo et al. 1990; White et al. 1986). Adebajo et al. (1990) compared 2ml of 0.5% lignocaine and 1ml of 80mg/ml triamcinolone hexacetonide to diclofenac (50mg three times daily). White et al. (1986) compared subacromial injection of 40mg triamcinalone acetonide to indomethacin (25mg four times daily). No significant differences were demonstrated between treatment groups at four and six weeks following treatment in either trial for any of the measured outcomes including pain, range of active abduction, function or global assessment. A systematic review of trials comparing corticosteroid injection to NSAIDs for shoulder pain of mixed duration in which the results of these two trials were pooled together with a third trial (Petri et al. 1987) for rotator cuff tendonitis also failed to find any benefit of subacromial steroid injection over NSAIDs with respect to improvement in pain, function or range of shoulder abduction at four or six weeks (Buchbinder et al. 2002).

There have been no other trials that have specifically compared corticosteroid injection to other modalities for acute shoulder pain. However systematic review of trials comparing corticosteroid injection to physical therapies for shoulder pain of mixed duration has yielded variable results (Buchbinder et al. 2002). Two of three trials comparing the efficacy of intra-articular steroid injection with passive joint mobilisation and exercises for adhesive capsulitis reported early differential benefit of steroid injection, although this benefit was no longer apparent by six months (van der Windt et al. 1998; Bulgen et al. 1984). A third study comparing local steroid injections to therapy mainly comprised of mobilisation found no difference between groups at any time (Dacre et al. 1989). An additional trial (Arslan and Celiker 2001) compared intra-articular corticosteroid injection to a combination of NSAIDs and physical therapy measures for adhesive capsulitis (mean duration of symptoms was greater than three months in both treatment groups). There were no differences between groups at two or 12 weeks (Buchbinder et al. 2002). The review also found one trial comparing intra-articular, sub-acromial and acromioclavicular steroid injections to exercise therapy, massage, and physical applications (no mobilisation techniques or manipulative techniques were allowed) and to manipulation (mobilisation and manipulation) for general shoulder pain (mixed diagnoses) (Winters et al. 1997a). At the end of treatment, steroid injections were more beneficial with respect to pain relief compared to the other interventions (WMD –2.30, 95%CI –4.10, –0.50; and WMD -3.40, 95%CI –5.46, –1.34, respectively) (Winters et al. 1997a).

Systematic review of trials with mixed duration of symptoms of shoulder pain yielded one trial that compared the frequency of adverse effects between intra-articular steroids and physical therapy groups for adhesive capsulitis and found no significant differences apart from facial flushing which was more common in the steroid injection group (RR = 9.0; 95%CI 1.18, 68.74) (Buchbinder et al. 2002, van der Windt et al. 1998).

Key Message

Subacromial corticosteroid injection for acute shoulder pain may improve pain at four weeks compared to placebo but this benefit is not maintained at 12 weeks. (Level I)

Exercises

Systematic review of trials of mixed duration of symptoms of shoulder pain found weak evidence from two trials suggesting that exercise may be effective for rotator cuff disease in both the short and longer-term (Green et al. 2002). One placebo-controlled trial of a supervised exercise regime in 56 participants with mixed shoulder disorders demonstrated significantly greater recovery (RR 7.74; 95%CI 1.97, 30.32), function (RR 1.53; 95%CI 0.98, 2.39) and range of abduction (RR for worsening range 0.33; 95%CI 0.11, 0.96) at one month (Ginn et al. 1997). A second trial, with a two and a half year follow-up period demonstrated sustained benefit from exercise over placebo with respect to function in rotator cuff disease (RR for good or excellent function 2.45; 95%CI 1.24, 4.86) (Brox et al. 1997).

Key Message

Exercises may improve shoulder pain compared to placebo in people with rotator cuff disease in both the short and longer term. (Level I)

Non-Steroidal Anti-Inflammatory Drugs

There were three placebo-controlled trials of non-steroidal anti-inflammatory drugs (NSAIDs) for acute shoulder pain (Ginsberg and Famaey 1991; Mena et al. 1986; Adebajo et al. 1990). All demonstrated a short-term benefit from NSAID compared to placebo. One cross-over trial of 30 participants compared 4% topical indomethacin spray to placebo for acute shoulder pain of less than three weeks duration (28 participants had 'periarthritis of the shoulder' which was not defined further and two participants had epicondylitis, site not specified) (Ginsberg and Famaey 1991). There was a statistically significant improvement favouring the active group with respect to all outcomes measured. Overall improvement at 14 days favoured the active group (26/30 versus 18/30 for the active and placebo groups respectively, $\chi^2 = 5.455$, $p < 0.025$). Two participants reported minor signs of local irritation that did not require interruption of treatment.

Another trial of 68 participants compared flurbiprofen (300mg daily in four divided doses; dose decreased if symptoms had improved sufficiently after Day 1 and Day 3) to placebo for acute 'bursitis or tendonitis' of the shoulder (defined as symptoms of no more than four days duration and localised tenderness over the shoulder area, limitation of motion, pain on motion, pain severity interfering with sleep and either normal xrays or periarticular calcification) (Mena et al. 1986). There was a reportedly statistically significantly greater proportion of participants in the active group with improvement according to investigators global assessments at all follow-up points (Day 1, 3 or 4, 7 and 14) and at Day 7 according to patients assessments (data not shown for patient assessment of overall improvement). There was a trend in a similar direction for other outcomes reported.

One trial of 60 participants compared diclofenac (50mg three times daily) (and placebo injection) to placebo (and to steroid injection) for rotator cuff disease of less than three months duration (Adebajo et al. 1990). Results favoured the NSAID group at four weeks: mean difference between groups in pain at four weeks was –2.25 (95%CI –3.6, –0.9) and mean difference between groups in range of abduction at four weeks was 41.4˚ (95%CI 18.09, 64.71). Systematic review of trials of mixed duration of symptoms of shoulder pain verified the results of trials performed in acute shoulder pain of a short-term benefit of NSAIDs (Green et al. 1998b).

There were four trials comparing one NSAID to another for acute shoulder pain (Vidal et al. 2001; Gotter 1987; Soave et al. 1982; Wielandts and Dequeker 1979). These included between 26 and 599 participants and were all performed using different NSAIDs: meloxicam versus piroxicam (Vidal et al. 2001), tenoxicam versus piroxicam (Gotter 1987), indoprofen versus indomethacin (Soave et al. 1982) and phenylbutazone versus fentiazac (Wielandts and Dequeker 1979). There were no appreciable differences in outcome between NSAIDs in any of the trials.

In general, NSAIDs may be associated with adverse effects, including gastrointestinal bleeding, renal dysfunction (particularly in older people), NSAID-induced asthma and impaired blood clotting (Bigos et al. 1994).

Key Messages

> Topical and oral non-steroidal anti-inflammatory drugs (NSAIDs) improve acute shoulder pain by a small to moderate degree for up to four weeks compared to placebo. (Level I)

> Serious adverse effects of NSAIDs include gastrointestinal complications (e.g. bleeding, perforation). (Level I)

Ultrasound (Therapeutic)

Ebenbichler et al. (1999) included 54 participants with radiologically verified calcific tendonitis and pain or restricted range of motion for less than four weeks and compared 24 treatments with therapeutic ultrasound to placebo. Immediately following the course of treatment there was a significant difference between groups in perceived recovery favouring ultrasound (RR 1.81; 95%CI 1.26, 2.60). At nine months following treatment this benefit was not maintained, however there continued to be a significantly greater benefit in terms of radiological appearance of the calcific tendonitis in the treated group (RR 3.74; 95%CI 1.26, 8.66).

There was no report or measurement of adverse effects in the use of ultrasound for acute shoulder pain.

Key Message

Ultrasound (therapeutic) may provide short-term pain relief in calcific tendonitis compared to placebo. (Level I)

Conflicting Evidence

Acupuncture

There was one randomised controlled trial of acupuncture for acute rotator cuff disease in a population of 52 athletes (Kleinhenz et al. 1999). Eight acupuncture sessions in four weeks were compared to the identical number of sessions of placebo ultrasound. At four weeks, there was a significant difference favouring acupuncture in Constant-Murley score (which incorporates pain, function and range of motion) (WMD = 10.83; 95%CI 2.46, 19.20) but no difference at four months (WMD = 3.53; 95%CI 0.74, 6.42). There was no difference between groups in proportion to short-term success of therapy (RR = 0.56; 95%CI 0.26, 1.17).

When data from this trial was combined with data from another trial in patients with mixed duration of symptoms (Berry et al. 1980), no benefit of acupuncture over placebo was demonstrated (Green and Buchbinder 2003).

Key Message

There is conflicting evidence of the effectiveness of acupuncture compared to placebo ultrasound for shoulder pain and function. (Level I)

Insufficient Evidence

Analgesics (Paracetamol or Compound Analgesics)

There is no evidence to either support or refute the efficacy of analgesia for acute shoulder pain. There are no randomised controlled trials of analgesia (e.g. paracetamol or compound analgesics) in acute shoulder pain or in shoulder pain of longer duration.

Key Message

There are no randomised controlled trials investigating the use of analgesics (paracetamol or compound analgesics) for acute or chronic shoulder pain. (No Level I or II studies)

Extracorporeal Shock Wave Therapy

There are no published randomised controlled trials investigating the value of extracorporeal shock wave therapy (ESWT) in the treatment of acute shoulder pain. Systematic review of ESWT for shoulder pain of mixed duration identified four trials, two for calcific tendonitis (one trial of unspecified pain duration and one trial involving more than six months of symptoms) and two for rotator cuff tendonitis (duration of symptoms at least three and six months) (Buchbinder et al. 2003a). Results of the two trials in rotator cuff tendonitis did not demonstrate any significant benefit of ESWT over placebo with respect to pain or function up to 12 weeks following therapy (Buchbinder et al. 2003a). The two trials in calcific tendonitis both reported benefit from different doses of ESWT. Transient hematomas and petechiae were reported to occur in both calcific tendonitis trials.

Key Messages

> There are no randomised controlled trials of Extracorporeal Shock Wave Treatment for acute shoulder pain. (No Level I or II studies)

> Trials conducted in populations with chronic shoulder pain show conflicting results for ESWT compared with placebo. (Level I)

Manual Therapy

One small trial of 14 participants compared shoulder joint mobilisation combined with 'comprehensive treatment' (hot packs, active exercise, stretching, soft tissue mobilisation and education) to comprehensive treatment alone in primary shoulder impingement syndrome (not defined) (Conroy and Hayes 1998). Three weeks following treatment there was a statistically significant difference between groups in pain favouring the addition of mobilisation (WMD –32.07mm on VAS; 95%CI –58.04, –6.10). There was however no significant difference between groups in range of elevation (WMD –7.28˚; 95%CI –25.74, 11.8).

There was no report or measurement of adverse effects in the use of manual therapy for acute shoulder pain.

Key Message

Shoulder joint mobilisation with combined treatments (hot packs, active exercise, stretching, soft tissue mobilisation and education) may improve acute shoulder pain in the short term compared to the combined treatments alone. (Level I)

Oral Corticosteroids

There are no published randomised controlled trials investigating the value of oral corticosteroids for acute shoulder pain. Systematic review of corticosteroids for shoulder pain of mixed duration identified one placebo-controlled trial and one trial comparing oral steroids to no treatment in adhesive capsulitis

(Green et al. 1998b). While neither trial reported any significant benefit from oral steroids, methodological weaknesses may have influenced trial outcomes in both studies.

Key Messages

> There are no randomised controlled trials investigating the use of oral corticosteroids for acute shoulder pain. (No Level I or II studies)

> Studies of mixed populations do not report significant benefit from oral corticosteroids compared with placebo or no treatment for adhesive capsulitis. (Level I)

Suprascapular Nerve Blocks

There are no published randomised controlled trials investigating the value of suprascapular nerve blocks in the treatment of acute shoulder pain (excluding trauma). Systematic review of suprascapular nerve blocks for shoulder pain of mixed duration identified three randomised controlled trials performed in both adhesive capsulitis and rotator cuff disease suggesting short-term benefit with respect to pain (Buchbinder et al. 2003b).

Key Messages

> There are no published studies investigating the value of suprascapular nerve blocks for acute shoulder pain. (No Level I or II studies)

> There is some evidence of short-term effect from suprascapular nerve blocks for chronic adhesive capsulitis and rotator cuff disease. (Level I)

Surgery

There are no published randomised controlled trials investigating the value of surgery in the treatment of acute shoulder pain (excluding trauma).

Key Message

There are no published randomised controlled trials investigating the effectiveness of surgery for acute shoulder pain, although studies exist for chronic populations. (No Level I or II studies)

Transcutaneous Electrical Nerve Stimulation (TENS)

One trial compared transcutaneous nerve stimulation (TENS) to therapeutic ultrasound in 50 female participants with acute shoulder pain (Shehab and Adham 2000). Following the intervention period (three to five times a week for 13 sessions) participants in the ultrasound group were significantly better than the TENS group with respect to pain and range of motion. There was no report or measurement of adverse effects in the use of TENS for acute shoulder pain.

Key Message

There is insufficient evidence for the use of TENS for acute shoulder pain. (Level I)

>References

Aaras A, Horgen G, Bjorset HH, Ro O, Walsoe H (2001). Musculoskeletal, visual and psychosocial stress in VDU operators before and after multidisciplinary ergonomic interventions. A six year prospective study: part II. Applied Ergonomics, 32: 559–571.

Adebajo AO, Nash P, Hazleman BL (1990). A prospective double blind dummy placebo controlled study comparing triamcinolone hexacetonide infection with oral diclofenac 50 mg TDS in patients with rotator cuff tendinitis. Journal of Rheumatology, 17: 1207–1210.

Albright J, Allman R, Bonfiglio RP, Conill A, Dobkin B, Guddione AA, Hasson S, Russo R, Shekelle P, Susman JL (2001). Philadelphia panel evidence based clinical practice guidelines. Physical Therapy, 81: 1719–1730.

Andrews JR, Carson WG, McLeod WD (1985). Glenoid labrum tears related to the long head of the biceps. American Journal of Sports Medicine, 13: 337–341.

Aprill C, Dwyer A, Bogduk N (1990). Cervical zygapophyseal joint pain patterns: II — a clinical evaluation. Spine, 15: 458–461.

Arslan S, Celiker R (2001). Comparison of the efficacy of local corticosteroid injection and physical therapy for the treatment of adhesive capsulitis. Rheumatology International, 21: 20–23.

Badcock LJ, Lewis M, Hay EM, McCarney R, Croft PR (2002). Chronic shoulder pain in the community: a syndrome of disability or distress? Annals of the Rheumatic Diseases, 61: 128–131.

Bain GI, Rudkin G, Comley AS, Heptinstall RJ, Chittleborough M (2001). Digitally assisted acromioplasty: the effect of interscalene block on this new surgical technique. Arthroscopy, 17: 44–49.

Balich SM, Sheley RC, Brown TR, Sauser DD, Quinn SF (1997). MR imaging of the rotator cuff tendon: inter-observer agreement and analysis of interpretive errors. Radiology, 204: 191–194.

Bamji AN, Erhardt CC, Price TR, Williams PL (1996). The painful shoulder: can consultants agree? British Journal of Rheumatology, 35: 1172–1174.

Bang M, Deyle G (2000). Comparison of supervised exercise with and without manual physical therapy for patients with shoulder impingement syndrome. Journal of Orthopaedic and Sports Physical Therapy, 30: 126–137.

Barber FA (2001). Coplaning of the acromioclavicular joint. Arthroscopy, 17: 913–917.

Barlow IW, Newman RJ (1994). Primary bone tumours of the shoulder: an audit of the Leeds Regional Bone Tumour Registry. Journal of the Royal College of Surgeons of Edinburgh, 39: 51–54.

Baslund B, Thomsen BS, Jensen EM (1990). Frozen shoulder: current concepts. Scandinavian Journal of Rheumatology, 19: 321–325.

Bateman JE (1983). Neurologic painful conditions affecting the shoulder. Clinical Orthopaedics and Related Research, 173: 44–54.

Bencardino JT, Beltran J, Rosenberg ZS, Rokito A, Schmahmann S, Mota J, Mellado JM, Zuckerman J, Cuomo F, Rose D (2000). Superior labrum anterior-posterior lesions: diagnosis with MR arthrography of the shoulder. Radiology, 214: 267–271.

Bennett WF (1998). Specificity of the Speed's test: arthroscopic technique for evaluating the biceps tendon at the level of the bicipital groove. Arthroscopy, 14: 789–796.

Bergenudd H, Nilsson B (1994). The prevalence of locomotor complaints in middle age and their relationship to health and socioeconomic factors. Clinical Orthopaedics and Related Research, 308: 264–270.

Berjano P, Gonzalez BG, Olmedo JF, Perez-Espana LA, Munilla G (1998). Complications in arthroscopic shoulder surgery. Arthroscopy, 14: 785–88.

Berry H, Fernandes L, Bloom B, Clark RJ, Hamilton EB (1980). Clinical study comparing acupuncture, physiotherapy, injection and oral anti-inflammatory therapy in shoulder cuff lesions. Current Medical Research and Opinion, 7: 121–126.

Bigos S, Bowyer O, Braen G, et al. (1994). Acute low back problems in adults. Clinical Practice Guideline no.14. AHCPR Publication No. 95–0642. Agency for Health Care Policy and Research, Public Health Service, US, Department of Health: Rockville MD.

Binder AI, Bulgen DY, Hazleman BL, Roberts S (1984a). Frozen shoulder: a long term prospective study. Annals of the Rheumatic Diseases, 43: 361–364.

Binder A, Hazleman BL, Parr E, Roberts S (1986). A controlled study of oral prednisolone in frozen shoulder. British Journal of Rheumatology, 25: 288–292.

Binder A, Parr E, Hazleman BL (1984b). Pulsed electromagnetic field therapy of persistent rotator cuff tendonitis. Lancet, 1: 696–698.

Binkert CA, Zanetti M, Gerber C, Hodler J (2001). MR arthrography of the glenohumeral joint: two concentrations of gadoteridol versus Ringer solution as the intraarticular contrast material. Radiology, 220: 219–224.

Biundo JJ, Mipro RC Jr, Djuric V (1995). Peripheral nerve entrapment, occupation-related syndromes, sports injuries, bursitis, and soft-tissue problems of the shoulder. Current Opinion in Rheumatology, 7: 151–155.

Bjelle A, Hagsberg M, Michaelsson G (1979). Clinical and ergonomic factors in prolonged shoulder pain among industrial workers. Scandinavian Journal of Work, Environment and Health, 5: 205–210.

Blanchard TK, Bearcroft PW, Constant CR, Griffin DR, Dixon AK (1999a). Diagnostic and therapeutic impact of MRI and arthrography in the investigation of full thickness rotator cuff tears. European Radiology, 9: 638–642.

Blanchard TK, Bearcroft PW, Maibaum A, Hazelman BL, Sharma S, Dixon AK (1999b). Magnetic resonance imaging or arthrography for shoulder problems: a randomised study. European Journal of Radiology, 30: 5–10.

Blazina ME, Satzman JS (1969). Recurrent anterior subluxation of the shoulder in athletics: a distinct entity. Journal of Bone and Joint Surgery, 51A: 1037–1038.

Blockey NJ, Wright JK, Kellgren JH (1954). Oral cortisone therapy in periarthritis of the shoulder: a controlled trial. British Medical Journal, June 26, 1455–1457.

Bogduk N, Simons DG (1993). Neck pain: joint pain or trigger points. In: Vaeroy H, Merskey H (eds). Progress in Fibromyalgia and Myofascial Pain. Elsevier: Amsterdam. pp 267–273.

Bongers PM (2001). The cost of shoulder pain at work. British Medical Journal, 322: 64–65.

Bonica JJ (1953). The Management of Pain. Lea and Febiger: Philadelphia.

Bonnici AV, Welsh RP (1993). Tendon injuries and nerve entrapment of the shoulder and elbow. Current Opinion in Orthopaedics, 4: 56–64.

Boone DC, Azen SP, Lin CM, Spence C, Baron C, Lee L (1978). Reliability of goniometric measurements. Physical Therapy, 58: 1355–1360.

Bostrom C, Harms-Ringdahl K, Nordemar R (1991). Clinical reliability of shoulder function assessment in patients with rheumatoid arthritis. Scandinavian Journal of Rheumatology, 20: 36–48.

Bottoni CR, Wilckens JH, DeBerardino TM, D'Alleyrand JC, Rooney RC, Harpstrite JK, Arciero RA (2002). A prospective, randomized evaluation of arthroscopic stabilization versus nonoperative treatment in patients with acute, traumatic, first-time shoulder dislocations. American Journal of Sports Medicine, 30: 576–580.

Brandt TD, Cardone BW, Grant TH, Post M, Weiss CA (1989). Rotator cuff sonography: a reassessment. Radiology, 173: 323–327.

Bredella MA, Tirman PF, Fritz RC, Wischer TK, Stork A, Genant HK (1999). Denervation syndromes of the shoulder girdle: MR imaging with electrophysiologic correlation. Skeletal Radiology, 28: 567–572.

Brenneke SL, Morgan CJ (1992). Evaluation of ultrasonography as a diagnostic technique in the assessment of rotator cuff tendon tears. American Journal of Sports Medicine, 20: 287–289.

Brosseau L, Casimiro L, Milne S, Robinson V, Shea B, Tugwell P, Wells G (2002). Deep transverse friction massage for treating tendinitis. In: The Cochrane Library; Issue 2. Oxford: Update Software.

Brown C (1983). Compressive, invasive referred pain to the shoulder. Clinical Orthopaedics and Related Research, 173: 55–62.

Brox A, Gjengdal E, Uppheim G, Bohmer AS, Brevik JI, Ljuggren AE, Staff PH (1999). Arthroscopic surgery versus supervised exercises in patients with rotator cuff disease (stage II: impingement syndrome): a prospective randomised controlled study in 125 patients with a 2 1/2 year follow up. Journal of Shoulder and Elbow Surgery, 8: 102–111.

Brox A, Staff P, Ljunggren A, Brevik J (1993). Arthroscopic surgery compared with supervised exercises in patients with rotator cuff disease. British Medical Journal, 307: 899–903.

Brox JI, Roe C, Saugen E, Vollestad NK (1997). Isometric abduction muscle activation in patients with rotator tendinosis of the shoulder. Achives of Physical Medicine and Rehabilitation, 78: 1260–1267.

Brulin C, Haag J, Sundelin G (2002). Psychosocial predictors for shoulder/neck and low back complaints among home care personnel. Advances in Physiotherapy, 3: 169–178.

Buchbinder R, Goel V, Bombardier C (1996a). Lack of concordance between the ICD-9 classification of soft tissue disorders of the neck and upper limb and chart review diagnosis: one steel mills experience. American Journal of Industrial Medicine, 29: 171–182.

Buchbinder R, Goel V, Bombardier C, Hogg-Johnson S (1996b). Classification systems of soft tissue disorders of the neck and upper limb: do they satisfy methodological guidelines? Journal of Clinical Epidemiology, 49: 141–149.

Buchbinder R, Green S, Youd J (2002). Corticosteroid injections for shoulder pain. In: The Cochrane Library; Issue 4. Oxford: Update Software.

Buchbinder R, Green S, Youd J (2003a). Extracorporeal shock wave therapy for shoulder pain. In: The Cochrane Library; Issue 1. Oxford: Update Software (in press).

Buchbinder R, Green S, Youd J (2003b). Suprascapular nerve blocks for shoulder pain. In: The Cochrane Library; Issue 1. Oxford: Update Software (in press).

Buess E, Friedrich B (2001). Synovial chondromatosis of the glenohumeral joint: A rare condition. Archives of Orthopaedic and Trauma Surgery, 121: 109–111.

Bulgen D, Binder A, Hazleman B, Dutton J, Roberts S (1984). Frozen shoulder: prospective clinical study with an evaluation of three treatment regimes. Annals of the Rheumatic Diseases, 43: 353–360.

Bunker TD, Reilly J, Baird KS, Hamblen DL (2000). Expression of growth factors, cytokines and matrix metalloproteinases in frozen shoulder. Journal of Bone and Joint Surgery, 82B: 768–773.

Burrows HJ (1951). Tenodesis of subclavius in the treatment of recurrent dislocation of the sterno-clavicular joint. Journal of Bone and Joint Surgery, 33B: 240.

Cailliet R (1981). Shoulder Pain (2nd Edition). FA Davis: Philadelphia.

Calis M, Akgün K, Birtane M, Karacan I, Calis H, Tüzün F (2000). Diagnostic values of clinical diagnostic tests in subacromial impingement syndrome. Annals of the Rheumatic Diseases, 59: 44–47.

Campbell CC, Koris MJ (1995). Etiologies of shoulder pain in cervical spinal cord injury. Clinical Orthopaedics and Related Research, 322: 140–145.

Carrillon Y, Noel E, Fantino O, Perrin-Fayolle O, Tran-Minh VA (1999). Magnetic resonance imaging findings in idiopathic adhesive capsulitis of the shoulder. Revue du rhumatisme (English ed.), 66: 201–206.

Carter B (2001). A pilot study to evaluate the effectiveness of Bowen technique in the management of clients with frozen shoulder. Complementary Therapies in Medicine, 9: 208–215.

Castagno AA, Shuman WP, Kilcoyne RF, Haynor DR, Morris ME, Matsen FA III (1987). Complex fractures of the proximal humerus: role of CT in treatment. Radiology, 165: 759–762.

Ceccherelli F, Bordin M, Gagliardi G, Caravello M (2001). Comparison between superficial and deep acupuncture in the treatment of the shoulder's myofascial pain: a randomised and controlled study. Acupuncture and Electro-therapeutics Research, 26: 229–238.

Chandnani V, Ho C, Gerharter J, Neumann C, Kursunoglu-Brahme S, Sartoris DJ, Resnick D (1992). MR findings in asymptomatic shoulders: a blind analysis using symptomatic shoulders as controls. Clinical Imaging, 16: 25–30.

Chiou HJ, Chou YH, Wu JJ, Hsu CC, Huang DY, Chang CY (2002). Evaluation of calcific tendonitis of the rotator cuff. Journal of Ultrasound in Medicine, 21: 289–295.

Chiou HJ, Chou YH, Wu JJ, Huang TF, Ma HL, Chang CY (2001). The role of high-resolution ultrasonography in management of calcific tendinitis of the rotator cuff. Ultrasound in Medicine and Biology, 27: 735–743.

Clunie G, Bomanji J, Ell PJ (1997). Technetium-99m MDP patterns in patients with painful shoulder lesions. Journal of Nuclear Medicine, 38: 1491–1495.

Clunie GP, Bomanji J, Renton P, Edwards JC, Ell PJ (1998). Technetium-99m human immunoglobulin imaging in patients with subacromial impingement or adhesive capsulitis. Clinical Rheumatology, 17: 419–421.

Coari G, Paoletti F, Iagnocco A (1999). Shoulder involvement in rheumatic diseases: sonographic findings. Journal of Rheumatology, 26: 668–673.

Cochrane AL (1977). Effectiveness and Efficiency. Random Reflections on Health Services. Cambridge University Press: Cambridge. pp 95–98.

Codman EA, Akerson IB (1931). The pathology associated with rupture of the supraspinatus tendon. Annals of Surgery, 93: 348–359.

Conroy, DE, Hayes, KW (1998). The effect of mobilization as a component of comprehensive treatment for primary shoulder impingement syndrome. Journal of Orthopaedic and Sports Physical Therapy, 28(1): 3–14.

Cousins MJ (1987). Visceral pain. In: Andersson S, Bond M, Mehta M, Swerdlow M (eds). Chronic Non-Cancer Pain: Assessment and Practical Management. MTP Press: Lancaster. pp 119–132.

Crass JR, Craig EV, Feinberg SB (1988). Ultrasonography of rotator cuff tears: a review of 500 diagnostic studies. Journal of Clinical Ultrasound, 16: 313–327.

Croft P, Pope D, Boswell R, Rigby A, Silman A (1994). Observer variability in measuring elevation and external rotation of the shoulder. British Journal of Rheumatology, 33: 942–946.

Croft P, Pope D, Silman A (1996). The clinical course of shoulder pain: prospective cohort study in primary care. British Medical Journal, 313: 601–602.

Culic V, Miric D, Eterovic D (2001). Correlation between symptomatology and site of acute myocardial infarction. International Journal of Cardiology, 77: 163–168.

Cummings SR, Nevitt MC, Browner WS Stone K, Fox KM, Ensrud KE, Cauley J, Black D, Vogt TM (1995). Risk factors for hip fractures in white women. New England Journal of Medicine, 332: 767–773.

D'Erme M, De Cupis V, De Maria M, Barbiera F, Maceroni P, Lasagni MP (1993). L'ecographia, la Risonza Magnetica e l'artrographia con doppio mezzo di contrasto della cuffia dei rotatori. La Radiologia medica, 86: 72–80.

Dacre J, Beeney N, Scott D (1989). Injections and physiotherapy for the painful, stiff shoulder. Annals of the Rheumatic Diseases, 48: 322–332.

Dahan THM, Fortin L, Pelletier M, Petit M, Vadeboncoeur R, Suissa S (2000). Double blind RCT examining the efficacy of bupivacaine suprascapular nerve blocks in frozen shoulder. Journal of Rheumatology, 27: 1464–1469.

Dal Conte G, Rivoltini P, Combi F (1990). Trattamento della periartrite calcarea di spalla con campi magnetici pulsanti: studio controllato. La Riabilitazione, 23: 27–33.

de Winter AF, Jans MP, Scholten RJ, Deville W, van Schaardenburg D, Bouter LM (1999). Diagnostic classification of shoulder disorders: interobserver agreement and determinants of disagreement. Annals of the Rheumatic Diseases, 58: 272–277.

Downing D, Weinstein A (1986). Ultrasound therapy of subacromial bursitis. Physical Therapy, 66: 194–199.

Duplay ES (1872). De la périarthritie scapulo-humerale et des raideurs de l'épaule qui en sont les conséquences. Archives Générales de Médicine, 20: 513–542.

Dwyer A, Aprill C, Bogduk N (1990). Cervical zygapophyseal joint pain patterns: I — a study in normal volunteers. Spine, 15: 453–457.

Ebenbichler GR, Erdogmus CB, Resch KL, Funovics MA, Kainberger F, Barisani G, Aringer M, Nicolakis P, Wiesinger GF, Baghenstanian M, Preisinger E, Fialka-Moser V (1999). Ultrasound therapy for calcific tendinitis of the shoulder. New England Journal of Medicine, 340: 1533–1538.

Ekberg K, Karlsson M, Axelson O, Bjorkqvist B, Bjerre-Kiely B, Malm P (1995). Cross-sectional study of risk factors for symptoms in the neck and shoulder area. Ergonomics, 38: 971–980.

England S, Farrell A, Coppock J, Struthers G, Bacon P (1989). Low laser therapy of shoulder tendonitis. Scandinavian Journal of Rheumatology, 18: 427–443.

English CJ, Maclaren WM, Court-Brown C, Hughes SPF, Porter RW, Wallace WA, Graves RJ, Pethick AJ, Soutar CA (1995). Relations between upper limb soft tissue disorders and repetitive movements at work. American Journal of Industrial Medicine, 27: 75–90.

Eustace JA, Brophy DP, Gibney RP, Bresnihan B, FitzGerald O (1997). Comparison of the accuracy of steroid placement with clinical outcome in patients with shoulder symptoms. Annals of the Rheumatic Diseases, 56: 59–63.

Evancho AM, Stiles RG, Fajman WA (1988). MR imaging diagnosis of rotator cuff tears. American Journal of Roentgenology, 151: 751–754.

Fankhauser F, Schippinger G (2002). Suprascapular nerve entrapment by a ganglion cyst: an alternative method of treatment. Orthopedics, 25: 87–88.

Farin PU, Kaukanen E, Jaroma H, Väätäinen U, Miettinen H, Soimakallio S (1996). Findings at ultrasound, double-contrast arthrogaphy, and computed tomography arthrography with surgical correlation. Investigative Radiology, 31: 387–393.

Feldman DE, Shrier I, Rossignol M, Abenhaim L (2002). Risk factors for the development of neck and upper limb pain in adolescents. Spine, 27: 523–528.

Fermand M, Hassen CS, Ariche L, Samuel P, Postel JM, Blanchard JP, Goldberg D (2000). Ultrasound investigation of the rotator cuff

after computed arthrotomography coupled to bursography. Joint Bone Spine, 67: 310–314.

Ferrante FM, Kaufman AG, Dunbar SA, Cain CF, Cherukuri S (1998). Sphenopalatine ganglion block for the treatment of myofascial pain of the head, neck, and shoulders. Regional Anesthesia and Pain Medicine, 23: 30–36.

Flannigan B, Kursunoglu-Brahme S, Snyder S, Karsel R, Del Pizzo W, Resnick D (1990). MR arthrography of the shoulder: comparison with conventional MR imaging. American Journal of Roentgenology, 155: 828–832.

Fraenkel L, Shearer P, Mitchell P, LaValley M, Feldman J, Felson DT (2000). Improving the selective use of plain radiographs in the initial evaluation of shoulder pain. Journal of Rheumatology, 27: 200–204.

Fredriksson K, Alfredsson L, Koster M, Thorbjornsson CB, Toomingas A, Torgen M, Kilbom A (1999). Risk factors for neck and upper limb disorders: results from 24 years of follow up. Occupational and Environmental Medicine, 56: 59–66.

Friedenberg ZB, Miller WT (1963). Degenerative disc disease of the cervical spine. Journal of Bone and Joint Surgery, 45A: 1171–1178.

Frisch H (1994). Systematic Musculoskeletal Examination. Springer-Verlag: Berlin. pp 24–25.

Fritts HM, Craig EV (1994). MRI of the shoulder. Seminars in Ultrasound, CT, and MRI, 15: 341–365.

Fukui S, Ohseto K, Shiotani M Ohno K, Karasawa H, Naganuma Y, Yuda Y (1996). Referred pain distribution of the cervical zygapophyseal joints and cervical dorsal rami. Pain, 68: 79–83.

Futatsaka M, Sakurai T, Matsumoto A (1985). Comparative study of vibration disease among operators of vibrating tools by factor analysis. British Journal of Industrial Medicine, 42: 260.

Gam AN, Johannsen F (1995). Ultrasound therapy in musculoskeletal disorders: a meta-analysis. Pain, 63: 85–91.

Gam AN, Schydlowsky P, Rossel I, Remvig L, Jensen EM (1998). Treatment of 'frozen shoulder' with distension and glucorticoid compared with glucorticoid alone. A randomized controlled trial. Scandinavian Journal of Rheumatology, 27(6): 425–30.

Gardner E (1948). The innervation of the shoulder joint. The Anatomical Record, 102: 1–18.

Gartsman GM, Brinker MR, Khan M, Karahan M (1998). Self-assessment of general health status in patients with five common shoulder conditions. Journal of Shoulder and Elbow Surgery, 7: 228–237.

Ginn KA, Herbert RD, Khouw W, Lee R (1997). A randomised controlled clinical trial of a treatment for shoulder pain. Physical Therapy, 77: 802–811.

Ginsberg F, Famaey JP (1991). Double blind randomised crossover study of the percutaneous efficacy and tolerability of a topical indomethacin spray versus placebo in the treatment of tendinitis. Journal of International Medical Research, 19: 131–136.

Goh GJM, Over KE, Daroszewska A, Whitehouse GH, Bucknall RC (1997). The value of arthrogaphy in steroid injection of the shoulder joint (Letter to the Editor). British Journal of Rheumatology, 36: 709–710.

Gotoh M, Hamada K, Yamakawa H, Yanagisawa K, Nakamura M, Yamazaki H, Ueyama Y, Tamaoki N, Inoue A, Fukuda H (2001). Interleukin-1-induced subacromial synovitis and shoulder pain in rotator cuff diseases. Rheumatology, 40: 995–1001.

Gotter G (1987). Comparative evaluation of tenoxicam and piroxicam in the treatment of humeroscapular periarthritis. European Journal of Rheumatology and Inflammation, 9: 95–97.

Green MR, Christensen KP (1994). Magnetic resonance imaging of the glenoid labrum in anterior shoulder instability. American Journal of Sports Medicine, 22: 493–498.

Green S, Buchbinder R (2003). Acupuncture for shoulder pain. In: The Cochrane Library, Issue 1; 2003. Oxford: Update Software (in press).

Green S, Buchbinder R, Forbes A, Bellamy N (1998a). A standardized protocol for measurement of range of movement of the shoulder using the Plurimeter-V inclinometer and assessment of its intrarater and interrater reliability. Arthritis Care and Research, 11: 43–52.

Green S, Buchbinder R, Glazier R, Forbes A (2002). Interventions for shoulder pain In: The Cochrane Library; Issue 2. Oxford: Update Software.

Green S, Buchbinder R, Glazier R, Forbes A (1998b). Systematic review of randomized controlled trials of interventions for painful shoulder: selection criteria, outcome assessment, and efficacy. British Medical Journal, 316:354–360.

Grey RG (1978). The natural history of 'idiopathic' frozen shoulder. Journal of Bone and Joint Surgery, 60A: 564.

Griggs SM, Ahn A, Green A (2000). Idiopathic adhesive capsulitis. Journal of Bone and Joint Surgery, 82A: 1398–1406.

Gross ML, Seeger LL, Smith JB, Mandelbaum BR, Finerman GAM (1990). Magnetic resonance imaging of the glenoid labrum. American Journal of Sports Medicine, 18: 229–234.

Gückel C, Nidecker A (1998). MR arthrographic findings in tenosynovitis of the long bicipital tendon of the shoulder. Skeletal Radiology, 27: 7–12.

Haake M, Deike B, Thon A, Schmitt J (2002). Exact focusing of extracorporeal shock wave therapy for calcifying tendinopathy. Clinical Orthopaedics and Related Research, 397: 323–331.

Harms SE, Morgan TJ, Yamanashi WS, Harle TS, Dodd GD (1984). Principles of nuclear magnetic resonance imaging. Radiographics, 4: 26–30.

Hassett G, Barnsley L (2001). Pain referral from the sternoclavicular joint: a study in normal volunteers. Rheumatology, 40: 859–862.

Hawkins RJ, Kennedy JC (1980). Impingement syndrome in athletes. American Journal of Sports Medicine, 8: 151–158.

Hijioka A, Suzuki K, Nakamura T, Hojo T (1993). Degenerative change and rotator cuff tears. Archives of Orthopaedic and Trauma Surgery, 112: 61–64.

Hirschhorn P, Schmidt JM (2000). Frozen shoulder in identical twins. Joint, Bone, Spine, 67: 75–76.

Horneij E, Hemborg B, Jensen I, Ekdahl C (2001). No significant differences between intervention programmes on neck, shoulder and low back pain: a prospective randomized study among home-care personnel. Journal of Rehabilitation Medicine, 33: 170–176.

Hoving JL, Buchbinder R, Green S, Forbes A, Bellamy N, Brand C, Buchanan R, Hall S, Patrick M, Ryan P, Stockman A (2002). How reliably do rheumatologists measure shoulder movement? Annals of the Rheumatic Diseases, 61: 612–616.

Hudak PL, Cole DC, Frank JW (1998). Perspectives on prognosis of soft tissue musculoskeletal disorders. International Journal of Rehabilitation Research, 21: 29–40.

Iannotti JP, Zlatkin MB, Esterhai JL, Kressel HY, Dalinka MK, Spindler KP (1991). Magnetic resonance imaging of the shoulder: sensitivity, specificity and predictive value. Journal of Bone and Joint Surgery, 73A: 17–29.

Invargarsson T, Hagglund G, Johnson R (1996). Anterior acromioplasty: a comparison of two techniques. International Orthopaedics, 290: 174–176.

Itoi E, Kido T, Sano A, Urayama M, Sato K (1999). Which is more useful, the 'full can test' or the 'empty can test', in detecting the torn supraspinatus tendon? American Journal of Sports Medicine, 27: 65–68.

Jacobsson L, Lindegärde F, Manthorpe R (1989). The commonest rheumatic complaints of over six weeks' duration in a

twelve-month period in a defined Swedish population. Scandinavian Journal of Rheumatology, 18: 353–360.

Jobe FW, Moynes DR (1982). Delineation of diagnostic criteria and a rehabilitation program for rotator cuff injuries. American Journal of Sports Medicine, 10: 336–339.

Johansson JA, Rubenowitz S (1994). Risk indicators in the psychosocial and physical work environment for work related neck, shoulder and low back symptoms: a study among blue and white collar workers in eight companies. Scandinavian Journal of Rehabilitation Medicine, 26: 131–142.

Johansson K, Oberg B, Adolfsson L, Foldevi M (2002). A combination of SR and clinicians' beliefs on interventions for subacromial pain. British Journal of General Practice, 52: 145–152.

Johnson MP, McClure PW, Karduna AR (2001). New method to assess scapular upward rotation in subjects with shoulder pathology. Journal of Orthopaedic and Sports Physical Therapy, 31: 81–89.

Jones DS, Chattopadhyay C (1999). Suprascapular nerve block for the treatment of frozen shoulder in primary care: a randomised controlled trial. British Journal of General Practice, 49: 39–41.

Jones G, Nguyen T, Sambrook PN, Kelly PJ, Gilbert C, Eisman JA (1994). Symptomatic fracture incidence in elderly men and women: the Dubbo Osteoporosis Epidemiology Study. Osteoporosis International, 4: 277–282.

Kaempffe FA (1995). Neoplasm as a cause of shoulder pain. Journal of Family Practice, 40: 480–485.

Kaergaard A, Andersen JH (2000). Musculoskeletal disorders of the neck and shoulders in female sewing machine operators: prevalence, incidence, and prognosis. Occupational and Environmental Medicine, 57: 528–534.

Kaneko K, De Mouy EH, Brunet ME (1995). Massive rotator cuff tears: screening by routine radiographs. Clinical Imaging, 19: 8–11.

Karatas GK, Meray J (2002). Suprascapular nerve block for pain relief in adhesive capsulitis: comparison of 2 different techniques. Archives of Physical Medicine and Rehabilitation, 83: 593–597.

Karjalainen K, Malmivaara A, van Tulder M, Roine R, Jauhiainen M, Hurri H, Koes B (2002). Multidisciplinary biopsychosocial rehabilitation for neck and shoulder pain among working age adults. In: The Cochrane Library; Issue 2. Oxford: Update Software.

Kelly BT, Kadrmas WR, Spear KP (1996). The manual muscle examination for rotator cuff strength: an electromyographic investigation. American Journal of Sports Medicine, 24: 581–588.

Kessell L, Watson M (1977). The painful arc syndrome: clinical classification as a guide to management. Journal of Bone and Joint Surgery, 59B: 166.

Ketola R, Toivonen R, Hakkanen M, Luukkonen R, Takala EP, Viikari-Juntura E (2002). Effects of ergonomic intervention work with VDUs. Scandinavian Journal of Work, Environment and Health, 28: 18–24.

Kim SH, Ha KI, Ahn JH, Kim SH, Choi HJ (2001). Biceps load test II: a clinical test for SLAP lesions of the shoulder. Arthroscopy, 17: 160–164.

Kivimaki J, Pohjolainen T (2001). Manipulation under anesthesia for frozen shoulder with and without steroid injection. Archives of Physical Medicine and Rehabilitation, 82: 1188–1190.

Klein MG, Whyte J, Esquenazi A, Keenan MA, Costello R (2002). A comparison of the effects of exercise and lifestyle modification on the resolution of overuse symptoms of the shoulder in polio survivors. Archives of Physical Medicine and Rehabilitation, 83: 708–713.

Kleinhenz J, Streitburger K, Windeler J, Gubbacher A, Mavridid G, Martin E (1999). Randomised clinical trial comparing the effects of acupuncture and a newly designed placebo needle in rotator cuff tendinitis. Pain, 83: 235–241.

Kuhlman JE, Fishman EK, Ney DR, Magid D (1988). Complex shoulder trauma: three-dimensional CT imaging. Orthopedics, 11: 1561–1563.

Kurol M, Rahme H, Hilding S (1991). Sonography for diagnosis of rotator cuff tear. Comparison with observations at surgery in 58 shoulders. Acta Orthopaedica Scandinavica, 62; 465–467.

Law TC, Chong SF, Lu PP, Mak KH (1998). Bilateral subacromial bursitis with macroscopic rice bodies: ultrasound, CT and MR appearance. Australasian Radiology, 42: 161–163.

Leclaire R, Bourgouin J (1999). Electromagnetic treatment of shoulder periarthritis: a randomised controlled trial of efficiency and tolerance of magnetotherapy. Archives of Physical Medicine and Rehabilitation, 72: 284–288.

Lee HS, Joo KB, Park CK, Kim YS, Jeong WK, Kim YS, Park DW, Kim SI, Park TS (2002). Sonography of the shoulder after arthrography (arthrosonography): preliminary results. Journal of Clinical Ultrasound, 30: 23–32.

Lee M, Haq A, Wright V, Longton E (1973). Periarthritis of the shoulder: a controlled trial of physiotherapy. Physiotherapy, 59: 312–315.

Leppala J, Kannus P, Sievanen H, Jarvinen M, Vuori I (1998). Adhesive capsulitis of the shoulder (frozen shoulder) produces bone loss in the affected humerus, but long-term bony recovery is good. Bone, 22: 691–694.

Lesprit E, Le-Huec JC, Moinard M, Schaeverbeke T, Chauveaux D (2001). Snapping scapula syndrome: conservative and surgical treatment. European Journal of Orthopaedic Surgery and Traumatology, 11: 51–54.

Liesdeck C, van der Windt DAWM, Koes BW, Bouter LM (1997). Soft-tissue disorders of the shoulder: a study of inter-observer agreement between general practitioners and physiotherapists and an overview of physiotherapeutic treatment. Physiotherapy, 83: 12–17.

Limb D, Collier A (2000). Impingement syndrome. Current Orthopaedics, 14: 161–166.

Lindh M, Norlin R (1993). Arthroscopic subacromial decompression versus open acromioplasty. A two-year follow up study. Clinical Orthopaedics and Related Research, 290: 174–176.

Linos A, Worthington JW, O'Fallon WM, Kurland LT (1980). The epidemiology of rheumatoid arthritis in Rochester, Minnesota: a study of the incidence, prevalence and mortality. American Journal of Epidemiology, 111: 97–98.

Litaker D, Pioro M, El Bilbeisi H, Brems J (2000). Returning to the bedside: using the history and physical examination to identify rotator cuff tears. Journal of the American Geriatric Society, 48: 1633–1637.

Lossos IS, Yossepowitch O, Kandel L, Yardeni D, Arber N (1998). Septic arthritis of the glenohumeral joint. A report of 11 cases and review of the literature. Medicine (Baltimore), 77: 177–187.

Lowe BD, Wurzelbacher SJ, Shulman SA, Hudock SD (2001). Electromyographic and discomfort analysis of confined-space shipyard welding processes. Applied Ergonomics, 32: 255–269.

Lundberg BJ (1969). The frozen shoulder. Acta Orthopaedica Scandinavica, suppl 119: 1–59.

Lundborg G, Rank F (1978). Experimental intrinsic healing of flexor tendons based upon synovial fluid nutrition. Journal of Hand Surgery, 3: 21–31.

MacDonald PB, Clark P, Sutherland K (2000). An analysis of the diagnostic accuracy of the Hawkins and Neer subacromial impingement signs. Journal of Shoulder and Elbow Surgery, 9: 299–301.

Macfarlane GJ, Hunt IM, Silman AJ (1998). Predictors of chronic shoulder pain: a population based prospective study. Journal of Rheumatology, 25: 1612–1615.

Mack LA, Gannon MK, Kilcoyne RF, Matsen FA III (1988). Sonographic evaluation of the rotator cuff: accuracy in patients without prior surgery. Clinical Orthopaedics and Related Research, 234: 21–27.

Mack LA, Matsen FA III, Kilcoyne RF, Davies PK, Sickler ME (1985). US evaluation of the rotator cuff. Radiology, 157: 205–209.

Manske PR, Gelberman RH, Vande Berg JS, Lesker PA (1984). Intrinsic flexor tendon repair: morphological study in vitro. Journal of Bone and Joint Surgery, 66A: 385–396.

Marcus M, Gerr F (1996). Upper extremity musculoskeletal symptoms among female office workers: associations with video display terminal use and occupational psychosocial stressors. American Journal of Industrial Medicine, 29: 161–170.

Matin P (1979). Appearance of bone scans following fractures: including immediate and long-term studies. Journal of Nuclear Medicine, 20: 1227.

Matsen FA III, Thomas SC, Rockwood CA Jr. (1990). Anterior glenohumeral instability. In: Rockwood CA Jr., Matsen FA III (eds). The Shoulder. WB Saunders: Philadelphia. pp 526–622.

Maurer KP, Schmidt D, Refior HJ, Pfahler M, Stäbler A (1997). Diagnosis of rotator cuff tears: prospective comparison of arthrography, nonenhanced MR imaging, and MR arthrography in 22 surgically proven cases. Orthopaedics, 5: 193–200.

McNeil BJ (1984). Value of bone scanning in neoplastic disease. Seminars in Nuclear Medicine, 14: 277–286.

Mena H, Lomen P, Turner L, Lamborn K, Brinn E (1986). Treatment of acute shoulder syndrome with flurbiprofen. American Journal of Medicine, 80 (Suppl 3A): 141–144.

Merkel KD, Fitzgerald RH, Brown ML (1984). Scintographic evaluation in musculoskeletal sepsis. Orthopedic Clinics of North America, 15: 401–416.

Merskey H (1979). Pain terms: a list with definitions and notes on usage recommended by the IASP Subcommittee on Taxonomy. Pain, 6: 249–252.

Merskey H, Bogduk N (eds). (1994) Classification of Chronic Pain. Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms (2nd Edition). IASP Press: Seattle.

Middleton WD, Reinus WR, Melson WR, Totty WG (1986). Ultrasonographic evaluation of the rotator cuff and biceps tendon. Journal of Bone and Joint Surgery, 68A: 440–450.

Middleton WD (1992). Ultrasonography of the shoulder. Radiology Clinics of North America, 30: 927–940.

Milgrom C, Schaffler M, Gilbert S, van Holsbeeck M (1995). Rotator-cuff changes in asymptomatic adults: the effect of age, hand dominance and gender. Journal of Bone and Joint Surgery, 77B: 296–298.

Miller CL, Karasick D, Kurtz AB, Fenlin JM (1989). Limited sensitivity of ultrasound for the detection of rotator cuff tears. Skeletal Radiology, 18: 179–183.

Miniaci A, Dowdy PA, Willits KR, Vellet AD (1995). Magnetic resonance imaging evaluation of the rotator cuff tendons in the asymptomatic shoulder. American Journal of Sports Medicine, 23: 142–145.

Miranda H, Viikari-Juntura E, Martikainen R, Takala EP, Riihimaki H (2001). A prospective study of work related factors and physical exercise as predictors of shoulder pain. Occupational and Environmental Medicine, 58: 528–534.

Moore ME, Berk SN (1976). Acupuncture for chronic shoulder pain. An experimental study with attention to the role of placebo and hypnotic susceptibility. Annals of Internal Medicine, 84: 381–384.

Murrell GAL, Walton JR (2001). Diagnosis of rotator cuff tears. Lancet, 357: 769–770.

Nahit ES, Pritchard CM, Cherry NM, Silman AJ, MacFarlane GJ (2001). The influence of work related psychosocial factors and psychological distress on regional musculoskeletal pain: a study of newly employed workers. Journal of Rheumatology, 28: 1378–1384.

Naredo E, Aguado P, De Miguel E, Uson J, Mayordomo L, Gijon-Banos J, Martin-Mola E (2002). Painful shoulder: comparison of physical examination and ultrasound findings. Annals of the Rheumatic Diseases, 61: 132–136.

National Health and Medical Research Council (1999a). A Guide to the Development, Implementation and Evaluation of Clinical Practice Guidelines. AGPS: Canberra.

National Health and Medical Research Council (1999b). Acute Pain Management: Scientific Evidence. Commonwealth of Australia: Canberra.

Needell SD, Zlatkin MB, Sher JS, Murphy BJ, Uribe JW (1996). MR imaging of the rotator cuff: peritendinous and bone abnormalities in an asymptomatic population. American Journal Roentgenology, 166: 863–867.

Neer CS II (1972). Anterior acromioplasty for the chronic impingement syndrome of the shoulder. A preliminary report. Journal of Bone and Joint Surgery, 54A: 41.

Neer CS II (1970). Displaced proximal humeral fractures: I. Classification and evaluation. Journal of Bone and Joint Surgery, 52A: 1077–1089.

Neer CS II (1983a). Impingement lesions. Clinical Orthopaedics and Related Research, 173: 70–77.

Neer CS II, Craig EV, Fukuda H (1983b). Cuff-tear arthropathy. Journal of Bone and Joint Surgery, 65A: 1232–1244.

Nevasier RJ, Nevasier TJ (1987). The frozen shoulder: diagnosis and management. Clinical Orthopaedics and Related Research, 223: 59–64.

Nicholson G (1985). The effects of passive shoulder mobilisation on pain and hypomobility associated with adhesive capsulitis of the shoulder. Journal of Orthopaedic and Sports Physical Therapy, 6: 238–246.

Nørregaard J, Krogsgaard MR, Lorenzen T, Jensen EM (2002). Diagnosing patients with long standing shoulder joint pain. Annals of the Rheumatic Diseases, 61: 646–649.

Norris TR, Green A (1993). Imaging modalities in the evaluation of shoulder disorders. In: Matsen FA III, Fu FH, Hawkins RJ (eds). The Shoulder: A Balance of Mobility and Stability. American Academy of Orthopaedic Surgeons: Rosemont, Illinois. pp 353–367.

Nykanen M (1995). Pulsed ultrasound treatment of the painful shoulder: a randomised double blind placebo-controlled study. Scandinavian Journal of Rehabilitation Medicine, 27: 105–108.

Ogata S, Uhthoff HK (1990). Acromial enthesopathy and rotator cuff tear. Clinical Orthopaedics and Related Research, 254: 39–45.

Oldervoll LM, Ro M, Zwart JA, Svebak S (2001). Comparison of two physical exercise programs for the early intervention of pain in the neck, shoulders and lower back in female hospital staff. Journal of Rehabilitation Medicine, 33: 156–161.

Ozaki J, Fujimoto S, Nakagawa Y, Masuhara K, Tamai S (1988). Tears of the rotator cuff of the shoulder associated with pathological changes of the acromion. Journal of Bone and Joint Surgery, 70A: 1224–1230.

Pal B, Quennell P, Hawes S (2000). A review of accident and emergency attendances for non-traumatic musculoskeletal complaints. Rheumatology International, 19: 171–175.

Palmer K, Walker-Bone K, Linaker C, Reading I, Kellingray S, Coggon D, Cooper C (2000). The Southampton examination

schedule for the diagnosis of musculoskeletal disorders of the upper limb. Annals of the Rheumatic Diseases, 59: 5–11.

Pancoast HK (1932). Superior pulmonary sulcus tumor: tumor characterized by pain, Horner's syndrome, destruction of bone and atrophy of hand muscles. Journal of American Medical Association, 99: 1391–1396.

Perron M, Malouin F (1997). Acetic acid iontophoresis and ultrasound for the treatment of calcifying tendinitis of the shoulder: a randomised controlled trial. Archives of Physical Medicine and Rehabilitation, 78: 379–384.

Petri M, Dobrow R, Neiman R, Whiting-O'Keefe Q, Seaman WE (1987). Randomized, double-blind, placebo-controlled study of the treatment of the painful shoulder. Arthritis and Rheumatism, 30: 1040–1045.

Phillips AC, Polisson RP (1997). The rational initial clinical evaluation of the patient with musculoskeletal complaints. American Journal of Medicine, 103: 7S–11S.

Plafki C, Steffen R, Willburger RE, Wittenberg RH (2000). Local anaesthetic injection with and without corticosteroids for subacromial impingement syndrome. International Orthopaedics, 24: 40–42.

Pope DP (2001) Association of occupational physical demands and psychosocial working environment with disabling shoulder pain. Annals of the Rheumatic Diseases, 60: 852–858.

Price CIM, Pandyan AD (2002). Electrical stimulation for preventing and treating post-stroke shoulder pain. In: The Cochrane Library; Issue 2. Oxford: Update Software.

Protzman RR (1980). Anterior instability of the shoulder. Journal of Bone and Joint Surgery, 62A: 909–918.

Punnett L, Fine LJ, Keyserling WM, Herrin GD, Chaffin DB (2000). Shoulder disorders and postural stress in automobile assembly work. Scandinavian Journal of Work, Environment and Health, 26: 283–291.

Quigley TB (1963). Indications for manipulation and corticosteroids in the treatment of stiff shoulders. Surgical Clinics of North America, 43: 1715–1720.

Rahme H, Solem-Bertoft E, Westerberg CE, Lundberg E, Sorensen S, Hilding S (1998). The subacromial impingement syndrome. A study of results of treatment with special emphasis on predictive factors and pain-generating mechanisms. Scandinavian Journal of Rehabilitation Medicine, 30: 253–262.

Reeves B (1975). The natural history of the frozen shoulder. Scandinavian Journal of Rheumatology, 4: 193–196.

Reid D, Saboe L, Chepeha J (1996). Anterior shoulder instability in athletes: comparison of isokinetic resistance exercises and an electromyographic feedback reeducation program: a pilot program. Physiotherapy Canada, 48: 251–256.

Reinus WR, Hatem SF (1998). Fractures of the greater tuberosity presenting as rotator cuff abnormality: magnetic resonance demonstration. Journal of Trauma, 44: 670–675.

Richardson A (1975). Ernest Fletcher Lecture. The painful shoulder. Proceedings of the Royal Society of Medicine, 68: 731–736.

Riddle DL, Rothstein JM, Lamb RL (1987). Goniometric reliability in a clinical setting: shoulder measurements. Physical Therapy, 67: 668–673.

Rissen D, Melin B, Sandsjo L, Dohns I, Lundberg U (2000). Surface EMG and psychophysical stress reactions in women during repetitive work. European Journal of Applied Physiology, 83: 215–222.

Ritchie LD, Sugawara S, Ishigami M, Satoh Y, Suzuki K (1995). Local action transcutaneous flurbiprofen (flurbiprofen LAT) in the treatment of acute musculoskeletal conditions: a review of European and Japanese experience. Journal of Orthopaedic Radiology, 8: 134–139.

Rizk TE, Pinals RS, Talaiver AS (1991). Corticosteroid injections in adhesive capsulitis: investigation of their value and site. Archives of Physical Medicine and Rehabilitation, 72: 20–22.

Roebuck DJ (1995). Diagnostic imaging: reversing the focus. Medical Journal of Australia, 162: 275.

Rompe JD, Zoellner J, Nafe B (2001). Shock wave therapy versus conventional surgery in the treatment of calcific tendinitis of the shoulder. Clinical Orthopaedics and Related Research, 387: 72–82.

Rothman RH, Parke W (1965). The vascular anatomy of the rotator cuff. Clinical Orthopaedics and Related Research, 41: 176.

Rowe CR, Zarins B (1981). Recurrent transient subluxation of the shoulder. Journal of Bone and Joint Surgery, 63A: 863–872.

Russe O, Gerhardt JJ, King PS (1976). An Atlas of Examination, Standard Measurements and Diagnosis in Orthopaedics and Traumatology (2nd Edition). Hans Huber: Berne.

Sambrook PN (1996). Osteoporosis. Medical Journal of Australia, 165: 332–336.

Saunders L (1995). The efficacy of low level laser therapy in supraspinatus tendinitis. Clinical Rehabilitation, 9: 126–134.

Saxton JM (2000). A review of current literature on physiological tests and soft tissue biomarkers applicable to work-related upper limb disorders. Occupational Medicine, 50: 121–130.

Schmitt J, Haake M, Tosch A, Hildebrand R, Deike B, Griss P (2001). Low-energy extracorporeal shock-wave treatment (ESWT) for tendinitis of the supraspinatus. A prospective, randomised study. Journal of Bone and Joint Surgery, 83: 873–876.

Scutellari PN, Orzincolo C (1998). Rheumatoid arthritis: sequences. European Journal of Radiology, 27: 531–538.

Seeger LL (1989a). Magnetic resonance imaging of the shoulder. Clinical Orthopaedics and Related Research, 244: 48–59.

Seeger LL (1989b). Physical principles of magnetic resonance imaging. Clinical Orthopaedics and Related Research, 244: 7–16.

Shaffer B, Tibone JE, Kerlan RK (1992). Frozen shoulder: a long term follow-up. Journal of Bone and Joint Surgery, 74A: 738–746.

Shehab D, Adham N (2000). Comparative effectiveness of ultrasound and transcultaneous electrical stimulation in treatment of periarticular shoulder pain. Physiotherapy Canada, 52(3): 208–210, 214.

Sher JS, Iannoti JP, Williams GR, Herzog RJ, Kneeland JB, Lisser S, Patel N (1998). The effect of magnetic resonance imaging on clinical decision making. Journal of Shoulder and Elbow Surgery, 7: 205–209.

Sher JS, Uribe JW, Posada A, Murphy BJ, Zlatkin MB (1995). Abnormal findings on magnetic resonance images of asymptomatic shoulders. Journal of Bone and Joint Surgery, 77A: 10–15.

Shibata Y, Midorikawa K, Emoto G, Naito M (2001). Clinical evaluation of sodium hyaluronate for the treatment of patients with rotator cuff tear. Journal of Shoulder and Elbow Surgery, 10: 209–216.

Sileghem A, Verstraeten A, Dequeker J (1991). Double-blind, randomized, parallel-group study of the efficacy and safety of proglumetacin and naproxen in periarthritis of the shoulder or elbow. Current Therapeutic Research, Clinical and Experimental, 50: 93–100.

Skov T, Borg V, Orhede E (1996). Psychosocial and physical risk factors for musculoskeletal disorders of the neck, shoulders, and lower back in salespeople. Occupational and Environmental Medicine, 53: 351–356.

Smith KL, Harryman DT II, Antoniou J, Campbell B, Sidles JA, Matsen FA III (2000). A prospective, multipractice study of shoulder function and health status in patients with documented

rotator cuff tears. Journal of Shoulder and Elbow Surgery, 9: 395–402.

Smith SP, Devaraj VS, Bunker TD (2001). The association between frozen shoulder and Dupuytren's disease. Journal of Shoulder and Elbow Surgery, 10: 149–151.

Snels IAK, Beckerman H, Lankhorst GJ, Bouter LM (2000a). Treatment of hemiplegic shoulder pain in the Netherlands: results of a national survey. Clinical Rehabilitation, 14: 20–27.

Snels IAK, Beckerman H, Twisk JWR, Dekker JHM, De-Koning P, Koppe PA, Lankhorst GJ, Bouter LM (2000b). Effect of triamcinolone acetonide injections on hemiplegic shoulder pain: a randomized clinical trial. Stroke, 31: 2396–2401.

Soave G, Bruni G, Lavezzari M, Parrinello L (1982). Indoprofen versus indomethacin in acute painful shoulder and other soft-tissue rheumatic complaints. Journal of International Medical Research, 10: 99–103.

Solomon DH, Bates DW, Schaffer JL, Horsky J, Burdick E, Katz JN (2001). Referrals for musculoskeletal disorders. Journal of Rheumatology, 28: 2090–2095.

Solomon DH, Schaffer JL, Katz JN, Horsky J, Burdick E, Nadler E, Bates DW (2000). Can history and physical examination be used as markers of quality? An analysis of the initial visit note in musculoskeletal care. Medical Care, 38: 383–391.

Spangehl MJ, Hawkins RH, McCormack RG, Loomer RL (2002). Arthroscopic versus open acromioplasty: a prospective, randomised, blinded study. Journal of Shoulder and Elbow Surgery, 11: 101–107.

Speed CA, Richards C, Nichols D, Burnet S, Wies JT, Humphreys H, Hazleman BL (2002). Extracorporeal shock-wave therapy for tendonitis of the rotator cuff. Journal of Bone and Joint Surgery British Volume, 84: 509–512.

Speed JS (1966). Personal communication (1952). In: Crenshaw AH, Kilgore WE. Surgical treatment of bicipital tenosynovitis. Journal of Bone and Joint Surgery, 48A: 1496–1502.

Sperber A, Hamberg P, Karlsson J, Sward L, Wredmark T (2001). Comparison of an arthroscopic and an open procedure for post-traumatic instability of the shoulder: a prospective, randomized multicenter study. Journal of Shoulder and Elbow Surgery, 10: 105–108.

Stiles RG, Otte MT (1993). Imaging of the shoulder. Radiology, 188: 603–613.

Struhl S (2002). Anterior internal impingement: An arthroscopic observation. Arthroscopy, 18: 2–7.

Sun KO, Chan KC, Lo SL, Fong DY (2001). Acupuncture for frozen shoulder. Hong Kong Medical Journal, 7: 381–391.

Szomor ZL, Wang MX, Kruller A, Murrell GAC, Farmer KM, Kirkham BW, Bonar F, Kirkham BW (2001). Differential expression of cytokines and nitric oxide synthase isoforms in human rotator cuff bursae. Annals of the Rheumatic Diseases, 60: 431–432.

Taverna E, Parrini M, Cabitza P (1990). Laserterapia IR versus placebo nel trattamento di alcune patologie a carcio dell'apparato locomotore. Minerva Ortopedica E Traumatologica, 41: 631–636.

Teefey SA, Hasan SA, Middleton WD, Patel M, Wright RW, Yamaguchi K (2000a). Ultrasonography of the rotator cuff. A comparison of ultrasonographic and arthroscopic findings in one hundred consecutive cases. Journal of Bone and Joint Surgery, 82: 498–504.

Teefey SA, Middleton WD, Bauer GS, Hildebolt CF, Yamaguchi K (2000b). Sonographic differences in the appearance of acute and chronic full-thickness rotator cuff tears. Journal of Ultrasound in Medicine, 19: 377–378.

Tempelhof S, Rupp S, Seil R (1999). Age-related prevalence of rotator cuff tears in asymptomatic shoulders. Journal of Shoulder and Elbow Surgery, 8: 296–299.

Torstensen AT, Hollinshead RM (1999). Comparison of magnetic resonance imaging and arthroscopy in the evaluation of shoulder pathology. Journal of Shoulder and Elbow Surgery, 8: 42–45.

Travlos J, Goldberg I, Boome RS (1990). Brachial plexus lesions associated with dislocated shoulders. Journal of Bone and Joint Surgery, 72B: 68–71.

Tsai JC, Zlatkin MB (1990). Magnetic resonance imaging of the shoulder. Radiologic Clinics of North America, 28: 279–291.

Tuite MJ, Turnbull JR, Orwin JF (1998). Anterior versus posterior, and rim-rent rotator cuff tears: prevalence and MR sensitivity. Skeletal Radiology, 27: 237–243.

Tyson LL (1995). Imaging of the painful shoulder. Current Problems in Diagnostic Radiology, 24: 110–140.

van der Heijden GJM (1999). Shoulder disorders: a state of the art review. In: Croft P, Brooks PM (eds). Bailliere's Clinical Rheumatology, 13: 287–309.

van der Heijden GJM, van der Windt DAW, De Winter AF (1997). Physiotherapy for patients with soft tissue shoulder disorders: a systematic review of randomised clinical trials. British Medical Journal, 315: 25–30.

van der Heijden GJM, van der Windt DAW, Kleijnen J, Koes BW, Bouter LM (1996). Steroid injections for shoulder disorders: a systematic review of randomised clinical trials. British Journal of General Practice, 46: 309–316.

van der Windt D, Croft P, Penninx B (2002). Neck and upper limb pain: more pain is associated with psychological distress and consultation rate in primary care. Journal of Rheumatology, 29: 564–569.

van der Windt DA, Koes BW, Boeke AJ, Deville W, DeJong BA, Bouter LM (1996). Shoulder disorders in general practice: prognostic indicators of outcome. British Journal of General Practice, 46: 519–523.

van der Windt DA, Koes BW, Deville W, Boeke AJ, DeJong BA, Bouter LM (1998). Effectiveness of corticosteroid injections versus physiotherapy for treatment of painful stiff shoulder in primary care: randomized trial. British Medical Journal, 317; 1292–1296.

van der Windt DA, van der Heijden GJ, Scholten RJ, Koes BW, Bouter LM (1995). The efficacy of non-steroidal anti inflammatory drugs (NSAIDS) for shoulder complaints. A systematic review. Journal of Clinical Epidemiology, 48: 691–704.

van der Windt DA, van der Heijden GJM, van den Berg SG, ter Riet G, de Winter AF, Bouter LM (1999). Ultrasound therapy for musculoskeletal disorders: a systematic review. Pain, 81: 257–171.

van Moppes FI, Veldkamp O, Roorda J (1995). Role of shoulder ultrasonography in the evaluation of the painful shoulder. European Journal of Radiology, 19: 142–146.

Vecchio PC, Hazleman BL, King RH (1993). A double blind trial comparing subacromial methylprednisolone and lignocaine in acute rotator cuff tendinitis. British Journal of Rheumatology, 32: 743–745.

Veldman PHJM, Goris RJA (1995). Shoulder complaints in patients with reflex sympathetic dystrophy of the upper extremity. Archives of Physical Medicine and Rehabilitation, 76: 239–242.

Vidal L, Kneer W, Baturone M, Sigmund R (2001). Meloxicam in acute episodes of soft-tissue rheumatism of the shoulder. Inflammation Research, 50: S24–29.

Viikari-Juntura E, Takala E, Riihimaki H, Martikainen R, Jappinen P (2000). Predictive validity of symptoms and signs in the neck and shoulders. Journal of Clinical Epidemiology, 53: 800–808.

Waling K, Jarvholm B, Sundelin G (2002). Effects of training on female trapezius myalgia: an intervention study with a 3-year follow-up period. Spine, 27: 789–796.

Walsh RM, Sadowski GE (2001). Systemic disease mimicking musculoskeletal dysfunction: a case report involving referred shoulder pain. Journal of Orthopaedic and Sports Physical Therapy, 31: 696–701.

Wang CJ, Ko JY, Chen HS (2001). Treatment of calcifying tendinitis of the shoulder with shock wave therapy. Clinical Orthopaedics and Related Research, 387: 83–89.

Weissberg DL, Resnick D, Taylor A, Becker M, Alazraki N (1978). Rheumatoid arthritis and its variants: analysis of scintiphotographic, radiographic and clinical examinations. American Journal of Rheumatology, 131: 665–673.

Welch WC (1994). Systemic malignancy presenting as neck and shoulder pain. Archives of Physical and Medical Rehabilitation, 75: 918–920.

Weldon III EJ, Richardson AB (2001). Upper extremity overuse injuries in swimming: a discussion of swimmer's shoulder. Clinics in Sports Medicine, 20: 423–438.

Welfling J, Cariot M, Labrousse CE (1964). Artrose de l'épaule et dérotation de la coiffe des rotateurs. Revue du rhumatisme et des maladies osteo-articulaires, 31: 629.

White A, Tuite J (1996). The accuracy and efficacy of shoulder injections in restrictive capsulitis. Journal of Orthopaedic Rheumatology, 9: 37–40.

White RH, Paull DM, Fleming KW (1986). Rotator cuff tendinitis: comparison of subacromial injection of a long acting corticosteroid versus oral indomethacin therapy. Journal of Rheumatology, 13: 608–613.

Wielandts L, Dequeker J (1979). Double blind trial comparing fentiazac with phenylbutazone in patients with tendinitis. Current Medical Research and Opinion, 6 (Suppl 2): 85–89.

Wiener SN, Seitz WH (1993). Sonography of the shoulder in patients with tears of the rotator cuff: accuracy and value for selecting surgical options. American Journal of Roentgenology, 160: 103–110.

Williams JG, Callaghan M (1990). Comparison of visual estimation and goniometry in determination of a shoulder joint angle. Physiotherapy, 76: 655–657.

Winters JC, Jorritsma W, Groenier KH, Sobel JS, Meyboom De Jong B, Arendzen HJ (1999a). Treatment of shoulder complaints in general practice: long term results of a randomised, single blind study comparing physiotherapy, manipulation, and corticosteroid injection. British Medical Journal, 318: 1395–1396.

Winters JC, Sobel JS, Groenier KH, Arendzen JH, Meyboom-de Jong B (1997a). Comparison of physiotherapy, manipulation and corticosteroid injection for treating shoulder complaints. British Medical Journal, May: 1320–1325.

Winters JC, Sobel JS, Groenier KH, Arendzen JH, Meyboom-de Jong B (1997b). The course of pain and the restriction of mobility in patients with shoulder complaints in general practice. Rheumatology International, 16: 219–225.

Winters JC, Sobel JS, Groenier KH, Arendzen JH, Meyboom-de Jong B (1999b). The long-term course of shoulder complaints: a prospective study in general practice. Rheumatology (Oxford, England), 38: 160–163.

Withrington RH, Girgis FL, Seifert MH (1985). A placebo-controlled trial of steroid injections in the treatment of supraspinatus tendinitis. Scandinavian Journal of Rheumatology, 14: 76–78.

Workman TL, Burkhard TK, Resnick D, Goff WB, Balsara ZN, Davis DJ, Lapoint JM (1992). Hill-Sachs lesion: comparison of detection with MRI, radiography, and arthroscopy. Radiology, 185: 847–852.

Worland RL, Jensen KL, Williams GR, Russell IJ, Rockwood CA Jr. (2000) Rotator cuff arthropathy (Letters). Journal of Bone and Joint Surgery, 82A: 1670–1671.

World Health Organisation (1986). International Classification of Impairments, Disabilities and Handicaps. WHO: Geneva.

Yanagisawa K, Hamada K, Gotoh M, Tokunaga T, Oshika Y, Tomisawa M, Hwan-Lee Y, Handa A, Kijima H, Yamazaki H, Nakamura M, Ueyama Y, Tamaoki N, Fukuda H (2001). Vascular endothelial growth factor (VEGF) expression in the subacromial bursa is increased in patients with impingement syndrome. Journal of Orthopaedic Research, 19: 448–455.

Yergason RM (1931). Supination sign. Journal of Bone and Joint Surgery, 131:60.

Yocum LA (1983). Assessing the shoulder. Clinics in Sports Medicine, 2: 281–289.

Zlatkin MB, Iannotti JP, Roberts MC, Esterhai JL, Dalinka MK, Kressel HY, Schwartz JS, Lenkinski RE (1989). Rotator cuff tears: diagnostic performance of MR imaging. Radiology, 172: 223–229.

Zuinen C (1993). Diclofenac/misoprostol vs diclofenac/placebo in treating acute episodes of tendinitis/bursitis of the shoulder. Drugs, 45: 17–23.

Chapter 8

Anterior Knee Pain

➔ *This document was developed by a multi-disciplinary group to provide the evidence for diagnosis and treatment of acute anterior knee pain, specifically patellofemoral pain, a benign condition of the anterior knee.*

Patellofemoral pain is a common condition diagnosed on the basis of features identified during clinical assessment. The incidence of patellofemoral pain in the general population is reported in some studies to be as high as one in four, with the proportion increasing in athletes (Levine 1979; Outerbridge 1964). The rate is around 7% in young active adults (Witvrouw et al. 2000), between 1% and 15% in army recruits (Almeida et al. 1999a,b; Heir and Glomsaker 1996; Jones et al. 1993; Kowal 1980; Milgrom et al. 1991; Schwellnus et al. 1990; Shwayhat et al. 1994) and between 2% and 30% of presentations to sports medicine clinics (Baquie and Brukner 1997; Clement et al. 1981; DeHaven and Lintner 1986; Derscheid and Feiring 1987; Devereaux and Lachman 1984; James et al. 1978; Matheson et al. 1989; Pagliano and Jackson 1987).

While patellofemoral pain may persist, regular activity provides relief in the majority of cases. Surgery appears to offer no advantage. The aim in management of patellofemoral pain is to:

- *Exclude potentially serious causes of acute knee pain.*
- *Promote effective self-management of symptoms through the provision of timely and appropriate advice.*

Definition of Patellofemoral Pain

The term 'patellofemoral' pain refers to pain predominantly experienced in the anterior aspect of the knee, in close proximity to the patellofemoral complex. The term does not infer anything more than the probable site of pain origin and is appropriate for practical purposes to classify anterior knee pain problems of otherwise unknown origin (Crossley et al. 2001).

The diagnosis of patellofemoral pain is based on two key elements:

- The area in which the pain is perceived.
- The exclusion of other causes of anterior knee pain.

In these guidelines, the term 'acute' refers to pain that has been present for less than three months (Merskey 1979); it does not refer to the severity or quality of pain. Chronic pain is pain that has been present for at least three months (Merskey and Bogduk 1994).

Scope

These guidelines describe the diagnosis and treatment of patellofemoral pain that is not attributable to a particular pathology. The following conditions are beyond the scope of the document:

- serious conditions: infection, tumour, fracture, neurological conditions, inflammatory arthropathies
- osteoarthritis and other specific conditions (e.g. Fat Pad Syndrome, Osgood-Schlatter Disease, Sinding-Larsen-Johannson Syndrome, plica syndromes, prepatellar and infrapatellar bursitis, tendonitis, complex regional pain syndromes, osteonecrosis)
- medial, lateral and posterior knee pain
- internal mechanical derangements (e.g. meniscal tear, cruciate ligament damage)
- conditions characterised by pain referred from other structures (e.g. hip)
- neuropathic pain
- pain in the anterior thigh and other regions of the knee

Guideline Development Process

This guideline for the management of anterior knee pain (patellofemoral pain) was developed using a combination of processes. An existing guideline developed in 1998 and updated in 2001 formed the basis for this document. Further updating has now occurred involving a process in line with current National Health and Medical Research Council (NHMRC) standards for guideline development (1999a).

The update of the existing work involved a review of the evidence on anterior knee pain published since the most recent update of the existing guidelines. A multi-disciplinary group identified, appraised and synthesised the available literature on diagnosis, prognosis and interventions for anterior knee pain. Studies were assessed against selection criteria and those meeting the criteria for inclusion were used to update the existing text of the guidelines. All studies assessed for this

update are included in either the Table of Included Studies or the Table of Excluded Studies (refer to Appendix E). Studies that were included in the existing guidelines are not described in these tables.

Relevant studies on areas related to diagnosis were identified in the literature search and used to update the sections on aetiology and prevalence, history, physical examination and ancillary investigations where possible. These sections are largely comprised of the existing work developed using a conventional literature review. Group members had the opportunity to evaluate the literature forming the basis of the existing guidelines, review the interpretation of the literature, nominate additional articles to undergo the appraisal process or request that an article be re-appraised.

Study Selection Criteria

The criteria oulined in the chart below guided the literature search and appraisal.

Search Strategy

Sensitive searches were performed; electronic searches were limited to adults, humans, and articles published in English in peer-reviewed journals. Where available, methodological filters were used. There were no hand searches conducted.

Searches for information on interventions for patellofemoral pain spanned the period from 2000 to 2002. This was based on the availability of a recently published systematic review on interventions for patellofemoral pain (Crossley et al. 2001). Searches for information on diagnosis and prognosis spanned the period from 1998 to 2002.

Articles that group members felt were important to the topic that did not appear in the search results were submitted to the review process.

The following databases were searched in October 2002:

- PubMed (Clinical Queries)
- CINAHL
- EMBASE — Physical and Rehabilitation Medicine
- The Cochrane Library, 2002, Issue 2

Access to CHIROLARS and PEDro was unavailable for this update.

Search Terms

- Knee pain .exp
- Patellofemoral joint .exp
- Anterior .tw
- Therapies .exp
- Diagnosis .exp
- Prognosis .exp
- Surgery .exp
- Drug therapy .exp
- Drug therapy .exp
- Patellofemoral pain .mp
- Treatment .mp
- Controlled trial
- Randomised
- Clinical trial
- Drug therapy .exp
- Aetiology
- Systematic review .tw

Study Selection Criteria

DIAGNOSIS	
The sections on Aetiology and Prevalence, History, Examination and Investigations comprise information from the existing draft (developed by conventional literature review) combined and updated with relevant articles appraised according to the following inclusion and exclusion criteria:	
Inclusion criteria	Systematic reviews, cross sectional studies Patellofemoral pain
Exclusion criteria	Chronic pain Specific diseases and conditions
PROGNOSIS	
Information from the existing draft was combined with relevant articles located and appraised according to the following inclusion and exclusion criteria:	
Inclusion criteria	Systematic reviews, cohort studies Patellofemoral pain
Exclusion criteria	Chronic pain
INTERVENTIONS	
A review of the literature was undertaken according to the following inclusion and exclusion criteria. The information was used to update the existing material:	
Inclusion criteria	Systematic reviews, randomised controlled trials Patellofemoral pain
Exclusion criteria	Chronic pain

Summary of Key Messages: Acute Pain Management

	EVIDENCE LEVEL
Management Plan	
It is recommended that the clinician and patient develop a management plan for acute musculoskeletal pain comprising the elements of assessment, management and review: • Assessment — Conduct a history and physical examination to assess for the presence of serious conditions; ancillary investigations are not generally indicated unless features of serious conditions are identified. • Management — Provide information, assurance and advice to resume normal activity and discuss other options for pain management as needed. • Review — Reassess the pain and revise the management plan as required.	CONSENSUS: Steering Committee
Non-Pharmacologic Interventions	
Simple interventions (providing information, assurance and encouraging reasonable maintenance of activity) may be used alone or in combination with other interventions for the successful management of acute musculoskeletal pain.	CONSENSUS: Steering Committee
Pharmacologic Interventions	
Specific pharmacologic interventions may be required to relieve pain; such agents can be used in conjunction with non-pharmacologic interventions.	CONSENSUS: Steering Committee; NHMRC 1999b
Paracetamol or other simple analgesics, administered regularly, are recommended for relief of mild to moderate acute musculoskeletal pain.	CONSENSUS: Steering Committee; NHMRC 1999b
Where paracetamol is insufficient for pain relief, a non-steroidal anti-inflammatory (NSAID) medication may be used, unless contraindicated.	CONSENSUS: Steering Committee; NHMRC 1999b
Oral opioids may be necessary to relieve severe musculoskeletal pain. It is preferable to administer a short-acting agent at regular intervals, rather than on a pain-contingent basis. Ongoing need for opioid analgesia is an indication for reassessment.	CONSENSUS: Steering Committee; NHMRC 1999b
Adjuvant agents such as anticonvulsants and antidepressants are not recommended in the management of acute musculoskeletal pain.	CONSENSUS: Steering Committee; NHMRC 1999b
Any benefits from muscle relaxants may be outweighed by their adverse effects, therefore they cannot be routinely recommended.	CONSENSUS: Steering Committee

Summary of Key Messages: Effective Communication

	EVIDENCE LEVEL
Clinicians should work with patients to develop a management plan so that patients know what to expect, and understand their role and responsibilities.	CONSENSUS: Steering Committee
Information should be conveyed in correct but neutral terms, avoiding alarming diagnostic labels; jargon should be avoided.	CONSENSUS: Steering Committee
Explanation is important to overcome inappropriate expectations, fears or mistaken beliefs that patients may have about their condition or its management.	CONSENSUS: Steering Committee
Printed materials and models may be useful for communicating concepts.	CONSENSUS: Steering Committee
Clinicians should adapt their method of communication to meet the needs and abilities of each patient.	CONSENSUS: Steering Committee
Clinicians should check that information that has been provided has been understood; barriers to understanding should be explored and addressed.	CONSENSUS: Steering Committee

Summary of Key Messages: Anterior Knee Pain

DIAGNOSIS	EVIDENCE LEVEL
Aetiology and Prevalence	
'Patellofemoral pain' is a general term used to describe idiopathic pain arising from the anterior knee/patellofemoral region that is of otherwise unknown origin.	CONSENSUS: Steering Committee
Anterior knee pain is commonly idiopathic; serious causes are rare.	*LEVEL IV: Kaempffe 1995; Ferguson et al. 1997; Kaandorp et al. 1995
Intrinsic risk factors for knee pain may include female gender, knee anatomy, joint laxity, muscle imbalance and prior injury. Extrinsic risk factors include occupation, sport and obesity.	*LEVEL IV: Kujala et al. 2001; Reider et al. 1981a,b; Witvrouw et al. 2000; Tanaka et al. 1989; Cooper et al. 1994
History	
The history provides information on possible causes of anterior knee pain and assists the identification of serious underlying conditions	CONSENSUS: Steering Committee
Physical Examination	
Although examination techniques lack specificity for diagnosing knee disorders, physical examination may assist the identification of serious conditions underlying anterior knee pain.	*LEVEL III, IV: Daniel 1991; Cook et al. 2001; Cushnagan et al. 1990; Biedert and Warnke 2001
Ancillary Investigations	
Indications for plain radiography are a history of trauma and: qualification under one of the Knee Rules, or sudden onset of severe pain, or alerting features of a serious condition.	*LEVEL III, IV: Chapman-Jones et al. 1998; Petit et al. 2001; Stiell et al. 1996; Seaberg and Jackson 1994; Bauer et al. 1995
Suspected fracture in the presence of a normal plain radiograph is an indication for CT scan.	CONSENSUS: Steering Committee
The presence of alerting features of a serious condition is an indication for the use of MRI.	CONSENSUS: Steering Committee
Swelling or potential rupture of anterior knee structures are indications for the use of ultrasound.	*LEVEL IV: Bianchi et al. 1994
Terminology	
The term 'patellofemoral pain' describes anterior knee pain for which there is no specific identifiable cause; it refers to the probable anatomical site of origin and is synonymous with retropatellar and patellofemoral joint pain.	CONSENSUS: Steering Committee
PROGNOSIS	**EVIDENCE LEVEL**
Multiple studies on a range of populations show a trend towards improvement with time; however, anterior knee pain persists to some degree in the majority of people.	*LEVEL IV: Nimon et al. 1998; Milgrom et al. 1996
INTERVENTIONS	**EVIDENCE LEVEL**
Evidence of Benefit	
Advice to Stay Active (Activation) — Maintenance of normal activity has a beneficial effect on patellofemoral pain compared to no treatment and to the use of patellofemoral orthoses.	LEVEL II: Finestone et al. 1993
Injection Therapy — There is evidence that injection therapy (treatment and placebo saline) is effective for the management of patellofemoral pain in the short term compared to no injection therapy.	LEVEL II: Kannus et al. 1992
Orthoses (Foot) — There is evidence that corrective foot orthoses in combination with quadriceps and hamstring exercises are effective compared to placebo insoles in women with patellofemoral pain.	LEVEL I: Based on a systematic review (Crossley et al. 2001) that located one RCT (Eng and Pierrynowski 1993)

Anterior Knee Pain continued

Exercises — A six-week regimen of quadriceps muscle retraining, patellofemoral joint mobilisation, patellar taping and daily home exercises significantly reduces patellofemoral pain compared to placebo in the short term. Eccentric quadriceps exercises produce better functional outcomes compared to standard quadriceps strengthening exercises.	LEVEL II: Based on one RCT (Crossley et al. 2002) LEVEL I: Based on a systematic review (Crossley et al. 2001) of eight RCTs
Conflicting Evidence	
Orthoses (Patellofemoral) — There is conflicting evidence that patellofemoral orthoses are effective compared to other interventions and to no treatment for patellofemoral pain.	LEVEL I: Based on two systematic reviews (Crossley et al. 2001; D'hondt et al. 2002)
Insufficient Evidence	
Acupuncture — There are no randomised controlled studies evaluating the effect of acupuncture for relief of patellofemoral pain.	No Level I or II evidence
Analgesics (simple and opioid) — There are no randomised controlled studies of the effectiveness of paracetamol or opioids versus placebo in the treatment of patellofemoral pain.	No Level I or II evidence
Electrical Stimulation — There are no randomised controlled studies of the effectiveness of electrical stimulation of the quadriceps muscle for patellofemoral pain. There is insufficient evidence that one form of electrical stimulation of the quadriceps muscle is superior to another for treating patellofemoral pain.	No Level I or II evidence LEVEL II: Callaghan et al. (2001)
Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) — There are no randomised controlled studies of the effectiveness of NSAIDs versus placebo in the treatment of patellofemoral pain. Different types of NSAIDs provide similar relief of patellofemoral pain after five days of use. Serious adverse effects of NSAIDs include gastrointestinal complications (e.g. bleeding, perforation).	No Level I or II evidence LEVEL II: Based on one RCT with limitations (Fulkerson and Folcik 1986) LEVEL I: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 2002)
Patellar Taping — There is insufficient evidence that patellar taping alone is effective in relieving patellofemoral pain, however it may be a useful adjunct to exercise therapy programs.	LEVEL I, II: Based on two systematic reviews (Crossley et al. 2001; Harrison et al. 2001) and one subsequent RCT (Crossley et al. 2002)
Progressive Resistance Braces — There is insufficient evidence that progressive resistance braces are effective in relieving patellofemoral pain compared to no treatment (this treatment is not routinely available in Australia).	LEVEL I: Based on a systematic review (Crossley et al. 2001) that located one RCT (Timm 1998)
Therapeutic Ultrasound — There is insufficient evidence that therapeutic ultrasound is more effective compared to ice massage for the treatment of patellofemoral pain.	LEVEL I: Based on a Cochrane Review (Brosseau et al. 2002b) and two meta-analyses (Gam and Johannsen 1995; van der Windt et al. 1999)
Evidence of No Benefit	
Laser Therapy — There is evidence that low-level laser therapy provides similar effect to sham laser in the management of patellofemoral pain.	LEVEL I: Based on a systematic review (Crossley et al. 2001) that identified one RCT (Rogvi-Hansen et al. 1991)

Note: * Indicative only. A higher rating of the level of evidence might apply (refer to the note in Chapter 1: Executive Summary, Limitations of Findings).

Research Agenda for Anterior Knee Pain

- The aetiology of patellofemoral pain.
- The diagnosis of patellofemoral pain.
- Well-designed, controlled studies on the effectiveness of specific interventions for patellofemoral pain.
- Psychosocial factors and their impact on anterior knee pain and chronic progression.

DIAGNOSIS

>Aetiology and Prevalence

Aetiology of Patellofemoral Pain

Pain in the anterior knee can arise from local conditions of the anterior knee or be referred from other knee structures and distant sites, such as the hip joint. Pain intrinsically derived from the anterior knee can arise from various disorders that affect the bones, joints, ligaments, muscles, adnexiae, nerves and vessels of the anterior knee (see Table 8.1).

There are many possible causes of anterior knee pain. The most likely site of origin is any structure in or around the patellofemoral complex. Knee pain is most commonly caused by intrinsic knee disorders; it is uncommon for pain referred from distant sites to be isolated to the knee.

In practice, it is not easy to identify the specific source of anterior knee pain. History, physical examination and conventional tests are often unhelpful in establishing a precise diagnosis. As a consequence, the term 'patellofemoral pain' is used to describe non-specific pain experienced in the anterior knee that cannot be confidently ascribed to a particular condition and that appears to derive from the patellofemoral joint.

Key Message

'Patellofemoral pain' is a general term used to describe idiopathic pain arising from the anterior knee/patellofemoral region that is of otherwise unknown origin. (Consensus)

There are numerous theories concerning the aetiology of patellofemoral pain. Current evidence suggests that the likely origin of patellofemoral pain may include the anterior synovium (Dye et al. 1998), infrapatellar fat pad (Aynaci et al. 2001; Morini et al. 1998) and retinacula (Kasim and Fulkerson 2000; Sanchis-Alfonso and Rosello-Sastre 2000; Sanchis-Alfonso et al. 2001). In the retinaculum, studies have concentrated on fibrosis, vascular and neural proliferation and neuromata (Kasim and Fulkerson 2000; Sanchis-Alfonso and Rosello-Sastre 2000; Sanchis-Alfonso et al. 2001). Subchondral bone mechanisms are often cited as a possible source of patellofemoral pain, however studies tend to concentrate on the relationship between subchondral bone pain and osteoarthritis rather than patellofemoral pain.

The pathogenesis of patellofemoral pain is unclear (Crossley et al. 2002). Malalignment leading to elevated patellofemoral joint stress has been promoted as a factor by some and refuted by others (Fulkerson 1989; Grelsamer and Klein 1998; Grelsamer 2000; Outerbridge 1961). Although it is assumed that the pain arises from the patellofemoral mechanism, in general this cannot be formally established. Different causes for pain may co-exist.

Research into mechanisms of pathogenesis has included:

- Muscle imbalances (Crossley et al. 2002; Lee et al. 2002)
- Maltracking and malalignment (Biedert and Warnke 2001; Jones et al. 1995; Sanchis-Alfonso and Rosello-Sastre 2000; Thomee et al.1995b; Witonski 2002)
- Patellofemoral joint stress (refuted) (Brechter and Powers 2002)
- Gender, muscle strength and motion (Csintalan et al. 2002)
- Loading and trauma (Thompson et al. 1993)
- Malalignment and ilio-tibial band tightness (Winslow and Yoder 1995).

Chondromalacia Patellae

Chondromalacia patellae refers to the state of the patellar articular cartilage. This term has been used interchangeably with patellofemoral pain (Kivimaki et al. 1994). In earlier classifications, identification of chondral damage by arthroscopy in a patient with patellofemoral pain would render a diagnosis of chondromalacia patellae (Bentley and Dowd 1984; Carson et al. 1984; Fulkerson and Hungerford 1990; Insall 1979; Kivimaki et al. 1994). However, several studies have shown poor correlation between articular cartilage damage and patellofemoral pain (Darracott and Vernon-Roberts 1971; DeHaven and Collins 1975; DeHaven et al. 1979; Hvid et al. 1981; Insall 1980; Leslie and Bentley 1978; Shino et al. 1993a,b). Cases with changes on arthroscopy consistent with chondromalacia patellae in asymptomatic patients have been described, as well as cases of pain and no changes (Carson et al. 1984; Fairbank et al. 1984). Additionally, cases of patellofemoral pain and abnormal xray have been found with normal articular cartilage (Goodfellow et al. 1976). The presence of severe cartilage damage, especially on the medial patellar facet, may not cause pain (Abernethy et al. 1978; Goodfellow et al. 1976; Meachim and Emery 1974). Softening and fibrillation of the patella can be a natural consequence of aging (Bennett et al. 1942; Collins and Meachim 1961; Owre 1936). The relationship between patellofemoral pain and chondromalacia patellae remains unclear (Kannus et al. 1999).

Table 8.1

Potential Causes of Anterior Knee Pain

Serious conditions	Fracture; Tumour; Infection; Inflammatory arthropathies; Osteonecrosis
Intrinsic mechanical conditions	Patella (patellar instability and dislocation; infrapatellar contracture syndrome; medial patellar subluxation); Patellar tendon (patellar tendonopathy; Osgood-Schlatter Disease; Sinding-Larsen-Johannson Syndrome); Quadriceps tendonopathy; Fat Pad Syndrome; Prepatellar and infrapatellar bursitis; Plicae; Osteochondritis dissecans; Sprains and strains
Referred pain	Somatic conditions (e.g. diseases of the hip joint)
Neurological conditions	Infrapatellar neuralgia

Serious Conditions Causing Anterior Knee Pain

Serious conditions of the anterior knee are those that manifest as anterior knee pain but which pose more serious health risks than common mechanical disorders of the knee apparatus. Serious conditions causing anterior knee pain, such as fractures, tumours and infections are rare and can be adequately screened through history and physical examination.

Fracture

The alerting features for fracture as the cause of acute anterior knee pain are trauma and age. In the general population, significant fractures occur only in people with a history of major trauma. Minor trauma is not a risk factor for fracture unless the patient has osteoporosis. In such cases, age greater than 50 years is a risk factor although the literature suggests that those with osteoporotic fractures following minor trauma tend to be substantially older than this limit (Scavone et al. 1981). Consumption of corticosteroids is another risk factor for osteoporosis (Blake and Fogelman 2002). Pathological fractures associated with cancer, Paget's disease and osteopaenia may occur after minimal trauma or even in the absence of trauma. Clinicians should be alert to the possibility of occult trauma in people with impaired memory or those with difficulty communicating.

Tumour

Primary or secondary tumours in and around the knee are rare, however the knee is the most commonly affected peripheral site. Of all areas of the appendicular skeleton, the distal femur is the most often affected, followed by the proximal tibia and then the proximal humerus (Kaempffe 1995). Precise figures for the prevalence of knee cancer have not been determined, but the pre-test probability of a patient in a primary care setting presenting with knee pain and having cancer as the cause is probably substantially less than 1%. Primary tumours in the knee are extremely rare. One study of a consecutive series of 587 patients identified eight cases (1.36%) of primary tumours of the patella in patients undergoing surgery for benign or malignant bone tumours (Ferguson et al. 1997).

Data on the prevalence of lower limb tumours are difficult to interpret because of referral bias. The following tumours have been described, however, frequency will vary with different age groups: synovial sarcoma, malignant fibrous histiocytoma, liposarcoma, malignant peripheral nerve sheath tumour and fibrosarcoma (Kransdorf 1995).

Infection

There are no data on the prevalence of infection as a cause of anterior knee pain. The pre-test probability in general practice of infection as the cause of knee pain is likely to be low. Septic arthritis should be considered in patients presenting with acute pain who have undergone recent knee surgery (Indelli et al. 2002), had a joint replacement (Kaandorp et al. 1995), have diabetes (Kaandorp et al. 1995), have rheumatoid arthritis (Kaandorp et al. 1995), have a skin infection (Kaandorp et al. 1995) or are intravenous drug users (Gupta et al. 2001).

The clinical features of septic arthritis have been evaluated prospectively. In a series of 75 subjects, 46 patients had underlying joint disease. Of these, 25 had rheumatoid arthritis and 15 had osteoarthritis. Fifty-six percent of cases involved the knee and 15% involved two or more joints. Clinical features included fever (64%) and leg ulcers (11%). Social deprivation (78%) and intravenous drug use (15%) were risk factors. The mortality rate was 11% (Gupta et al. 2001). Osteomyelitis is most common in the rapidly growing distal femur and proximal tibia (Unkila-Kallio et al. 1993) and may present with knee pain. Plain radiography can be negative in the early stages of this disease; MRI provides the best information about the extent of the disease (Poyhia and Azouz 2000).

Key Message

Anterior knee pain is commonly idiopathic; serious causes are rare. (*Level IV)

Other Specific Conditions Causing Anterior Knee Pain

Inflammatory Arthropathies

The knee can be affected by the inflammatory arthropathies, such as rheumatoid arthritis, psoriatic arthritis, crystal arthritis and reactive arthritis. These conditions are characterised by joint effusion and should be considered in the presence of joint swelling. The differential diagnosis of inflammatory arthropathies may be difficult in their very early stages; a combination of vigilance and consideration of the diagnostic possibilities is required. In the case of crystal arthropathies, synovial fluid analysis is indicated.

Neurological Conditions

It is uncommon for a neurological condition to present primarily as pain isolated to the anterior knee. In most cases neurological pain will be distributed more widely than that usually associated with mechanical knee impairments. However, neurological and somatic pain may co-exist.

Infrapatellar Neuralgia

Injury to the infrapatellar branch of the saphenous nerve is thought to produce pain and tenderness over the anteromedial aspect of the knee (Detenbeck 1972; House and Ahmed 1977; Senegor 1991; Swanson 1983; Worth et al. 1984). It is diagnosed most commonly after previous meniscectomy and vascular surgery.

The condition typically presents with neurogenic features, including sympathetic manifestations. Therefore, local symptoms can include burning and severe, shooting pain triggered by light contact. Night pain is common. Clinical features include local allodynia, hyperalgesia, hyperaesthesia, hypoaesthesia, temperature change, colour change and sweating.

Complex Regional Pain Syndromes

Complex regional pain syndromes (CRPS) may encompass the knee and may be precipitated by peripheral nerve injury or following a relatively trivial local musculoskeletal injury (Armadio 1988; Campa et al. 2001; Kelly et al. 1994; O'Brien et al. 1995; Schwartzman and McLellan 1987). Pain out of proportion to the clinical findings is considered by some to be the most reliable diagnostic criteria for CRPS (Cooper et al. 1989; Merskey and Bogduk 1994; Seale 1989).

Fat Pad (Hoffa's) Syndrome

Although the fat pad has been implicated as a common cause of anterior knee pain there is insufficient evidence to prove this. There are no clear clinical features. However pain and swelling of the infrapatellar fat pad is sometimes referred to as Hoffa's disease. The condition is considered to be impingement and inflammation of the infrapatellar fat pad (Krebs and Parker 1994).

The clinical features include tenderness on palpation and pain in the retro- and infrapatellar regions aggravated by movement of the knee. Physical signs are considered to be tenderness and swelling over the anterior knee, deep to the patellar tendon.

The differential diagnosis of swelling in this region includes benign and malignant tumours including myxoid liposarcoma (Lundy et al. 1997), pigmented villonodular synovitis (Palumbo et al. 1994) and chondroma (Krebs and Parker 1994).

Osteonecrosis

Osteonecrosis is generally an idiopathic condition, usually found in the medial femoral condyle (Ahlback et al. 1968). It presents with local pain and tenderness usually with quite sudden onset. In a case series of 19 patients with atraumatic patellar osteonecrosis, only one patient presented with anterior knee pain localised to the patella (Baumgarten et al. 2001).

Prepatellar and Infrapatellar Bursitis

Bursitis is a natural response to bursal trauma. The bursae most commonly associated with anterior knee pain are the pre- and infrapatellar bursae. Either may be impaired by a single traumatic insult of sufficient magnitude; the more common mechanism of injury is repetitive trauma such as that involved in kneeling. Gout is another potential cause of patellar bursitis. Infection is uncommon. The prepatellar bursa is usually associated with kneeling and leaning forwards, or being on all fours, while the infrapatellar bursa is more likely to be affected by upright kneeling. Prepatellar bursitis presents with anterior knee pain aggravated by kneeling and climbing stairs and is associated with features of inflammation, especially tenderness and swelling anterior to the patella. Infrapatellar bursitis presents in a similar way, however the pain, tenderness and swelling is at the level of the lower border of the patella or in the region of the patellar tendon.

Plica Syndromes

Plicae are embryonic vestiges of synovial tissue that are present in most knees. They vary in size to a considerable extent and may be implicated as causes of acute anterior knee pain (Matsusue et al. 1994). The mechanism of pain generation is uncertain. It is believed that a plica may become swollen and painful as a result of haemorrhage (Mital and Hayden 1979) or inflammation (Klein 1983) after trauma. Further mechanical pain may be induced as the inflamed plica is stretched across a femoral condyle (usually the medial) when the knee flexes. Plicae are described as palpable in 70% of cases (Johnson et al. 1993) and are diagnosed definitively by MRI and arthroscopy. Controversy exists as to the extent to which plicae cause symptoms. Some consider the plica to be a common source of anterior knee pain (Matsusue et al. 1994; Nottage et al. 1983; Reid et al. 1980), especially in adolescents (Dugdale and Barnett 1986; Fairbank et al. 1984) whilst others consider the syndrome to be over-diagnosed (Broom and Fulkerson 1986; Lupi et al. 1990).

Patellar Instability and Dislocation

Patellar instability encompasses all disorders in which the patella subluxes or dislocates from its normal position. The clinical features of patellofemoral instability depend on the degree of instability (Dugdale and Barnett 1986). Predisposing factors for recurrent dislocation of the patella, which have been identified but not substantiated, include an abnormally shallow trochlear sulcus (Fulkerson and Hungerford 1990), shallow patellar depth (Malghem and Maldague 1989) and hereditary ligamentous laxity (Carter and Sweetnam 1958). A high Q angle is thought to be a predisposing factor but no statistical evidence has been published. Pain associated with subjective or objective evidence of instability and dislocation may not relate to a specific entity. In this case, anterior knee pain is considered patellofemoral pain until proven otherwise.

Medial patellar subluxation is generally iatrogenic, occurring after a lateral release. It presents with anterior knee pain on passive subluxation or dislocation of the patella in a medial direction. A case series of reconstruction of the lateral patellotibial ligament reported that 68% had functional improvement (Hughston et al. 1996).

Infrapatellar Contracture Syndrome

This condition is described as a delayed post-traumatic reaction (2–8 weeks). The presenting symptoms include anterior knee pain and the signs include loss of knee mobility and voluntary guarding. Although the authors class this as infrapatellar contracture syndrome, the condition appears similar to complex regional pain syndrome (Ellen et al. 1999).

Patellar Tendonopathy

Patellar tendonopathy is anterior knee pain due to tendonous micro-tears of the patellar tendon, usually at the infrapatellar region of the patellar tendon. Other sites of involvement are the insertion of the quadriceps tendon and the tibial insertion of the patellar tendon (Blazina et al. 1973). Patellar tendonopathy is also called 'Jumper's knee' and 'patellar tendonitis'. Terms such as 'incomplete patellar ligament tear' and 'chronic micro-tearing of the patellar ligament' have also been proposed (el Khoury et al. 1992). Patellar tendonopathy occurs most commonly in athletes, especially in those who participate in activities that involve intense rapid quadriceps contraction, the prime example being jumping sports including volleyball, high jump and long jump (Maurizio 1963). Playing on concrete and the amount of time spent on physical training are other risk factors (Ferretti 1986).

Patellar tendonopathy is generally diagnosed on the basis of clinical features including well-localised pain and tenderness in association with peripatellar tendonous structures. However, these features are not universal and lack validity data. In a series of 172 individuals (Ferretti et al. 1985), pain was localised at the lower pole of the patella in 65% of cases, at the insertion of the quadriceps tendon into the patella in 25% and the tibial tuberosity in 10%. The pain was bilateral in 23% of cases. The cardinal physical feature was local tenderness. Local swelling was present in 14%; quadriceps wasting was present in 63% and radiological change at the point of tenderness was present in 8% of cases.

Osgood-Schlatter Disease

This condition is defined as traction apophysitis of the tibial tuberosity, the lower point of attachment of the extensor apparatus (Osgood 1903). Osgood-Schlatter Disease is an apophysitis. The condition occurs during the growth phase of the knee and typically affects adolescents. The presenting features are local pain and tenderness over the tibial tuberosity, often accompanied by marked swelling.

Sinding-Larsen-Johannson Syndrome

Anterior knee pain may be due to traction apophysitis of the lower pole of the patella, known as Sinding-Larsen-Johannson syndrome (Sinding-Larsen 1921). Adolescents are typically affected and findings may include local distal patellar tenderness and characteristic fragmentation of the lower pole of the patella on radiography.

Quadriceps Tendon

Complete rupture of the quadriceps femoris tendon is a well-described injury, occurring with peak incidence in the sixth

decade and more commonly affecting males (O'Shea et al. 2002). Bilateral simultaneous quadriceps tendon rupture is uncommon, usually accompanying disease, especially renal disease (Shah 2002b; Hansen et al. 2001). It occurs in association with sporting activity (Shah and Jooma 2002; Bikkina et al. 2002). As it is frequently misdiagnosed (Shah 2002a) and often accompanied by other diseases, clinicians should be aware of its existence in acute anterior knee pain (Kelly et al. 2001). When there is sudden onset of anterior knee pain, rupture of quadriceps tendon should be considered; however no studies evaluating the validity of clinical signs were located.

Other uncommon conditions affecting the quadriceps tendon and producing anterior knee pain include synovial osteochondromatosis (Langguth et al. 2002), painful cysts (Siebert et al. 1999) and in post-knee surgery patients, the patellar clunk syndrome (Lucas et al. 1999) and synovial entrapment (Pollock et al. 2002).

Conditions Referring Pain to the Anterior Knee

Pain may be referred to the anterior knee by a number of mechanisms. Hip disease, especially in children, may present with primary anterior knee pain. It is generally considered that the most common error in misdiagnosis of knee pain is to neglect examination of the hip joint. Neurological disorders affecting the femoral nerve and mid and lower lumbar nerve roots may also present with anterior knee pain. Consequently, pain in the anterior knee does not necessarily imply a local source.

Somatic structures that have innervation in common with components of the knee may also refer pain to the knee. Knee or distal thigh pain is the primary complaint in 15% of patients presenting with slipped capital femoral epiphysis (Matava et al. 1999). It is possible that knee pain may derive from other proximal disease, including femoral lymphadenopathy or pelvic disorder. However, it is unlikely that such conditions would present with knee pain alone.

Prevalence of Causes of Anterior Knee Pain

Data in the Aetiology section are summarised in Table 8.2 to demonstrate the prevalence of a number of conditions as a cause of patellofemoral pain. These provide a guide to the probabilities of particular conditions underlying clinical presentations.

Aetiological Risk Factors for Patellofemoral Pain

It is apparent that knee disorders in general are substantially related to activity and injury. Obesity, female sex (Outerbridge 1964) and iliotibial band tightness in ballet dancers (Winslow and Yoder 1995) have been identified as risk factors for patellofemoral pain.

Table 8.2

Prevalence of Conditions Presenting as Anterior Knee Pain

Prevalence	Condition
Rare causes	Tumour
	Infection
	Neurological conditions
Uncommon causes	Fracture
	Osteonecrosis
	Inflammatory arthropathies
	Pain referred to the knee
Common causes	Mechanical conditions

Note: As there is limited evidence available, Table 8.2 was developed through consensus.

Intrinsic Risk Factors

Intrinsic risk factors for knee disorders may include gender, knee anatomy, joint laxity, muscle imbalance, prior injury and personality. However, the higher lower limb injury rates in women may be explained by gender differences in symptom reporting (Almeida et al. 1999a). Shortened quadriceps, altered vastus medialis obliquus muscle response time, decreased explosive time and patellar hypermobility are risk factors for patellofemoral pain (Witvrouw et al. 2000).

A number of studies investigating validity have produced conflicting results. In an early study, patellofemoral pain was not associated with joint mobility, Q angle, genu valgum or femoral anteversion (Fairbank et al. 1984). Malalignment features that have been associated with patellofemoral pain include increased Q angle (Reider et al. 1981a); an infacing patella with palpable lateral patellofemoral bands (Reider et al. 1981b); hypermobility (al Rawi and Nessan 1997); anteversion, measured as the difference between the axis of the head-neck and the axis of posterior condyles (Eckhoff et al. 1994); and changes in the patellofemoral joint relationship during the last 10˚ of active extension (Brossmann et al. 1993).

Predisposing factors found in comparative studies include increased height, increased leg length difference, increased passive mediolateral patellar movement, increased knee laxity (Kujala et al. 2001), reduced quadriceps strength and increased medial tibial intercondylar distance (Milgrom et al. 1991). The relationship of the tibial tubercle to the femoral trochlear groove has been found to be a valid indicator of patellofemoral pain (Jones et al. 1995; Muneta et al. 1994), with a sensitivity of 91%, specificity of 88% and likelihood ratio of 7.6 in one study (Brown and Quinn 1993). Foot pronation has only been linked to patellofemoral pain in uncontrolled studies (Clement et al. 1981; James et al. 1978).

Although an early study cited joint laxity as a risk factor for knee injury (Nicholas 1970), subsequent studies have found no such relationship (Godshall 1975; Grana and Moretz 1978; Jackson et al. 1978; Kalenak and Morehouse 1975; Moretz et al. 1982).

Extrinsic Risk Factors

Obesity

Obesity has been implicated in the incidence and progression of knee osteoarthritis, particularly in females (Felson 1990; Leach et al. 1973). Obesity has been related to knee pain and disability; however, it has not been specifically looked at in patellofemoral pain (McAlindon et al. 1992).

Occupation and Sport

Knee disorders occur more often in occupations where the lower limbs are more heavily loaded (Ekstrom et al. 1983; Lawrence and Aitken-Swan 1952) including shipyard workers, firemen, farm labourers and construction workers (Anderson and Felson 1988; Kivimaki et al. 1992; Lindberg and Montgomery 1987; Tanaka et al. 1989). Bursitis and other anterior soft tissue changes occur more often in workers who kneel frequently (Tanaka et al. 1989; Thun et al. 1987; Watkins et al. 1958). The most common lesions in kneeling workers are meniscal lesions (Holibkov et al. 1985) osteoarthritis (Kasch and Enderlein 1986) and prepatellar bursitis (Sharrad 1964). In these occupations, weak knee extensors occur more frequently, but whether this is cause or effect is unknown (Kivimaki et al. 1994). The relationship

between patellofemoral pain and work is less convincing (Mbaruk 1980). Occupations that involve frequent bending at the knee are a risk factor for anterior knee pain (Cooper et al. 1994) and for the development of osteoarthritis of the knee, particularly in males (Hunter et al. 2002).

A prospective randomised study found that the volume of vigorous physical training may be an aetiological factor for exercise-related injuries. The type of training, particularly running and abrupt increases in training volume, may further contribute to injury risk (Almeida et al. 1999b).

Key Message

Intrinsic risk factors for knee pain may include female gender, knee anatomy, joint laxity, muscle imbalance and prior injury. Extrinsic risk factors include occupation, sport and obesity. (*Level IV)

>History

A detailed and appropriate pain history is crucial in the assessment of a person with anterior knee pain. History is a practical means to detect clinical features of a serious condition.

History comprises the pain history together with broader enquiry into general medical and psychosocial history. When a serious condition is suspected, conventional algorithms should be implemented for the confirmation and management of that condition. Some elements of the pain history specific to knee pain are presented below. For detail of all of the elements of a pain history, refer to Chapter 2: Acute Pain Management.

Pain History

Presenting Complaint

It is important to establish that an individual is indicating the presence of pain in the anterior knee. It is common for anterior knee pain to occur in the presence of other symptoms including locking, giving-way, crepitus, popping, clicking, snapping and swelling. Thus, the history pertinent to the knee should include other relevant local inquiries, as symptoms other than pain relate to the knee.

Site and Distribution

Whilst it is possible for patellofemoral pain to spread, the presence of pain outside the confines of the anterior knee should alert the clinician to consider other causes of anterior knee pain, either local or referred.

Although a classification system based on the site of pain is not foolproof, the site of pain indicated by the patient is a guide to the likely site of pain origin.

Intensity

The Visual Analog Scale (VAS) has been assessed in a study on patellofemoral pain; it was found that subjects tended not to use the whole range of the linear scale and the Rasch analysis was used to convert the readings to an interval scale (Thomee et al. 1995a). Refer to Chapter 2: Acute Pain Management for discussion of pain intensity measurement.

Onset (Precipitating Event)

A primary consideration in the management of acute anterior knee pain is the presence or absence of trauma. A traumatic onset suggests the possibility of fracture, microfracture, bone bruise, ligamentous disruption, meniscal disruption and chondral damage.

Trauma also includes the notion of repetitive strain. In the context of the knee, such conditions occur in both sporting and occupational activities and involve repetitive activities such as running, kicking and kneeling. Such injuries may occur to any knee structure. Typical examples are stress fractures of the patella, and tibio-femoral condyles, chondral desiccation, bursitis of the prepatellar bursa in occupations that involve kneeling and tendon injuries of the patellar or hamstring tendons in running.

Spontaneous onset of explosive pain may be an indication of osteonecrosis, infection or fracture, or of internal derangement of ligament or meniscal structures.

Precipitating and Aggravating Factors

Sporting activities, particularly running and jumping, appear to be a major precipitating factor in the genesis of patellofemoral pain (McKenzie et al. 1985). Knee pain when ascending and descending stairs is commonly attributed to the patellofemoral joint and patellar tendon mechanism. However, pain aggravated by such activities cannot be considered 'patellofemoral pain' unless all other possible causes of anterior knee pain are excluded. Pain aggravated by kneeling suggests anterior knee disorders including prepatellar bursitis and patellofemoral osteoarthritis in older age groups.

Periodicity

Clinicians should be aware that the relationship between variations in pain during the day or over time and any particular condition have not been formally studied. Morning stiffness is said to be a feature of inflammatory disorders. Pain and stiffness that worsens after rest invites a consideration of inflammatory causes; pain at rest or unchanged by activity should prompt a further consideration of serious conditions. Pain that is worse and particularly severe at night should raise suspicion of a serious underlying cause. Pain that is worse during the day is consistent with many forms of mechanical pain as it is progressively aggravated by activity and compounded by fatigue.

Local Associated Features

Knee Joint Swelling

Knee joint swelling is most often related to local causes, although consideration must always be given to the possibility of a systemic process.

In the absence of a history of trauma, an acutely swollen knee with symptoms of less than 24 hours duration suggests such entities as septic arthritis, crystal arthritis, haemarthrosis, rheumatoid arthritis and seronegative spondyloarthritis. Local anterior knee swelling should alert the clinician to the possibility of local causes of anterior knee pain, such as bursitis, infection, inflammation and cancer.

Following trauma, swelling is more likely due to haemarthrosis or serous effusion. The causes of swelling are broadly classified into inflammatory and mechanical:

- Inflammatory symptoms may include morning stiffness, rest pain, night pain and relief on walking.
- Mechanical symptoms include pain on weight bearing and pain that worsens as the day progresses (Brand and Muirden 1987).

These relationships have not been formally studied. The above features may involve the entire knee joint or be confined to the anterior knee.

Other Features

Locking is generally considered due to impingement of an abnormally located structure between the joint surfaces on movement. When locking and anterior knee pain co-exist, consideration should be given to a single local cause such as

patellofemoral articular cartilage derangement, loose bodies, fat pad fibrosis and adhesions (Finsterbush et al. 1989).

Block to extension similarly implicates impingement. Apart from the more common causes listed above, rare causes include infrapatellar plicae (Kim and Choe 1996; Kim et al. 2002) and intra-articular ganglia (Yasuda and Majima 1988).

Popping is a not uncommon symptom that can occur in association with and subsequent to knee trauma (Cooper 1999). It does not imply any particular pathology (Noyes et al. 1980; Crites et al. 1998; Dupont 1997; McNair et al. 1990).

Clicking is a common symptom that can occur at the time of injury or on subsequent occasions. Whilst suggestive of abnormal anatomy, it lacks formal study.

Giving way is traditionally ascribed to internal damage to the knee and/or muscle weakness. The symptom is not specific for anterior cruciate ligament rupture. When formally investigated, limb collapse at time of injury has a sensitivity of 90% but specificity of only 33% and likelihood ratio of 1.34 for anterior cruciate ligament rupture (Noyes et al. 1980). It can be difficult to differentiate between giving way related to a muscular reflex and bony giving way, as in patellar subluxation or dislocation.

Snapping may be due to the gracilis and semitendonosus tendon passing over the medial tibial condyle (Bae and Kwon 1997).

Alerting Features of Serious Conditions (see Table 8.3)

Features alerting to the possibility of a serious condition may be identified during clinical assessment. While the predictive values of these alerting features have not been tested specifically in relation to patellofemoral pain, their presence in conjunction with anterior knee pain should prompt further investigation.

Key Message

The history provides information on possible causes of anterior knee pain and assists the identification of serious underlying conditions. (Consensus)

>Physical Examination

The object of physical examination is to identify features of a clinical presentation that help to establish the nature of the problem and, if possible, its cause. Although the examination findings may provide the clinician with further information about the symptoms described, the lack of proven reliability of individual physical tests is a significant problem. Despite this, physical examination is important to identify any alerting features of serious conditions.

There is no test for patellofemoral pain and tests for patellar tracking aberration and other patellofemoral malalignment problems lack reliability and/or validity. In particular, evaluation of the physical signs in patients with and without patellofemoral pain has demonstrated that:

- There are no differences in the knees of those with symptoms and those without symptoms.
- Lower extremity alignment is similar in the two groups (e.g. Q angle and leg–heel alignment measures) (Thomee et al. 1995b).

Physical examination of the knee may include inspection, palpation and assessment of movement.

Inspection

Inspection of the knee may reveal fixed or reducible deformities, bony, articular, bursal or other soft tissue swelling, muscle wasting and features of inflammation.

It is traditional to evaluate morphology such as genu varum, genu valgum, excurvatum, torsional alignment, patellar alignment, pes planus, pelvic tilt and obliquity. However, the diagnostic significance of any of these features has not been determined.

Palpation

Palpation may be performed firstly with light pressure for conformity and temperature, secondly for tissue induration and effusion and thirdly for tenderness (Feagin and Cooke 1989).

Acute Knee Joint Swelling

Knee effusions may be the result of trauma, overuse or systemic disease; they are an alerting feature of serious conditions. The most common traumatic causes of knee effusion are ligamentous, osseous and meniscal injuries and overuse syndromes. Non-traumatic aetiologies include arthritis, infection, crystal deposition and tumour (Johnson 2000).

Table 8.3

Alerting Features of Serious Conditions Associated with Anterior Knee Pain

Feature or Risk Factor	Condition
Major trauma	Fracture or tendon and ligament rupture, osteonecrosis
Sudden onset of pain (alerting feature for such entities as fracture and osteonecrosis)	
Minor trauma (if > 50 years, history of osteoporosis and taking corticosteroids)	
Fever, night sweats, signs of inflammation (large, warm effusion)	Infection (e.g. septic arthritis), crystal arthritis
Risk factors for infection (e.g. underlying disease process, immunosuppression, penetrating wound)	
Past history of malignancy	Tumour
Age > 50	
Failure to improve with treatment	
Unexplained weight loss	
Pain at multiple sites	
Pain at rest	
Night pain	

The key elements of this aspect of examination involve determining:

- if the swelling is articular (i.e. arising from within the joint cavity) or extra-articular (i.e. arising from soft tissue structures around the joint)
- if there is any possibility of infection
- if there is any evidence of a poly-articular problem.

If the swelling is not associated with trauma, examination must include a general physical examination with emphasis on assessing for signs of infection, regional lymphadenopathy and examination of other joints and bursae (Brand and Muirden 1987).

Extra-articular swelling can be due to bursae, meniscal and other cysts, ganglia, and other bony or soft tissue problems. If the swelling is articular and post-traumatic, the most likely findings are a serous effusion or haemarthrosis.

Haemarthrosis is most frequently associated with substantial internal knee trauma. It usually presents as a painful, tense, generalised, knee joint swelling arising within a few hours of injury. It is often warm to touch. The condition is common in association with damage to the anterior cruciate ligament (ACL) and it frequently occurs after patellar dislocation. The incidence of ACL rupture in acute traumatic haemarthrosis has been reported between 62% and 85% (Adalberth et al. 1997; Butler and Andrews 1998; Daniel et al. 1994; Hardacker et al. 1990; Noyes et al. 1980; Woods and Chapman 1984).

In contrast to the adult population, children with haemarthrosis have a lower incidence of anterior cruciate ligament tears and a greater incidence of osteochondral fracture of the lateral femoral condyle or patella (half of which may not be seen on plain radiography) (Maffulli et al. 1997; Matelic et al. 1993).

Patellar Ballotment

Patellar ballotment can be used to elicit an effusion. A plica may be felt in the area extending superiorly from the anteromedial joint line (Hardacker et al. 1980).

Tenderness

Palpation should be performed systematically, carefully addressing each of the structures and tissues around the joint. Tenderness should be defined in terms of:

- anatomical location in relation to the joint (lateral, medial, anterior, posterior), and
- the structure or tissue involved, if possible (bone, joint line, bursa, ligament, tendon or skin).

It should be recognised that when several structures overlie one another it is not possible to validly ascribe tenderness to a particular structure. Under these circumstances, tenderness is best described in terms of its general anatomical location. For tenderness to be ascribed to a particular structure it should be the only structure palpated or it should be palpable from at least three dimensions, such that it is tender not only upon pressing the structure but also upon selectively squeezing it.

It is traditional to regard focal tenderness at one or more points as a key clinical finding, particularly if stimulation of these points reproduces the typical pain.

Tenderness related to a joint line is said to suggest either local (e.g. medial collateral ligament) or intra-articular (e.g. meniscal tear) pathology.

Tenderness related to a focal area of bone is said to suggest bony pathology and tenderness related to one of the soft tissues is said to suggest somatic impairment involving muscles, tendons or ligaments.

Apart from patellar tendonopathy, there are no data for the reliability of such findings on palpation of the knee and no firm evidence for their validity as indicators of specific knee problems. Additionally, joint line tenderness has been shown to have no validity in the diagnosis of meniscal injury (Fowler and Lubliner 1989).

Palpation in patellar tendonopathy has been found to be reliable for a single examiner (Cook et al. 2001). In association with anterior knee symptoms, palpation revealing moderate or severe pain is correlated with patellar tendonopathy seen on ultrasound (Cook et al. 2001).

Other Signs

Other signs may be elicited by palpation. Apparent alterations of skin sensitivity include hypoaesthesia, suggesting neurological deficit and hyperaesthesia, suggesting neuropathic pain. Apparent alteration of bony landmarks, soft tissue conformation and muscle tone may suggest mechanical problems; palpable deformities of bones and other tissues alert to the potential for a serious underlying condition. The reliability and validity of such findings are unknown.

The bursae should be palpated for local swelling, temperature change and tenderness. Other soft tissue swellings that might be encountered include meniscal or popliteal cysts and ganglia.

Assessment of Movement

The active and passive movements of the knee may be tested by assessing the range of movement and challenging the restraints to movement. Various associated signs, such as blocking and crepitus, may be elicited during movement tests. However, these findings should be interpreted with caution as most tests do not stand up to research scrutiny.

Ranges of Movement

The ranges of active and passive movements can be assessed according to various conventions (Russe et al. 1976); however the reliability and validity of such tests have not been established.

Blocking and/or Locking

Blocking and locking is a loss of movement of the joint and has been correlated with internal mechanical problems, particularly meniscal pathology (Shakespeare and Rigby 1983). Blocking may also be caused by 'mechanical' factors, such as the interposition of some osteochondral (loose body), meniscal or ligamentous fragments between condyles and tibial plateau or by non-mechanical ('functional') factors, such as the pain associated with capsular-ligamentous structure injuries or with intraosseous bruises involving the synovia (Perin et al. 1997).

Crepitus

Crepitus is defined as the crackling sound or sensation detectable during joint motion. It is a cardinal feature of osteoarthritis (Altman et al. 1986). However, simple attempts to elicit this physical sign, such as placing a hand over the patella during passive movements, lack reliability (Bergquist et al. 1993; Cushnaghan et al. 1990; Ike and O'Rourke 1995; Jones et al. 1992). Although crepitus is generally considered unreliable and invalid (Cushnaghan et al. 1990; Hart et al. 1991; Jones et al. 1992) 'transmitted bony crepitus' may be a better assessment protocol (Ike and O'Rourke 1995).

Patellar Apprehension Test

A particular finding on restraint testing is called the 'apprehension sign'. The test is performed with the knee in 0˚ extension and then in 30˚ flexion. A lateral stress is applied to the patella during the movement. The apprehension sign is 'positive' when the examiner challenges the restraints in a particular direction of movement and the patient responds by guarding and becoming apprehensive. The sign is described as an indicator of instability of the patellofemoral mechanism. Previous experience of pain on subluxation or dislocation of the joint may cause apprehension when the examiner moves the joint in the direction in which it is unstable because the restraints are impaired. The apprehension sign has been described often as associated with instability but no data have been produced to substantiate the relationship.

Q Angle

The 'Q angle' is defined as the acute angle between the line connecting the anterior superior iliac spine and the midpoint of the patella and the line connecting the tibial tubercle with the same reference point on the patella (Horton and Hall 1989), although many subtle alterations in methodology have been used (Aglietti et al. 1983; Brown et al. 1984; Hvid and Andersen 1982; O'Donohue 1980). The Q angle is generally less than 10˚ in men and 15˚ in women (Millbauer and Patel 1986). The reliability of Q angle measurement is good (Horton and Hall 1989; Caylor et al. 1993); however the validity for the detection of patellofemoral pain has been refuted (Biedert and Warnke 2001; Caylor et al. 1993; Thomee et al. 1995b).

Patellofemoral Alignment

When formally studied (Fitzgerald and McClure 1995), four patellofemoral alignment tests, medial/lateral displacement, medial/lateral tilt, medial/lateral rotation and anterior tilt were found to be unreliable with kappa scores between 0.10 and 0.36. Additionally, the lateral pull test and patellar tilt test were found to have fair intrarater (Kappa 0.0.39–0.50) and poor interrater reliability (Kappa 0.20–0.35) (Watson et al. 2001). Validity has not been tested.

Reliability and Validity of Physical Tests

There are no valid or reliable physical examination features of patellofemoral pain. Reliability is poor for general knee examination. One study demonstrated that there was significant discrepancy in estimation of joint displacement by a group of experienced knee surgeons using standardised tests, concluding that there is a clear need for improvement of inter-examiner reliability (Daniel 1991). Also, examination of people with knee arthritis (Dervin et al. 2001) and the examination of the patella by the lateral pull test and tilt test (Watson et al. 2001) have been found to be unreliable.

The only sign that has been found reliable for a single examiner is patellar tendon palpation for tenderness (Cook et al. 2001), however as this is a common finding the sign has limited diagnostic utility. The results of physical tests challenging the restraints of the patella cannot be interpreted specifically and their reliability and validity must be considered unproven. However in certain cases extreme laxity of the medial patella retinaculum allows frank lateral dislocation of the patellar at the time of examination.

Summary of Clinical Features of Patellofemoral Pain

Studies have determined that there are numerous clinical features that may occur in subjects with patellofemoral pain. Physical examination may help exclude some specific causes of anterior knee pain, however it does not carry any further diagnostic weight.

Some of the clinical features that may be associated with potential patellofemoral pain are summarised below:

- People presenting with patellofemoral pain are typically young and pain onset is often vague and insidious.
- Pain has an aching quality and is felt anteriorly or anteromedially and tends to be poorly localised.
- It is not uncommon for people with patellofemoral pain to describe a sensation of 'instability'.
- It is frequently aggravated by activities such as walking and running uphill and climbing stairs and also by loading (e.g. squatting, sitting for prolonged periods with the knees flexed, rising from a sitting position).
- Bilateral knee pain is common.
- Patellofemoral pain can be associated with mild swelling, crepitus, snapping and clicking.
- Clinical findings may vary from no abnormality detected to such findings as mild effusion, crepitus, tenderness over the medial or lateral peripatellar regions, anterior knee pain on active or passive movements and pain on patellar glide.

Key Message

Although examination techniques lack specificity for diagnosing knee disorders, physical examination may assist the identification of serious conditions underlying anterior knee pain. (*Level III, Level IV)

>Ancillary Investigations

Medical Imaging

There has been a trend towards preoccupation with the detection of morphological lesions via conventional xray, bone scan, computed tomography (CT) and more recently magnetic resonance imaging (MRI), with the belief that such abnormalities constitute the source of knee pain.

With the exception of serious conditions, the detailed correlation between most morphological change and pain is not established. Changes seen on imaging, therefore, should not be assumed to be the cause of the presenting problem.

Because of its greater resolution of soft tissues and intraosseous tissues, MRI is superior to CT for the demonstration of conditions such as cysts, infections and tumours. However, these conditions rarely cause anterior knee pain. Their low pre-test probability does not justify the use of MRI as a screening test in those presenting with anterior knee pain unless there are alerting features of serious conditions noted during the clinical assessment.

Plain Radiography

Plain radiography demonstrates the structure of bones and to a limited extent, the structure of joints. It will not demonstrate lesions that do not affect bones and has a limited sensitivity even for lesions that do affect bones. Consequently, plain radiography serves poorly as a diagnostic test to detect the cause of anterior knee pain and as a screening test to detect occult lesions.

Non-Traumatic Knee Pain

Conventional films have a limited role in the primary diagnosis of acute non-traumatic knee pain. There is no evidence that they can diagnose the source of knee pain, including patellofemoral pain. There is no indication for plain xray in those presenting with acute anterior knee pain in the absence of features suggesting a serious cause. Even in cases with clinical features significant enough to warrant surgery, conventional plain radiography does not add substantially to the diagnostic process (O'Shea et al. 1996). Chapman-Jones et al. (1998) demonstrated that plain radiography in cases of non-specific knee pain has a high probability of a negative result irrespective of any anatomical derangement.

A study (Morgan et al. 1997) on the use of xrays in general practice found that 50% of knee xrays were ordered according to the Royal College of Radiologists guidelines (1995), in which the indications for xray are locking or restricted movement. Of the 1153 xrays ordered, 50% fell within the guidelines, 90% were normal or showed degenerative changes and 13% led to a change in management.

The concerns about the guidelines were that some diagnoses would be missed in the presence of persistent pain or effusion (e.g. foreign body or Brodie's abscess) and that in the presence of locking, a normal xray would compromise treatment because of false reassurance. However, these other conditions occurred very infrequently (Brodie's abscess 1/1153; foreign body 2/1153).

This study noted that despite the apparent lack of utility of plain radiography in many instances, clinicians were unlikely to change their referral patterns because they still found the results to be of use in clinical discussion and decision-making. An additional issue was the pressure from patients for imaging.

It has been recognised that clinicians need both information and time to explain the indications for imaging. Peer comparisons, educational intervention and positive physician feedback utilising Percentage Abnormal Results can help to rationalise the use of plain xrays (Wigder et al. 1999).

Infection

In cases of suspected prosthetic joint infection, the best single test is joint aspiration (sensitivity 67–75%, specificity 95–100%) (Levitsky et al. 1991; Virolainen et al. 2002). Bone scan provides sensitivity of 33% and specificity of 65% (Levitsky et al. 1991). Plain radiography does not improve diagnostic certainty if bone scanning is also used concurrently (Levitsky et al. 1991; Virolainen et al. 2002).

Tumour

Tumours are rare causes of knee pain, including anterior knee pain. Osteoid osteoma, which can present with referred pain to the knee, is frequently missed on plain radiography. However, bone scan and MRI are reliable detection techniques (Georgoulis et al. 2002). If a bone tumour is suspected, MRI is indicated either alone or subsequent to screening by plain radiography (Dickinson et al. 1997; Meyer et al. 2002).

Fracture

The rare conditions osteonecrosis, transient osteoporosis and regional migratory osteoporosis can present with spontaneous, severe, non-traumatic, weight-bearing knee pain. In such presentations, plain radiography is indicated (Glockner et al. 1998; Crespo et al. 2001).

Traumatic Knee Pain

A history of trauma raises the possibility of fracture (McConnochie et al. 1990), however the overall detection rate of fractures is low even when the index of suspicion is high (Petit et al. 2001).

The indications for conventional xray have been defined and evaluated for acute traumatic knee injuries and are outlined in the following Rules:

- The Ottawa Knee Rule
- The Pittsburgh Knee Rule
- The Bauer Rule.

These Rules apply to all patients presenting with knee pain, not to anterior knee pain in particular.

Ottawa Knee Rule

A review of all conventional xrays taken in an emergency department revealed that about 92% were negative for fracture (Stiell et al. 1995). As a consequence, the 'Ottawa Knee Rule' was developed following a multi-centre trial (Stiell et al. 1996). This rule has been validated and found to be reliable (Stiell et al. 1997; Wasson and Sox 1996).

The rule states that a conventional xray is required for acute knee injury in the presence of any of these findings:

- Age 55 years or older
- Isolated tenderness of patella
- Tenderness at head of fibula
- Inability to flex to 90˚
- Inability to bear weight both immediately post-injury or in the emergency department (described as 'unable to transfer weight twice onto each lower limb regardless of limping').

This rule has not been validated when clinical assessment is unreliable, for example, in head injury, drug or alcohol intoxication, paraplegia and diminished limb sensation.

The Ottawa Knee Rule has been studied on a sample (N = 234) of children (2–18 years). Twelve of thirteen fractures were detected (sensitivity 92%; 95%CI 64%, 99%) using the Rule. If implemented, the Rule would have lead to a 46% reduction in knee xrays. It was suggested that the Rule needs further modification before it is used on children (Khine et al. 2001).

The Ottawa Knee Rule is cost-effective when considering the likelihood and cost of radiography, missed fracture, lost productivity and medicolegal actions as defined by published data and an expert panel (Nichol et al. 1999).

The ability of triage nurses to interpret the Ottawa Knee Rule was assessed in a community emergency department. Reliability of examination was assessed by Kappa with comparison to emergency physician findings. Demographic reliability was high (age = 0.94); physical examination was moderately reliable (fibular head tenderness = 0.4; isolated patellar tenderness = 0.68; inability to bend knee to 90 degrees = 0.73; inability to bear weight = 0.76). The sensitivity of nurse interpretation of the Ottawa Knee Rule for fracture was 70%, specificity 33%, with a likelihood ratio of 1.04. The sensitivity of emergency physician interpretation was 100%, specificity 25%, with a likelihood ratio of 1.33. It was concluded that the agreement between triage nurses and emergency physicians was fair to good. However, specific training in assessment is recommended for nurses engaged in triage of patients with acute knee trauma (Kec et al. 2003).

Pittsburgh Knee Rule

The algorithm for the Pittsburgh Knee Rule (Seaberg and Jackson 1994) is as follows:

For people of any age presenting with acute knee pain, xrays are taken only when there is a history of a fall or blunt trauma (any injury involving a direct blow or mechanical force applied to the knee). When this is the case, the following rules apply:

- All patients aged 11 or younger and those aged 51 and older are xrayed.
- Of those remaining, only those who cannot walk four weight-bearing steps in the emergency department are xrayed. Weight-bearing ability is the ability to bear weight fully on the toe pads and heels for four full steps.

Training is necessary in order for the rule to be accurately implemented (Szucs et al. 2001).

The Bauer Rule

In the Bauer Rule, the inability to bear weight combined with the presence of an effusion or an ecchymosis was initially found to be 100% sensitive and specific for the detection of a fracture (Bauer et al. 1995).

Comparison of Ottawa, Pittsburgh and Bauer Rules

The Pittsburgh Knee Rule has been prospectively compared to the Ottawa Knee Rule and found to be more specific without any loss of sensitivity (Seaberg et al. 1998). In the 745 cases where the Pittsburgh Rule could be applied, there were 91 fractures (12.2%). The use of the Pittsburgh Rule missed one fracture, yielding a sensitivity of 99%, specificity of 60% and likelihood ratio of 2.5. The Ottawa inclusion criteria were met by 750 patients (a total of 87 fractures, or 11.6%). The Ottawa Rule missed three fractures yielding a sensitivity of 97%, specificity of 27% and likelihood ratio of 1.3.

In a comparison and evaluation of the Bauer and Ottawa Rules (Richman et al. 1997) use of each of these Rules would have led to a radiographic evaluation of 22 of the 26 cases with knee fractures (sensitivity = 84.6%, specificity = 48.9%). This study demonstrated that neither Rule was 100% sensitive.

No Rule has 100% sensitivity, however the Pittsburgh Knee Rule (unable to walk four steps) is easy to apply, has greatest predictive value and a Likelihood Ratio of 2.5.

Other than establishing the presence of serious bony conditions, plain xray in the presence of trauma seems to have no valid or reliable role in establishing significant patellofemoral mechanical contributions. However, the Knee Rules indicate that under certain conditions knee xrays should be taken, although no Rule has clear benefit over another until proven otherwise.

Key Message

Indications for plain radiography are a history of trauma and: qualification under one of the Knee Rules, or sudden onset of severe pain, or alerting features of a serious condition. (*Level III, Level IV)

Computed Tomography (CT)

There is no specific role for static computed tomography (CT) scanning in the diagnosis of patellofemoral pain. However, it does have a significant role in the assessment of complex fractures, especially tibial plateau fractures. Other uses include CT arthrography, which can be used to detect patellofemoral articular cartilage irregularity, patellar tracking abnormality and osteochondral fractures and fragments (Gray et al. 1997) and ultrafast CT, which has been used for dynamic evaluation of the patellofemoral joint (Stanford et al. 1988).

Key Message

Suspected fracture in the presence of a normal plain radiograph is an indication for CT scan. (Consensus)

Radionucleotide Scan

The role of bone scanning in the assessment of anterior knee pain is confined to circumstances where the index of suspicion of a serious condition is high. Bone scanning appears to be a highly sensitive, relatively non-specific and relatively non-invasive method of assessing certain knee disorders. It is a sensitive screening device for conditions such as stress fractures, occult fractures, osteochondritis dissecans and bone tumours (Lee and Sartoris 1995).

In the assessment of long bone osteomyelitis and infected joint replacement, bone scan studies have been assessed (Larikka et al. 2001; Joseph et al. 2001; Palestro et al. 2002) and various protocols have been compared. For example, in one study the use of monoclonal antibody study and bone scanning was the most accurate, improving sensitivity over bone scan alone from 38% to 85%; in both the specificity was 100% (Palestro et al. 2002).

Magnetic Resonance Imaging (MRI)

A virtue of magnetic resonance imaging (MRI) is its ability to reveal rare disorders that are undetectable or poorly resolved by other means or for which other imaging modalities lack specificity. It has an established role in the detection of serious conditions such as malignancies or osteomyelitis when alerting features are present and in the detection of internal derangements, particularly anterior cruciate ruptures and meniscus tears. It has an emerging role in the detection of articular cartilage defects.

Cine MRI is emerging as a possible diagnostic tool for use in the evaluation of patellofemoral pain but its role remains unclear (McNally et al. 2000; McNally 2001). Issues of cost and availability reduce the utility of MRI.

Key Message

The presence of alerting features of a serious condition is an indication for the use of MRI. (Consensus)

Ultrasound

There is a limited role for ultrasound in the assessment of anterior knee pain. It is indicated for determining the cause of painful swelling that appears to be extra-articular. The benefits of ultrasound are that it is relatively non-invasive, freely available, well accepted by patients, inexpensive and useful for dynamic evaluation (Richardson et al. 1988; van Holsbeeck and Introcaso 1992). Limitations include reliability, which is largely operator dependent, the small size of the field and the inability to evaluate bone.

Tendon Lesions

Ultrasound has a role in the assessment of tendon lesions, particularly partial and complete quadriceps rupture (Bianchi et al. 1994). It may be useful for differentiating between cellulitis, soft tissue abscess and septic arthritis in a patient presenting with a confusing clinical picture (Jacobson and van Holsbeeck 1998). It can also be useful for assessing possible causes of medial, lateral and posterior knee pain such as cysts

and bursitis including differentiating between popliteal cyst and other local swellings such as aneurysm, nerve sheath tumour and ganglia (Jacobson and van Holsbeeck 1998). Mourad et al. (1988) found ultrasound to be more accurate than CT scan for chronic patellar tendonitis in nine patients in which the results were compared to histological examination.

Key Message

Swelling or potential rupture of anterior knee structures are indications for the use of ultrasound. (*Level IV)

Arthrography

Arthrography does not have any application in the assessment of anterior knee pain.

Other Ancillary Investigations

Refer to Appendix C: Ancillary Investigations.

>Terminology

Patellofemoral Pain

Patellofemoral pain is a term used to describe anterior knee pain of unclear aetiology. It is a descriptive term denoting the site of pain (i.e. pain located in close proximity to the patellofemoral complex) and not the nature and circumstances of the pathological process underlying the pain.

Patellofemoral pain can be considered synonymous with such terms as 'retropatellar pain' and 'patellofemoral joint pain'.

Criterion standards for the diagnosis of patellofemoral pain do not exist, however it is possible that specific clinico-pathological correlations will be discovered in the future. A number of specific conditions associated with pain experienced in the anterior knee have already been determined, as outlined in the Aetiology and Prevalence section. Thus, diagnoses such as supra-, pre- and infrapatellar bursitis, patellar tendonopathy, fat pad impingement and plica impingement can be made (although, a rigorous search for the aetiology of pain in some of these diagnoses reveals shortcomings).

Key Message

The term 'patellofemoral pain' describes anterior knee pain for which there is no specific identifiable cause; it refers to the probable anatomical site of origin and is synonymous with retropatellar and patellofemoral joint pain. (Consensus)

PROGNOSIS

Natural History

Studies on the natural history of patellofemoral pain report that in general it is a benign condition that may improve or persist over time; serious disability is uncommon. However, the available studies have methodological limitations.

Nimon et al. (1998) followed a small series of adolescent females (mean age 15.5 years) for an average of 16 years (range 14–20) and demonstrated improvement in 73% over that time. An earlier case series (Karlson 1939) noted improvement in 79% of patients at 3 to 20 years follow up.

Studies show that pain persists: 35% with pain persisting at 6 years (Milgrom et al. 1996); 71% with pain persisting at 1 to 4 years (soldiers) (Robinson and Darracott 1970); 86% at 1 to 20 years (army recruits) (Karlson 1939); and 95% at 2 to 8 years (Sandow and Goodfellow 1985). Total or near total recovery was noted in 22% at 16 years (Nimon et al. 1998), 70% at 3 years (Kannus and Nittymaki 1994), 81% at 12 years (Jensen and Albrektsen 1990) and 85% at 11 years (Karlsson et al. 1996). Severe long-term pain was experienced by 6% at 12 years (Jensen and Albrektsen 1990) and 8% at 6 years (Milgrom et al. 1996). See Table 8.4.

The natural history of anterior knee pain is unclear, suggesting methodological flaws. The fundamental flaws include the operationalisation of the term 'patellofemoral pain', the disparity among the groups under study and the outcome measures used.

Key Message

Multiple studies on a range of populations show a trend towards improvement with time; however, anterior knee pain persists to some degree in the majority of people. (*Level IV)

INTERVENTIONS

The search for studies on interventions for anterior knee pain was limited to randomised controlled trials (RCTs) and systematic reviews of RCTs. Other study designs testing these and other interventions exist, however they are not covered in this document. All of the RCTs on interventions located in the search involved mixed acute and (predominantly) chronic populations.

It is important to note that a lack of evidence (i.e. insufficient evidence) does not mean that a particular intervention

Table 8.4

Natural History of Patellofemoral Pain: Summary of Study Results

Total or near total recovery	70% at 3 years	85% at 11 years	81% at 12 years	22% at 16 years (girls), total recovery
Improvement	79% at 3 to 20 years follow-up	73% at 16 years follow-up (range 14–20)		
Persisting pain	71% at 1 to 4 years (soldiers)	95% at 2 to 8 years	35% at 6 years	86% at 1 to 20 years (army recruits)
Severe long term pain	8% at 6 years	6% at 12 years		

Note: Based on data from: Nimon et al. (1998); Karlson (1939); Milgrom et al. (1996); Robinson and Darracott (1970); Sandow and Goodfellow (1985); Kannus and Nittymaki (1994); Jensen and Albrektsen (1990); Karlsson et al. (1996).

has no place in the management of anterior knee pain, however, it is preferable to employ interventions for which there is evidence of benefit, where appropriate. Management decisions should be based upon knowledge of the existing evidence, consideration of individual patient needs and clinical judgment.

The criteria formulated to categorise the following interventions and the definitions of the levels of evidence are described in Chapter 9: Process Report.

Adverse effects have not specifically been investigated during this review, however information has been included in the text where adverse effects have been described in the cited material.

Evidence of Benefit

Advice to Stay Active (Activation)

Activity is required for the maintenance of the load-resistant properties of most tissues. Muscles, tendons, ligaments and other soft tissues all tend to lose their physiological resistance to applied loads and to atrophy if not used for regular load-bearing. Conversely, rest reduces the forces that give rise to mechanical nociception when applied to particular tissues.

As patellofemoral pain is noted most frequently during activity, such as climbing stairs, running and jumping, it might seem prudent to advise relative rest from such aggravating activities. A study of 59 male army recruits with patellofemoral pain compared the use of a knee brace versus an elastic sleeve versus no treatment. The 'no treatment' group, who were not allowed to rest or take non-steroidal anti-inflammatory drugs (NSAIDs), had less pain (p = 0.04) compared to the two groups managed with different types of patellofemoral orthoses (Finestone et al. 1993). The results suggest that maintenance of physical activity aids recovery from patellofemoral pain.

The apparent conflict between activity-related pain and the need for regular activity gives rise to controversies about appropriate responses to pain and behaviours associated with pain, about pain tolerance and motivation to recover and about 'fitness' (especially for work tasks) and 'deconditioning'. Encouraging activity in subjects with chronic knee pain has positive benefits in terms of psychological distress and physical dysfunction (Hopman-Rock et al. 1997). Whether the situation is similar in acute patellofemoral pain has not been established.

Key Message

Maintenance of normal activity has a beneficial effect on patellofemoral pain compared to no treatment and to the use of patellofemoral orthoses. (Level II)

Injection Therapy

A systematic review by Arroll et al. (1997) identified one RCT of injection therapy involving intramuscular glycosaminoglycan polysulphate in patients with proven patellar articular cartilage damage. Therefore, conclusions cannot be transferred to patellofemoral pain in general.

Kannus et al. (1992) compared two intra-articular injection groups with a control no-injection group in the treatment of chronic patellofemoral pain. The intra-articular injection groups received either local anaesthetic (lignocaine) and glycosaminoglycan polysulphate or local anaesthetic and physiologic saline. All groups received NSAID medication and performed isometric exercises of the quadriceps muscles. At six-weeks, the two injection groups fared better than the 'no injection' control group for pain relief but at six-months the groups were equal, with full recovery occurring in 63% of the control group, 77% of the glycosaminoglycan polysulphate group and 81% of the saline group (Kannus et al. 1992).

No studies were identified for injection therapy involving corticosteroids with and without local anaesthetic for patellofemoral pain syndrome. Limited evidence available in osteoarthritis of the knee suggests possible short-term pain reduction with these agents but there is no evidence for long-term pain relief (Clinical Evidence 2002).

Key Message

There is evidence that injection therapy (treatment and placebo saline) is effective for the management of patellofemoral pain in the short term compared to no injection therapy. (Level II)

Orthoses (Foot)

In-shoe orthotic devices are thought to reduce patellofemoral pain by preventing excessive pronation of the foot (D'hondt et al. 2002).

In their systematic review, Crossley et al. (2001) described a study by Eng and Pierrynowski (1993) comparing corrective shoe orthoses versus placebo insoles in women with rearfoot varus; both groups received concurrent exercises (comprising quadriceps femoris and hamstring strengthening and stretching exercises). At eight weeks, there was significantly less pain during aggravating activities in the group wearing corrective shoe orthoses. The effectiveness of shoe orthoses as a monotherapy is yet to be determined.

Key Message

There is evidence that corrective foot orthoses in combination with quadriceps and hamstring exercises are effective compared to placebo insoles in women with patellofemoral pain. (Level I)

Physical Therapy

Physical therapy comprises conservative interventions for patellofemoral pain such as muscle strengthening or realignment.

In their systematic review, Crossley et al. (2001) included eight studies (Witvrouw et al. 2000; Clark et al. 2000; Harrison et al. 1999; Thomee 1997; Stiene et al. 1996; Eburne and Bannister 1996; Kowall et al. 1996; McMullen et al. 1990) evaluating different physical therapy techniques for patellofemoral pain. None of the studies compared the chosen treatment to a placebo control. Five of the studies reported that eccentric quadriceps exercises were more effective, particularly in relation to functional outcomes, than standard quadriceps strengthening exercises. Two studies (Clark et al. 2000; Harrison et al. 1999) comparing education and advice versus exercises produced conflicting results. Clark et al. (2000) reported no difference in pain outcomes between those undergoing exercise and those not exercising (effect size for pain = 0.18; 95%CI –1.17, 0.82). Harrison et al. (1999) compared a McConnell-style program of patellar taping, mobilisation and eccentric quadriceps biofeedback versus a program of supervised standard quadriceps exercises and patellar mobilisation versus a standard home exercise program. They reported a significant reduction in pain in the McConnell program group compared to the supervised exercises but no difference between the McConnell program and the home exercise program (effect size for pain = –0.45; 95%CI –1.20, 0.27). Overall, Crossley et al. (2001) concluded that exercises might be effective in reducing pain associated with patellofemoral pain however

there was no strong evidence that one physical intervention was superior to another.

A recently published randomised controlled trial (Crossley et al. 2002) compared a six week treatment regimen consisting of quadriceps muscle retraining, patellofemoral joint mobilisation, patellar taping and daily home exercises with a placebo arm consisting of sham ultrasound, light application of a non-therapeutic gel and placebo taping. On completion of the treatment course, the treatment group showed significantly greater improvement in pain than the placebo group. Further studies are needed to evaluate the efficacy of individual components of combined therapy programs.

Key Messages

> A six-week regimen of quadriceps muscle retraining, patellofemoral joint mobilisation, patellar taping and daily home exercises significantly reduces patellofemoral pain compared to placebo in the short term. (Level II)

> Eccentric quadriceps exercises produce better functional outcomes compared to standard quadriceps strengthening exercises. (Level I)

Conflicting Evidence

Orthoses (Patellofemoral)

Patellofemoral orthoses are thought to reduce patellofemoral pain by influencing patellar tracking. Orthotic devices included knee straps, braces, sleeves and patellar taping techniques (D'hondt et al. 2002).

Crossley et al. (2001) identified two RCTs on the use of patellofemoral orthoses; both involved military populations and had methodological limitations (Finestone et al. 1993; Miller et al. 1997). Finestone et al. (1993) compared an elastic sleeve versus a knee brace versus no treatment and reported no effect from the patellofemoral brace compared to the other groups. Miller et al. (1997) found no difference in pain outcomes between a knee brace versus an infrapatellar strap versus no brace.

The Cochrane Review by D'hondt et al. (2002, last updated in 2002) included five RCTs; two of these (Miller et al. 1997; Timm 1998) described orthotic devices (excluding patellar taping). Miller et al. (1997) compared the Cho-pat® knee strap plus exercises versus the Palumbo® knee brace plus exercises versus exercise alone. There were no statistically significant differences in pain between the groups. Timm (1998) compared the Protonics™ knee brace with no treatment and reported a statistically significant reduction in pain in the treatment group.

D'hondt et al. (2002) concluded that it was inappropriate to draw conclusions on the use of knee orthotics for the treatment of patellofemoral pain as all five studies had methodological limitations.

Key Message

There is conflicting evidence that patellofemoral orthoses are effective compared to other interventions and to no treatment for patellofemoral pain. (Level I)

Insufficient Evidence

Acupuncture

Crossley et al. (2001) located one study comparing four weeks of acupuncture treatment versus no treatment (Jenson et al. 1999) in their systematic review of conservative treatments for patellofemoral pain. Jenson et al. (1999) reported significant improvement in function at one year in the acupuncture group compared to the 'no treatment' group, however Crossley et al. (2001) caution that the placebo effect associated with acupuncture may affect the outcome, highlighting the need for controlled studies.

Key Message

There are no randomised controlled studies evaluating the effect of acupuncture for relief of patellofemoral pain. (No Level I or II studies)

Analgesics (Simple and Opioid)

No placebo-controlled trials were identified for the use of paracetamol or opioid medications in patellofemoral pain.

Key Message

There are no randomised controlled studies of the effectiveness of paracetamol or opioids versus placebo in the treatment of patellofemoral pain. (No Level I or II studies)

Electrical Stimulation

Although the origin of patellofemoral is unknown, it is thought that weakened quadriceps muscles or an imbalance in the strength of the vastus lateralis and vastus medialis obliquus muscles may lead to malalignment of the patella, causing pain (Dursun et al. 2001).

Callaghan et al. (2001) conducted a small pilot study comparing the effect of two types (sequential versus mixed frequency) of electrical stimulation on the rehabilitation of the quadriceps muscle in patients with patellofemoral pain. There was no significant difference between the two methods of muscle stimulation; pain was not a primary outcome measure.

Key Messages

> There are no randomised controlled studies of the effectiveness of electrical stimulation of the quadriceps muscle for patellofemoral pain. (No Level I or II studies)

> There is insufficient evidence that one form of electrical stimulation of the quadriceps muscle is superior to another for treating patellofemoral pain. (Level II)

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)

No placebo-controlled trials were identified for patellofemoral pain and NSAIDs. Clinical Evidence (2002) notes that systematic reviews of NSAIDs in a variety of acute and chronic musculoskeletal conditions have found no important differences in efficacy between different NSAIDs or doses but have identified differences in toxicity related to increased doses and to the nature of the NSAID (Bigos et al. 1994; van Tulder et al. 2002).

Fulkerson and Folcik (1986) reported similar relief of patellofemoral pain from diflusinal compared to naproxen after five days use; however, there was no placebo comparison group in the study.

Key Messages

> There are no randomised controlled studies of the effectiveness of NSAIDs versus placebo in the treatment of patellofemoral pain. (No Level I or II studies)

> Different types of NSAIDs provide similar relief of patellofemoral pain after five days of use. (Level II)

> Serious adverse effects of NSAIDs include gastrointestinal complications (e.g. bleeding, perforation). (Level I)

Patellar Taping

The purpose of patellar taping is to centralise the patella within the trochlear groove to improve patellar tracking (Crossley et al. 2001), however the mechanism responsible for improving pain remains unknown (Harrison et al. 2001).

Crossley et al. (2001) identified two studies (Clark et al. 2000; Kowall et al. 1996) on patellar taping in their systematic review of physical interventions for patellofemoral pain syndrome. Clark et al. (2000) compared pain outcomes in those undergoing patellar taping versus no taping; there was no difference between the groups at three months. Kowall et al. (1996) compared standard quadriceps exercises versus quadriceps exercises and patellar taping. There were no differences in pain observed, however the study involved taping during treatment sessions rather than sustained taping, which is the norm in clinical settings.

A review article (Harrison et al. 2001) identified four studies on patellar taping; pain was an outcome measure in two of these, one of which (Handfield and Kramer 2000) was not described in the Crossley et al. (2001) review. This study investigated the effect of patellar taping using the McConnell technique versus no taping on pain and peak torque during isokinetic concentric quadriceps testing. Subjects were tested with the knee taped and untaped, with a 30-minute rest period between taped and untaped conditions. Pain scores were significantly lower ($p < 0.01$) when the knee was taped.

Subsequently, Crossley et al. (2002) conducted a trial comparing a six-week regimen of physical therapy interventions (comprising quadriceps muscle retraining, patellofemoral joint mobilisation, patellar taping and daily home exercises) versus sham ultrasound, application of a non-therapeutic gel and placebo taping in the control arm. At three months, there was a significant reduction in pain in the physical therapy group compared to the placebo arm. However, further studies are required to determine the efficacy of patellar taping as a single therapy in patellofemoral pain.

No harms were reported for taping however local skin irritation from prolonged taping is a potential problem. Taping is relatively simple and inexpensive in comparison to other interventions.

Key Message

There is insufficient evidence that patellar taping alone is effective in relieving patellofemoral pain, however it may be a useful adjunct to other physical therapy programs. (Level I, II)

Progressive Resistance Brace

Crossley et al. (2001) located one RCT (Timm 1998) on the use of a progressive resistance brace for quadriceps strengthening versus no treatment of patellofemoral pain. Timm (1998) reported significant improvement in pain using the device, however the study was not placebo-controlled and had other methodological limitations. This treatment is not routinely available in Australia.

Key Message

There is insufficient evidence that progressive resistance braces are effective in relieving patellofemoral pain compared to no treatment (This treament is not routinely available in Australia). (Level I)

Therapeutic Ultrasound

A Cochrane Review by Brosseau et al. (2002b) on the effect of therapeutic ultrasound on patellofemoral pain located only one RCT (Antich et al. 1986) that met their inclusion criteria and allocated a score of 1/5 for methodological quality. The study compared a number of interventions, including ice massage versus a combination of ultrasound and ice massage (three minutes of ultrasound followed by two minutes of ice massage) using the contralateral knee as a control ($N = 29$). The combination therapy was not significantly superior to cryotherapy alone in the treatment of patients with patellofemoral pain. It was concluded that there is currently no evidence to support the use of ultrasound for the treatment of patellofemoral pain. No harms were reported.

Two meta-analyses on the use of therapeutic ultrasound for musculoskeletal pain have been conducted, both concluding there is a lack of evidence to support or refute the use of therapeutic ultrasound. Gam and Johannsen (1995) pooled data on 13 RCTs comparing ultrasound with sham ultrasound for the treatment of musculoskeletal pain; three of the studies involved subjects with osteoarthritis of the knee. van der Windt et al. (1999) included the Antich et al. (1986) study in their systematic review but were unable to pool data on the small number of studies involving osteoarthritis of the knee.

Key Message

There is insufficient evidence that therapeutic ultrasound is more effective compared to ice massage for the treatment of patellofemoral pain. (Level I)

Evidence of No Benefit

Laser Therapy

One study (Rogvi-Hansen et al. 1991) comparing low-level laser therapy to sham laser was identified in the Crossley et al. (2001) systematic review. The study reported no difference in pain levels between the groups (effect size = –0.44; Standard Deviation –1.18, 0.30).

Key Message

There is evidence that low-level laser therapy provides similar effect to sham laser in the management of patellofemoral pain. (Level I)

>Economic Implications

A formal economic evaluation has not been performed for these recommendations, nor were any economic evaluations of guidelines for knee pain identified following searches of computer data bases (The Cochrane Library, PubMed) of published literature.

With regards to individual components of this guideline's recommendations for the diagnosis and treatment of anterior knee pain, only the Knee Rules for xray diagnosis of a fracture were subject to a cost-effectiveness evaluation (Nichol et al. 1999). The evaluation was performed in Canada and concluded that xray ordering could be reduced by as much as 46% if Knee Rules were applied in the Emergency Department and that patient health outcomes were not adversely affected. These Rules need further evaluation in the Australian setting but it is likely that they would reduce routine xray ordering, thereby reducing costs.

With regards to other recommendations, most are more likely to lead to cost savings rather than generation of increased costs. Examples of these are:

- Minimising ancillary investigations unless serious conditions are expected should minimise direct costs.

- Advice to stay active should maintain productivity and minimise indirect costs.
- Conservative measures such as foot orthoses and exercises to improve muscle strength appear to be effective and relatively simple and inexpensive to administer.
- Limiting medication use to simple analgesics as first line therapy should reduce pharmaceutical expenditure on acute knee pain.

>References

Abernethy PJ, Townsend PR, Rose RM, Radin EL (1978). Is chondromalacia patellae a separate clinical entity? Journal of Bone and Joint Surgery, 60B: 205–210.

Abrahams S, Kern JH (2001). Anterior knee pain: plica syndrome, the forgotten pathology? Physiotherapy, 87: 523–528.

Adalberth T, Roos H, Lauren M, Akeson P, Sloth M, Jonsson K, Lindstrand A, Lohmander LS (1997). Magnetic resonance imaging, scintigraphy, and arthroscopic evaluation of traumatic hemarthrosis of the knee. American Journal of Sports Medicine, 25: 231–237.

Aglietti P, Insall JN, Cerulli G (1983). Patellar pain and incongruence. I: Measurement of incongruence. Clinical Orthopaedics and Related Research, 176: 217–224.

Ahlback S, Bauer GC, Bohne WH (1968). Spontaneous osteonecrosis of the knee. Arthritis and Rheumatism, 11: 705–733.

Al Rawi Z, Nessan AH (1997) Joint hypermobility in patients with chondromalacia patellae. British Journal of Rheumatology, 36: 1324–1327.

Almeida SA, Trone DW, Leone DM, Shaffer RA, Patheal SL, Long K (1999a). Gender differences in musculoskeletal injury rates: a function of symptom reporting? Medicine and Science in Sports and Exercise, 31: 1807–1812.

Almeida SA, Williams KM, Shaffer RA, Brodine SK (1999b). Epidemiological patterns of musculoskeletal injuries and physical training. Medicine and Science in Sports and Exercise, 31: 1176–1182.

Altman R, Asch E, Bloch D, Bole G, Borenstein D, Brandt K, Christy W, Cooke TD, Greenwald R, Hochberg M et al. (1986). Development of criteria for the classification and reporting of osteoarthritis. Classification of osteoarthritis of the knee. Arthritis and Rheumatism, 29: 1039–1049.

Anderson JJ, Felson DT (1988). Factors associated with osteoarthritis of the knee in the first national Health and Nutrition Examination Survey (HANES I). Evidence for an association with overweight, race, and physical demands of work. American Journal of Epidemiology, 128: 179–189.

Antich TJ, Randall CC, Westbrook RA, Morrissey MC, Brewster CE (1986). Physical therapy treatment of knee extensor mechanism disorders: comparison of four treatment modalities. Journal of Orthopaedic and Sports Physical Therapy, 8: 255–259.

Amadio PC (1988). Pain dysfunction syndromes. Journal of Bone and Joint Surgery, 70A: 944–949.

Arroll B, Ellis-Pegler E, Edwards A, Sutcliffe G (1997). Patellofemoral pain syndrome: a critical review of the clinical trials on non-operative therapy. American Journal of Sports Medicine, 25: 207–212.

Aynaci O, Ahmetoglu A, Reis A, Turhan AU (2001). Synovial hemangioma in Hoffa's fat pad (case report). Knee Surgery, Sports Traumatology, Arthroscopy, 9: 355–357.

Bae DK, Kwon OS (1997). Snapping knee caused by the gracilis and semitendonosus tendon. A case report. Bulletin (Hospital for Joint Diseases [New York, NY]), 56: 177–179.

Baker MM, Juhn MS (2000). Patellofemoral pain syndrome in the female athlete. Clinics in Sports Medicine, 19: 315–329.

Baquie P, Brukner P (1997). Injuries presenting to an Australian sports medicine centre: a 12-month study. Clinical Journal of Sport Medicine, 7: 28–31.

Bauer SJ, Hollander JE, Fuchs SH, Thode HC (1995). A clinical decision rule in the evaluation of acute knee injuries. Journal of Emergency Medicine, 13: 611–615.

Baumgarten KM, Mont MA, Rifai A, Hungerford DS (2001). Atraumatic osteonecrosis of the patella. Clinical Orthopedics, 383: 191–196.

Bennett GA, Waine H, Bauer W (1942). Changes in the knee joint at various ages with particular reference to the nature and development of degenerative joint disease. Oxford University Press: Oxford.

Bentley G, Dowd G (1984). Current concepts of etiology and treatment of chondromalacia patellae. Clinical Orthopaedics and Related Research, 189: 209–228.

Bergquist SR, Hannan MT, Neuhauser KB, Wieland DA, Felson DT (1993). Lack of validity and reliability of physical exam components of the American College of Rheumatology osteoarthritis diagnostic criteria. Arthritis and Rheumatism, 36 (suppl 9): S58.

Bianchi S, Zwass A, Abdelwahab IF, Banderali A (1994). Diagnosis of tears of the quadriceps tendon of the knee: value of sonography. American Journal of Roentgenology, 162: 1137–40.

Biedert RM, Warnke K (2001). Correlation between the Q angle and the patella position: a clinical and axial computed tomography evaluation. Archives of Orthopaedic and Trauma Surgery, 121: 346–349.

Bigos S, Bowyer O, Braen G, et al. (1994). Acute low back problems in adults. Clinical Practice Guideline no.14. AHCPR Publication No. 95-0642. Agency for Health Care Policy and Research, Public Health Service, US, Department of Health: Rockville MD.

Bikkina RS, Chaljub G, Singh H, Allen SD (2002). Magnetic resonance imaging of simultaneous bilateral quadriceps tendon rupture in a weightlifter: case report. Journal of Trauma, 52(3): 582–584.

Blake GM, Fogelman I (2002) Bone densitometry, steroids and osteoporosis. Current Opinion in Nephrology and Hypertension, 11: 641–647.

Blazina ME, Karlan RK, Jobe FW (1973). Jumper's knee. Orthopedic Clinics of North America, 4: 665–678.

Brand C, Muirden KD (1987). When the patient has a swollen knee. Patient Management, 149–160.

Brechter JH, Powers CM (2002). Patellofemoral joint stress during stair ascent and descent in persons with patellofemoral pain. Gait Posture, 16: 115–123.

Broom MJ, Fulkerson JP (1986). The plica syndrome: a new perspective. Orthopedic Clinics of North America, 17: 279–281.

Brosseau L, Casimiro L, Milne S, Robinson V, Shea B, Tugwell P, Wells G (2002a). Deep transverse friction massage for treating tendonitis. In: The Cochrane Library; Issue 2. Oxford: Update Software.

Brosseau L, Casimiro L, Robinson V, Milne S, Shea B, Judd M, Wells G, Tugwell P (2002b). Therapeutic ultrasound for treating patellofemoral pain syndrome. In: The Cochrane Library; Issue 2. Oxford: Update Software.

Brossmann J, Muhle C, Schroder C, Melchert UH, Bull CC, Spielmann RP, Heller M (1993). Patellar tracking patterns during active and passive knee extension: evaluation with motion-triggered cine MR imaging. Radiology, 187: 205–212.

Brown DE, Alexander AH, Lichtman DM (1984). The Elmslie-Trillat procedure: evaluation in patellar dislocation and subluxation. American Journal of Sports Medicine, 12: 104–108.

Brown TR, Quinn SF (1993). Evaluation of chondromalacia of the patellofemoral component with axial magnetic resonance imaging. Skeletal Radiology, 22: 325–328.

Butler JC, Andrews JR (1998). The role of arthroscopic surgery in the evaluation of acute traumatic hemarthrosis of the knee. Clinical Orthopaedics and Related Research, 228: 150–152.

Callaghan MJ, McCarthy CJ, Al-Omar A, Oldham JA (2000). The reproducibility of multi-joint isokinetic and isometric assessments in a healthy and patient population. Clinical Biomechanics, 15: 678–683.

Callaghan MJ, Oldham JA, Winstanley J (2001). A comparison of two types of electrical stimulation of the quadriceps in the treatment of patellofemoral pain syndrome. A pilot study. Clinical Rehabilitation, 15: 637–646.

Campa J, Broadnax W, Broderick J (2001) Neuropathic and sympathetically maintained pain complicating trauma or surgery to the knee. Neurology, 41: 165.

Carson WG Jr, James SL, Larson RL, Singer KM, Winternitz WW (1984). Patellofemoral disorders: physical and radiographic evaluation. Part I: Physical examination. Clinical Orthopaedics and Related Research, 185: 165–177.

Carter C, Sweetnam R (1958). Familial joint laxity and recurrent dislocation of the patella. Journal of Bone and Joint Surgery, 664–667.

Caylor D, Fites R, Worrell TW (1993). The relationship between quadriceps angle and anterior knee pain syndrome. Journal of Orthopaedic and Sports Physical Therapy, 17: 11–6.

Cesarelli M, Bifulco P, Bracale M (2000). Study of the control strategy of the quadriceps muscles in anterior knee pain. IEEE Transactions on Rehabilitation Engineering, 8: 330–341.

Chapman-Jones D, Paterson A, Johnston S (1998). Plain radiography of the knee: a useful diagnostic modality for patients with non-specific knee pain: a retrospective study of plain radiography of the knee in comparison with MRI in patients with non-specific knee pain. Radiography, 4: 183–187.

Clark DI, Downing N, Mitchell J et al. (2000). Physiotherapy for anterior knee pain: a randomized controlled trial. Annals of the Rheumatic Diseases, 59: 700–704.

Clement DB, Taunton SE, Smart GW, McNicol KL (1981). A survey of overuse running injuries. The Physician and Sportsmedicine, 9: 47–58.

Clinical Evidence. June 2002, 7. BMJ: London.

Cohen ZA, Roglic H, Grelsamer RP, Henry JH, Levine WN, Mow VC, Ateshian GA (2001). Patellofemoral stresses during open and closed kinetic chain exercises: an analysis using computer simulation. American Journal of Sports Medicine, 29: 480–487.

Collins DH, Meachim G (1961). Sulfate fixation by human articular cartilage compared in the knee and shoulder joints. Annals of the Rheumatic Diseases, 20: 117.

Cook JL, Khan KM, Kiss ZS, Purdam CR, Griffiths L (2001). Reproducibility and clinical utility of tendon palpation to detect patellar tendonopathy in young basketball players. British Journal of Sports Medicine, 35: 65–69.

Cooper C, McAlindon T, Coggon D, Eggar P, Dieppe P (1994). Occupational activity and osteoarthritis of the knee. Annals of the Rheumatic Diseases, 53: 90–93.

Cooper DE (1999). Snapping popliteus tendon syndrome. A cause of mechanical knee popping in athletes. American Journal of Sports Medicine, 27: 671–674.

Cooper DE, DeLee JC, Ramamurthy S (1989). Reflex sympathetic dystrophy of the knee. Treatment using continuous epidural anesthesia. Journal of Bone and Joint Surgery, 71A: 365–369.

Crespo E, Sala D, Crespo R, Silvestre A (2001). Transient osteoporosis. Acta Orthopaedica Belgica, 67: 330–337.

Crites BM, Lohnes J, Garrett WE Jr (1998). Snapping popliteal tendon as a source of lateral knee pain. Scandinavian Journal of Medical Science Sports, 8: 243–244.

Crossley K, Bennell K, Green S, Cowan S, McConnell J (2002). Physical therapy for patellofemoral pain. American Journal of Sports Medicine, 30: 857–865.

Crossley K, Bennell K, Green S, McConnell J (2001). A systematic review of physical interventions for patellofemoral pain syndrome. Clinical Journal of Sport Medicine, 11: 103–110.

Csintalan RP, Schulz MM, Woo J, McMahon PJ, Lee TQ (2002) Gender differences in patellofemoral joint biomechanics. Clinical Orthopaedics, 402: 260–269.

Cushnaghan J, Cooper C, Dieppe P, Kirwan J, McAlindon TE, McCrae F (1990). Clinical assessment of osteoarthritis of the knee. Annals of the Rheumatic Diseases, 49: 768–770.

D'hondt NE, Struijs PA, Kerkhoffs GM, Verheul C, Lysens R, Aufdemkampe G, Van Dijk CN (2002) Orthotic devices for treating patellofemoral pain syndrome. Cochrane Library; Issue 2. Oxford: Update Software.

Daniel DM (1991). Assessing the limits of knee motion. The American Journal of Sports Medicine, 19: 139–147.

Daniel DM, Stone ML, Dobson BE, Fithian DC, Rossman DJ, Kaufman KR (1994). Fate of the ACL-injured patient. A prospective outcome study. American Journal of Sports Medicine, 22: 632–644.

Darracott J, Vernon-Roberts B (1971). The bony changes in "chondromalacia patellae". Rheumatology and Physical Medicine, 11: 175–179.

Davies AP, Costa ML, Donnell ST, Glasgow MM, Shepstone L (2000). The sulcus angle and malalignment of the extensor mechanism of the knee. Journal of Bone and Joint Surgery, 82B: 1162–1166.

DeHaven KE, Collins HR (1975). Diagnosis of internal derangements of the knee. The role of arthroscopy. Journal of Bone and Joint Surgery, 57A: 802–810.

DeHaven KE, Dolan WA, Mayer PJ (1979). Chondromalacia patellae in athletes. Clinical presentation and conservative management. American Journal of Sports Medicine, 7: 5–11.

DeHaven KE, Lintner DM (1986). Athletic injuries: comparison by age, sport, and gender. American Journal of Sports Medicine, 14: 218–24.

Derscheid GL, Feiring DC (1987). A statistical analysis to characterize treratment adherence of the 18 most common diagnoses seen at the sports medicine clinic. Journal of Orthopaedic and Sports Physical Therapy, 9: 40–46.

Dervin GF, Stiell IG, Wells GA, Rody K, Grabowski J (2001). Physicians' accuracy and interrator reliability for the diagnosis of unstable meniscal tears in patients having osteoarthritis of the knee. Canadian Journal of Surgery, 44: 267–274.

Detenbeck LC (1972). Infrapatellar traumatic neuroma resulting from dashboard injury. Journal of Bone and Joint Surgery, 54A: 170–172.

Devereaux MD, Lachmann SM (1984). Patello-femoral arthralgia in athletes attendong a Sports Injury Clinic. British Journal of Sports Medicine, 18: 18–21.

Dickinson FL, Harper WM, Finlay DB (1997). Knee tumours—duration and nature of symptoms prior to investigation. British Journal of Radiology, 70: 635–637.

Dugdale TW, Barnett PR (1986). Historical background: patellofemoral pain in young people. Orthopedic Clinics of North America, 17: 211–219.

Dupont JY (1997). Synovial plicae of the knee. Controversies and review. Clinics in Sports Medicine, 16: 87–122.

Dursun N, Dursun E, Kilic Z (2001). Electromyographic biofeedback-controlled exercise versus conservative care for patellofemoral pain syndrome. Archives of Physical Medicine and Rehabilitation, 82: 1692–1695.

Dye SF, Vaupel GL, Dye CC (1998). Conscious neurosensory mapping of the internal structures of the human knee without intraarticular anaesthesia. Amercian Journal of Sports Medicine, 6: 773–777.

Eburne J, Bannister G (1996), The McConnell regimen versus isometric quadriceps exercises in the management of anterior knee pain. A randomized prospective controlled trial. Knee, 3: 151–153.

Eckhoff DG, Montgomery WK, Kilcoyne RF, Stamm ER (1994). Femoral morphometry and anterior knee pain. Clinical Orthopaedics and Related Research, 302: 64–68.

Ekstrom H, Engholm G, Nyqvist B, Wallenquist A (1983). Knabesvar som arbetsmedicinisk problem. Bygghalsans forskningsstiftelse: Stockholm.

el Khoury GY, Wira RL, Berbaum KS, Pope TL Jr, Monu JU (1992). MR imaging of patellar tendonitis. Radiology, 184: 849–854.

Ellen MI, Jackson HB, DiBiase SJ (1999). Uncommon causes of anterior knee pain: a case report of infrapatellar contracture syndrome. American Journal of Physical Medicine and Rehabilitation, 78: 376–380.

Eng JJ, Pierrynowski MR (1993). Evaluation of soft foot orthotics in the treatment of patellofemoral pain syndrome. Physical Therapy, 73: 62–70.

Fairbank JC, Pynsent PB, van PJ, Phillips H (1984). Mechanical factors in the incidence of knee pain in adolescents and young adults. Journal of Bone and Joint Surgery, 66B: 685–693.

Feagin JA Jr, Cooke TD (1989). Prone examination for anterior cruciate ligament insufficiency. Journal of Bone and Joint Surgery, 71B: 863.

Felson DT (1990). The epidemiology of knee osteoarthritis: results from the Framington Osteoarthritis Study. Seminars in Arthritis and Rheumatism, 20: 42–50.

Ferguson PC, Griffin AM, Bell RS (1997). Primary patellar tumors. Clinical Orthopedics, 336: 199–204.

Ferrari JD, Bach BR Jr (1998). Knee pain in adults: when to manage, when to refer: fracture, suspicion of infection, and locking call for immediate referral. Journal of Musculoskeletal Medicine, 15: 52–63.

Ferretti A, Puddu G, Mariani PP, Neri M (1985). The natural history of jumper's knee. Patellar or quadriceps tendonitis. International Orthopaedics, 8: 239–242.

Ferretti A (1986). Epidemiology of jumper's knee. Sports Medicine, 3: 289–295.

Finsterbush A, Frankl U, Mann G (1989). Fat pad adhesion to partially torn anterior cruciate ligament: a cause of knee locking. American Journal of Sports Medicine, 17: 92–95.

Finestone A, Radin EL, Lev B, Schlamkovitch N, Wiener M, Milgrom C (1993). Treatment of overuse patellofemoral pain. Prospective randomized controlled clinical trial in a military setting. Clinical Orthopaedics and Related Research, 293: 208–210.

Fitzgerald GK, McClure PW (1995). Reliability of measurements obtained with four tests for patellofemoral alignment. Physical Therapy, 75: 84–90.

Fouts D, Welker MJ, Haines DJ (1999). Diagnosis and treatment of common knee injuries. Family Practice Recertification, 21: 62–76.

Fowler PJ, Lubliner JA (1989). The predictive value of five clinical signs in the evaluation of meniscal pathology. Arthroscopy, 5: 184–186.

Fulkerson JP (1989). Evaluation of the preipatellar soft tissues and retinaculum in patients with patellofemoral pain. Clinics in Sports Medicine, 8:197–202.

Fulkerson JP (2002). Diagnosis and treatment of patients with patellofemoral pain. American Journal of Sports Medicine, 30: 447–455.

Fulkerson JP, Arendt EA (2000). Anterior knee pain in females. Clinical Orthopaedics and Related Research, 372: 69–73.

Fulkerson JP, Folcik MA (1986). Comparison of diflunisal and naproxen for relief of anterior knee pain. Clinical Therapeutics, 9 (Suppl C): 59–61.

Fulkerson JP, Hungerford DS (1990). Disorders of the patellofemoral joint (2nd Edition). Baltimore, MD.

Gam AN, Johannsen F (1995). Ultrasound therapy in musculoskeletal disorders: a meta-analysis. Pain, 63: 85–91.

Georgoulis AD, Papageorgiou CD, Moebius UG, Rossis J, Papadonikolakis A, Soucacos PN (2002). The diagnostic dilemma created by osteoid osteoma that presents as knee pain. Arthroscopy, 18: 32–37.

Glockner JF, Sundaram M, Pierron RL (1998). Radiologic case study. Transient migratory osteoporosis of the hip and knee. Orthopedics, 21: 594–596.

Godshall RW (1975). The predictability of athletic injuries: an eight year study. American Journal of Sports Medicine, 3: 50–54.

Goodfellow J, Hungerford DS, Woods C (1976). Patello-femoral joint mechanics and pathology. 2. Chondromalacia patellae. Journal of Bone and Joint Surgery, 58B: 291–299.

Gotlin RS (2000). Effective rehabilitation for anterior knee pain. Journal of Musculoskeletal Medicine, 17: 421–432.

Grana WA, Moretz JA (1978). Ligamentous laxity in secondary school athletes. Journal of the American Medical Association, 240: 1975–1976.

Gray SD, Kaplan PA, and Dussault RG (1997). Imaging of the knee. Current status. Orthopedic Clinics of North America, 28: 643–658.

Grelsamer RP (2000). Current concepts review. Patellar malalignment. Journal of Bone and Joint Surgery, 82A: 1639–1650.

Grelsamer RP, Klein JR (1998). The biomechanics of the patellofemoral joint. The Journal of Orthopaedic and Sports Physical Therapy, 28: 286–297.

Gupta MN, Sturrock RD, Field MA (2001). Prospective 2-year study of 75 patients with adult-onset septic arthritis. Rheumatology (Oxford), 40: 24–30.

Handfield T, Kramer J (2000). Effect of McConnell taping on perceived pain and knee extensor torques during isokinetic exercise performed by patients with patellofemoral pain syndrome. Physiotherapy Canada, 4: 39–44.

Hansen L, Larsen S, Laulund T (2001). Traumatic bilateral quadriceps tendon rupture. Journal of Orthopedic Science, 6: 187–188.

Hardacker WT, Carrett WE, Jr., Bassett FH3 (1990). Evaluation of acute traumatic hemarthrosis of the knee joint. Southern Medical Journal, 83: 640–644.

Hardacker WT, Whipple TL, Bassett FH3 (1980). Diagnosis and treatment of the plica syndrome of the knee. Journal of Bone and Joint Surgery, 62A: 221–225.

Harrison E, Magee D (2001). Patellofemoral pain syndrome: the ongoing challenges in etiology, diagnosis, and management. Critical Reviews in Physical and Rehabilitation Medicine, 13: 105–129.

Harrison E, Sheppard MS, McQuarrie AM (1999). A randomized controlled trial of physical therapy treatment programs in patellofemoral pain syndrome. Physiotherapy Canada, 51: 93–100, 106.

Hart DJ, Spector TD, Brown P, Wilson PW, Doyle DV, Silman AJ (1991). Clinical signs of early osteoarthritis; reproducibility and relation to x-ray changes in 541 women in the general population. Annals of the Rheumatic Diseases, 50: 467–70.

Hayes KW, Petersen CM (2001). Reliability of assessing end-feel and pain and resistance sequence in subjects with painful shoulders and knees. The Journal of Orthopaedic and Sports Physical Therapy, 31: 432–445.

Heir T, Glomsaker (1996). Epidemiology of musculoskeletal injuries among Norwegian conscripts undergoing basic military training. Scandinavian Journal of Medicine Science in Sports, 6:186–191.

Herrington L (2000). The effect of patellofemoral joint taping. Critical Reviews in Physical and Rehabilitation Medicine, 12: 271–276.

Holibkov A, Machalek AL, Holabka R, Chromek Z (1985). Lesions of the knee joint menisci in miners. Acta Universitatis Palackianae Olomucensis Facultatis Medicae, 123: 147–168.

Hopman-Rock M, Odding E, Hofman A, Kraaimaat FW, Bijlsma JW (1997). Differences in health status of older adults with pain in the hip or knee only and with additional mobility restricting conditions. Journal of Rheumatology, 24: 2416–23.

Horton MG, Hall TL (1989). Quadriceps femoris muscle angle: normal values and relationships with gender and selected skeletal measures. Physical Therapy, 69: 897–901.

House JH, Ahmed K (1977). Entrapment neuropathy of the infrapatellar branch of the saphenous nerve. American Journal of Sports Medicine, 5: 217–224.

Hughston JC, Flandry F, Brinker MR, Terry GC, Mills JC3 (1996). Surgical correction of medial subluxation of the patella. American Journal of Sports Medicine, 24: 486–491.

Hunter DJ, March L, Sambrook PN (2002). Knee osteoarthritis: the influence of environmental factors. Clinical and Experimental Rheumatology, 20: 93–100.

Hvid I, Andersen LI (1982). The quadriceps angle and its relation to femoral torsion. Acta Orthopaedica Scandinavica, 53: 577–579.

Hvid I, Andersen LI, Schmidt H (1981). Chondromalacia patellae. The relation to abnormal patellofemoral joint mechanics. Acta Orthopaedica Scandinavica, 52: 661–666.

Ike RW, O'Rourke KS (1995). Compartment-directed physical examination of the knee can predict articular cartilage abnormalities disclosed by needle arthroscopy. Arthritis and Rheumatism, 38: 917–925.

Indelli PF, Dillingham M, Fanton G, Schurman DJ (2002). Septic arthritis in postoperative anterior cruciate ligament reconstruction. Clinical Orthopaedics and Related Research, 398: 182–188.

Insall J (1979), Chondromalacia patellae: patellar malalignment syndrome. Orthopedic Clinics of North America, 10: 117–27.

Insall J (1980). Current concepts review: patellar pain. Journal of Bone and Joint Surgery, 64A: 933–936.

Irrgang JJ, Anderson AF, Boland AL, Harner CD, Kurosaka M, Neyret P, Richmond JC, Shelborne KD (2001). Development and validation of the International Knee Documentation Committee Subjective Knee Form. American Journal of Sports Medicine, 29: 600–613.

Jackson DW, Jarrett H, Bailey D, Kausek J, Swanson J, Powell JW (1978). Injury prediction in the young athlete: a preliminary report. American Journal of Sports Medicine, 6: 6–14.

Jacobson JA, van Holsbeeck MT (1998). Musculoskeletal ultrasonography. Orthopedic Clinics of North America, 29: 135–167.

James SL, Bates BT, Osternig LR (1978). Injuries to runners. American Journal of Sports Medicine, 6: 6–14.

Jensen DB, Albrektsen SB (1990). The natural history of chondromalacia patellae. A 12-year follow-up. Acta Orthopaedica Belgica, 56: 503–506.

Jenson R, Gotheson O, Liseth K et al. (1999). Acupuncture treatment of patellofemoral pain syndrome. Journal of Alternative and Complementary Medicine, 5: 521–527.

Johnson DP, Eastwood DM, Witherow PJ (1993). Symptomatic synovial plicae of the knee. Journal of Bone and Joint Surgery, 75A: 1485–1496.

Johnson MW (2000). Acute knee effusions: a systematic approach to diagnosis. American Family Physician, 61: 2391–2400.

Jones A, Hopkinson N, Pattrick M, Berman P, Doherty M (1992). Evaluation of a method for clinically assessing osteoarthritis of the knee. Annals of the Rheumatic Diseases, 51: 243–245.

Jones BH, Cowan DN, Tomlinson JP, Robinson JR, Polly DW, Frykman PN (1993). Epidemiology of injuries associated with physical training among young men in the army. Medicine and Science in Sports and Exercise, 25: 197–203.

Jones RB, Barlett EC, Vainright JR, Carroll RG (1995). CT determination of tibial tubercle lateralization in patients presenting with anterior knee pain. Skeletal Radiology, 24: 505–509.

Joseph TN, Mujtaba M, Chen AL, Maurer SL, Zuckerman JD, Maldjian C, Di Cesare PE (2001). Efficacy of combined technetium–99m sulfur colloid/indium-111 leukocyte scans to detect infected total hip and knee arthroplasties. Journal of Arthroplasty, 16: 753–758.

Kaandorp CJ, Van Schaardenburg D, Krijnen P, Habbema JD, van de Laar MA (1995). Risk factors for septic arthritis in patients with joint disease. A prospective study. Arthritis and Rheumatism, 38: 1819–1825.

Kaeding C, Tomczak RL (2001). Running injuries about the knee. Clinics in Podiatric Medicine and Surgery, 18: 307–318.

Kaempffe FA (1995). Neoplasm as a cause of shoulder pain. The Journal of Family Practice, 40: 480–485.

Kalenak A, Morehouse CA (1975). Knee stability and knee ligament injuries. Journal of the American Medical Association, 234: 1143–1145.

Kannus P, Natri A, Nittymaki S, Jarvinen, M (1992). Effect of intrarticular glycosaminoglycan polysulphate treatment on patellofemoral pain syndrome. A prospective randomized double blind trial comparing glycosaminoglycan polysulphate with placebo and quadriceps muscle exercises. Annals of the Rheumatic Diseases, 35: 1053–1061.

Kannus P, Nittymaki S (1994). Which factors predict outcome in the nonoperative treatment of patellofemoral pain syndrome? A prospective follow-up study. Medicine and Science in Sports and Exercise, 26: 289–296.

Kannus P, Natri A, Paakkala T, Jarvinen M (1999). An outcome study of chronic patellofemoral pain syndrome. Journal of Bone and Joint Surgery, 81(A): 355–363.

Karlson S (1939). Chondromalacia patellae. Acta Orthopaedica Scandinavica, 83:381.

Karlsson J, Thomee R, Sward L (1996). Eleven year follow-up of patello-femoral pain syndrome. Clinical Journal of Sport Medicine, 6: 22–26.

Kasch J, Enderlein G (1986). Kniegelenksschaden im Schiffbau. Beitrage zur Orthopadie und Traumatologie, 33: 48–49.

Kasim N, Fulkerson JP (2000). Resection of clinically localized segments of painful retinaculum in the treatment of selected patients with anterior knee pain. American Journal of Sports Medicine, 28: 811–814.

Kec RM, Richman PB, Szucs PA, Mandell M, Eskin B (2003). Can emergency department triage nurses appropriately utilize the Ottawa knee rules to order radiographs?-An implementation trial, Academic Emergency Medicine, 10: 146–150.

Kelly MA, Gotlin RS, Lewis L (1994). Reflex sympathetic dystrophy. In: Scott WS (ed). The Knee. Mosby Yearbook: St. Louis. pp 365–77.

Kelly BM, Rao N, Louis SS, Kostes BT, Smith RM (2001). Bilateral, simultaneous, spontaneous rupture of quadriceps tendons without trauma in an obese patient: a case report. Archives of Physical Medicine and Rehabilitation, 82: 415–418.

Khine H, Dorfman DH, Avner JR (2001). Applicability of Ottawa knee rule for knee injury in children. Pediatric Emergency Care 17: 401–404.

Kim SJ, Choe WS (1996). Pathological infrapatellar plica: a report of two cases and literature review. Arthroscopy, 12: 236–239.

Kim SJ, Kim JY, Lee JW (2002). Pathologic infrapatellar plica. Arthroscopy, 18: E25.

Kivimaki J, Riihimaki H, Hanninen K (1992). Knee disorders in carpet and floor layers and painters. Scandinavian Journal of Work, Environment and Health, 18: 310–316.

Kivimaki J, Riihimaki H, Hanninen K (1994). Knee disorders in carpet and floor layers and painters. Part II: Knee symptoms and patellofemoral indices. Scandinavian Journal of Rehabilitation Medicine, 26: 97–101.

Klein W (1983). The medial shelf of the knee. A follow-up study. Archives of Orthopaedic and Trauma Surgery, 102: 67–72.

Kowall DM (1980). Nature and causes of injuries in woman resulting from an endurance training program. American Journal of Sports Medicine, 8: 265–269.

Kowall MG, Kolk G, Nuber GW et al. (1996). Patellar taping in the treatment of patellofemoral pain. A prospective randomized study. American Journal of Sports Medicine, 24: 61–66.

Kransdorf MJ (1995) Malignant soft tissue tumors in a large referral population: distribution of diagnosis by age, sex, and location. American Journal of Roentgenology, 164: 129–134.

Krebs VE, Parker RD (1994). Arthroscopic resection of an extrasynovial ossifying chondroma of the infrapatellar fat pad: end-stage Hoffa's disease? Arthroscopy, 10: 301–304.

Kujala UM, Kvist M, Osterman K, Friderg O, Aalto T (2001). Factors predisposing army conscripts to knee exertion injuries incurred in a physical training program. Clinical Orthopaedics and Related Research, 210: 203–211.

Lam PL, Ng GYF (2001). Activation of the quadriceps muscle during semisquatting with different hip and knee positions in patients with anterior knee pain. American Journal of Physical Medicine and Rehabilitation, 80: 804–808.

Langguth DM, Klestov A, Denaro C (2002). Synovial osteochondromatosis. Internal Medicine Journal, 32: 419–420.

Larikka MJ, Ahonen AK, Junila JA, Niemela O, Hamalainen MM, Syrjala HP (2001). Improved method for detecting knee replacement infections based on extended combined 99mTc-white blood cell/bone imaging. Nuclear Medicine Communications, 22: 1145–1150.

Lawrence JS, Aitken-Swan J (1952). Rheumatism in miners. Part 1: Rheumatic complaints. British Journal of Industrial Medicine, 9: 1–18.

Leach RE, Baumgard S, Broom J (1973). Obesity: its relationship to osteoarthritis of the knee. Clinical Orthopaedics and Related Research, 93: 271–273.

Lee TQ, Sandusky MD, Adeli A, McMahon PJ (2002) Effects of simulated vastus medialis strength variation on patellofemoral joint biomechanics in human cadaver knees. Journal of Rehabilitation Research and Development, 39: 429–438.

Lee TQ, Yang BY, Sandusky MD, McMahon PJ (2001). The effects of tibial rotation on the patellofemoral joint: assessment of the changes in in situ strain in the peripatellar retinaculum and the patellofemoral contact pressures and areas. Journal of Rehabilitation Research and Development, 38: 463–469.

Lee YU, Sartoris DJ (1995). Imaging of the knee. Current Opinion in Orthopaedics, 6: 56–65.

Leslie IJ, Bentley G (1978). Arthroscopy in the diagnosis of chondromalacia patellae. Annals of the Rheumatic Diseases, 37: 540–547.

Levine J (1979). Chondromalacia patellae. The Physician and Sportsmedicine, 7: 41–49.

Levitsky K A, Hozack W J, Balderston R A, Rothman R H, Gluckman S J, Maslack M M, Booth R E Jr (1991). Evaluation of the painful prosthetic joint. Relative value of bone scan, sedimentation rate, and joint aspiration. Journal of Arthroplasty, 6: 237–244.

Lindberg H, Montgomery F (1987). Heavy labor and the occurrence of gonarthrosis. Clinical Orthopaedics and Related Research, 214: 235–236.

Livingston LA, Mandigo JL (1999). Bilateral Q angle asymmetry and anterior knee pain syndrome. Clinical Biomechanics, 14: 7–13.

Lucas TS, DeLuca PF, Nazarian DG, Bartolozzi AR, Booth RE Jr. (1999). Arthroscopic treatment of patellar clunk. Clinical Orthopedics, 367: 226–229.

Lundy DW, Aboulafia AJ, Otis JB, Fuller TR, Ogden JA (1997). Myxoid liposarcoma of the retropatellar fat pad. American Journal of Orthopedics, 26: 287–289.

Lupi L, Bighi S, Cervi PM, Limone GL, Massari L (1990). Arthrography of the plica syndrome and its significance. European Journal of Radiology, 11: 15–18.

Maffulli N, Chan KM, Bundoc RC, Cheng JC (1997). Knee arthroscopy in Chinese children and adolescents: an eight-year prospective study. Arthroscopy, 13: 18–23.

Malghem J, Maldague B (1989). Depth insufficiency of the proximal trochlear groove on lateral radiographs of the knee: relation to patellar dislocation. Radiology, 170: 507–510.

Matava MJ, Patton CM, Luhmann S, Gordon JE, Schoenecker PL (1999). Knee pain as the initial symptom of slipped capital femoral epiphysis: an analysis of initial presentation and treatment. Journal of Pediatric Orthopedics, 19: 455–460.

Matelic TM, Aronsson DD, Boyd DW, Jr., LaMont RL (1993). Acute hemarthrosis of the knee in children. American Journal of Sports Medicine, 23: 668–671.

Matheson GO, Macintyre JG, Taunton JE, Clement DB, Lloyd-Smith R (1989). Musculoskeletal injuries associated with physical activity in older adults. Medicine and Science in Sports and Exercise, 21: 379–385.

Matsusue Y, Yamamuro T, Hama H, Kuzuoka K, Ueo T, Thomson N (1994). Symptomatic type D (separated) medial plica: clinical features and surgical results. Arthroscopy, 10: 281–285.

Maurizio E (1963). La tendonite rotulea nei giocatori di pallavolo. Arch Soc Tosco Umbra Chir, 24: 443–452.

Mbaruk A (1980). Traumatische chondropathia patellae, ihre versicherungsmedizinische Bedeurung bei Beductachtung als Unfallfolge-Therapie. Beitrage zur Orthopadie und Traumatologie, 27: 217–226.

McAlindon T, Cooper C, Kirwan J, Dieppe P (1992). Knee pain and disability in the community. British Journal of Rheumatology, 31: 189–192.

McConnochie KM, Roghmann KJ, Pasternack J, Monroe DJ, Monaco LP (1990). Prediction rules for selective radiographic assessment of extremity injuries in children and adolescents. Pediatrics, 86: 45–57.

McKenzie DC, Clement DB, Taunton JE (1985). Running shoes, orthotics and injuries. Sports Medicine, 2: 334–347.

McMullen W, Roncarati A, Koval P (1990). Static and isokinetic treatments of chondromalacia patella: a comparative investigation.

Journal of Orthopaedic and Sports Physical Therapy, 12: 256–266.

McNair PJ, Marshall RN, Matheson JA (1990). Important features associated with acute anterior cruciate ligament injury. New Zealand Medical Journal, 103: 537–539.

McNally EG, Ostlere SJ, Pal C, Phillips A, Reid H, Dodd C (2000). Assessment of patellar maltracking using combined static and dynamic MRI. European Radiology, 10: 1051–1055.

McNally EG (2001). Imaging assessment of anterior knee pain and patellar maltracking. Skeletal Radiology, 30: 484–495.

Meachim G, Emery IH (1974). Quantitative aspects of patello-femoral cartilage fibrillation in Liverpool necropsies. Annals of the Rheumatic Diseases, 33: 39–47.

Mears SC, Cosgarea AJ (2001). Surgical treatment options in patellofemoral disorders. Current Opinion in Orthopaedics, 12 (2): 167–173.

Merskey H (1979). Pain terms: a list with definitions and notes on usage recommended by the IASP Subcommittee on Taxonomy. Pain, 6: 249–252.

Merskey H, Bogduk N (eds) (1994). Classification of Chronic Pain. Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms (2nd Edition). IASP Press: Seattle.

Meyer S, Reinhard H, Graf N, Puschel W, Ziegler K, Schneider G (2002). The importance of conventional radiographs in the diagnosis of osteosarcoma. Klin Padiatr, 214: 58–61.

Milgrom C, Finestone A, Eldad A, Shlamkovitch N (1991). Patellofemoral pain caused by overactivity. A prospective study of risk factors in infantry recruits. Journal of Bone and Joint Surgery, 73A: 1041–1043.

Milgrom C, Finestone A, Shlamkovitch N, Giladi M, Radin E (1996). Anterior knee pain caused by overactivity: a long term prospective followup. Clinical Orthopaedics and Related Research, 331: 256–260.

Millbauer DL, Patel SJ (1986). Roentgenographic examination of the knee. In: Nicholas JA HE (ed). The Lower Extremity and Spine in Sports Medicine. St. Louis. pp 801–840.

Miller MD, Hinkin DT, Wisnowski JW (1997). The efficacy of orthotics for anterior knee pain in military trainees. A preliminary report. American Journal of Knee Surgery, 10: 10–13.

Mital MA, Hayden J (1979). Pain in the knee in children: the medial plica shelf syndrome. Orthopedic Clinics of North America, 10: 713–722.

Moretz JA, Walters R, Smith L (1982). Flexibility as a predictor of knee injuries in college football players. The Physician and Sportsmedicine, 10: 93–97.

Morgan B, Mullick S, Harper WM, Finlay DB (1997). An audit of knee radiographs performed for general practitioners. The British Journal of Radiology, 70: 256–260.

Morini G, Chiodi E, Centanni F, Gattazzo D (1998). Hoffa's disease of the adipose pad: magnetic resonance versus surgical findings. La Radiologia Medica, 95: 278–85.

Mourad K, King J, Guggiana P (1988). Computed tomography and ultrasound imaging of jumper's knee-patellar tendinitis. Clinical Radiology, 39: 162–165.

Muneta T, Yamamoto H, Ishibashi T, Asahina S, Furuya K (1994). Computerized tomographic analysis of tibial tubercle position in the painful female patellofemoral joint. American Journal of Sports Medicine, 22: 67–71.

National Health and Medical Research Council (1999a). A Guide to the Development, Implementation and Evaluation of Clinical Practice Guidelines. AGPS: Canberra.

National Health and Medical Research Council (1999b). Acute Pain Management: Scientific Evidence. Commonwealth of Australia: Canberra.

Neptune RR, Wright IC, Van den Bogert AJ (2000). The influence of orthotic devices and vastus medialis strength and timing on patellofemoral loads during running. Clinical Biomechanics, 15: 611–618.

Nichol G, Stiell IG, Wells GA, Juergensen LS, Laupacis A (1999). An economic analysis of the Ottawa knee rule. Annals of Emergency Medicine, 34: 438–447.

Nicholas JA (1970). Injuries to knee ligaments. Relationship to looseness and tightness in football players. Journal of the American Medical Association, 212: 2236–2239.

Nimon G, Murray D, Sandow M, Goodfellow J (1998). Natural history of anterior knee pain: a 14 to 20 year follow-up of non-operative management. Journal of Pediatric Orthopedics, 18: 118–122.

Nottage WM, Sprague NF, Auerbach BJ, Shahriaree H (1983). The medial patellar plica syndrome. American Journal of Sports Medicine, 11: 211–214.

Noyes FR, Bassett RW, Grood ES, Butler DL (1980). Arthroscopy in acute traumatic hemarthrosis of the knee. Incidence of anterior cruciate tears and other injuries. Journal of Bone and Joint Surgery, 62A: 687–695.

O'Brien SJ, Ngeow J, Gibney MA, Warren RF, Fealy S (1995). Reflex sympathetic dystrophy of the knee. Causes, diagnosis, and treatment. American Journal of Sports Medicine, 23: 655–659.

O'Donohue DH (1980). Treatment of acute dislocations of the patella. In: Funk FJ Jr. (ed). Symposium on the athlete's knee. CV Mosby Co: St Louis, MO.

Osgood RB (1903). Lesions of the tibial tubercle occurring during adolescence. The Boston Medical and Surgical Journal, 114–117.

O'Shea K, Kenny P, Donovan J, Condon F, McElwain JP (2002). Outcomes following quadriceps tendon ruptures. Injury, 33: 257–260

O'Shea KJ, Murphy KP, Heekin RD, Herzwurm PJ (1996). The diagnostic accuracy of history, physical examination, and radiographs in the evaluation of traumatic knee disorders. American Journal of Sports Medicine, 24: 164–167.

Outerbridge RE (1961). The etiology of chondromalacia patellae. Journal of Bone and Joint Surgery, 43B: 752–757.

Outerbridge RE (1964). Further studies on the etiology of chondromalacia patellae. Journal of Bone and Joint Surgery, 46B: 179–190.

Owre A (1936). Chondromalacia of the patella. Acta Chirurgica Scandinavica, 77 (suppl).

Pagliano JW, Jackson DW (1987). A clinical study of 3,000 long distance runners. Annals of Sports Medicine, 3: 88–91.

Palestro CJ, Kipper SL, Weiland FL, Love C, Tomas MB (2002). Osteomyelitis: diagnosis with (99m) Tc-labeled antigranulocyte antibodies compared with diagnosis with (111) In-labeled leukocytes — initial experience. Radiology, 223: 758–764.

Palumbo RC, Matthews LS, Reuben JM (1994). Localized pigmented villonodular synovitis of the patellar fat pad: a report of two cases. Arthroscopy, 10: 400–403.

Perin B, Nardacchione R, Bonaga S, Angelini F, Girotto A, Giovagnoni A, Valeri G (1997). Acute knee block. Assessment with magnetic resonance, correlated with arthroscopy. La Radiologia Medica, 93: 40–44.

Petersen CM, Hayes KW (2000). Construct validity of Cyriax's selective tension examination: association of end-feels with pain at the knee and shoulder including commentary by Flynn TW and Atkins E with author response. The Journal of Orthopaedic and Sports Physical Therapy, 30: 512–527.

Petit P, Sapin C, Henry G, Dahan M, Panuel M, Bourliere-Najean B, Chaumoitre K, Devred P (2001). Rate of abnormal osteoarticular

radiographic findings in pediatric patients. American Journal of Roentgenology, 176: 987–990.

Pollock DC, Ammeen DJ, Engh GA. (2002). Synovial entrapment: a complication of posterior stabilized total knee arthroplasty. Journal of Bone and Joint Surgery, 84A: 2174–2178.

Powers CM (1998). Literature review. Rehabilitation of patellofemoral joint disorders: a critical review. Journal of Orthopaedic and Sports Physical Therapy, 28: 345–354.

Poyhia T, Azouz EM (2000) MR imaging evaluation of subacute and chronic bone abscesses in children. Journal of Paediatric Orthopaedics, 30: 763–768.

Reid GD, Glasgow M, Gordon DA, Wright TA (1980). Pathological plicae of the knee mistaken for arthritis. Journal of Rheumatology, 7: 573–576.

Reider B, Marshall JL, Koslin B, Ring B, Girgis FG (1981a). The anterior aspect of the knee joint. Journal of Bone and Joint Surgery, 63A: 351–356.

Reider B, Marshall JL, Warren RF (1981b). Clinical characteristics of patellar disorders in young athletes. American Journal of Sports Medicine, 9: 270–274.

Richardson ML, Selby B, Montana MA, Mack LA (1988). Ultrasonography of the knee. Radiologic Clinics of North America, 26: 63–75.

Richman PB, McCuskey CF, Nashed A, Fuchs S, Petrik R, Imperato M, Hollander JE (1997). Performance of two clinical decision rules for knee radiography. Journal of Emergency Medicine, 15: 459–463.

Rillman P, Oswald A, Holzach P (2000). Fulkerson's modified Elmslie-Trillat procedure for objective patellar instability and patellofemoral pain syndrome. Swiss Surgery, 6: 328–334.

Robinson AR, Darraccott J (1970). Chondromalacia patellae. Annals of Physical Medicine, 10: 290.

Rogvi-Hansen B, Ellitsgaard N, Funch M et al. (1991). Low level laser treatment of chondromalacia patellae. International Orthopaedics, 15: 359–361.

Roush MB, Sevier TL, Wilson JK, Jenkinson DM, Helfst RH, Gehlsen GM, Basey AL (2000). Anterior knee pain: a clinical comparison of rehabilitation. Clinical Journal of Sport Medicine, 10: 22–30.

Russe O, Gerhardt JJ, King PS (1976). An Atlas of Examination, Standard Measurements and Diagnosis in Orthopaedics and Traumatology (2nd Edition). Hans Huber: Berne.

Salsich GB, Brechter JH, Farwell D, Powers CM (2002). The effects of patellar taping on knee kinetics, kinematics, and vastus lateralis muscle activity during stair ambulation in individuals with patellofemoral pain. Journal of Orthopaedic and Sports Physical Therapy, 32: 3–10.

Sanchis-Alfonso V, Rosello-Sastre E (2000). Immunohistochemical analysis for neural markers of the lateral retinaculum in patients with isolated symptomatic patellofemoral malalignment. A neuroanatomic basis for anterior knee pain in the active young patient. American Journal of Sports Medicine, 28: 725–731.

Sanchis-Alfonso V, Rosello-Sastre E, Monteagudo-Castro C, Esquerdo J (1998). Quantitative analysis of nerve changes in the lateral retinaculum in patients with isolated symptomatic patellofemoral malalignment. A preliminary study. American Journal of Sports Medicine, 26: 703–709.

Sanchis-Alfonso V, Rosello-Sastre E, Revert F (2001). Neural growth factor expression in the lateral retinaculum in painful patello-femoral malalignment. Acta Orthopaedica Scandinavica, 72: 146–149.

Sandow MJ, Goodfellow JW (1985). The natural history of anterior knee pain in adolescents. Journal of Bone and Joint Surgery, 67B: 36–38.

Scavone JG, Latshaw RF, Weidner WA (1981). AP and lateral radiographs: an adequate lumbar spine examination. American Journal of Roentgenology, 136: 715–717.

Schwartzman RJ, McLellan TL (1987). Reflex sympathetic dystrophy. A review. Archives of Neurology, 44: 555–561.

Schwellnus MP, Jordaan G, Noakes TD (1990). Prevention of common overuse injuries by the use of shock absorbing insoles. A prospective study. American Journal of Sports Medicine, 18: 636–641.

Seaberg DC, Jackson R (1994). Clinical decision rule for knee radiographs. American Journal of Emergency Medicine, 12: 541–543.

Seaberg DC, Yealy DM, Lukens T, Auble T, Mathias S (1998). Multicenter comparison of two clinical decision rules for the use of radiography in acute, high-risk knee injuries. Annals of Emergency Medicine, 32: 8–13.

Seale KS (1989). Reflex sympathetic dystrophy of the lower extremity. Clinical Orthopaedics and Related Research, 243: 80–85.

Selfe J, Harper L, Pedersen I, Breen-Turner J, Waring J (2001). Four outcome measures for patellofemoral joint problems: Part 1. Development and validity. Physiotherapy, 87: 507–515.

Selfe J, Harper L, Pedersen I, Breen-Turner J, Waring J (2001). Four outcome measures for patellofemoral joint problems: Part 2. Reliability and clinical sensitivity. Physiotherapy, 87: 516–522.

Senegor M (1991). Iatrogenic saphenous neuralgia: successful therapy with neuroma resection. Neurosurgery, 28: 295–298.

Shah MK (2002a). Simultaneous bilateral rupture of quadriceps tendons: analysis of risk factors and associations. Southern Medical Journal, 95: 860–866.

Shah MK (2002b). Simultaneous bilateral quadriceps tendon rupture in renal patients. Clinical Nephrology, 58: 118–121.

Shah M, Jooma N (2002). Simultaneous bilateral quadriceps tendon rupture while playing basketball. British Journal of Sports Medicine, 36: 152–153.

Shakespeare DT, Rigby HS (1983). The bucket-handle tear of the meniscus. A clinical and arthrographic study. Journal of Bone and Joint Surgery, 65B: 383–387.

Sharrad WJ (1964). Pressure effects on the knee in kneeling miners. British Journal of Industrial Medicine, 21: 309–324.

Shino K, Nakagawa S, Inoue M, Horibe S, Yoneda M (1993a). Deterioration of patellofemoral articular surfaces after anterior cruciate ligament reconstruction. American Journal of Sports Medicine, 21: 206–211.

Shino K, Nakata K, Horibe S, Inoue M, Nakagawa S (1993b). Quantitative evaluation after arthroscopic anterior cruciate ligament reconstruction. Allograft versus autograft. American Journal of Sports Medicine, 21: 609–616.

Shwayhat AF, Linenger JM, Hofherr LK, Slymen DJ, Johnson CW. (1994). Profiles of exercise history and overuse injuries among United States Navy Sea, Air, and Land (SEAL) recruits. American Journal of Sports Medicine, 22: 835–840.

Siebert CH, Kaufmann A, Niedhart C, Heller KD (1999). The quadriceps tendon cyst: an uncommon cause of chronic anterior knee pain. Knee Surgery, Sports Traumatology, Arthroscopy, 7(6): 349–351.

Sinding-Larsen MF (1921). A hitherto unknown affectation of the patella in children. Acta Radiologica, 1: 171–173.

Stanford W, Phelan J, Kathol MH et al. (1988). Patellofemoral joint motion: evaluation by ultrafast computed tomography. Skeletal Radiology, 17: 487–492.

Stiell IG, Greenberg GH, Wells GA, McDowell I, Cwinn AA, Smith NA, Cacciotti TF, Sivilotti ML (1996). Prospective validation of a decision rule for the use of radiography in acute knee injuries. Journal of the American Medical Association, 275: 611–615.

Stiell IG, Wells GA, Hoag RH, Sivilotti ML, Cacciotti TF, Verbeek PR, Greenway KT, McDowell I, Cwinn AA, Greenberg GH, Nichol G, Michael JA (1997). Implementation of the Ottawa Knee Rule for the use of radiography in acute knee injuries. Journal of the American Medical Association, 278: 2075–2079.

Stiell IG, Wells GA, McDowell I, Greenberg GH, McKnight RD, Cwinn AA, Quinn JV, Yeats A (1995). Use of radiography in acute knee injuries: need for clinical decision rules. Academic Emergency Medicine, 2: 966–973.

Stiene HA, Brosky T, Reinking MF et al. (1996). A comparison of closed kinetic chain and isokinetic joint isolation in patients with patellofemoral dysfunction. Journal of Orthopaedic and Sports Physical Therapy, 24: 136–141.

Suter E, McMorland G, Herzog W, Bray R (1999). Decrease in quadriceps inhibition after sacroiliac joint manipulation in patients with anterior knee pain. Journal of Manipulative and Physiological Therapeutics, 22: 149–153.

Suter E, McMorland G, Herzog W, Bray R (2000). Conservative lower back treatment reduces inhibition in knee-extensor muscles: a randomized controlled trial. Journal of Manipulative and Physiological Therapeutics, 23: 76–80.

Swanson AJ (1983). The incidence of prepatellar neuropathy following medial meniscectomy. Clinical Orthopaedics and Related Research, 181: 151–153.

Szucs PA, Richman PB, Mandell M (2001). Triage nurse application of the Ottawa Knee Rule. Academic Emergency Medicine, 8: 112–116.

Tanaka S, Lee ST, Halperin WE, Thun M, Smith AB (1989). Reducing knee morbidity among carpetlayers. American Journal of Public Health, 79: 334–335.

Tang SFT, Chen-C K, Hsu R, Chou-S W, Hong-W H, Lew HL (2001). Vastus medialis obliquus and vastus lateralis activity in open and closed kinetic chain exercises in patients with patello-femoral pain syndrome: an electromyographic study. Archives of Physical Medicine and Rehabilitation, 82: 1441–1445.

Thomee R (1997). A comprehensive treatment approach for patellofemoral pain syndrome in young women. Physical Therapy, 77: 1690–1703.

Thomee R, Grimby G, Wright BD, Linacre JM (1995a). Rasch analysis of Visual Analog Scale measurements before and after treatment of Patellofemoral Pain Syndrome in women. Scandinavian Journal of Sports Medicine, 27: 145–151.

Thomee R, Renstrom P, Karlsson J, Grimby G (1995b) Patellofemoral pain syndrome in young women. I. A clinical analysis of alignment, pain parameters, common symptoms and functional activity level. Scandinavian Journal of Sports Medicine 5: 237–244.

Thompson RC, Vener MJ, Griffith HJ et al. (1993). Scanning electron microscope and magnetic microscope and magnetic resonance imaging studies of injuries to the patellofemoral joint after transaxial loading. Journal of Bone and Joint Surgery, 75A: 704–713.

Thun M, Tanaka S, Smith AB, Halperin WE, Lee ST, Luggen ME, Hess EV (1987). Morbidity from repetitive knee trauma in carpet and floor layers. British Journal of Industrial Medicine, 44: 611–620.

Timm KE (1998). Randomised controlled trial of Protonics on patellar pain, position and function. Medicine and Science in Sports and Exercise, 30: 665–670.

Tobin S, Robinson G (2000). The effect of McConnell's vastus lateralis inhibition taping technique on vastus lateralis and vastus medialis obliquus activity. Physiotherapy, 86: 173–183.

Unkila-Kallio L, Kallio MJ, Petola H (1993) Acute haematogenous osteomyelitis in children in Finland. Finnish Study Group. Annals of Medicine, 25: 545–549.

van der Windt DAWM, van der Heijden GJ, van den Berg SGM, ter Riet G, deWinter AF, Bouter LM (1999). Ultrasound for musculoskeletal disorders: a systematic review. Pain, 81: 257–271.

van Holsbeeck M, Introcaso JH (1992). Musculoskeletal ultrasonography. Radiologic Clinics of North America, 30: 907–925.

van Tulder MW, Scholten RJPM, Koes BW, Deyo RA (2002). Non-steroidal anti-inflammatory drugs for low back pain. In: The Cochrane Library; Issue 2. Oxford: Update Software.

Virolainen P, Lahteenmaki H, Hiltunen A, Sipola E, Meurman O, Nelimarkka O (2002). The reliability of diagnosis of infection during revision arthroplasties. Scandinavian Journal of Surgery, 91: 178–81.

Wasson JH, Sox HC (1996). Clinical prediction rules. Have they come of age? Journal of the American Medical Association, 275: 641–642.

Watkins JT, Hunt TA, Fernandez RH, Edmonds OP (1958). A clinical study of beat knee. British Journal of Industrial Medicine, 15: 105–109.

Watson CJ, Leddy HM, Dynjan TD, Parham JL (2001). Reliability of the lateral pull test and tilt test to assess patellar alignment in subjects with symptomatic knees: student raters. Journal of Orthopaedic and Sports Physical Therapy, 31: 368–374.

Wigder HN, Cohan Ballis SF, Lazar L, Urgo R, Dunn BH (1999). Successful implementation of a guideline by peer comparisons, education, and positive physician feedback. Journal of Emergency Medicine, 17: 807–810.

Winslow J, Yoder E (1995). Patellofemoral pain in female ballet dancers: correlation with iliotibial band tightness and tibial external rotation. Journal of Orthopaedic and Sports Physical Therapy, 22: 18–21.

Witonski D (2002). Dynamic magnetic resonance imaging, Clinics in Sports Medicine 21: 403–415.

Witvrouw E, Lysens R, Bellemans J, Cambier D, Vanderstraeten G (2000). Intrinsic risk factors for the development of anterior knee pain in an athletic population: a two-year prospective study. Amercian Journal of Sports Medicine, 28: 480–489.

Wolfe F, Lane NE (2002). The long-term outcome of osteoarthritis: rates and predictors of joint space narrowing in symptomatic patients with knee osteoarthritis. Journal of Rheumatology, 29: 139–146.

Woods GW, Chapman DR (1984). Repairable posterior meniscocapsular disruption in anterior cruciate ligament injuries. American Journal of Sports Medicine, 12: 381–385.

Worth RM, Kettelkamp DB, Defalque RJ, Duane KU (1984). Saphenous nerve entrapment. A cause of medial knee pain. American Journal of Sports Medicine, 12: 80–81.

Yasuda K, Majima T (1988). Intra-articular ganglion blocking extension of the knee: brief report. Journal of Bone and Joint Surgery 70B: 837.

Yeung EW, Yeung SS (2001). A systematic review of interventions to prevent lower limb soft tissue running injuries. British Journal of Sports Medicine, 35: 383–389.

Chapter 9

Process Report

➔ *This document provides a review of the scientific evidence on the diagnosis, prognosis and management of acute musculoskeletal pain. The planning and conduct of the evidence review (i.e systematically locating, appraising and summarising the evidence) was based on the National Health and Medical Research Council of Australia (NHMRC) toolkit series for guideline development (1999, 2000a,b,c).*

>Overview

The material in this document builds on the work of members of the Australasian Faculty of Musculoskeletal Medicine (AFMM). In 1998, the AFMM drafted a series of evidence-based guidelines for the National Musculoskeletal Medicine Initiative (NMMI) for use by general practitioners specialised in musculoskeletal medicine. A uni-disciplinary approach to the literature search and the selection and interpretation of studies was employed. The authors of the original draft guidelines were involved in this project to update their work.

Planning was undertaken to integrate new material and new requirements for the development of evidence-based guidelines with the existing work. Consideration was also given to developing an end product for use by multiple health care disciplines. Measures were taken to ensure that the process of updating and enhancing the original work was in line with current standards for guideline development. Every attempt has been made to make this effort transparent.

Multi-Disciplinary Involvement

Five multi-disciplinary review groups were formed to systematically identify, appraise and interpret the literature on the diagnosis, prognosis and treatment of acute musculoskeletal pain.

The involvement of multiple disciplines in the project enabled the groups to develop a document free from the bias of a particular profession. The aim was to promote consistency in the approach to patient care, based on evidence, by all disciplines involved in the management of acute musculoskeletal pain.

Target Audiences

This document provides the evidence base for summary publications that have been developed for the following groups:

- Clinicians including general practitioners, physiotherapists, chiropractors, osteopaths and specialists who see, on referral, people with acute musculoskeletal pain, including rheumatologists, orthopaedic surgeons, pain specialists, rehabilitation specialists, sports medicine specialists
- Health consumers and patients

It is acknowledged that there are other clinician groups involved in the care of people with acute musculoskeletal pain. For practical purposes, this document targets clinicians who invoice for services. There was no distinction made with respect to professional discipline in the literature search, appraisal and development of guideline statements; thus the information is relevant to all clinicians.

>Evidence Review Process

A review protocol was developed based on the National Health and Medical Research Council (NHMRC) Toolkit series (1999, 2000a,b,c), the Cochrane Reviewer's Handbook (2001), and the NHS Centre for Reviews and Dissemination guidelines (2001).

Five multi-disciplinary groups applied the protocol to review and analyse the scientific literature to update the content of existing guidelines on the management of acute low back, thoracic spine, neck, shoulder and anterior knee pain.

The process consisted of:

- An evaluation of existing guidelines in the five topic areas.
- A systematic search for new evidence to update existing material.
- Critical appraisal of new studies that met selection criteria.
- Data analysis (description of the results of new studies and formulation of key messages to highlight the main points).
- Development of a management plan for acute musculoskeletal pain.
- Public consultation and independent review.

Evaluation of Existing Guidelines

Guidelines on knee, shoulder, low back and neck pain developed by other groups were obtained to determine whether they could be readily adapted for use. However, they did not specifically address acute pain, were comprised of a mix of consensus and evidence based statements or required updating. There were no existing guidelines for the management of acute thoracic spinal pain.

The decision was made to update guidelines developed by members of the Australasian Faculty of Musculoskeletal Medicine (AFMM) for the National Musculoskeletal Medicine Initiative (NMMI). The authors (Professor Nikolai Bogduk, Dr Wade King, Dr David Vivian and Dr Michael Yelland) participated together with other review group members in this project.

The existing guidelines were initially developed and periodically updated by the original authors using a process of conventional literature review. The most recent work was circulated to the review groups. Group members had the opportunity to evaluate the literature forming the basis of the existing guidelines, review its interpretation, nominate additional articles to undergo the appraisal process or request that an article be re-appraised.

In addition, an evaluation of each of the guidelines was undertaken by the groups using the AGREE instrument

(2001) for evaluating clinical practice guidelines. Areas identified for improvement included the use of a systematic process to update the work and the need to document the guideline development process.

Search for New Evidence

This update encompasses the findings of new and old literature searches. Where details of the previous literature searches were available, these have been provided. The specific strategy, including selection criteria, databases searched, dates and search terms is detailed at the beginning of each chapter.

The reference sections contain references to studies cited in the existing guidelines together with the references added during this update.

Study Types

A search for systematic reviews and recent primary research was undertaken to find evidence on the diagnosis, prognosis and treatment of acute low back, thoracic spine, neck, shoulder and anterior knee pain. Evidence was sought for different study types according to the three study questions (i.e. diagnosis, prognosis, interventions) explored.

Where systematic reviews were available, the primary studies were checked to determine whether they met the inclusion criteria established for this review. Details of individual studies in systematic reviews are not recorded in the tables of included and excluded studies.

The criteria for selecting the study types for each question was based on the NHMRC Toolkit (2000b), adapted below in Table 9.1 to include levels of evidence (see Table 9.2). The type of study chosen is detailed in the study selection criteria section in each of the five topics.

Levels of Evidence: Definitions

Levels of evidence I–IV in Table 9.2 were developed by the NHMRC to describe studies of interventions. For the purposes of these guidelines, this system was also applied to studies in other domains (i.e. aetiology, risk factors, prognosis). In such cases, the level of evidence applied to the cited studies is indicative only and may not be appropriate or accurate. Under other evidence rating systems, higher levels of evidence may apply.

Limitations of the Search Strategy

Limitations include:

- Search terms may not have identified all relevant studies.
- Difficulty in obtaining articles (not all articles requested were accessible).
- Inability to access the chiropractic database MANTIS due to licensing requirements.
- Inability to access PEDro during the search period.

Table 9.1

Ideal Study Types for Clinical Questions

Question	Study Type	Level of Evidence
Intervention	Systematic review	I
	Randomised controlled trial	II
	Cohort study	III-2
	Case-control study	III-2
Diagnostic test/performance	Systematic review	*I
	Cross-sectional study	*III-3
	Case series	*IV
Prediction and prognosis	Systematic review	*I
	Cohort/survival study	*III-2

Note: * These levels of evidence have been developed primarily for intervention studies. Adapted from National Health and Medical Research Council (2000). How to Review the Evidence: Systematic Identification and Review of the Scientific Literature. Canberra: NHMRC.

Table 9.2

Levels of Evidence

Level of Evidence	Study Design
I	Evidence obtained from a systematic review of all relevant randomised controlled trials.
II	Evidence obtained from one or more properly designed randomised controlled trials.
III-1	Evidence obtained from well-designed pseudo randomised controlled trials (alternate allocation or some other method).
III-2	Evidence obtained from comparative studies (including systematic reviews of such studies) with concurrent controls and allocation not randomised (cohort studies), case-control studies, or interrupted time-series with a control group.
III-3	Evidence obtained from comparative studies with historical control, two or more single arm studies, or interrupted time series without parallel control group.
IV	Evidence obtained from case series, either post-test or pre-test/post-test.
Consensus	In the absence of scientific evidence and where the executive committee, steering committee and review groups are in agreement, the term 'consensus' has been applied.

Note: Adapted from National Health and Medical Research Council (1999). A Guide to the Development, Implementation and Evaluation of Clinical Practice Guidelines. Canberra: NHMRC.

- Reliance on the process undertaken by Clinical Evidence (2002) to derive conclusions on interventions for acute low back and acute neck pain.

No attempt was made to translate articles in foreign languages, to hand search journals or to seek unpublished studies and conference proceedings.

There was some variation in the time parameters of the searches conducted for the five topic areas. This was the result of a number of factors including the results of the evaluations of the existing guidelines by review group members, the search strategy used for the existing guidelines, and the date the existing work was last updated.

The risk of failing to include important studies was offset by the multi-disciplinary nature of the process and the opportunity for group members to note the absence of seminal articles. In such cases, the articles were retrieved and critically appraised.

Critical Appraisal Process

The five review groups developed study selection criteria and viewed the search results (title and abstract) in relation to the criteria. The full text of articles appearing to meet the criteria was retrieved for critical appraisal. Specific information on the search strategy and study selection criteria is included in the five topics.

A process for critical appraisal was distributed to all review groups. Standard data collection forms were developed incorporating the selection criteria and the relevant quality checklists for primary research on interventions, diagnosis and prognosis (NHMRC 2000b). A data collection form was designed to evaluate the quality of systematic reviews (based on Bury and Mead 1998). Two people independently appraised the articles and their results were compared. In cases where there was disagreement between reviewers, a third reviewer appraised the article.

No attempt was made to re-appraise studies cited in the existing guidelines, however where the interpretation was questioned, the article was obtained and subjected to the critical appraisal process.

Tables of Included and Excluded Studies

The results from the data collection forms were entered onto a database. Critically appraised studies were included if they met all of the inclusion criteria, none of the exclusion criteria and the study was relevant to the development of guidelines for clinical practice. Studies that were excluded (i.e. did not meet all inclusion criteria) appear in the Tables of Excluded Studies with a brief explanation of the reason for exclusion. Refer to Appendix E: Tables of Included and Excluded Studies.

Studies that were obtained and reviewed prior to this update (i.e. to formulate the existing draft guidelines) and studies cited in Clinical Evidence (2002) are not included in the Tables of Included and Excluded Studies.

Data Analysis and Key Messages

A summary of the results of the critical appraisals (entered into the Tables of Included Studies) was used to update the text of the existing guidelines, using quantitative terms where possible.

Major points were drawn from the text to formulate key messages. Due to the paucity of evidence specifically on acute musculoskeletal pain, many of the key messages are consensus views rather than evidence-based.

Development of a Management Plan for Acute Musculoskeletal Pain

The management plan was based on the key messages derived from the evidence review on the diagnosis, prognosis and treatment of acute musculoskeletal pain. The sections on diagnosis, prognosis and interventions are summarised below. Further detail on the study selection criteria is provided in the introductory sections of the low back, thoracic spine, neck, shoulder and anterior knee pain guidelines.

Diagnosis

The term diagnosis expresses what is known about the presenting condition after clinical assessment (comprising a history and physical examination). This section contains information from the existing guidelines supplemented with evidence from recent studies. Systematic reviews, cross-sectional studies, case studies and case series were located using the search strategy outlined in the Introduction.

Aetiology and Prevalence

Attempting to identify the underlying cause of pain by progressively ruling out possible causes may be useful for chronic conditions. However in the case of acute musculoskeletal pain, the evidence suggests that this approach is likely to be confounded by the unreliability of clinical methods and the variation in the understanding and description of clinical entities.

The purpose of this section is to inform the reader of the rare or serious conditions that may be associated with acute musculoskeletal pain and to highlight the low prevalence of such conditions. The management of specific conditions is beyond the scope of these guidelines.

History

Eliciting a history provides clinicians with information on the subjective aspects of a condition. Information should be sought with the clear aim of providing a better outcome than can be expected from the natural history of the condition. While the history carries little diagnostic weight, it is an important component of clinical assessment as it aids the identification of potentially serious conditions.

This section outlines how to assess musculoskeletal pain when eliciting a history. Chapter 2 (Acute Pain Management) provides further detail on conducting a pain assessment.

Examination

The primary purpose of physical examination is to elicit objective information on the physical features of a presenting condition. Information can be obtained through inspection, palpation and movement. It is important to be aware of the limitations of physical assessment as specific clinical tests often lack reliability and validity and thus lack diagnostic utility. There is a need for a thorough examination of the musculoskeletal system in the presence of pain and other musculoskeletal symptoms. In addition, there is a need to assess for psychosocial and occupational factors that may influence recovery.

Ancillary Investigations

Investigations are indicated when the history and physical examination reveal alerting features ('red flags') of potentially serious causes of pain. In the absence of such features, the use of investigations for acute musculoskeletal pain often lacks utility. However, when alerting features of serious conditions are present, ancillary investigations should be considered.

A table of investigations based on alerting features is included in Appendix C: Ancillary Investigations.

In assessing the diagnostic utility of investigations, aspects of safety, reliability, validity, clinical significance and cost require consideration. The safety and cost-effectiveness of any test are relative to improvements in treatment and outcome that are likely to result. Those benefits in turn depend on diagnostic accuracy, which is a product of reliability and validity. In each case, evidence of reliability and validity is crucial to any decision to undertake investigation.

These aspects are presented below in relation to imaging:

- Safety — Issues include the exposure to ionising radiation. An imaging test is not justified unless it is likely to yield information that will improve management and the risks are outweighed by the potential benefit.
- Reliability — Issues related to the extent to which the results of an investigation are reproducible. The limitations of the equipment used, the skill of the operator in selecting views and apparatus settings, and the interpretation of the images all have an impact on the findings.
- Validity — Refers to the extent to which images show what they are intended to show. Reliability is a component of validity so if a test is not reliable its results cannot be valid. Other factors contributing to the validity of an imaging test are the sensitivity and specificity for showing particular changes and the clinical significance of any changes shown. Sensitivity and specificity data have to be assessed for external validity; they are only applicable to a particular index condition if they were generated from a well-designed study of a representative population with similar complaints.
- Clinical significance — Imaging cannot demonstrate pain but may (or may not) demonstrate changes possibly associated with pain. The interpretation of the image is based on the judgment of a radiologist and a clinician.
- Cost — Investigations should be effective in terms of cost and outcome. There is little justification for investigations if the results are unlikely to alter management. Cost information is included in Appendix B.

Terminology

In the absence of alerting features of serious conditions, terms to describe episodes of acute musculoskeletal pain are provided. These terms express what is known about the presenting condition after clinical assessment (history and physical examination).

Prognosis

Prognosis is influenced by risk factors, the natural history of the condition and the treatment regime. The term natural history describes the usual course of a condition if no treatment is undertaken.

Knowledge of the factors influencing prognosis provides a rational basis both for understanding the condition and its likely effects and for decisions about appropriate interventions at any given stage of the condition.

In general, the prognosis of acute musculoskeletal pain is favourable. Barriers to recovery include internal and external risk factors. These differ for different conditions and should be identified early so that measures can be implemented to improve the prognosis.

The section is comprised of information from the existing guidelines updated with evidence from recent studies. Cohort studies and systematic reviews were located using the search strategy outlined in each topic.

Interventions

Systematic reviews and randomised controlled trials (i.e. Level I and II evidence) were sought to determine the efficacy of interventions for acute musculoskeletal pain. The search strategy is outlined in each topic area.

While there was a paucity of evidence, it is important to note that this does not necessarily mean that a particular intervention is not efficacious or beneficial. There are limits to scientific investigation and in addition, evidence for interventions may exist in study types excluded from this evidence review.

Because effect sizes were not always available, criteria were developed to categorise the findings. Each intervention is categorised (refer Table 9.3) and the level of evidence (refer Table 9.2) provided. Interventions are arranged alphabetically within each category.

In the case of acute low back and neck pain, the Clinical Evidence text (2002) was used as the basis for updating the evidence on interventions. The titles and abstracts of studies cited in Clinical Evidence were checked to determine whether they met the inclusion criteria for this review. Studies that met the criteria were considered in the analysis, however their results are not recorded in the Tables of Included Studies. Primary studies and systematic reviews published after the search date in the Clinical Evidence text were located and appraised, with the results appearing in the Tables of Included and Excluded Studies (Appendix E).

In cases where there were no studies of populations meeting the definition of acute pain, studies involving mixed acute and chronic populations were included in the analysis.

Table 9.3

Criteria for Categorising Interventions

Category	Criteria
Evidence of Benefit	Interventions for which there is evidence of a clinically significant beneficial effect compared to placebo, natural history or to other interventions that have demonstrated a beneficial effect vs. placebo or natural history.
Conflicting Evidence	Interventions for which there have been a number of similar controlled trials that have achieved conflicting results.
Insufficient Evidence	Interventions for which there have been no controlled trials or those for which an effect has been demonstrated in a general sense but not in all specific regions of musculoskeletal pain (e.g. NSAIDs) or those interventions that have not been tested against placebo.
Evidence of No Benefit	Interventions that have demonstrated no effect vs. placebo or natural history and have confidence intervals that exclude a clinically important benefit.

A literature search for harm associated with interventions was not specifically conducted although harms were sometimes documented in the studies appraised. Clinical Evidence (2002) provided information on harm associated with interventions for acute low back pain and these are noted in the key messages.

>Economic Implications

The search for studies on the cost of interventions was limited to the Cochrane Library database. A number of articles were located comparing the cost of interventions for low back pain and these were critically appraised.

In addition, a list of the costs of services and treatments described in the document has been appended (Refer Appendix B: Table of Unit Costs) as a guide.

>Consultation Process

The draft guidelines were circulated to members of the review groups, steering committee and executive committee for approval.

The draft document was made available for a period of public consultation, advertised in the Weekend Australian and via press releases to the general and medical media. In addition, specific groups identified by the steering committee for targeted consultation were approached independently to review the document. A web page was developed to provide electronic access and feedback submission. A list of those contributing feedback is provided in Appendix D.

>Health Consumers

The objective in reviewing the evidence on management of acute musculoskeletal pain is to improve the quality and consistency of information and care provided to consumers, with the goal of improving health outcomes. The project aims to promote partnership in decision-making between patients and clinicians by making the results of this evidence review widely available.

A representative of Consumers' Health Forum of Australia has been actively involved in this project as a member of the steering committee and a review group. A number of consumer groups and lay organisations were approached to contribute their comments on the draft guidelines.

>Dissemination and Implementation

The aim in producing evidence-based guidelines is to facilitate the integration of clinical expertise and the values and beliefs of consumers with the best available evidence. An effective strategy for dissemination and implementation is required to achieve this.

The transfer of research evidence into clinical practice is a slow process requiring integration of the following elements:

- Good information
- Good access to the information
- Supportive environments
- Evidence-based promotion of knowledge uptake using methods to promote knowledge uptake that have been proven in the literature (NHMRC 2000a).

The processes of guideline development, dissemination and implementation are closely linked. The involvement of multiple disciplines in this evidence review was an important step in linking these domains. A multi-faceted dissemination strategy including distribution of the information to consumers and multi-disciplinary clinician groups will enable uptake of the information by the target audiences.

Dissemination Strategies

- Electronic access to the evidence review.
- Publication and distribution of an evidence summary for clinicians. This will be available electronically.
- Publication and distribution of information sheets for patients. These will available electronically.
- Publication and distribution of a management plan for acute musculoskeletal pain. This will be available electronically.
- CD-ROM of the evidence review, evidence summary and other publications to be made available at meetings and other events.
- A marketing strategy will highlight the availability of the respective documents to consumer and clinician groups.

Implementation Strategies

Clinical practice guidelines will only be successful if the information is incorporated into practice decisions.

To this end, the literature on implementation strategies was reviewed. This comprised a search of the Cochrane database of reviews, use of the NHMRC toolkit series for guideline development (1999, 2000) and consideration of the results of the 'Final Report of a Consultancy to Develop an Implementation Strategy for Evidence-Based, Best Practice Clinical Practice Guidelines for General Practice in Australia' (RACGP 2000).

Evidence on the effectiveness of implementation strategies is limited and there is little data on their cost-effectiveness. It is also difficult to generalise the findings to different settings and groups. The use of active rather than passive modes of delivery appears to be a successful approach, however the cost is prohibitive.

The development of strategies that address barriers to implementation is another approach. Barriers include the physical form of the material, lack of awareness, personal characteristics of those in the target audience, structural constraints (organisational, economic), and consumer-related barriers (NHMRC 2000a).

Strategies to manage barriers:

- Multi-disciplinary approach to guideline development
- Involvement of a consumer representative
- Production of a range of physical formats for different target groups
- Broad dissemination, and a range of means to access the information
- Publication of the results in professional journals and the general media
- Endorsement by professional and lay associations
- Approval by the NHMRC

Revision Strategy

In the past, regularly scheduled review and revision dates for guidelines were proposed as a means to facilitate the continual updating of information. This approach, however, is potentially resource intensive.

Recently it has been suggested that the rate of progress in a particular field and the rapidity with which new information is

becoming available is a valid and sustainable approach to guideline revision. Shekelle et al. (2001) provide a set of principles based on changes in performance or evidence as a means for determining when guidelines should be reviewed and updated:

- When there are changes in the evidence on the harms and benefits of interventions.
- When changes in important outcomes become evident.
- When new interventions become available.
- When there is evidence that current practice is optimal.
- When changes in societal values occur.
- When changes occur in the availability of health care resources.

It is suggested that the decision to revise a guideline should be made by a multi-disciplinary committee.

One approach involves combining expert opinion and knowledge with a search of the literature:

- Send a questionnaire to the steering committee and review group members asking if there are new interventions, new outcomes, new data on harms and/or benefits, or no longer a need for an evidence review.
- Supplement this approach with a literature search.

Key Message

It is recommended that funding be made available to conduct a simple survey of members of the existing multi-disciplinary steering and review committees, annually, together with a literature search of the relevant electronic databases as a means to determine the need for revision of this document. (Consensus)

>Legal Implications

Every attempt has been made to locate the most recent evidence. Judgment is necessary when applying evidence in a clinical setting. It is important to note that weak evidence does not necessarily mean that a practice is unadvisable, but may reflect the insufficiency of evidence or the limitations of scientific investigation.

These guidelines are intended to act as a guide to practice. The ultimate decision of what to do rests with the practitioner and the consumer and depends on individual circumstances and beliefs (NHMRC 1999).

>References

AGREE Collaboration (2001). Appraisal of Guidelines for Research and Evaluation (AGREE) Instrument. http://www.agreecollaboration.org

Bury T, Mead J (1998). Evidence-based healthcare: a practical guide for therapists. Oxford: Butterworth-Heinemann.

Clinical Evidence (June 2002) 7: 1018–1031. BMJ: London.

Cochrane Collaboration. The Cochrane Reviewer' Handbook: Version 4.1. URL: www.cochrane.org.au

Royal Australian College of General Practitioners (2000). Final report of a consultancy to develop an implementation strategy for evidence-based best practice clinical practice guidelines for general practice in Australia. June 2000.

National Health and Medical Research Council (1999). A Guide to the Development, Implementation and Evaluation of Clinical Practice Guidelines. Canberra: NHMRC

National Health and Medical Research Council (2000a). How to Put the Evidence into Practice: Implementation and Dissemination Strategies. Canberra: NHMRC

National Health and Medical Research Council (2000b). How to Review the Evidence: Systematic Identification and Review of the Scientific Literature. Canberra: NHMRC.

National Health and Medical Research Council (2000c). How to Use the Evidence: Assessment and Application of Scientific Evidence. Canberra: NHMRC

NHS Centre for Reviews and Dissemination (2001). Undertaking Systematic Reviews of Research on Effectiveness. The University of York.

Shekelle P, Eccles MP, Grimshaw JM, Woolf SH. (2001). When should clinical practice guidelines be updated? British Medical Journal, 323: 155–157.

Appendix A

Glossary of Terms

➔ *This glossary contains definitions obtained from a range of sources.*

Acute Pain
'Acute' pain refers to the duration of pain rather than to its severity. Bonica (1953) defined 'acute' pain as pain that is likely to resolve spontaneously within a relatively short time. Merskey (1979) subsequently specified the time frame for acute pain as pain of less than three months duration.

Chronic Pain
The International Association for the Study of Pain (IASP) defines chronic pain as pain that has persisted for longer than three months (Merskey and Bogduk 1994).

Clinician
In this document the term 'clinician' refers to health care providers who receive a fee for service independently (i.e. general practitioners, physiotherapists, chiropractors, osteopaths, specialist medical consultants). This list is not exhaustive in relation to clinicians who participate in the care of people with musculoskeletal pain.

Cognitive Behavioural Therapy
A cognitive behavioural approach involves helping people achieve their desired goals through specifying the steps required and systematically reinforcing progress. It is critical that the client and therapist work in partnership with shared responsibilities. This approach is often incorporated with exercise and activity restoration interventions. More complex cases are likely to require cognitive behavioural therapy (CBT), which is a more sophisticated and specialised application of this approach.

Confidence Interval
The 95% confidence interval of a measure is the range across which values of that measure might fall in 95% of instances, if the observation were to be repeated by others.

Consumer
In this document the term 'consumer' is used in cases where a person is acting independently of a clinician. Where a person is receiving care from a clinician, the term 'patient' is used instead.

Effect Size
Effect-size is a measure of how much the outcome of one treatment is better than the outcome of another treatment. It is calculated as the difference between the mean outcome values expressed as a proportion of the standard deviation of the control group (or the pooled standard deviation of both groups). For example, a score of 2 indicates that the magnitude of the difference is on average two standard deviations. Cohen (1988) has suggested that an effect-size of 0.5 or more is 'large'; 0.3–0.5 is 'moderate'; 0.1–0.3 is 'small'; and less than 0.1 is negligible. See Cohen J (1988). Statistical power analysis for the behavioural sciences. (2nd ed). New Jersey: Lawrence Erlbaum.

Efficacy
The efficacy of a therapeutic intervention is its rate of successful outcomes when applied under ideal conditions. Efficacy is expressed as number-needed-to-treat (NNT).

Health Practitioner
In this document the term 'health practitioner' refers to health care providers who receive a fee for service independently (i.e. general practitioners, physiotherapists, chiropractors, osteopaths, specialist medical consultants). This list is not exhaustive.

Intervention
An intervention will generally be a therapeutic procedure such as treatment with a pharmaceutical agent, surgery, a dietary supplement, a dietary change or psychotherapy. Some other interventions are less obvious, such as early detection (screening), patient educational materials, or legislation. The key characteristic is that a person or their environment is manipulated in order to benefit that person (NHMRC 2000).

Kappa Score
See Reliability

Likelihood Ratio (LR)
The likelihood ratio is the extent to which a diagnostic test increases the likelihood of a condition being diagnosed beyond the prevalence of that condition. Algebraically, the likelihood ratio is defined as the sensitivity of the test divided by (1 – specificity). Multiplying the pre-test odds of a condition being present by the likelihood ratio of the test yields the post-test odds of the diagnosis being correct. For example, if the pre-test odds of a person having a condition are one in four and there is a positive test with a likelihood ratio of 2, the chance of the person having the condition are doubled to one in two. If the LR is one, a positive test result adds nothing to the diagnostic information. Likelihood ratios can be calculated as positive and negative. Positive likelihood ratios reflect the ability of a test to establish a particular diagnosis. Negative likelihood ratios reflect the ability of a test to exclude that diagnosis. Only positive LRs are quoted in these guidelines.

Manipulation (Spinal)
Manual therapy technique in which loads are applied to the spine using short- or long-lever methods. The spinal joint to which the technique is applied is moved to its end range of voluntary motion, followed by application of a single high-velocity, low amplitude thrust. Spinal manipulation is usually accompanied by an audible pop or click.

Manual Therapy
The application of physical techniques, which includes but is not limited to, massage, spinal manipulation and mobilisation.

Massage
A mechanical form of therapy in which the soft tissue structures of the low back are pressed and kneaded, using the hand or a mechanical device. Many different types of massage are performed, including but not limited to, acupressure, deep-tissue therapy, friction massage, Swedish massage, myofascial release, shiatsu, reflexology, craniosacral therapy, trigger and pressure point therapy.

Mobilisation
Mobilisation is the passive application of repetitive, rhythmical, low velocity movements of varying amplitudes applied within the joint range of motion. The technique includes methods of a singular or repetitive movement and/or stretching of the spinal joints.

Number Needed to Treat (NNT)
The number need to treat is the number of patients with a particular outcome who must be treated before one patient can be claimed to have achieved that outcome as a result of the effects of the intervention.

Odds Ratio (OR)
Ratio of odds of the outcome in the treatment group to the corresponding odds in the control group (NHMRC 2000)

***p*-value**
The probability (obtained from a statistical test) that the null hypothesis is incorrectly rejected.

Pain
Pain is 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).

Pain, Recurrent
Recurring episodes of pain may be labelled as 'recurrent pain' and classified as acute or chronic depending on the duration of the episode.

Patient
In this document the term 'consumer' is used in cases where a person is acting independently of a clinician. Where a person is receiving care from a clinician, the term 'patient' is used instead.

Randomised Controlled Trial
An experimental comparison study in which participants are allocate to treatment/intervention or control/placebo groups using a random mechanism to allocate them to either group. When there is equal chance of allocation to either the treatment or the control group, allocation bias is eliminates (NHMRC 2000).

'Red Flags'
The term 'red flags' refers to clinical (i.e. physical) features that may alert to the presence of serious but relatively uncommon conditions or diseases requiring urgent evaluation. Such conditions include tumours, infection, fractures and neurological damage. Screening for serious conditions occurs as part of the history and physical examination and should occur at the initial assessment and subsequent visits. Alerting features of serious conditions are covered in detail in the specific guideline topics.

Referred Pain
Referred pain is pain perceived in a region remote from its origin.

Reliability
Also called reproducibility, reliability involves the consistency or dependability of observations. It is an expression of the stability of the observation when tested over time under different conditions and by different investigators. It is usually assessed in terms of the Kappa (κ) score, which is sometimes qualified by its standard error or its 'base rate' (the prevalence of the index condition in the population studied).

Kappa Score	Agreement
0.8–1.0	Very Good
0.6–0.8	Good
0.4–0.6	Moderate
0.2–0.4	Fair
0.0–0.2	Poor

Note: From Cohen J (1960). A coefficient of agreement for nominal scales. Educational and Psychological Measurement, 20: 37–46. Reproduced with permission from??

Relative Risk (RR)
The ratio of proportions in the treatment and control groups with the outcome. It expresses the risk of the outcome in the treatment group relative to the risk of the outcome in the control group (NHMRC 2000).

Sensitivity
Is the ability of a test to detect a condition when it is known by other means to be present; it is expressed as a decimal or a percentage and sometimes qualified by a confidence interval. Sensitivity is inflated by false positive results.

Specificity
Is the ability of a test to establish correctly that a particular condition is absent; it too is expressed as a decimal or a percentage and sometimes qualified by a confidence interval. Specificity is inflated by false negative results.

Systematic Review
The process of systematically locating, appraising and synthesising evidence from scientific studies in order to obtain a reliable overview (NHMRC 2000).

Validity
The validity of measurement is an expression of the degree to which a measurement measures what it purports to measure; it includes construct and content validity (NHMRC 2000).

'Yellow Flags'
The term 'yellow flags' was introduced to identify psychosocial factors that may increase the risk of chronicity and that should be assessed at the initial and subsequent consultations, particularly when progress is slower than expected. The presence of psychosocial factors is a prompt for further detailed assessment and early intervention. The areas to evaluate include:

- attitudes and beliefs about pain
- behaviours
- compensation issues
- diagnostic and treatment issues
- emotions
- family
- work

Red flags and yellow flags are not mutually exclusive and intervention may be required for both clinical and psychosocial risk factors.

Appendix B

Table of Unit Costs

(November 2002)

➔ *This table has been provided as a guide to the relative costs of services and interventions discussed in these guidelines. The fees for services are indicative or relative only, and influenced by anti-competition policy on fees and costs. The table is not meant as an endorsement of any particular therapy for acute musculoskeletal pain and includes examples only. No formal cost-benefit analysis was performed.*

Unit Costs (November 2002)

	MBS ITEM NO.	FEE ($)	85% REBATE
Service*			
General practitioner initial subsequent	 36 (Level A, B, C) 23 (Level A, B, C)	 55.95 29.45	 47.60 25.05
Surgeon initial subsequent	 104 105	 69.35 34.80	 58.95 29.60
Consultant physician initial subsequent	 110 116	 122.35 61.25	 104.00 52.10
Physiotherapist initial subsequent		 Mean 50.00 Mean 40.00	
Chiropractor initial subsequent		 60.00–100.00 40.00	
Osteopath initial subsequent		 100.00 60.00–80.00	
Acupuncture	173	21.65	18.45
Interventions			
Massage therapy (1 hour)	(average from Cost Study)	49.00 (range 40–60)	
TENS Machine buy hire Replacement gel pads Battery (9v)		 120–220 60–68/month 16.00 5.60	

Unit Costs continued

	MBS ITEM NO.	FEE ($)	85% REBATE
Injection therapy			
injection into joint/synovial cavity	50124	24.00	20.40
injection under image intensifier into 1 or more zygapophyseal or costotransverse joints or 1 or more primary posterior rami of spinal nerves	39013	88.55	75.30
drug — Kenacort 40			
Depo-Medrol			
acal anaesthetic	From RNSH pharmacy	10.05/ea 3.10/ea 0.25/ea	
Course of NSAID therapy			
Celebrex 200mg 1 or 2/day for 2 weeks (1 pkt of 30)	PBS 8440F	32.09	
Voltaren 50mg 2 or 3/day for 2 weeks (1 pkt of 50)	PBS 1300K	13.65	
Pain killers???			
Panadol (500mg x 24)	RRP	4.25	
Panamax (500mg x 100)	PBS 1746X	7.72	
Panadeine (pkt 24)	RRP	7.25	
Panadeine Forte	PBS 1215Y	7.12	
Endone — tablet 5mg	PBS 2622B	10.38	
Ordine — 5mg/ml	PBS 2123R	18.67	
Investigations			
Xray			
plain film of thoracic spine	58103	51.95	44.20
lumbosacral region	58106	72.55	61.70
cervical spine	58100	63.30	53.85
spine (four regions)	58108	125.30	106.55
knee	57521	40.90	34.80
shoulder	57700	38.15	32.45
CT — no IV contrast medium			
thoracic spine	56221	228.00	193.80
lumbosacral region	56223	228.00	193.80
cervical spine	56220	228.00	193.80
two regions	56233	228.00	193.80
three regions	56237	228.00	193.80
knee/shoulder	56619	209.00	177.65
CT — with IV contrast medium			
thoracic spine	56225	333.80	283.75
lumbosacral region	56226	333.80	283.75
cervical spine	56224	333.80	283.75
two regions	56234	333.80	283.75
three regions	56238	333.80	283.75
knee/shoulder	56625	317.90	270.25
MRI (fee the same for all regions)		475.00	419.40
Arthrography	59751	131.15	111.50
Ultrasonography			
knee	55828	99.90	84.95
shoulder	55808	99.90	84.95

Note: * These refer to MBS fees only. Table prepared by Marita Cross, Project Officer, NHMRC Arthritis Cost Study, University of Sydney, Department of Rheumatology, Royal North Shore Hospital.

Physiotherapy Fees

No guidelines available from Australian Physiotherapy Association. The mean cost is $50 for an initial consultation and treatment, and $40 for follow up treatments.

Chiropractic Fees

From Chiropractic Association — as a guideline they recommend $60–100 for initial visit and $40 for subsequent visits.

Osteopath

From Osteopathic Association — guidelines — $100 initial visit, $60–80 for subsequent visits.

Massage

Amount in table above from average paid in 2001 and 2002 by 7 people in Arthritis Cost Study for 1-hour massage.

TENS

Prices from Gillespies Hire, Artarmon — to buy $220, to hire $68 for 4 weeks then $12/week. MediRent, Matraville — to buy (Proten, without timer) $120, to hire $60/month; gel pads $16, uses 9v battery. Masters Medical — to buy $165–$495, electrodes $18, recharger kit $70.

Appendix C

Ancillary Investigations

➔ *The presence of alerting clinical features of serious conditions is an indication for ancillary investigations. The principal first line investigations are summarised below. The table is intended as a general guide only.*

Appropriate Investigations for Possible Serious Causes of Acute Musculoskeletal Pain

SUSPECTED CONDITION (and alerting clinical features)		REGION OF PAIN: Lumbar Spinal	Cervical Spinal	Thoracic Spinal	Shoulder	Knee
Fracture History of significant trauma History of minor trauma in association with corticosteroid use, age over 50, history of osteoporosis History or previous fracture or metabolic disease Positive for Canadian C-spine rule Positive for Ottawa Knee Rule	All cases	Plain radiography				
	Stress of pars interarticularis	Bone scan				
Infection Fever Sweating Risk factors for infection (invasive medical procedure, indwelling device, injection, injecting drug use, trauma to skin or mucous membrane, immunosuppressive disease or treatment, diabetes mellitus, alcoholism)	All cases	ESR, FBC, CRP				
	Spinal	MRI				
	Osteomyelitis				MRI	
	Joint				Aspiration, Culture and Microscopy	
Tumour Palpable mass Past history of malignancy Age > 50 years Failure to improve with treatment Unexplained weight loss Pain not relieved by rest	Myeloma	IEPG, Serum protein electrophoresis				
	Prostate	PSA				
	All cases	First line: ESR, CRP Second line: MRI				
Crystal arthritis Joint effusion					Aspiration, Microscopy	
Aneurysm Cardiovascular risk factors Anticoagulants Transient ischaemic attacks Bruits Recent history of torsion to neck Absence of musculoskeletal signs	Vertebral, Carotid		MRA			
	Aortic	Ultrasound				
Osteonecrosis Immunosuppression Renal dialysis Use of corticosteroids Diabetes, alcoholism					MRI	

Note: ESR: erythrocyte sedimentation rate; FBC: full blood count; CRP: C-reactive protein; MRI: magnetic resonance imaging; IEPG: immunoelectrophoretogram; MRA: magnetic resonance angiography.

Appendix D

Consultation

➔ *The draft guidelines were submitted for public comment. The comments received have been incorporated to improve the content and format of the document. A list of the individuals and organisations who provided comment is as follows:*

1. Australian Physiotherapy Association
2. Australian Rheumatology Association
3. Diana Bainbridge
4. Les Barnsley
5. Nikolai Bogduk (Australasian Faculty of Musculoskeletal Medicine)
6. Melanie Cantwell (Consumers' Health Forum of Australia)
7. Keith Charlton
8. Chiropractors' Association of Australia
9. Chiropractic Education Australia Ltd
10. Paul Clarke
11. Milton Cohen (Australia and New Zealand College of Anaesthetists, Faculty of Pain Medicine)
12. Myles Coolican
13. Margaret Crowe (Australian Rheumatology Health Professionals Association)
14. Megan Davidson (School of Physiotherapy, Latrobe University)
15. Phillip Donato (Chiropractors' Association of Australia)
16. Jan Elsner (Sapience Consulting)
17. RL Galley
18. Phillip Giles
19. Roger Goucke (Australian Pain Society)
20. Jay Govind (Australasian Faculty of Musculoskeletal Medicine)
21. Health Consumers Council of WA
22. Alison Hogg
23. Julia Hush (Rehab One Physiotherapy)
24. Wade King (Pain Medicine)
25. Bridget Kirkham (Arthritis Foundation of Australia)
26. Christopher Maher (School of Physiotherapy, The University of Sydney)
27. Scott Masters (Australasian Faculty of Musculoskeletal Medicine)
28. John Murtagh (Royal Australian College of General Practitioners)
29. Michael Nicholas (Pain Management and Research Centre, The University of Sydney)
30. Andrew Nunn
31. CA Richardson (School of Health and Rehabilitation Sciences, The University of Queensland)
32. Patricia Roach (School of Physiotherapy, The University of Queensland)
33. Stephen Robbins (Australian Osteopathic Association)
34. Royal Australian and New Zealand College of Radiologists
35. Royal College of Nursing, Australia
36. Andrew Skinner
37. Ian Steven (WorkCover South Australia)
38. JR Taylor
39. Janney Wale (consumer)
40. Peter Werth (Chiropractic and Osteopathy College of Australia)
41. Victor Wilk (Australasian Faculty of Musculoskeletal Medicine)
42. Michael Yelland (Department of General Practice, The University of Queensland)

Appendix E

Tables of Included and Excluded Studies

➔ *A list of the tables of included and excluded studies is as follows:*

EFFECTIVE COMMUNICATION — Table of Included Studies

STUDY	METHODS	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY	NOTES
Abenheim 1995	Random sample of workers compensated for back and neck injury looks at prognostic consequences of making an initial medical diagnosis of work-related back injuries. Medical records were retrieved for the diagnosis. Follow-up occurred at 2 years and recorded duration of compensated absence from work	*n* = 2500; 2147 met criteria of more than 1 day off but no compensation in previous 2 years for back pain	N/A	13.9% had no diagnosis within 7 days (6% of these went on to become chronic); 43.8% had > one diagnosis in first week; only 8–9% were given a specific diagnosis. Older workers more likely to be given a specific diagnosis as well as those from the primary sector. Workers receiving a specific diagnosis were 4.9 times more likely to become chronic; older subjects and those receiving $50+ compensation were also predictors. Occupation was not a predictor. Older workers given specific diagnosis for their back injury 10 times more likely to become chronic than younger non-specific back pain patients	Selection bias minimised = Y Adjusted for confounders = Y (age) Measurement bias minimised = data extractors were blinded, the outcome measure was independent (computer database) Drop out rate = U Overall bias rating = moderate	Specific diagnoses (i.e. neck, thoracic or low back pain) are not described in the 8-9% with a specific diagnosis
Burton 1999	Randomised controlled trial in primary care setting in 5 general practices and 1 osteopathic practice in England. 86% of eligible patients agreed to participate	Participants had new acute or recurrent episode of low back pain with current duration of < 3 months, and no lost time from work. Intervention group: *n* = 83 (41 F; 42 M); mean age 42.6 + 10.8; Control group: *n* = 79 (48 F; 31 M); mean age 44.7 + 12.2 years	Novel education booklet vs traditional educational booklet. Novel booklet provided advice to remain active, assurance that nothing serious was wrong, encouragement to have a positive attitude and discussion of patient's involvement and responsibilities. Traditional booklet advised against activity if in pain, described spinal injury and damage, encouraged passivity	Significant reduction in mean belief scores at 2/52 and 3/12: $p = 0.02$, and at 12/12 $p = 0.05$. For clinically important improvement, at 2/52 RR = 2.52 (1.17–5.40) ; at 3/12 RR = 1.53 (1.05-2.23); at 12/12 RR = 1.47 (1.02–2.11). Disability and pain scores improved in both groups from baseline, with no significant difference between groups	Adequate allocation concealment = U Blinding = double blinded plus outcome assessor blinded ITT analysis = U LTFU < 15% = N (78% followed) Bias = Moderate	Doesn't describe method of randomisation
Croft 1998	Consecutive cohort of new presentations. South Manchester LBP cohort	*n* = 490 presentations to GP for back pain over 1 year. Mean duration of pain 3 weeks. M = 203; F = 287 Excluded if presented in past 3/12		59% of subjects did not consult again for their back pain in the next 12/12; 32% had repeat consultation but this occurred within the first 3/12. Patients > 30 years were 3 times more likely to have repeat consults than younger low back pain patients. 25% stated fully recovered at 12/12	Selection bias minimised = N Adjusted for confounders = Y Measurement bias minimised = U Drop out rate = 20% Overall bias rating = Moderate	A subsample of the non-interviewed group (*n* = 44) were followed (not stated how chosen) and found to have better outcomes than those who did participate (full recovery at 3/12 was 33% compared with 21% in the study sample). Therefore the estimates of poor outcome stated by the authors may be overestimates
Deyo 1987	RCT. Stratified by number of prior episodes and employment status	Low risk subjects from consecutive patients presenting to walk in clinic of public hospital. 81% were Hispanic. Study compared immediate xray (*n* = 43; F24, M19, mean age 34.3 years, mean duration of pain 12.6 days) vs education (*n* = 49; F24, M25, mean age 32.5 years, mean duration of pain 16.1 days	Immediate lumbosacral spine xray vs education (5 minutes brief advice) with xray after 3/52 if unimproved	50% of eligible enrolled. Follow-up at 3/52 was 92% and at 3/12 was 83%. No significant difference in any of the self reported measures of pain, satisfaction with care, functional ability, days off work. Overall 88.4% of xray group had an xray vs 29.3% of the education group over 3/12	Adequate allocation concealment: = U Blinding = N ITT analysis = Y LTFU < 15% = Y (17%) Bias = Moderate	Patients also randomised to receive 2 days vs 7 days of bed rest but these groups were combined for this report and analysis

EFFECTIVE COMMUNICATION — Table of Included Studies continued

STUDY	METHODS	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY	NOTES
Indahl 1995	Quasi-randomised study	Patients with > 8 weeks sick leave with a sick leave certificate; Interventions group: *n* = 463; mean age 46 years; 176 F; 287 M); Control group: *n* = 512; mean age 44.3 years; 202 F; 310 M	Conventional care versus mini-back school (advice to be active, education about pain, assurance). All subjects were fully examined including xray and CT scan. The interventions group were followed up at 3/12 and 12/12; there was no follow-up visit of the control group members. Interventions group received clinical examination plus mini back school × 2 hours first day and 1 hour on individual basis after 2 weeks; then 3/12 and 12/12 outpatients appointment for follow-up	Return to work (RTW) was the primary outcome measure. At 200 days 70% had RTW in the intervention group vs 40% RTW in the control group. $p = 0.000$	Adequate allocation concealment: = N (intervention group given even number and control group given odd numbers) Blinding = N ITT analysis = Y LTFU < 15% = no LTFU Bias = moderate	Difficult to determine method of randomisation. Patients with subacute pain 8–12 weeks. No documentation of conventional care
Indahl 1998	Five year follow-up of 500 subjects from the original cohort (Indahl 1995 study)	Interventions group = 245 (156 M; 89 F; mean age 68.6 years + 15); Control group = 244 (150 M; 94 F; mean age 68.8 + 13.3). Norway National Insurance Office provided the follow-up data	As above (mini-back school vs conventional care)	RTW at 5 years: Interventions group = 81% and Control group = 65%. Permanent disability (i.e. no RTW): Interventions group = 19%; Control group = 34%. 58% of the Interventions group had at least one recurrence; 48% of the control group had at least one recurrence of LBP. However the control group were more likely to have 2 or more recurrences (69%) compared with the intervention group (49%), $p < 0.03$	Adequate allocation concealment: = N Blinding = insurance assessor blinded; patients in control group unaware of involvement in the study; data processing and statistical analysis done by an independent observer ITT analysis = Y LTFU < 15% = N Bias = moderate	Patients with subacute pain of 4–12 weeks duration. A follow-up of only 500 patients from the original study but not clear how these were selected from the 1995 study population. No documentation of conventional care
McGuirk 2001	Open-label, non-randomised trial of 13 special (i.e. staffed by specialised GPs with post-graduate training in musculoskeletal medicine) urban and rural clinics who agreed to abide by evidence-based guidelines	Participants had LBP for less than 12 weeks with pain present for at least several days. Workers' compensation recipients excluded. 437/547 participated out of those eligible. Median duration of pain approximately 2 weeks	Treatment × 3 months according to evidence-based guidelines for acute low back pain versus usual care	1.4% found to have features alerting to the presence of serious conditions associated with their LBP	Adequate allocation concealment: = N (N/A) Blinding = unblinded ITT analysis = Y LTFU < 15% = Y Bias = high (non-randomised)	

ACUTE LOW BACK PAIN — Table of Included Studies (Diagnosis)

STUDY	METHODS	PARTICIPANTS	TEST	RESULTS	QUALITY SUMMARY	NOTES
Hollingworth 2002	An observational/descriptive study of the impact of guidelines for lumbar spine radiography on GP practice; provides useful background information				N/A	Useful for additional information and for summary of radiography findings
Kendrick 2001	RCT to test hypothesis that radiography of lumbar spine in people with acute low back pain is not associated with improved clinical outcomes or satisfaction with care	Median duration of low back pain was 10 weeks and the median number of days away from work was 14. 50% had pain duration of less than 12 weeks; pain duration ranged from at least 6 weeks to less than 6 months. Participants identified by medical record review	Primary outcome, Roland Morris scale; secondary VAS, EuroQol, satisfaction with care, use of health services. Randomised to xray (*n* = 210; 186 had it) and non-xray (*n* = 211)	At 3 months, more pain and disability and medical attention in xray group (RR 1.26, 95% CI 1.0–1.6) and had lower overall health status score and borderline higher Roland Morris pain scores. Satisfaction was greater at 9 months in those receiving xray but not at 3/12. Overall, 80% of patients would want xray if given the choice. An abnormal finding on xray made no difference to the outcome by the Roland Score.	Adequate allocation concealment = Y ITT analysis = Y LTFU < 15% = Y (93.6% at completion; follow-up at 3/12 and 9/12) Blinded = N Bias rating = Moderate Groups similar at baseline	
Kerry 2002	Randomised unblinded controlled trial, prospective with an observational arm to enable comparisons	No xray (*n* = 76, mean age 44, past hx back pain 57%, bad pain 20%). Xray (*n* = 65, mean age 44, past history of back pain 54%, bad pain 22%). Patients from UK general practices. First presentation for low back pain	Randomised to either xray or no xray (observational). Questionnaires provided at baseline, 6 weeks and 1 year	xray group had no effect on physical functioning, pain or disability but had small improvement in psychosocial wellbeing at 6 weeks and 1 year RR: 87%, 73%, 67%	Adequate allocation concealment = Y ITT analysis = Y LTFU < 15% = N (30% drop out rate but greater than 90% medical record follow-up at 1 year) Blinded = patient not blinded; self-reporting measures Bias rating = Moderate Adjusting for confounders	
Knutson 2002	Cross-sectional survey of leg length asymmetry, foot rotation and pelvic crest unlevelling; examiner was blinded	74 volunteers of the community at various social gatherings; 50 F, 24 M; mean age 36 years (11–65); 82% had had back pain in the past; Community based		VAS of pain; pelvic level and foot rotation were not good predictors of self-reported pain; postural leg length inequality positive likelihood of 2.24 and negative likelihood ratio of 0.49; sensitivity 65%; specificity 71%	Selection bias minimised = U Measurement bias minimised = Y Verification bias minimised = U Compared to reference standard = N Confounding avoided = N Self-report of pain Bias rating = Moderate	Mixed population of volunteers; 78.7% had pain < 2 months and many had recurring pain; and definition of back pain was unclear
Leboeuf-Yde 2002	Population based sample of volunteers from a twin register	2927 twin pairs; 94 twin pairs in this substudy. Participants had a chiropractic examination with the aim of determining the prevalence of positive motion palpation findings (so-called fixations and spontaneous pain response). research lab at a university hospital in Denmark		Sensitivity low (< 60%); Specificity < 80%. Concluded that motion palpation was not a good method to detect people with low back pain	Selection bias minimised = N Measurement bias minimised = N Verification bias minimised = Y Reference standard = Y Confounding avoided = N Bias = moderate	

ACUTE LOW BACK PAIN — Table of Excluded Studies (Diagnosis)

STUDY	REASON
Borenstein 2001	Descriptive view — opinion of single author. Mostly discussing chronic low back pain. Qualitative review focused on herniated disc related low back pain
Haijiao 2001	Not a diagnostic test study. No information on duration of pain
Hollenberg 2002	MRI study, did not specify duration of back pain. Primarily adolescent population all with clinical diagnosis of sport-related stress injury to spine
Hunter 2001	Concerned with chronic/persistent pain. Review includes neck and carpal tunnel and non-specific low back pain
Kilpikoski 2002	Chronic pain subjects (average duration of pain = 14 years)
McNally 2001	Patient group not well defined; other back pain sites; pain > 6 weeks but cannot isolate the less than 12 week group; select group unresponsive to therapy; descriptive study of MRI findings only
Patel 2000	Qualitative review. No original data
Yelland 2001	Not concerned with low back pain only. Doesn't meet inclusion criteria. Does not specifically deal with acute pain — no duration given
Zuberbier 2001	No specific information on patient spectrum. Review is at least partly based on studies of chronic pain subjects (based on the reference list)

ACUTE LOW BACK PAIN — Table of Included Studies (Prognosis)

STUDY	METHOD	PARTICIPANTS	RESULTS	QUALITY SUMMARY	NOTES
Abenheim 1995	Random sample of workers compensated for back and neck injury looks at prognostic consequences of making an initial medical diagnosis of work-related back injuries *medical records were retrieved for the diagnosis *f/u × 24/12 and recorded duration of compensated absence from work	*n* = 2500; 2147 met criteria of more than 1 day off but no compensation in previous 2 years for back pain	13.9% had no diagnosis within 7 days (6% of these went on to become chronic); 43.8% had > one diagnosis in first week; only 8–9% were given a specific diagnosis. Older workers more likely to be given a specific diagnosis as well as those from the primary sector. Workers receiving a specific dx were 4.9 times more likely to become chronic; older subjects and those receiving $50+ compensation were also predictors. Occupation was not a predictor *older workers given specific diagnosis for their back injury 10 times more likely to become chronic than younger non-specific back pain patients	Selection bias minimised = Y Adjusted for confounders = Y (age) Measurement bias minimised = data extractors were blinded, the outcome measure was independent (computer database) Drop out rate = U Overall bias rating = moderate	Specific diagnoses (i.e. neck, thoracic or low back pain) are not described in the 8–9% with a specific diagnosis
Croft 1998	Consecutive cohort of new presentations. The South Manchester LBP cohort	*n* = 490 presentations to GP for back pain over 1 year. Mean duration of pain 3 weeks. M = 203; F = 287. Excluded if presented in past 3/12	59% of subjects did not consult again for their back pain in the next 12/12; 32% had repeat consultation but this occurred within the first 3/12. Patients > 30 years were 3 times more likely to have repeat consults than younger low back pain patients. 25% stated fully recovered at 12/12	Selection bias minimised = N Adjusted for confounders = Y Measurement bias minimised = U Drop out rate = 20% Bias rating = moderate	A subsample of the non-interviewed group (*n* = 44) were followed (not stated how chosen) and found to have better outcomes than those who did participate (full recovery at 3/12 was 33% compared with 21% in the study sample). Therefore the estimates of poor outcome stated by the authors may be overestimates
Fransen 2002	Prospective cohort study	1440 claimants (compensation cohort) with 854 (59.3%) providing data. 33.6% had prior back pain in past 6 months. 75% in high risk groups. 26% female/74% male with 28% > 46 years. 43.4% with prior ACC claim. 62% had radiating leg pain. 56.5% had severe/crippling pain at onset/baseline	At 3/12 23.9% of claimants still receiving compensation. Determinants of chronicity were: severe radiating leg pain OR 2.1 (1.3–3.5); BMI obese vs. normal OR 1.8 (1.2–3); moderate Oswestry OR 3.3 (1.5–7.0); Severe Oswestry OR 5.1 (2.4–10.6); Extreme Oswestry OR 4.6 (2.2–9.7); GHQ-28 (> 6) OR 2.8 (2–3.87); no light duties OR 1.84 (1.28–2.66); lifting for > 3/4 of the day OR 1.85 (1.25–2.75) were all independent determinations of chronicity. Psychosocial characteristics were not significant in this study including APGAR, job satisfaction, life events (apart from new family members = less likely to be claimants at 3 months)	Selection bias minimised = U Probably consecutive Adjusted for confounders = Y Measurement bias minimised = Y Drop out rate = 60.7% Bias rating = Low	Possibly has to be excluded by reference to radiating pain however it is not clear if this is somatic referral (which would be included) or radicular pain (which wouldn't). Not clear that they are reviewing acute onset low back pain. Requires caution using univariate models. 84% have leg pain. Generalisability is restricted to workers covered by non-adversarial claims systems. High prevalence had radiating pain that made it a more select group simple self-report like GHQ-28 with depression and anxiety-distress items more predictable
Fritz 2001	Longitudinal cohort study on the role of fear-avoidance beliefs in acute low back pain: relationships with current and future disability and work status	78 participants in clinical trials comparing physical therapy treatments in work related low back pain; 62% male, mean age 37.4 years; 50% prior history low back pain; baseline Oswestry 42.9 to 26.4 at 4 weeks	CESD depression score; fear avoidance beliefs scale; WaddellI non-organic; physical impairment; pain rating; work subscale (from FABQ); physical activity (from FABQ); predictors of poorer function (i.e. Higher Oswestry) at 4 weeks; initial Oswestry and treatment group R2 0.23 FABQ work subscale explained additional 7%; · Pain ratings were not predictors (R2 0.3) *p* = 0.009 change; Predictors of RTW only FABQ work was significantly independent predictor (OR 1.17) (1.04–1.31) but treatment group highly suggestive (OR 3.19; 0.95–10.76). Increased R2 0.26 to 0.39 (13%); *p* = 0.003 *fear-avoidance beliefs are present in patients with acute low back pain and may be a factor in the transition from acute to chronic *screening for fear-avoidance beliefs may help identify patients at risk of prolonged disability and work absence	Measurement bias minimised = U Drop out rate = U Bias rating = Low Selection bias minimised = U Adjusted for confounders = Y	Include because is ALBP, a RCT, but ? whether they analysed the results correctly. Setting: may not be typical of GP practice. f/u limited to only 4 weeks. Subjects may have different expectations and outcomes as part of a clinical trial. Their FABQ levels were higher than previously published. Small study done within a work based clinical trial of physical therapies for LBP suggest fear-avoidance belief related to work but not physical activity in general was an independent predictor of poor outcome (more desirability, less likely to RTW) at 4 weeks

ACUTE LOW BACK PAIN — Table of Included Studies (Prognosis) continued

STUDY	METHOD	PARTICIPANTS	RESULTS	QUALITY SUMMARY	NOTES
Fritz 2000	Longitudinal cohort study based on record review. The use of nonorganic signs and symptoms as a screening tool for RTW in patients with acute low back pain	69; same cohort as Fritz et al. 2001 Fear avoidance; non-organic signs (0–7)/non-organic symptoms (0:5). Setting = referral to physiotherapist	No inter or intrarater reliability for the subjective predictor variables. Nonorganic signs, symptoms or a combination thereof were unable to predict who would RTW at 4 weeks with full duties. Likelihood ratios showing no consistent direction with increasing scores and < 3.00 maximum and general < 1.0 Lack of association may be due to the small sample size and small numbers in each of the categories. Measurement variability also a problem. Likelihood of returning to full duties: 0 = 0.43; 1 = 2.00; 2 = 3.11; 3 = 0.5; 4 = 3.0. None of the non-organic tests served as effective screening tool	Measurement bias minimised = U Drop out rate = None Bias rating = Low Selection bias minimised = Y Adjusted for confounders = Y	Concerns that it includes (or doesn't exclude) some people with nerve root compression and not clear how many and it is a small sample. It does involve ALBP. This is not necessarily representative of pts presenting to GP practice
Hurley 2001a	Prospective cohort study	Consecutive sample of 118 subjects recruited from physiotherapy departments in Northern Ireland. Patients referred by medical practitioner for treatment of LBP. 56% had current pain < 3 months	76% available at 12/12 follow-up. 86% experienced a recurrence in the 12 months follow-up indicating that they were a particularly severe group. The authors evaluated the ability of the ALBPSQ (a biopsychosocial risk profile score) to predict chronicity. Although the score was correlated with pain and disability at 12/12, sensitivity (53%) and specificity (60%) for predicting recurrences were low. A high score had a high sensitivity (100%) for predicting work loss at 12 months but a lower specificity (61%)	Measurement bias minimised = N Drop out rate = 24% Bias rating = moderate Selection bias minimised = U Adjusted for confounders = N LTFU = 24%. This could be most useful in the setting of a normal score whereby one could rule out work loss (SnNOUT)	Include because within 12 week period and results presented separately by duration of pain. Claimant cohort so not necessarily representative of patients presenting to GP. Those who were older and had a heavy work index and more severe injury were less likely to RTW. If employed > 1 year prior to injury, more likely to RTW
Krause 2001	Retrospective cohort study to determine the association of psychosocial factors with duration of work disability	Claimant cohort with acute LBP. *n* = 433 (70% male; mean age 37.3 years). 33% had previous back LTI	High physical and psychological job demands and low supervisory support are each associated with about 20% lower RTW rates during all phases of disability whereas high control is associated with a 30% higher RTW rate. Multivariate analysis adjusted for psychosocial injury, workload and employment factors. Significant factors: heavy work index: $p = 0.000$; more severe injury: $p = 0.000$; age: $p = 0.000$; pre-injury employment > 1 year: $p = 0.01$. Not significant: job strain, work flexibility, supervisor support, co-worker support, previous LTI back injury, vibration index, posture index, gender, union, employer size	Measurement bias minimised = U Drop out rate = 0 — Retrospective Bias rating = Low Selection bias minimised = U Adjusted for confounders = U	
Linton 1998	Prospective cohort study. Looked at accumulated leave at 6/12 and whether a screening tool could predict this	Recruited from 19 health clinics in Sweden; included acute/subacute back and neck pain. *n* = 142 eligible; 137 participated *mean age 42.6 years (22–63); F = 65%; 93% Swedish; 55% back, 55% should, 53% neck pain	At 6/12 62% had not had any sick leave; 18% required short term leave (1–30 days) and 20% had long term leave. Factors associated with 73% of chronic pain sufferers: belief that one should not work with current pain levels (fear avoidance); perceived chance of working within 6/12; light work 'problems with work function'; stress; previous number of sick leave days	LTFU < 15% = Y Bias = high (non-randomised) Adequate allocation concealment: = N (N/A) Blinding = unblinded ITT analysis = Y	Selection criteria acute/subacute (< 3 months). (limited studies of acceptable quality on which to base results)
Linton 2001	A systematic review of the literature on the role of psychosocial workplace factors on back pain		There was strong evidence that job satisfaction, monotonous work, work relations, work demands, stress, perceived ability to work influence the development of future episodes of back pain and its disability	Number of studies = 21 Quality assessment = Y Data pooled = N Heterogeneity = Y	

ACUTE LOW BACK PAIN — Table of Included Studies (Prognosis) continued

STUDY	METHOD	PARTICIPANTS	RESULTS	QUALITY SUMMARY	NOTES
McGuirk 2001	Open-label, non-randomised trial of 13 special (i.e. staffed by specialised GPs with post-graduate training in musculoskeletal medicine) urban and rural clinics who agreed to abide by evidence-based guidelines	Participants had LBP for less than 12 weeks with pain present for at least several days. Workers' compensation recipients excluded. 437/547 participated out of those eligible. Median duration of pain approximately 2 weeks	Treatment × 3 months according to evidence-based guidelines for acute low back pain versus usual care. 1.4% found to have features alerting to the presence of serious conditions associated with their LBP. Full recovery at 3/12 for special clinic arM = 67%; at 6/12 = 70%; at 12/12 = 71%. Full recovery at 3/12 for usual care arM = 49%; at 6/12 = 64%; at 12/12 = 56%. Recurrences at 6/12 for usual care = 16%; for special clinic = 16%. Recurrences at 12/12 for usual care = 27%; for special clinic = 7%	Adequate allocation concealment = N Blinding = N ITT analysis = Y LTFU < 15% = U Bias = High	
Pincus 2002	Systematic review of prospective cohort studies in acute or sub-chronic low back pain, minimum 12 month f/u. Measured at least one psychosocial variable at baseline	Identified 25 studies. 18 independent cohorts	Only two got three star rating. Six met acceptability criteria re: methodology, psychological measurement and statistical analysis. Says not enough evidence to say that fear-avoidance is a marker of chronicity. Distress effect size 0.4, OR 3 and somatisation varying effect size 0.2 and 0.6 at 12/12 and 0.9 at 24/12 *fear avoidance beliefs, passive coping strategies and personality (MMPI hysteria subscale) did not appear to be independent predictors of unfavourable outcome	Number of studies= 25 Quality assessment = Y Data pooled = Y Heterogeneity = U	
Pulliam 2001	Designed as a prospective study and only presented as a cross sectional analysis with low response rate and modelling	57/425 screened; divided into 28 low and 29 high risk of chronic back pain disability based on Gatchel	Predictors of being in high risk group but not predictors of chronicity because follow-up study has not been conducted. Low positive SNAP for adaptive and non-adaptive, high workaholism, avoidance coping strategies and AXIS 1 disorders (affective, anxiety, somatoform, substance abuse)	Measurement bias minimised = N Drop out rate = not a follow-up study Bias rating = High Selection bias minimised = N Adjusted for confounders = N	Acute LBP pts < 10 weeks duration attending orthopaedic practices but no information about diagnoses/red flags except fibromyalgia or cancer. Poor quality/very high risk of bias. Non consecutive sampling. Unrepresentative sample. Unsure how representative the sample was. Not a follow-up study
Schiottz-Christensen 1999	Prospective cohort study in general practice in Denmark. 330 GPs invited to participate; 130 agreed, 75 enrolled. Range recruited (1–41; median 6.5)	524 LBP < 14 days; excluded back pain in last 6 months (but 70% had pain before) and red flags; 43% on sick leave at index consultation; 60% low back only and other had some radiation; 29–46 years, 62% male, 97% employed or employable; followed at 1, 6, 12 months	50% of those on sick leave were back at work within 8 days; at 12/12 only 2% still on sick leave (14.8% had some sick leave during the f/u); 46% (41–49%; *n* = 229) continued to c/o LBP i.e. not completely recovered. Previous sick leave OR 2.30 (1.3–3.9; 95%CI) and GP global assessment most likely to develop CLBP OR 10.40 (2.2–49.1; 95% CI) or poor outcome. Those with GP global assessment of likelihood to develop CLBP + ve SLR had more sick days and sick leave in the first month but beyond that no obvious difference	Measurement bias minimised = N Drop out rate = 4% Bias rating = Low Selection bias minimised = U Adjusted for confounders = Y	GP practice with 4% drop out rate. Good natural history article even though include people with nerve root problems (although these people are separated). Were not able to identify objective factors at first visit to GP that strongly predict prognosis (i.e. being on sick leave) *global assessment is an interesting area to explore. Higher prevalence of subjects still having problems — may be influenced by the way they worded the question
Shaw 2001	Systematic review to synthesise findings from available studies of prognostic factors for acute OLBP disability	22 studies met criteria for inclusion	Prognostic factors: low workplace support; personal stress; shorter job tenure; prior episodes; heavier occupations with no modified duties; delayed reporting; severity of pain; functional impact; radicular findings; extreme symptom report. Physicians can decrease OLBP disability by using standard questionnaires, improving communication with patients and employees, specifying RTW accommodations, employing behavioural approaches to pain and disability management	Number of studies= 22 Quality assessment = N Data pooled = N Heterogeneity = Y	Included as a source reference but with caveat that involves a mixture of study designs, there is no individual study or quality review and no raw data provided to calculate OR etc.

ACUTE LOW BACK PAIN — Table of Included Studies (Prognosis) continued

STUDY	METHOD	PARTICIPANTS	RESULTS	QUALITY SUMMARY	NOTES
Truchon 2000	Systematic review of three electronic databases (Medline, PsycInfo, Sociofile) plus review of reference lists 1987–1999. Looked at prospective studies of LBP < 12 weeks	Reviewed 18 articles that met criteria: looking at occupational factors that influence LBP	Factors that are useful predictors were: prior history of significant LBP, preoccupation with health, negative attitudes and outlook, passive coping strategies. Potential predictors of LBP were: locus of control, work environment, job satisfaction, compensation, family situation, personality. Probably not predictors: pain intensity in acute phase, medical assessment of severity in acute phase, age, gender, ethnicity, education, work task	Number of studies = 18 Quality assessment = U Data pooled = N Heterogeneity = Y	Not clear whether articles double-read
Werneke 2001	Prospective evaluation of patients at 1 year after discharge from physiotherapy services to determine if centralisation phenomenon is a prognostic factor for CLBP and disability *secondary to acute onset of NSLBP and neck pain *setting: American private health clinic	223 pts with LBP (original cohort described elsewhere); mean age 37.8 (9.9); 52% male; 18.2% multiple sites; 30.7% leg pain at intake; 79% high pain at intake and mean 13.3 + 9.6 days pain; 10% workers comp. Lot of psychosocial factors (depressive (47%), somatisation (45%), fear-avoidance beliefs (45%), perceived disability (63%); central and partial reduction (77.3%); non-centralisation (22.7%)	Drop out rate 16% at 12/12 *assessed pain intensity, RTW status, sick leave at work, activity interference at home, and continued use of health care *univariate analysis: Overt pain behaviour (2, 3, 4); Perceived disability at discharge (1–5); Pain pattern classification (1–5); Leg pain at intake (2, 3); Pain at intake (2, 4); Non-organic physical signs (1); Fear of work activities, multiple sites, payer (2) *Dependent (outcomes at 12 months): see TABLE *pain pattern classification (non-centralisation) and leg pain at intake were the strongest predictive variables of chronicity	Measurement bias minimised = N Drop out rate = 4% Bias rating = Low Selection bias minimised = U Adjusted for confounders = Y	Not much detail on patient population but gives reference to baseline study, Spine 1999 *unsure how referred initially

ACUTE LOW BACK PAIN — Table of Excluded Studies (Prognosis)

STUDY	REASON
BenDebba 2002	Chronic LBP mean 2.52 years for current episode. Non-randomised — describing interventions. Chronic study population (average pain duration = 10 years)
Dionne 2001	No knowledge of duration of pain. Is it a yellow flag? Low educational status. Studies not grouped or analysed by duration of LBP. Acute LBP studies included in review pre-date the years of literature update
Hemmila 2002	Described as patients with back pain for at least 7 weeks — mean deviation actually around 7 years; also subgroup of acute patients excluded after randomisation
Hudak 1998	A critical appraisal of the research; not primary research
Kaiser 2001	Subjects selected from rehab scheme, which is activated after healing and medical treatment is complete and not if 'likely to return to work within a reasonable time'. No specific mention of duration of pain. Includes those with herniated discs but no subgroup results presented
Kjellman 2001	Inadequate description/definition of duration of pain at baseline (50% had sick leave of 28 days or more); primary focus is comparison of pain episodes/impact on ADLs between pain sites (back pain vs neck/shoulder); back group included sciatica disc problems
Kuch 2001	Can't identify methods for systematic review. Can't dissect out results for LBP. Deals with risk factors for chronic pain rather than risk factors for progression from acute — chronic pain
Kummel 2001	Not only concerned with acute LBP. Not a systematic review or coherent study. Letter reviewed from B Kimmel (595) Spine. Patients were probably in chronic phase when first evaluated in referral — not an inception cohort. Analysis based on retrospective chart review
Linton 2001	Broad review of back and neck pain and which interventions prevent neck and back pain. Does not meet inclusion criteria
Linton 2002	Looks at effect of abuse on pain; does not meet inclusion criteria for acute pain
Loisel 2002	Includes thoracic spine pain, subacute rather than acute pain, radiating pain, neurological signs and surgery — contaminated sample
Lundin 2001	Study deals with onset of LBP at follow-up in a cohort who had back xrays at baseline approx. 10 years earlier. Not acute LBP. Seems high rate of self-reported pain. Different definition of pain — could be whole spine
McIntosh 2000b	No description of duration of LBP; no description of consumer population and no prognosis; no results given
Musgrave 2001	Risk factor for pain onset rather than acute — chronic. No description of duration of pain. Pain conditions not defined by duration but by recency/frequency and pain related disability
O'Sullivan 2000	Commentary — not original
Shannon 2001	Duration of LBP uncertain and discusses risk factors for onset of LBP; low numbers
Stig 2001	Uncontrolled intervention. Most subjects chronic. Results not shown separately for acute/chronic
Torp 2001	Risk factors for onset rather than progression. Deals with risk factors for having musculoskeletal pain 1 year after baseline data collection. Also pain not categorised on overall duration but on pain in last 30 days
Trainor 2002	Qualitative review. No original data. Discusses risk factors for development of LBP in athletes

ACUTE LOW BACK PAIN — Table of Included Studies (Interventions)

STUDY	METHODS	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY	NOTES
Browning 2001	Meta-analysis of cyclobenzaprine and back pain	Mixture of acute and chronic back and neck pain; 11 of the 14 studies only included ALBP (sensitivity analysis showed no difference between studies for acute and chronic LBP	Compares cyclobenzaprine to placebo, not other medications. Used variable doses of cyclobenzaprine from 10-60mg daily between trials	Cyclobenzaprine has a moderate effect on global symptoms (OR 4.7 95%CI 2.7, 8.1) NNT 2.7 (2.0–4.2) and back pain by (effect size 0.38–0.58) day 14 but trials were of dubious quality and heterogeneity significant. Five domains of pain were improved (local pain, muscle spasm, tenderness to palpation, range of motion, ADL). Local pain improvement days 1–4 0.41 (0.29, 0.53) and day 5–9 0.38 (0.21–0.56) and days > 9 0.44 (0.24–0.64). Efficacy was greatest in the first few days declining after the first week. Cyclo group experienced more adverse effects. Drowsiness $p < 0.001$, dry mouth $p = 0.02$, dizziness = 0.04, nausea $p = 0.70$, any 53% compared with 28% in placebo $p = 0.002$	Number of RCTs = 14 Quality assessment = Y Data pooled = Y Heterogeneity = Y Applicability of results = not available in Australia	Publication bias evident; many of the studies reviewed were conducted by pharmaceutical companies. Excluded non-English papers. Harms detailed. Quality of studies often poor, significant heterogeneity, publication bias, adverse effects of cyclo common. Patient rating of pain and global symptoms was only measured in one trial
Furlan 2002	Massage for LBP; a systematic review of RCT, quasi-RCT or CCT for massage for NSLBP. Cochrane Review	Patients with acute, subacute and chronic non-specific LBP	Any type of massage (M) using hands or mechanical device used to treat low back pain	Cannot conclude that massage is beneficial for ALBP (< 4 weeks) pts (only 1 low quality study). Moderate evidence that massage improves pain intensity and pain quality in subacute (4–12 weeks) LBP pts; Costs considered if required. M < manipulation, TENS; M = corsets; M = exercises; M > relaxation therapy, acupuncture, self-care education, sham laser. M in combination with exercise and education was significantly better (pain and function) than exercises in the short-term	Number of RCTs = 8 (2/8 included mixed subacute/chronic; 1/8 included only ALBP patients) Quality assessment = Y, blinded Data pooled = N Heterogeneity = Y	Meets inclusion criteria but the results are not so relevant for ALBP. Populations differed in duration of pain, interventions and age. Costs — cost of treatment but potential for long-term savings
Hagen 2002	Bed rest and advice to stay active: a systematic review. Included quasi-randomised studies	9 (n = 1435 subjects in total). Trials included patients with acute low back pain with or without radiation of pain below the knee	Bed rest compared with advice to stay active	There is not an important difference in the effects of bed rest compared with advice to remain active in the treatment of ALBP, with and without radiating pain	Number of RCTs = 9 Quality assessment = Y (5/9 studies low risk of bias) Data pooled = Some outcomes only Heterogeneity = Y	Includes sciatica therefore a heterogeneous group but correct duration of pain. Separate results not presented for acute simple LBP vs sciatica; excluded red flag conditions. No analysis of harms or costs. Multiple databases up to December 1998
Hides 2001	RCT; randomisation method not described	First episode of unilateral mechanical low back pain of less than 3 weeks presenting to emergency department	Intervention group (n = 20) received 4 weeks of twice weekly isometric exercises with real time ultrasound imaging. Control group (n = 19) received advice to stay active and resume 'normal activities as tolerated' plus medications etc.	There was reduced multifidus muscle size on the affected side in the short term (results reported in earlier study). In the control group, 84% had a recurrence compared with 30% in the treatment group at 12 months. Mean number of recurrent pain episodes in control group = 4.2 + 3.4 increased compared with trt group = 2.3 + 2	Adequate allocation concealment = U Blinding = Y (assessors only) ITT analysis = U LTFU < 15% = Y (0 at 1 year; < 15% at 3 years) Bias = Moderate Recall bias (patients not blinded; main outcome measure self-report)	

ACUTE LOW BACK PAIN — Table of Included Studies (Interventions) continued

STUDY	METHODS	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY	NOTES
Hilde 2002	Advice to stay active; systematic review	4 (*n* = 491 subjects in total). Included acute, subacute & chronic LBP with & without radiation below the knee; excluded red flag conditions	Advice to stay active as a single treatment for acute low back pain with and without sciatica.	Advice to stay active has small beneficial effects for people with acute non-specific back pain and little or no effect for those with sciatica; there is no evidence that advice to stay active is harmful	Number of RCTs = 4 Quality assessment = Y (2/4 studies showed risk of bias) Data pooled = N Heterogeneity = Y	Group was heterogeneous but results were presented separately; Medline search 1966-1998; other databases up to 1998 Review limited by excluding studies which included co-interventions which may be typical of clinical practice
Hsieh 2002	RCT looks at the effect of four conservative treatments for subacute low back pain	200 pts in an outpatient physiotherapy clinic in the USA. Assessed at baseline, after 3/52 of therapy, 6/12 after therapy completed. Subjects had lbp > 3 weeks and < 6 months for current episode or pain free × 2/12.	Back school vs. myofascial therapy, joint manipulation and combined MFT and JM. Back school comprised education, and body mechanics for daily activities and supervised home program	No significant difference by ANOVA at 3 weeks and 6/12 for VAS pain score and Roland Morris Disability Score. Combined JM/MFT as effective as JM or MFT alone. Back school as effective as JM and MFT	Adequate allocation concealment = U Blinding = Y (examiners) ITT analysis = Y LTFU <15% = Y (8% at 3/52, 11% at 6/12) Bias = Low	Includes some pts with pain > 3 months, mean duration of pain 10.7–11.5 (+ 6–7 weeks). No control group
Hurley 2001b	RCT investigating interferential electrode placement in acute low back pain compared with control treatmeng. Single blind, randomised controlled trial with 3/12 follow-up	*N* = 59. 48 remaining at 3/12. GP and self-referrals to the study in O/P physiotherapy departments, hospital and university settings	Interferential therapy electrode technique 1. IFT in painful areas with back book vs 2. IFT in spinal nerve area and back book vs 3. control and back book only	1) MPQ mean values 5.0 2.0 and 3.0; $p = 0.0592$ 2) RMDQ mean values 2.0, 1.0 and 1.0; $p = 0.049$ 3) EQ — 5D mean 0.80, 0.80 and 1.0; $p = 0.054$. There is a trend toward improvement in pain and disability scores with IFT over the spinal nerve area compared with painful area or control	Adequate allocation concealment = Y Blinding = N, U ITT analysis = Y LTFU < 15% = N (20% LTFU at ??/12) Bias = High	65% had LBP < 3 months duration. Sample size eligible consumer only 60. *p* values borderline
Karjalainen 2002	Cochrane Review (last updated 1999)	2 RCTs (*n* = 233 subjects in total)	Effectiveness of multi-disciplinary rehabilitation for subacute (> 4 week and < 12 weeks) back pain among working age adults; intervention was a physician consultation plus either a psychological, vocational or social intervention, or combination of these	Moderate evidence of positive effects of multi-disciplinary rehabilitation for subacute low back pain; workplace visit increases the effectiveness	Number of RCTs = 2 Quality assessment = Y (Both studies low quality, but data missing on several quality assessment criteria) Data pooled = N Heterogeneity = Y	These interventions are costly; more high quality trials needed. Subacute LBP (between 4–12 weeks duration). Red flag conditions excluded. Medline search 1966–1998; other databases up to 1998
Little 2001	RCT factorial trial to assess effectiveness of a booklet of physician advice to take regular exercise	*N* = 311. 8 doctors from 6 practices randomised 311 pts with a new episode of back pain. Aberdeen pain and function scale used. Patients randomised to 1 of 4 groups. Pain and function scores received from 239 (77%) of pts	1. Receive a detailed self-mgt booklet 2. Advice to take regular exercise 3. Both one and two or 4. Neither	Follow-up at 1 week: authors concluded that pain and functional outcomes can be improved with the use of either advice to exercise ($p = 0.076$) or provision of a booklet ($p = 0.049$), but the effect was reduced if both of these interventions were used ($p = 0.98$). Reviewers conclude that result for exercise was not significant; Follow-up at 3 weeks — no significant difference in pain/function between all groups.	Adequate allocation concealment = N Blinding = Y (data collectors) ITT analysis = U LTFU < 15% = N (23% LTFU) Bias = Moderate	

ACUTE LOW BACK PAIN — Table of Included Studies (Interventions) continued

STUDY	METHODS	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY	NOTES
Maher 1999	Systematic review evaluating 'activity prescription' for non-specific low back pain (NSLBP)	Acute (< 6 weeks) and subacute (6 weeks to 3 months) NSLBP. 9 RCTs for exercise therapy for acute NSLBP	'Activity prescription', i.e. bed rest, advice to alter activity levels and exercise	9 RCTs for exercise therapy for acute NSLBP. Exercise therapy less effective than NSAIDS, encouraging normal activity, spinal manipulative therapy, medical care, bed rest, no treatment and less intense exercise 7 RCTs on bed rest for acute NSLBP and 6 of 7 trials suggest bed rest harmful 2 RCTs for activity prescription (exercise) for subacute NSLBP found good results for patients who remain off work at 6 weeks	Number of RCTs = 9 Quality assessment = Y Data pooled = Y Heterogeneity = Y	Search of Medline & CINAHL (up to 1997)
Nadler 2002	Prospective, randomised single blind comparative efficacy trial	*N* = 371 F = 216; M = 155. with acute low back pain. 11 settings/sites	1. Heat wrap *n* = 113; 2. Acetaminophen 4000mg per day *n* = 113; 3. Ibuprofen 1200mg per day *n* = 106; 4. Oral placebo *n* = 20; 5. Untreated back wrap *n* = 19. Study involved 2 days treatment and 2 days f/u	Outcomes discussed: pain, muscle stiffness, lateral trunk flexibility. At day 2, significantly better for group 1 than 2 ($p = 0.0001$) or 3 ($p = 0.0007$) but only looked at results for 2 days — no discussion of 6 week, 3 months	Adequate allocation concealment = Y (oral placebo, unheated wrap) Blinding = Y (participants) ITT analysis = by patient record LTFU < 15% = Y Bias = Low/moderate	F/U 2 days only. Patients equivalent at baseline
Nelemans 2002	Cohcrane review of steroid injection therapy	21 RCTs. 'Benign' LBP of at least 4 wks duration; included studies with chronic pain subjects		Only one RCT met the criteria for this review (Garvey et al.)	Number of RCTs = 21 Quality assessment = Y Data pooled = Y (some outcomes only) Heterogeneity = N	Mixture of subacute and chronic; looks at facet injections. Medline & Embase 1966-1996. Extensive critical commentary on this review in Cochrane database
Palangio 2002	RCT — combination hydrocodone and ibuprofen vs oxcodone and acetaminophen. Computer-randomised, double-blind, intention to treat analysis and <15%follow-up	*N* = 197; mean age 45y. Adults with moderate to severe acute LBP requiring opioid or opioid-nonopioid combination therapy, enrolled within 48 hours of onset pain, self-rated pain mod-severe	Hydrocodone 7.5mg and ibuprofen 200mg (HC/IB) vs. combination oxycodone 5mg and acetaminophen 325mg (OX/AC)	No difference between groups in daily pain relief and SF-36 scores at 8 days	Adequate allocation concealment = Y Blinding = Y (double) ITT analysis = Y LTFU < 15% = Y (HC/IB = 14.7%; OX/AC = 15.3%) Bias = Low	Follow-up = 8 days. No difference between groups at baseline
Pengel 2002	Systematic review and meta-analysis of RCTs	Subacute (6 weeks to 3 months) nonspecific LBP with or without leg referral. Also used alternate definition of subacute (7 days to 6 months)	Manipulation, back schools, exercise, advice, TENS, massage, corset, cognitive behavioural treatment, coordination of primary care	Greater efficacy of advice on RTW compared to usual medical care. No high quality evidence was found for any other intervention. When using alternate definition for subacute, high quality evidence for exercise and manipulation and TENS may be effective	Number of RCTs = 13 Quality assessment = Y Data pooled = Y Heterogeneity = Y	Lack of a uniform definition for subacute LBP is a problem
Pohjolainen 2000	RCT — randomised, blinded to category, well designed	104 (5 withdrew); F = 52; M = 52. Median age 42 (19–63). Finnish outpatient clinic. Patients had < 4weeks duration LBP with no sciatica or radicular pain	Compares nimesulide (Cox-2 selective anti-inflammatory) with ibuprofen	Significant improvement in Oswestry score at 10 days for Cox 2 group (10.0 for nimesulide and 16.5 for ibuprofen — $p < 0.026$). There was no significant difference in pain relief between groups	Adequate allocation concealment = Y Blinding = Y ITT analysis = Y LTFU < 15% = Y Bias = Low	Excluded LBP > 4 weeks duration, sciatica or radiating pain, pregnancy. Reviewer got different values than author on *t*-test analysis

ACUTE LOW BACK PAIN — Table of Included Studies (Interventions) continued

STUDY	METHODS	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY	NOTES
Rozenberg 2002	RCT — randomised to normal activity or 4 days bed rest	278 multi-centre ambulatory patients. Bedrest = 137; activity = 140	4 days bed rest vs. normal activity (as much activity as pain would allow). All received analgesics and muscle relaxants	No difference in pain intensity functional disability and vertebral stiffness between groups at all outcome points (1 week, 1 month and 3 months). Prescription for bed rest should be limited when the physical demands of the job are similar to those for daily life activities	Adequate allocation concealment = Y Blinding = N ITT analysis = Y LTFU < 15% = Y (2.1%) Bias = Moderate	Blinding not done. Physician-investigator conducting outcomes measurement. Groups not equivalent at baseline — group on bed rest had significantly shorter duration of LBP. f/u = 3/12
Stam 2001	RCT	161 patients in 19 GP practices in UK. SRL = 83; CCC = 78. M = 87; F = 74.	Spiroflar (SRL) homeopathic gel vs. cremol capsici (CCC)	No significant difference in VAS pain after 1 week	Adequate allocation concealment = U Blinding = data collector ITT analysis = Y LTFU < 15% = Y Bias = Low	Participants not blinded. No placebo or control. Equivalent at baseline. f/u = 1/52
van Tulder 2002f	Cochrane Review	Mixed settings, hospital, outpatient and general practice	Oral NSAIDS vs. placebo; NSAIDs vs. paracetamol/acetaminophen; NSAIDs vs. narcotic analgesics/muscle relaxants; NSAIDs vs. other non-drug therapies; NSAIDs vs. (NSAIDs plus muscle relaxants); NSAID vs. (NSAID plus Vit B). NSAIDs vs. placebo, 9/11 included trials analysed separately for acute LBP. A quantitative analysis of placebo-controlled trials on acute LBP was performed	There is no difference in pain intensity between NSAIDs and placebo (3/9 trials but significant heterogeneity $p < 0.001$) but increased global improvement (RR = 1.24 95%CI 1.10, 1.41 6/9 trials) and reduced need for additional analgesia (RR = 1.29 95%CI 1.05, 1.57 3/9 trials). There is conflicting evidence that NSAIDs more effective than paracetamol; Moderate evidence that NSAIDs no more effective than other drugs, physio or spinal manipulation; Moderate evidence that muscle relaxants to not provide additional benefits to NSAIDs; Conflicting evidence about additional benefit of Vit B with NSAIDs	Number of RCTs = 51 (total); 9 ALBP specific trials of NSAID vs placebo analysed separately Quality assessment = Y Data pooled = Y Heterogeneity = N	Includes trials where non-specific back pain may be associated with sciatica mixed chronic/acute LBP. Acute LBP analysed separately
van Tulder 2000	Exercise therapy for LBP. Systematic review	10 trials on acute LBP included	Eight different exercise therapies included compared with numerous controls (other conservative treatment, usual care, manual therapy, back school and NSAIDs)	Strong evidence that exercise therapy is not more effective for acute LBP than other active treatments, inactive treatments or placebo. However, 8 different types of exercise therapy were combined when making this conclusion	Number of RCTs = 10 Quality assessment = Y Data pooled = Y Heterogeneity = Y Due to heterogeneity, difficult to interpret and apply results	Included acute and chronic and radiating pain. Problems with pooling different types of exercise therapies; care needs to be taken with interpretation. Heterogeneity likely to be significant
van Tulder 2002a	Cochrane Review of acupuncture				Number of RCTs = 11 (only one met the criteria for this update) Quality assessment = Y Data pooled = N Heterogeneity = Y	Mix of acute and subacute (12 weeks or less) and chronic with results pooled; poor quality studies to review
van Tulder 2002d	Cochrane Review of exercise therapy for low back pain			The evidence summarised in this systematic review does not indicate that specific exercises are effective for the treatment of acute low back pain	Number of RCTs = 39 (12 studies on acute low back pain; only 1 RCT met the criteria for this update Quality assessment = Y Data pooled = N Heterogeneity = Y	

ACUTE LOW BACK PAIN — Table of Included Studies (Interventions) continued

STUDY	METHODS	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY	NOTES
van Tulder 2002c	Cochrane Review of lumbar supports				Number of RCTs = 6 (studies on use of lumbar support for treatment) Quality assessment = Y Data pooled = N Heterogeneity = Y	
van Tulder 2002b	Back schools for non-specific LBP. Cochrane Review	2 trials (*n* = 217, *n* = 100)	Two trials included (*n* = 217, *n* = 100). Results not pooled. Back school = education and skills program including supervised exercises	Limited evidence for effectiveness of back schools for acute LBP, reduced pain, reduced recurrence and reduced sick leave. One high quality study compared one back school session to McKenzie exercises and reported better short and long term outcomes for McKenzie. Other study (low quality ranking from reviewers) showed no differences between back schools and physiotherapy	Number of RCTs = 2/15 were LBP specific Quality assessment = Y (blinded) Data pooled = N Heterogeneity = N	Mixed populations with radiating pain, or unspecified. Acute LBP analysed separately. Question feasibility, cost

ACUTE LOW BACK PAIN — Table of Excluded Studies (Interventions)

STUDY	REASON
Anonymous 2001a	This is an editorial only. Not a systematic review
Anonymous 2001b	Is for chronic low back pain and is only an editorial. Editorial comment only
Blomberg 1994	Did not meet inclusion criteria due to inclusion of patients with leg pain and herniated discs
Brockow 2001	Majority of patients back pain > 3 months (i.e. 137/140). Only 34/140 had LBP. Others had neck, or neck plus back pain
Cardon 2002	Involved children and preventive interventions. Primary prevention. Not an RCT
Chrubasik 2001	59% of patients had LBP > 3 months and results not presented separately for those < 3 months. Lack of definition re: 6 months susceptibility
Cooperstein 2001	Not LBP intervention. Description of chiropractic techniques. Describes manipulative techniques
Del Mar 2001	Systematic review of reviews — only one study included on LBP (1996), so should already be included. This is a one-page summary of a review, which is not specific to Acute NSLBP
Drezner 2001	Narrative Review. Not a systematic review. Not specific to acute LBP
Ernst 2000	Not a systematic review. A literature review on the effects of manipulation
Ernst 2001	None of included RCT were ALBP
Fitz-Ritson 2001	Narrative review
Frank 2001	Narrative review
Garfin 2002	Not a systematic review. Lit review of exercise therapy
Glomsrod 2001	Preventive strategy
Haigh 2001	Not a systematic review. Not reviewing acute non-specific LBP
Hawk 1999	Not acute LBP. Not a RCT
Hemmila 2002	Chronic LBP (> 7 weeks); patients with pain < 3 months not reported separately. Chronic lower back pain
Horneij 2001	Not acute LBP. Prevention study
Hurwitz 2002	Mostly chronic LBP. Duration of pain greater than 3 months in most participants. Majority (60%) had sciatica
Jacobson 2002	Not an RCT (before and after study). Chronic LBP
Johanning 2000	No methods, inclusion criteria included — narrative review. Not a systematic review
Kovacs 2002	Subjects have sub-acute or chronic LBP and data is not presented separately for pain < 3 months (our definition of acute LBP). Included patients with chronic back pain > 12 weeks and those with referred pain
Larsen 2002	Preventive strategy; orthoses
Linton 2001	Preventive strategy involves both back and neck pain and does not meet criteria for inclusion
Linton 2001	Addressing primary prevention

ACUTE LOW BACK PAIN — Table of Excluded Studies (Interventions) continued

STUDY	REASON
Loisel 2001	Study is for subacute LBP (> 6 weeks) and data not presented separately for acute LBP (< 3 months by our definition). Not an RCT
Lorig 2002	Chronic LBP
Maier-Riehle 2001	Chronic LBP (mean 7.0 years)
Newswanger 2000	Letter to the editor; not primary research
Newton-John 2001	Not an RCT. Describes a process
Onorato 2001	Subacute/chronic LBP
Pentinnen 2002	Chronic LBP
Quittan 2002	Narrative review. Not a systematic review, lit review
Ramey 2001	Review not systematic — not only LBP — looks at acupuncture for many conditions
Rossignol 2000	Likely to have substantial bias. More of an implementation study
Schreiber 2001	Participants only included if pain > 3 months duration; included neck and LBP
Sheppard 2001	Review of RCT only (chronic pain). Critical appraisal and is CLBP
Smith 2002	Narrative Review. Not a systematic review. Not specific to acute LBP
Strand 2001	Mean duration of back pain in participants is 10 years (+ 10 years)
Susman 2001	This is an editorial only. Not RCT
van Dieen 2001	Lit review — not RCT — narrative — healthy subjects
van Tulder 2002e	Addresses behavioural treatment for chronic LBP only
Veneema 2000	Does not distinguish acute LBP from chronic
Von Korff 2001	Not an RCT. Describes a process
Yassi 2001	Addressing primary prevention and is not a study of acute LBP. Back pain neck pain (summary of a review therefore exclude)

ACUTE LOW BACK PAIN — Table of Included Studies (Cost Effectiveness)

AUTHOR	TITLE	COMMENTS
Malmivaara 1995	The treatment of low back pain: bed rest, exercises, or ordinary activity?	RCT comparing group 1: rapid mobilisation with back extension exercises (*n* = 52) vs Group 2: usual activities and avoiding bed rest (*n* = 67) vs Group 3: bed rest group (*n* = 67) *f/u at 3/52, 12/52 *LTFU 11% *no ITT analysis; no one blinded; good allocation concealment *measured costs of treatment and indirect costs with time absent from work and cost of home help *outcomes: at 12/52 median sick days in Group 1 = 5; Group 2 = 4; Group 3 = 6 *at 12 weeks all were back at work — this is generalisable to general practice *total cost (USD, 1992): Group 1 = 397.00; Group 2 = 168.00; Group 3: 234.00 *author conclusion: cost-benefit analysis that Group 2 would be most economical *cochrane reviewer conclusion: small sample with high LTFU and self-reported outcomes
Cherkin1998	A comparison of physical therapy, chiropractic manipulation, and provision of an educational booklet for the treatment of patients with lower back pain	RCT Group 1: educational booklet *n* = 66; Group 2: chiropractic *n* = 122; Group 3: McKenzie method *n* = 133 *2 year f/u; interviewer blinded to subject assignment up to 4 weeks * ITT analysis: Group 2 had < severe pain at 4 weeks than Group 1 (*p* = 0.02); trend for less severe symptoms in Group 3 (*p* = 0.06) *greater dysfunction in booklet group at 1 year but not marked and no difference in other outcomes measured *mean cost of episode of care (USD, 1995): Group 1 = 153.00; Group 2 = 429.00; Group 3 = 437.00 *conclusion: given the limited benefits and high cost it seems unwise to refer all patients for McKenzie therapy or chiropractic *could not identify predictive factors that would identify responders/good outcome *no incremental cost ratios done
Moffett 1999	Randomised controlled trial of exercise for low back pain: clinical outcomes, costs, and preferences	RCT; *n* = 187 *Group 1: received community based exercise program (*n* = 89) vs Group 2: usual primary care management which may/may not involve referral to physiotherapist (*n* = 98) *looked at direct medical cost to the NHS and included indirect costs (lost work days) and at clinically important improvement *f/u at 6/52, 6/12, 12/12 *at 12/12 group 1: 64% (54–74) had clinically important improvement (no raw data shown) * Group 2: had 35% (25–45) clinically important improvement *RR 0.55 NNT 3.4 — only 5 people needed to do the exercise program for one more person to have clinically important improvement *exercise program more clinically effective and more cost effective: cost of care Group 1: 360.15 (*SD* 582.77) UK pounds (1996) vs. Group 2: 508.43 (*SD* 1108.79) UK pounds (1996) *cost of days off work: Group 1: 378 days off in 12/12 (cost = 17,010) vs group 2: 607 days off work in 12/12 (cost = 27,315)

ACUTE LOW BACK PAIN — Table of Excluded Studies (Cost Effectiveness)

STUDY	TITLE	COMMENTS
Carey 1995	The outcomes and costs of care for acute low back pain among patients seen by primary care clinicians, chiropractors, and orthopaedic surgeons	Observational study only — not a CEA — not an RCT — patients had significantly different demographic and prognostic characteristics — age, ethnic, income
Mitchell 1994	Effectiveness and cost-effectiveness of employer-issued back belts in areas of high risk for back injury	Prevention study — not an RCT — lots of confounding not continued for heavyweights and previous injury had to wear belts
Sinclair 1997	The effectiveness of an early active intervention programme for workers with soft-tissue injuries: the early claimant cohort study	Don't know duration of back pain; cohort study not RCT
Skargren 1998	1-year follow-up comparison of the cost and effectiveness of chiropractic and physiotherapy as primary management for back pain: subgroup analysis, recurrence and additional health care utilisation	Neck and back included together — don't know if they adjusted for these as subgroups
Skargren 1997	Cost and effectiveness analysis of chiropractic and physiotherapy treatment for low back and neck pain: 6 month follow-up	Have neck and back included; cannot tell if have done subgroups and cannot determine duration of pain

ACUTE THORACIC PAIN — Table of Included Studies (Diagnosis)

STUDY	METHOD	PARTICIPANTS	RESULTS	REFERENCE STANDARD	QUALITY SUMMARY
Bryner 1989	Dolorimeter applied to region of transverse processes of each thoracic vertabrae from T1 to T12 greater than 50 newtons regarded as normal if no flinching or pain reported from the subject	60 students divided into asymptomatic and symptomatic of pain in the thoracic spine region. Tenderness assessed using a dolorimeter. Pain of at least 3 weeks duration or no pain in previous 8 weeks. Setting: chiropractic teaching clinic	Tenderness can be assessed using a dolorimeter. No significant difference in levels of tenderness between the two groups at the mallelous control point. The mastoid point can not be considered a reliable control point. There are significant overall and individual differences in levels of tenderness between symptomatic and asymptomatic subjects. $p < 0.001$; X2 = 35.2	no	Selection bias minimised = U Measurement bias minimised = U Verification bias minimised = U Compared to reference standard = N Confounding avoided = U Bias rating = Low Marginally useful as a diagnostic test — specificity around 50%+ at most levels. Data has been reworked by KC. Data are aggregated for the test when it is really 12 tests/at each vertebral level. Low bias
Christensen 2002	3 palpation procedures for the detection of spinal biomechanic dysfunction in the upper 8 segments of the thoracic spine. A repeated measures design was used in all sub-studies. Measured levels of intra and interobserver agreement for tenderness on palpation of T1–8 with subjects with suspected angina	*N* = 107 subjects; 22 dropouts. 43 subjects 42 controls; 29 and 27 respectively were enrolled in the interobserver reliability protocol and 14 and 15 respectively participated in the intraobserver protocol. Setting: Dept of Nuclear Medicine, Denmark	Using a clinically acceptable definition of 'expanded agreement' as agreement to within one vertebral level achieved good kappa scores of 0.63–0.77 for intraobserver agreement and kappa scores of 0.67–0.70 for interobserver agreement	no	Selection bias minimised = U Measurement bias minimised = Blinded Verification bias minimised = Y Compared to reference standard = N Confounding avoided = Y Bias rating = Low Talks to chronic pain but demonstrates reasonable agreement for pain diagnosis. Low bias
Delamarter 1990	Discusses primary osseous neoplasm of the thoracic and lumbar spine. Case series of 29 adults and children with these rare tumors	*N* = 29 adults and children with primary osseous lesions between 1965 and 1982	Back pain was the most common complaint in 86% of the patients and neurologic symptoms or deficits were present in 55%. Neoplasms were predominantly benign in children, malignant in adults. Most common finding was generalised paravertebral muscle spasm with tenderness to palpation. Laminectomies and concomitant stabilisation procedures all resulted in stable spines in the 8 patients with malignant lesions	no	N/A
Fukui 1997	Investigated the patterns of pain induced by distending the cervical and lumbar zygapophyseal joints	*N* = 15 patients with back pain suspected to be of zygapophyseal origin	Pain distribution maps were drawn: pain in suprascapular region was referred from C7–T1 and T1–2; pain in the superior angle of the scapula from C7–T1 and T1–2; pain in the midscapular region from C7–T1, T1–2, T2–3; pain in paravertebral region from T11–12. The referred pain distribution for C7–T2–3 showed significant overlap	no	Selection bias minimised = N Measurement bias minimised = N Verification bias minimised = Y Compared to reference standard = N Confounding avoided = N Bias rating = High
Kleineman 1978	Describes the clinical course of metastatic cancer of the spinal column in a 2 year retro-spective study	*N* = 77 patients with documented metastatic cancer to the spinal column. Ages 30–85 (mean 62); M = 43%; F = 57% *30/77 had thoracic spine involvement	Average length of time from onset of symptoms to diagnosis of metastatic disease was 4 months in patients without neurological disfunction. Intractable day and night pain was the most consistent clinical symptom in all cases, regardless of area of spine involved. Of the thoracic spine, 28/30 had evidence of metastases on plain films. 43% had interscapular and dorsal pain, 11% had girdle pain, 39% had signs of neurological deficit, 7% had left anterior anginal chest pain	no	N/A

ACUTE THORACIC PAIN — Table of Included Studies (Diagnosis) continued

STUDY	METHOD	PARTICIPANTS	RESULTS	REFERENCE STANDARD	QUALITY SUMMARY
Ozaki Toshifumi 2002	Imaging survey. In all 22 a bone scan was done; 17 patients CT was done, in 10 patients who had surgery MRI was used for diagnosis of the tumor. CT-guided resection or coagulation is advisable for lesions that are not the spine. Radiotherapy should be reserved for patients in whom complete removal of the tumor is impossible and the tumor continues to grow	*N* = 22. Of the 22, 6 had tumors located in T-spine, 17 of the 22 had scoliosis. Average duration between onset of pain and surgery was 16.6 months in 1980's and 8.6 months in 1990's. Patient age range between 8 and 49 years. Average age 16.8 years with osteoid osteoma and 19.8 years with osteoblastoma. Of the 22, 13 had osteoblastoma; 9 were male and 4 were female. Of the 9 with osteoma, 4 were male and 5 were female. Some patients had neurologic symptoms. All patients had open resection of the lesion. Most patients presented with pain and spinal stiffness. Setting: Department of Orthopaedics, Germany	Series of 22 cases of spinal osteoid osteoma and osteoblastoma, of which 6 were in the thoracic spine (Ozaki 2002). Of these only 2 cases had pain for 3 months or less, 1 with an osteoid osteoma presenting with scoliosis and the other with osteoblastoma presenting with spinal palsy. Amongst the cases with longer durations of pain, scoliosis, spastic diplegia and leg pain were amongst the presenting symptoms. Although the sample was small, they suggest that painful scoliosis and long tract neurological signs in the lower limbs should raise the possibility of primary thoracic spinal malignancy	no	Selection bias minimised = U Measurement bias minimised = N/A Verification bias minimised = N/A Compared to reference standard = N/A Confounding avoided = N/A Bias rating = Moderate Osteoblastomas and osteoid osteomas of the spine are are bone-forming tumors. Osteoblastomas have more aggressive characteristics. *The curative approach is operative removal or deactivation of the nidus with coagulation. If the treament is not complete, pain persists and additional intervention is necessary
Patel 1991	Describes the events leading to #, features of acute pain, previous medical history, VAS in patients with osteoporotic vertebral fractures	*n* = 30 patients with acute painful vertebral compression # seen within 14 days of the event. F = 26; M = 4. Women had early menopause (mean age 44; 30–54 yrs)	Fractures occurred between the fourth thoracic and fifth lumbar vertebra; there was a predilection for T8, T12, L1 and L4. Commonest complaint was spontaneous acute back pain; the majority had pain localised to the site of fracture. Local percussion increased pain in all patients but did not necessarily accurately locate the site of #. Some patients c/o nausea, chest pain and abdominal pain	Xray with bone scanning to verify presence of recent fracture	Selection bias minimised = Y Prevalence of a range of symptoms and signs Measurement bias minimised = N Verification bias minimised = N/A Compared to reference standard = Y Confounding avoided = U Bias rating = Moderate The study had a bias toward those vertebral # causing severe pain

ACUTE THORACIC PAIN — Table of Excluded Studies (Diagnosis)

STUDY	REASON
Comeax 2001	Examination of normal subjects without pain. Not relevant in primary care. Based on osteopathic palpatory skills. Study on normal subjects without pain — not been reviewed
Haneline 2000	Referred pain from thoracic spine. Does not meet inclusion criteria
Kandabarow 1997	Expert opinion, not primary research
Keating 2001	Investigates non-symptomatic subjects
LaBan 1994	Does not meet inclusion criteria. Deals with chest wall pain arising from fractures of the ribs and sternum
Mitra 1996	Chronic thoracic spinal pain
O'Neill 1999	Does not meet selection criteria
Quast 1987	Not primary research and not a systematic review; unsuitable references, too old for possible red flag value. Narrative review and 2 case studies
Raine 1994	Subject had dorsal spine pain. Pain excluded in entry criteria
Sward 1991	Dissection of pain not specified. Convenience samples for both cases and controls lead to a high chance of selection bias
Triano 1999	Case report from a non-refereed journal with uncontrolled blocks for confirmation of diagnosis. Describes pain referred from the thoracic spine. Not relevant in primary care
Wise 1992	Does not meet inclusion criteria
Wood 1999	Chronic pain
Yelland 2002	Does not meet criteria

ACUTE THORACIC PAIN — Table of Excluded Studies (Prognosis)

STUDY	REASON
Cockerill 2000	Cross sectional study with high risk or bias. Based on survey but pain distinction not specified
Evans 2000	Can't be used for prognosis — it's a cross-sectional survey. Pain > 12 weeks — representative of primary care. Not relevant
Harreby 1995	Study examines risk factors for low back pain. This study is about the progression of risk factors for LBP. Based on children not adults
Hudak 1998	Review of criteria for assessing studies of prognosis of soft tissue musculoskeletal disorders in general — not specific to thoracic spine
Musgrave 2001	Study of risk factors. Doesn't distinguish thoracic pain from other back pain
Norlander 1997	Neck and shoulder pain not thoracic spinal pain. Not relevant in primary care
Norlander 1996	Neck and shoulder pain not thoracic spinal pain. Not relevant in primary care
Vanichkachorn 2000	This is a narrative review article by two authors. May provide a useful perspective
Wedderkopp 2001	Not an adult population. Study of back pain in children and adolescents only

ACUTE THORACIC PAIN — Table of Included Studies (Interventions)

STUDY	METHODS	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY	NOTES
Schiller 2001	RCT	*N* = 30. 15 to control group and 15 to intervention	Spinal manipulation versus non-functional ultrasound	Outcomes measured on Oswestry, NRS, McGill Pain Questionnaire. SM has greater benefits than ultrasound: $p < .025$ between the SM group and the placebo group for % of pain, right lateral flexion and left lateral flexion after the treatment period. The result was maintained at 1-month follow-up	Adequate allocation concealment = U Blinding = N ITT analysis = analysis by treatment received LTFU < 15% = U (? None) Bias = Moderate	Small sample size. Type II error. Pre-intervention pain duration not described. Groups equivalent at base-line. Follow-up for 4/52

ACUTE THORACIC PAIN — Table of Excluded Studies (Interventions)

STUDY	REASON
Giles 1999	Chronic pain. Part of the data relates to thoracic spine (for pain duration > 12 weeks) and compares three therapies
Haldeman S 1993	Does not meet criteria. Not T spine, not RCT, not pain duration < 12 weeks

ACUTE NECK PAIN — Table of Included Studies (Diagnosis)

STUDY	METHOD	PARTICIPANTS	RESULTS	QUALITY SUMMARY	NOTES
Gordon 2002	Randomised population based study to determine frequency, duration and prevalence of waking C-spine pain and stiffness by gender and age		Incidence of 18% cervical spine pain on awakening — more frequent among females and decline in pain with age; males = peak prevalence in 40–59 year group	Selection bias minimised = Y Measurement bias minimised = Y Verification bias minimised = Y Compared to reference standard = N/A Confounding avoided = Y Bias rating = Moderate	Authors note that there is little validated advice re the mgt and prevention of people waking with such symptoms *Chronicity is not defined Telephone survey
Johnson 1997	Value of C-spine radiographs in pts presenting with vague, non-localising and non-specific neck pain		Not a cost effective method of diagnosing cause of cervical pain	Selection bias minimised = U Measurement bias minimised = U Verification bias minimised = U Compared to reference standard = U Confounding avoided = U Bias rating = Moderate	Include for information on the fact that millions of dollars are spent unnecessarily on c xrays *does not mention duration of pain
Mihmanli 2001	Case report of inflammation of vertebral bone associated with acute calcific tendonitis of the longus colli muscle			N/A	Case report not research study but useful for differential diagnosis
Muffoletto 2001	Case studies: hematogenous pyogenic facet joint infection of the subaxial cervical spine	Two cases reported		N/A	Case report not research study but useful for differential diagnosis; very rare condition. Use for additional information only.
Nolla 2002	Case series of pyogenic vertebral osteomyelitis (spontaneous) *back or neck pain reported in all cases with mean duration of 48 days + 40 days *pain was mechanical in 25% of patients	64 patients		N/A	Include for useful descriptive data on incidence of O/M in neck
Ring 1994	Case series (× 5) of acute calcific retropharyngeal tendonitis			Selection bias minimised = U Measurement bias minimised = Y Verification bias minimised = Y Compared to reference standard = Y Confounding avoided = N/A Bias rating = Low	Include as it provides descriptive data and reassuring data on an unusual differential diagnosis
Sandmark 1995	To assess sensitivity and specificity of 5 manual tests for pain provocation in the neck for use in epidemiological studies	Sample of 75 men randomly selected — asymptomatic and symptomatic for neck pain	Palpation of facet joints had highest sens (82%) and the upper limb tension test the highest specificity (94%) *tests of c-spine rotation and cervical flexion/extension were not sufficiently sensitive	Selection bias minimised = Y Measurement bias minimised = Y Verification bias minimised = Y Compared to reference standard = N Self report of neck pain was the crite-rion standard Confounding avoided = N/A Bias rating = Low	Could be used in the revised section on physical examination
Wang 1999	Flexion-extension xrays in acutely injured may not be useful due to the decreased CROM and there are reports of serious neurological injuries occur-ring with the use of such xrays			Selection bias minimised = Y Measurement bias minimised = N/A Verification bias minimised = test was criterion standard Compared to reference standard = N/A Confounding avoided = Y Bias rating = Low	Algorithm on page115–6 may be useful; suitable to base guidelines on why not to take Flexion-extension xrays

ACUTE NECK PAIN — Table of Excluded Studies (Diagnosis)

STUDY	REASON
Gordin 2001	Descriptive review of procedures for chronic neck pain
Bono 2000	Opinion piece. No raw data. Mostly chronic
Demure 2000	Does not present the results for neck separately from 'neck/shoulder'
Hudak 1998	Opinion piece. Not a study or a review of data. **Could be used, if wanted, for its scoring system for articles on prognosis
Jull 1988	Chronic neck pain
Jull 1997	Current pain — but chronicity history (chronic) not known
Kligman 2000	Case report. No evidence of causative link to neck pain. Could be incidental finding
Stolk-Hornsveld 1999	Abstract — headache — insufficient data provided
Suttner 2002	Chronic. Single case-report not a study
Walsh 2000	Descriptive study only. Chronicity not defined
Werneke 1999	Not relevant in primary care

ACUTE NECK PAIN — Table of Included Studies (Prognosis)

STUDY	METHOD	PARTICIPANTS	RESULTS	QUALITY SUMMARY	NOTES
Ariens 2001	3 year prospective cohort study to see whether work related job demands are risk factors for neck pain	Males and females from 34 companies in the NL. 1334 workers met the criteria but 26.8% loss to follow-up	Neck pain recorded at 1 and 3 years; work factors assessed at baseline. Low co-worker support was found to be predictive of neck pain RR = 2.4 (1.1–5.3) and also high job demands RR = 2.1(1.3–3.6)	Selection bias minimised = Y Adjusted for confounders = Y Measurement bias minimised = Y Drop out rate = 26.8% authors note this could have biased the results of the study Bias rating = Low-moderate	The ascertainment of neck pain was unreliable. Authors note number of sources of bias including the healthy worker effect and not adjusting for prior neck pain experience
Borchgrevink 1997	Prospective cohort study of the relationship between personality and psych symptoms and long lasting physical symptoms were assessed in 88 people with neck sprain in MVA. Patients completed the MCMI-1 at intake and then 6/12 later. Assessed neck pain, neck stiffness and headache, taking prev symptoms into account	88 (41M/47F) pts > MVA recruited from ER if MRI done in 4 days of injury *nearly all covered by insurance *Norway, university hospital	Follow-up of 1 year in total: at 5 clinical consultations during first 6/12 and once at 12/12 following MVA *Results do not support the view that pre-morbid personality traits predict outcome for neck sprain pts	Selection bias minimised = Y Adjusted for confounders = Y Measurement bias minimised = Y Drop out rate = 11% Bias rating = Low	Concern re limited info about the way in which the reference standard was determined and whether assessors had access to the baseline results, including the psych profile assessment. Small numbers
Borghouts 1998	Systematic review of observational studies and RCTs to identify the clinical course and prognosis of non-specific neck pain. Prognosis determined by overall % of recovery		A higher severity of pain and a previous history of neck pain seem to be associated with a worse prognosis. Appears to be no association between localisation and a worse outcome and some indication of no association between xray findings of degenerative discs/joints and worse prognosis. There is not much data on the course of acute neck pain — more so for pts with pain > 6 months	Number of RCTs = 23 Quality assessment = Y Data pooled = N Heterogeneity = Y	
Brison 2000	Prospective f/u study of factors assoc with development of WAD > rear end collisions. Aim to describe the natural course of symptoms among patients from acute stages of injury — 2 years post injury	*n* = 352 mostly female of people presenting to ER post rear end collision; minimal data 'noise' due to low rate of litigation/compensation (Canadian setting)	61% experienced WAD post-collision with decline to 37% at 3/12	Selection bias minimised = Y Adjusted for confounders = N Measurement bias minimised = Y Drop out rate = 6 months = 5%; 12 months = 30%; 24 months = 64% Bias rating = Low	
Fredriksson 2002	Prospective case control study (part of a pop'n health survey) looks at the influence of exposure time to different work environmental conditions and the incidence of neck and shoulder pain		Associations between several exposures in the work environment and seeking care for neck or shoulder pain were found *induction period for neck or shoulder pain may be short	Selection bias minimised = Y Adjusted for confounders = Y Measurement bias minimised = Y Drop out rate = U Bias rating = Low	Worth considering if we are looking at aetiology of neck pain even though pain was of greater duration than 3 months
Karlsborg 1997	Prospective study of 39 patients with acute whiplash injury *f/u at 14 days, 1 month and 7 months post-injury; MRI of brain and cervical spine done at 1/12 post-injury and 6/12 if abnormalities were found	Age range 22–59 years *included if acute whiplash injury within past 14 days *n = 43 consecutive patients; 39 participated in study *38/39 had been in rear end collisions *neck pain present in all patients	Total recovery rate (asymptomatic) was 29% at 7/12 *correlation between MRI and clinical findings was poor *poor outcome was related more to stressful life events than clinical findings *85% resumed work within 1/12	Selection bias minimised = Y Adjusted for confounders = Y Measurement bias minimised = U Drop out rate = 0 Bias rating = Low	
Kasch 2001	Observational study looking at people with acute whiplash injury to assess sensitivity and specificity of 5 possible predictors of handicap following injury; looking at risk of developing chronic whiplash syndrome	*n* = 141 recruited over 1 year in Denmark *involved in rear end collision and seen within 2–4 days of injury in ER *excluded those with chronic neck pain	Initial pain intensity did not predict long term handicap after injury, nor did the type of therapy chosen by pt following injury *poor prognosis related to reduced CROM and high initial VAS score (sens and spec were 73% and 91%)	Selection bias minimised = Y Adjusted for confounders = U Confounders not formally searched for but do not appear present from the raw data Measurement bias minimised = Y Drop out rate = 6% at 12/12 Bias rating = Low	Discusses WAD only — not non-specific neck pain. Confounders not formally searched for but do not appear present from the raw data *measurement of CROM and determination of outcome made by same investigator

ACUTE NECK PAIN — Table of Excluded Studies (Prognosis)

STUDY	REASON
Hudak 1998	A perspective; not primary research or a review
Palmer 2001	Other neurological /pain presentations
Schrader 1996	Interpretation may exceed the comparative data; methodological limitations
Trinkoff 2002	Descriptive study of incidence in nurses. Not study of acute, non-specific cervical pain — chronically some > 6 months
Virani 2001	Survey of opinion only; not study of non-specific acute pain — chronic sufferers > 6 months

ACUTE NECK PAIN — Table of Included Studies (Interventions)

STUDY	METHODS	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY	NOTES
Bonk 2000	Prospective RCT of activity vs collar and the natural history of whiplash in Germany	Recruited from hospital ER within 3 days of accident. Active therapy *n* = 47; collar *n* = 50; control = approx 50	Group 1. active therapy (AT) 3 wks vs Group 2. collar (CT) 3 wks vs asymptomatic control group. All allowed to use ample analgesia	AT resulted in significantly faster recovery rate from acute whiplash injury at 3/52 compared to CT. At 6/52 there was no difference between AT and healthy controls and at 12/52 CT did not differ from healthy controls. AT posture advice and exercises had better outcomes than passive modalities. COST: AT 15/47 took time off work; CT 22/50 took time off work	Adequate allocation concealment = U Blinded = N ITT analysis = by treatment received LTFU < 15% = Y Bias = moderate AT and CT groups similar at baseline	Randomisation method not discussed. Unblinded treatment and unblinded f/u at 12/52. Matched controls
Gross 2002a	Cochrane systematic review on patient education for mechanical neck disorders	RCTs involving patients *n* = 271 with mechanical neck pain. Inception cohort was from primary care in all 3 studies		Conclusion was that patient education using individualised or group instructional strategies has not been shown to be beneficial in reducing pain for mechanical neck disorders	Systematic review = Y Number of RCTs = 3 Quality assessment = Y Data pooled = Y Heterogeneity = Y Applicability of results = Y	Two studies involved people with chronic neck pain; one with acute whiplash injury. Mix of poor and high quality trials. Mechanical neck disorders not the same as acute neck pain
Gross 2002b	Cochrane review to assess CCT and RCT of physical medicine modalities for mechanical neck disorders	13 trials included. Excluded patients with neurological deficit	Electromagnetic therapy, laser therapy, exercise, traction, acupuncture, heat/cold, electrotherapies, cervical orthoses and cognitive behavioural rehab strategies	Some support for EMT in reducing pain. Some data against the use of laser therapy: 3 articles using laser therapy did not differ from a placebo ($p = 0.20$) for 6–10 sessions of treatment. Little information available from trials to support the use of these modalities for mechanical neck pain	Systematic review = Y Number of studies= 13 Quality assessment = Y Data pooled = Y Heterogeneity = Y Applicability of results = Y	Unable to find separate analysis of acute neck pain and concerned over issues raised by Cochrane editorial committee re heterogeneity. The review covers too many modalities that probably cannot be combined; highlights the lack of data in this area. Trial quality good
Gross 2002c	Systematic review of manual therapy for mechanical neck disorders	20 RCTs		Multimodal care consisting of mobilisation/massage or manipulation and exercise alone or exercise in combination with thermal modalities and infrequent collar use appears more effective than application of these therapies alone, specific .physical medicine modalities and continous collar use with rest for pain reduction. Treatment advantages for pain ranged from 6% to 41%	Systematic review = Y Number of RCTs = 20 Quality assessment = Y Data pooled = N Heterogeneity = Y Applicability of results = Y	Search date December 1999
Hoving 2002	RCT with blinded outcome assessments	Patients with non-specific neck pain referred by GPs, age 18–70 years, pain or stiffness in the neck × 2 weeks or more, no treatment for neck pain in previous 6 months. 74% had neck pain of < 3 months	Compared manual therapy (MT), physical therapy (PT) and usual care (UC) by a GP. Intervention period = 6 weeks. Allowed co-interventions were restricted to analgesia and exercises at home	Data collected at 3 weeks and 7 weeks. At 7 weeks success rate twice as high for MT as for UC and higher for PT than for UC but not statistically significant. ITT analysis — adjusting for covariates including duration of neck pain did not greatly influence results. Patients receiving MT had less absence from work than PT or UC	Adequate allocation concealment = Y Blinded = outcome assessor ITT analysis = Y LTFU < 15% = Y Bias = Moderate	Mixed sample (acute and chronic). No separate analysis of acute and chronic neck pain. Contentious principal outcome measure
Karjalainen 2002	Multi-disciplinary biopsychosocial rehab for neck and shoulder pain among working age adults: a Cochrane Review				Systematic review = Y Number of RCTs = 2 (1 acute) Quality assessment = Y Data pooled = N Heterogeneity = N/A Applicability of results = Y	Include but must disregard study by Jensen which dealt only with chronic neck pain. The review highlights the lack of available data

ACUTE NECK PAIN — Table of Included Studies (Interventions) continued

STUDY	METHODS	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY	NOTES
McKinney 1989	Single blind prospective randomised trial to determine whether manipulative physiotherapy or patients ability to perform home exercise program influence reduction in pain and ROM	Receiving physio *n* = 66; receiving analgesia and a c- collar *n* = 33. Recruited from an ER in Belfast	Group 1: rest and advice to mobilise after 2 weeks; Group 2: active out-patient physiotherapy (hydrotherapy, short wave diathermy, application of heat and cold, traction, active and passive repetitive movements and education on posture and home exercises); Group 3: education on mobilisation and posture	Patients receiving out-patient physiotherapy and those receiving advice on home exercises Groups 2 and 3) had similar pain outcomes to each other at 1 and 2 months and had significantly less neck pain ($p < 0.01$) and greater cervical movement ($p < 0.01$) at 1 and 2 months vs patients receiving rest (Group 1)	Adequate allocation concealment = Y Blinded = Y single ITT analysis = N LTFU < 15% = N (> 20%) Bias = Moderate	
McLean 2001	RCT to determine the effect of 'microbreaks' on muscle activation behaviour, perceived discomfort and on worker productivity	*n* = 15 pain free at inception	Random assignment to three experimental groups (microbreaks at 20 minute intervals, 40 minute intervals, and at own discretion)	Regularly scheduled microbreaks reduced subjective discomfort ($p < 0.05$) in the neck and other areas of the body and has no detrimental effect on productivity. This was true particularly when breaks taken at 20 minute intervals	Adequate allocation concealment = N Blinded = N ITT analysis = N LTFU < 15% = Y Bias = Moderate	Although unblended, the results of the study are biologically plausible, and support a simple intervention that may be useful in reducing neck discomfort
Mealy 1986	RCT looking at early cervical passive mobilisation of acute whiplash injuries	*N* = 61(31 active trt and 30 matched controls). Recruited from ER, Dublin	Rest and initial cervical passive mobilisation with a soft collar vs early active cervical passive mobilisation	At 8 weeks a significant improvement in actively treated group in terms of CROM ($p < 0.05$) and pain intensity ($p < 0.0125$)	Adequate allocation concealment = Y Blinded = N/U blinding of data collectors not specifically stated ITT analysis = Y LTFU < 15% = N (16%) Bias = Moderate	Follow-up at 8/52
Peeters 2001	Systematic review (Cochrane-style) to assess efficacy of conservative treatment for whiplash (same as Verhagen 2002)			Because of heterogeneity of the 11 included studies, no statistical pooling was possible. The review indicates that active treatment showed a beneficial effect on pain. Concluded that active treatment better than passive (rest/immobility)	Systematic review = Y Number of RCTs = 11 Quality assessment = Y Data pooled = N Heterogeneity = Y Applicability of results = Y	No discussion of duration of pain. Pre-1998 publications
Provinciali 1996	RCT — single blind prospective trial looking at multi-modal treatment to prevent late whiplash syndrome	60 patients enrolled following rear end collision within past 60 days or less. Subjects were diagnosed with 'cervico-encephalic syndrome'. Subjects well-matched	Multi-modal treatment (MMT) consisting of postural training, manual technique, psychological support vs control treatment using physical agents such as electrical and sonic modalities (TENS and U/S)	Outcomes evaluated: CROM, pain, self rating of outcome and time interval between injury and RTW. Greater improvement in MMT group over control for all outcome measures except neck mobility. Pain intensity/VAS score significantly lower in treatment group at both 1 and 6 month f/u ($p < 0.001$)	Adequate allocation concealment = U Blinded = Y single (data collectors) ITT analysis = Y LTFU < 15% = Y Bias = Low	Unable to say specifically which intervention resulted in pain reduction. Follow-up at 1/12 and 6/12.Excluded patients with symptom exaggeration with the intention of enhancing financial rewards
Verhagen 2002	Cochrane Review assessing efficacy of conservative treatment for whiplash			Because of heterogeneity of the 11 included studies, no statistical pooling was possible. The review indicates that active treatment showed a beneficial effect on pain. Concluded that active treatment better than passive (rest/immobility)	Systematic review = Y Number of RCTs = 11 Quality assessment = Y Data pooled = N Heterogeneity = Y Applicability of results = Y	10 of the RCTs related to acute pain

ACUTE NECK PAIN — Table of Excluded Studies (Interventions)

STUDY	REASON
Brockow 2001	Majority had pain > 3 months — fails inclusion criteria. Chronic neck pain and back pain
Dabbs 1995	Narrative view, not experimental or systematic review; Not a scientific paper. A narrative of reviewed papers including back pain
Ernst 2002	Does not satisfy any inclusion criteria. SR-observational report citing case control studies. Prospective studies and a systematic review; a commentary only
Horneij 2001	Not acute neck pain; Outside scope of guidelines
Hoving 2001	Not a review of primary studies; criteria based appraisal of review articles on neck pain; not confined to acute neck pain. Good article for concordance
Ketola 2002	Could provide GPs with advice re ergonomic mgt in workplace; can't determine whether chronic or acute pain
Linton 2001	Not acute pain. Included back, neck or upper back pain. Also out of the scope of this project
Munchau 2001	Not neck pain — not an experimental study; not non-specific neck pain; inclusion criteria not fulfilled
Schreiber 2001	Chronic subjects in study
Skargren 1997	No separate analysis of acute (< 3 months) neck pain. Fails inclusion criteria. Neck and back pain not differentiated
Sterling 2001	Not acute pain; Neck pain > 3 months
Sterner 2001	A retrospective cohort study. Fails inclusion criteria; Chronic symptoms > 3 months
Wheeler 1998	Chronic neck pain
Wood 2001	Not acute neck pain; No control group — test two versions of application of cervical manipulation — predominantly chronic patients

ACUTE SHOULDER PAIN — Table of Included Studies (Diagnosis)

STUDY	METHOD	PARTICIPANTS	RESULTS	REFERENCE STANDARD	QUALITY SUMMARY	NOTES
Bennett 1998	Speeds test evaluated in a prospective study to determine sensitivity, specificity, PPV and NPV	*n* = 45; 31 male, 14 female *variety of diagnoses including acute and chronic	PPV = 23%; NPV = 83%; sensitivity = 90%; specificity = 13.8% *concluded that Speeds test is a nonspecific but sensitive test for macroscopic labral/biceps pathology	Speeds test versus arthroscopy	Selection bias minimised = Y All Pts between Oct 94–Feb 95 Measurement bias minimised = Y Gold Standard arthroscopy Verification bias minimised = Y Compared to reference standard = Y Confounding avoided = U Bias rating = Moderate	Duration of pain is unstated however this is not relevant in the case of diagnostic tests
Blanchard 1999a	RCT comparing MRI and arthrography for diagnostic impact in shoulder patients *f/u 10/12	Patients referred from a rheumatology clinic *29 shoulders randomised to MRI; 24 to arthrography	MRI and arthrography had similar therapeutic impact although MRI was associated with a significant shift towards surgical intervention		Selection bias minimised = U Measurement bias minimised = Y Verification bias minimised = U Compared to reference standard = Y Confounding avoided = U Bias rating = Moderate	Duration of pain is unstated however this is not relevant in the case of diagnostic tests
Fraenkel 2000	Improving the selective use of plain radiographs *descriptive study, not analytic *prospective f/u of patients for 3/12	Patients presenting to ER with shoulder pain, excluding 'red flag' conditions *206 had shoulder xray of which 88% were TU	Findings suggest that specific clinical criteria can identify patients who do not need xray as part of initial mgt of shoulder pain *of the 60% of patients presenting with shoulder pain, findings suggest that only 20% of these xrays revealed significant abnormalities (primarily #s or dislocations) *revealed only 1/135 in the low risk groups had a TI radiograph — this person had a lytic lesion and history of lymphoma *results suggest that patients without a precipitating fall, no swelling or deformity on exam might not need xray, and those with no pain or swelling but have sustained a fall may also be managed without xray if no pain at rest and normal ROM	Plain xray	Selection bias minimised = U Measurement bias minimised = Y Verification bias minimised = Y Compared to reference standard = Y Confounding avoided = U Bias rating = Low	Useful for better utilisation of xrays; pain duration unknown and half patients had no xray, however 88% TI and 66% TU had pain < 1 day; some patients had specific conditions and unknown pain duration so not a pure sample; low risk of bias
Green 1998a	Standardised protocol for measurement of ROM shoulder using plurimeter-V inclinometer and assessment of inter/intrarater reliability	Mean duration of symptoms = 16.5 months (9–24 months). 3 of the 6 patients had history of shoulder trauma; 4 had previous shoulder surgery	This study demonstrated that one or more active movements of the shoulder would probably fulfill the requirement of an outcome measure, with total shoulder flexion and abduction, external rotation in neutral, and hand behind back possibly the most reliable active movements of the shoulder		Selection bias minimised = Y Measurement bias minimised = Y Verification bias minimised = Y Compared to reference standard = Y Confounding avoided = Y Bias rating = Low	Subjects not acute; useful reference re the reliability of physical examination
Litaker 2000	Retrospective chart review looking at how well bedside history and physical exam predict arthrography results in older patients suspected of having rotator cuff tears	*n* = 448 consecutive patients with suspected RCTs	Weakness with external rotation; age > 65 years; and night pain were predictors of rotator cuff tears		Selection bias minimised = Y Prevalence noted obtusely Measurement bias minimised = Y Verification bias minimised = Y Compared to reference standard = Y Confounding avoided = Y Bias rating = Low	Mixed data; useful as a reference to the validity of medical history taking
MacDonald 2000	Looked at the diagnostic accuracy of Hawkins and Neer subacromial impingement signs				Selection bias minimised = Y Measurement bias minimised = Y Verification bias minimised = Y Compared to reference standard = Y Confounding avoided = U Bias rating = Low	Included as a reference to the validity of physical examination

ACUTE SHOULDER PAIN — Table of Included Studies (Diagnosis) continued

STUDY	METHOD	PARTICIPANTS	RESULTS	REFERENCE STANDARD	QUALITY SUMMARY	NOTES
Naredo 2002	Compared clinical diagnosis established by physical examination with high frequency ultrasonographic findings in patients with shoulder pain. All underwent exam and U/S was carried out 1 week later by a third examiner with no knowledge of the clinical findings	*n* = 31 with first flare of shoulder pain. All had clinical diagnosis of periarticular disorders. Excluded patients with chronic pain or previous trauma	Clinical assessment showed low accuracy in diagnosis of periarticular shoulder lesions through physical examination; U/S should be used when possible to aid diagnosis and treatment		Selection bias minimised = Y Measurement bias minimised = Y Verification bias minimised = U Compared to reference standard = Y Confounding avoided = U Bias rating = Moderate	Reference to validity of physical examination
Norregaard 2002	Diagnosing patients with long-standing shoulder joint pain — to examine the interobserver agreement of commonly used clinical tests and diagnoses in patients with shoulder pain *U/S exam was performed followed by clinical exam performed randomly by orthopod or rheumatologist who had no knowledge of pt history	86 consecutive patients with long-standing shoulder pain of at least 2, 3 and 6 months duration (mean symptom duration = 25 months *they either had steroid injection without lasting effect or this was not indicated	Accuracy of clinical tests and diagnoses in comparison with arthoscopic findings was low and only slightly better when the results of U/S became available in addition to clinical examination	Arthroscopic examination	Selection bias minimised = Y Measurement bias minimised = Y Verification bias minimised = Y Compared to reference standard = Y Confounding avoided = U Bias rating = Low	Although duration of pain is mixed, study included as a reference
Pal 2000	Clinical audit of practice in emergency department (QA exercise to determine how people with shoulder pain were managed)				N/A	Include as a reference to the difficulty in clinical diagnosis
Reinus 1998	Case series of patients with subtle greater tuberosity fractures who were sent for MRI because of possible rotator cuff tear	*n* = 6; age range 27–51, mean age 40.5 *5/6 reported direct trauma to their shoulders suggestive of #; 1/6 had shoulder pain after fracturing an ankle in a fall	Authors conclude that in these cases MRI detected subtle # of the greater tuberosity in people suspected of having rotator cuff tears and in whom no # was visible on plain xray		N/A	For use as reference to diagnosis of red flags
Szomor 2001	Differential expression of cytokines and nitric oxide synthase isoforms in rotator cuff bursae *subacromial bursal samples were collected at open surgery	*n* = 17; Sydney Australia	There was a consistent pattern of cytokine mRNA expression in the subacromial bursal samples *the study was unable to detect correlation between expression levels of cytokines or NOS isoforms and patient age, symptom duration and shoulder pain scores		Selection bias minimised = U Measurement bias minimised = Y Verification bias minimised = Y Compared to reference standard = Y Independent pathology tests Confounding avoided = Y Bias rating = Low	Reference to bursitis mechanism
Teefey 2000b	Study to identify differences in the sonographic appearance of acute and chronic full-thickness rotator cuff tears	*n* = 28 consecutive patients examined following arthoscopy to identify full thickness tears; 24 with acute RC tear and 20 with chronic reviewed for tear width, location and presence and distribution of fluid	A mid-substance location and presence of fluid was more commonly associated with acute tear; non-visualised cuff and absence of any fluid more commonly associated with chronic tear		Selection bias minimised = Y Measurement bias minimised = Y Verification bias minimised = Y Retrospectively & independently Compared to reference standard = Y Confounding avoided = Y Bias rating = Low	
Torstensen 1999	Looked at utility of MRI as a diagnostic tool in comparison with arthroscopy.				Selection bias minimised = N Measurement bias minimised = U Verification bias minimised = U Compared to reference standard = Y Confounding avoided = N Bias rating = Moderate to High	Pain duration not mentioned; a range of conditions were involved; useful for reference only

ACUTE SHOULDER PAIN — Table of Excluded Studies (Diagnosis)

STUDY	REASON
Bencardino 2000	Chronic shoulder pain. Not relevant in primary care
Binkert 2001	Exclude — not relevant to these guidelines; doesn't meet selection criteria
Blanchard 1999b	No pain data/small numbers
Bredella 1999	Patient group not relevant
Bunker 2000	May be useful as a pathological reference only
Carrillon 1999	High chronic pain — specific diagnosis
Clunie 1998	Chronic pain > 4/12
Coari 1999	Doesn't meet any inclusion criteria
Culic 2001	Myocardial infarction. Article does not deal with acute shoulder pain
Fermand 2000	Chronic medicinal pain
Gartsman 1998	Not relevant; looks at patient perceptions of shoulder conditions on general health status
Johnson 2001	Needs review
Kim 2001	Apparent selection bias therefore exclude
Law 1998	Case report of chronic pain (5 year duration)
Lee 2002	Chronic only
Murrell 2001	Does not meet criteria; data appears incomplete
Saxton 2000	Chronic — no shoulder specific data — review paper — no data
Scutellari 1998	Review paper — not acute
Struhl 2002	Chronic except one subject with 2/12 duration of symptoms. No differentiation between anterior and posterior impingment
Teefey 2000a	Chronic
Viikari-Juntura 2000	Irrelevant to these guidelines; mixed data on neck and shoulder
Worland 2000	Doesn't meet any inclusion criteria
Yanagisawa 2001	Chronic pain. Study related to the mechanics of pain (subacromial)

ACUTE SHOULDER PAIN — Table of Included Studies (Prognosis)

STUDY	METHOD	PARTICIPANTS	RESULTS	QUALITY SUMMARY	NOTES
MacFarlane 1998	Prospective cohort study over 3 years to determine the natural hx of shoulder symptoms in the general pop'n and whether long term outcome can be predicted on the basis of clinical (pain related) and individual (host related) factors	*n* = 92; cross-section of UK population	Baseline factors that were predictive of continuing symptoms in follow-up were s/s > 1 year duration; pain on day of initial exam; high score on General Health Questionnaire, seeing a GP re the pain; psychological stress	Selection bias minimised = Y Adjusted for confounders = Y Measurement bias minimised = Y Drop out rate = 32% Bias rating = Low-medium	Include because goes to natural history; useful as a reference only
Miranda 2001	Prospective study of work-related risk factors and physical exercise as predictors of shoulder pain among forestry workers *questionnaire administered to 7000 employees with 75% response rate	Subjects estimated the number of days they had pain in past 12 months and described what sort of activities they did	Conclude that shoulder pain the result of multiple factors; physical exercise appears to have more protective than impairing effects on the shoulders *1 year incidence of shoulder pain was 14% *awkward work postures, obesity, heavy physical work and mental stress were risk factors	Selection bias minimised = Y Adjusted for confounders = U Measurement bias minimised = U Drop out rate = U Bias rating = Moderate	Mixture of acute and chronic subjects *gives some idea of predictors of pain and factors that are associated with chronicity; useful as a reference only
Nahit 2001	Questionnaire given to newly employed workers to determine presence of shoulder etc pain present in past month lasting for > 1 day	1081 subjects recruited; 20% reported shoulder pain	High levels of stress were associated with increased likelihood of pain, particularly job demand and control	N/A	General point that psychosocial stress at work is associated with more pain and i ndicated need to assess this at first presentation although not specifically based on acute pain. Use for reference only
Punnett 2000	Cohort study conducted in auto-assembly plant to evaluate risk of shoulder disorders associated with non-neutral postures *used questionnaire to rate discomfort *10 month study period	Cases identified prospectively over 10 month period from workers reporting shoulder c/o *93 potential cases (89%) were interviewed and 79 (85%) of these met the study case definition	The risk increased as the proportion of the work cycle exposed increased and with use of hand-held tools	Selection bias minimised = Y Adjusted for confounders = U Measurement bias minimised = Y Drop out rate = 1 Bias rating = Low	
Solomon 2000	Examined medical records to determine whether treating physicians documented key history and exam findings to determine whether documentation linked with patient satisfaction and clinical outcome		There was no association between documentation and 3-month pain relief or functional status	N/A	Mixed data and prognostic value of history taking/examination. 50% had pain > 12 weeks *may be of use in the section on medical history; useful as a reference only
Solomon 2001	To examine the factors that influence referral of patients with musculoskeletal pain and whether referral influences outcome. Follow-up 12/12	41% (65/160) of patients presenting with knee or shoulder pain were referred. 29% were referred to an orthopod; 8% to a rheumatologist; 4% to both	Referral was not associated with improvement of pain or function and may be associated with worse outcomes among patients with shoulder pain *referral for shoulder c/o was associated with significantly less improvement in clinical outcomes than non-referral ($p = 0.02$)	N/A	Mixed data and prognostic value of referral. Not specifically acute pain; useful as a reference only
Tempelhof 1999	Looked at the prevalence of rotator cuff tears in asymptomatic shoulders using U/S	No previous history of shoulder pain or dysfunction		N/A	Talks to prevalence of tears in the general population; useful as a reference only
Tuite 1998	To determine the relative distribution of the locations of rotator cuff tears and sensitivity of of anterior versus posterior tears on MR images	*n* = 110 consecutive patients who had MR and full/partial/rim rent tear of cuff at arthroscopy	In patients less than 36 years old, most partial and small full-thickness tears are centred in the anterior half of the supraspinatus	Selection bias minimised = Y Adjusted for confounders = Y Measurement bias minimised = U Arthroscopic evaluation guided by MR findings: not independently Drop out rate = U Bias rating = Moderate	Likely to be a chronic population due to the diagnosis is made at arthroscopy; useful as a reference only

ACUTE SHOULDER PAIN — Table of Included Studies (Prognosis) continued

STUDY	METHOD	PARTICIPANTS	RESULTS	QUALITY SUMMARY	NOTES
van der Windt 1996	Shoulder disorders in general practice — what are the prognostic indicators of outcome *prospective f/u study (observational) by 11 Dutch GPs	*n* = 349 pts with new episodes of shoulder pain with f/u at 1, 3, 6, 12 months	At 1/12 23% showed complete recovery; at 3/12 44% fully recovered; at 12/12 59% fully recovered; 41% had persistent symptoms after 12/12	Selection bias minimised = Y Adjusted for confounders = Y Measurement bias minimised = Y Drop out rate = 4% Bias rating = Low	
Winters 1999a	Prospective study in general practice of the long term course of shoulder complaints *assessed diagnostic category and fluctuations in pain *all were given same treatment with NSAID during first 2 weeks of inclusion *after this, the GP prescribed therapies tailored to the patients	All patients with shoulder complaints presenting to 4 practices in the Netherlands over a 6 month period were followed for up to 18/12 **n* = 101	The character of the symptoms changed considerably in the first week *51% had recurrent complaints after 26/52 (assessed at visit) and 41% after 12–18 months (assessed via survey)	Selection bias minimised = Y Adjusted for confounders = U Measurement bias minimised = N Drop out rate = 7% Bias rating = Moderate	Included for information on long term course of shoulder pain; although has mixed data; useful as a reference only

ACUTE SHOULDER PAIN — Table of Excluded Studies (Prognosis)

STUDY	REASON
Albright 2001	Chronic pain
Berjano 1998	Not specifically acute pain. Duration of symptoms unknown. Not relevant in primary care. Looks at complication not diagnosis or prognosis. Study on implication
Brulin 2002	Follow-up study — issue of interest — pain — 5 years previously
Chiou 2002	As per all exclusion criteria. Not specifically acute pain patients
Feldman 2002	Can't separate local causes of shoulder pain from those referred
Fredriksson 1999	No baseline shoulder data. No specificity of data, diagnosis
Goh 1997	Retrospective. Duration of pain not specified
Griggs 2000	As per all exclusion criteria. Majority of patients chronic
Hirschhorn 2000	Not relevant patient group. Case report
Hudak 1998	No data. Review article
Johansson 1994	Diagnosis by questionnaire. Not specified for acute or chronic. 12-month period — no indication of site preparation
Kaergaard 2000	Data mixed — neck and shoulder.
Leppala 1998	Chronic capsulitis > 3/12. Chronic disease
Lowe 2001	Eight subjects recruited; 4/8 had musculoskeletal pain in the past 12/12; results do not differentiate for shoulder pain
Rahme 1998	Chronic patients > 1 year duration
Smith 2001	Chronic pain — association between two chronic conditions
Winters 1999b	50% of sample acute — 25% chronic — 25%

ACUTE SHOULDER PAIN — Table of Included Studies (Interventions)

STUDY	METHOD	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY
NSAIDS vs Placebo					
Adebajo 1990	Randomised, controlled trial	60 patients. Inclusion criteria: symptoms < 3 months and rotator cuff tendonitis defined by pain exacerbated by resisted movement, on abduction with a painful arc or external rotation; active range frequently limited by pain and passive range always > active range of movement; normal glenohumeral range of passive movement Exclusion criteria: Systemic inflammatory arthropathy; recent peptic ulceration or gastrointestinal bleeding or sensitivity to NSAID or triamcinolone; shoulder injection within previous 3 months; glenohumeral arthritis, acromioclavicular arthritis, bicipital tendonitis or a suspected rotator cuff tear (weak arm elevation, positive 'drop arm sign' or a high riding humerus seen radiologically); local infection. NSAIDs stopped at least 1 week before study entry	Group 1(20 patients): 50 mg diclofenac 3 times a day for 28 days + subacromial injection of 3ml of 0.5% lignocaine Group 2 (20 patients): diclofenac placebo tablets + subacromial injection of 2ml 0.5% lignocaine & 1ml of 80mg/ml triamcinolone hexacetomide Group 3 (20 patients): diclofenac placebo tablets + subacromial injection of 3ml 0.5% lignocaine All patients instructed in pendulum and wall climbing exercises to perform at home	**NSAID vs Placebo** At 4 weeks, significant difference between groups in pain favours NSAID (mean difference between groups –2.25 (95% CI –3.6, –0.9); significant difference between groups in range of abduction favours NSAID (mean difference between groups 41. degrees (95% CI 18.09, 64.71) **Injection vs Placebo** At 4 weeks, significant difference between groups favours injection (mean difference between groups 3.6 (95% CI 1.55, 5.65); significant difference between groups in range of abduction favours injection (mean difference between groups 45 degrees (95% CI 19.12, 70.88). **NSAID vs Injection** At 4 weeks, no significant difference between groups in pain (mean difference between groups –1.35 (95% CI –3.3, 0.6); no significant difference between groups in range of abduction (mean difference between groups –3.6 (95% CI –22.86, 15.66)	Adequate allocation concealment = Y Blinding = Y (participants and outcome assessors blinded) ITT analysis = Y LTFU < 15% = Y (No loss to follow-up) Bias rating = Low
Ginsberg 1991	Randomised double blind crossover trial	*n* = 30 (2 with epicondylitis; 28 with peri-arthritis of the shoulder)*	Group 1: 4% indomethacin spray (approx 5mg dose/spray) 3–5 times/day for 14 days, then placebo for 14 days * Group 2: placebo for 14 days, followed by 4% indomethacin spray for 14 days	At 14 days: significant difference between groups. Overall improvement in NSAID group 26/30 versus 18/30 in placebo group favours NSAID Two patients in the NSAID group reported minor cutaneous irritation with the spray	Adequate allocation concealment = U Blinding = Y ITT analysis = U LTFU < 15% = No mention of any patients lost to follow-up Bias rating = Low Trial sponsored by pharmaceutical company
Mena 1986	Randomised double blind placebo-controlled trial	*n* = 68 Inclusion criteria: Ages 14–70 years with acute bursitis or tendonitis of the shoulder and maximum duration of symptoms = 4 days	Group 1: Placebo NSAID plus physiotherapy Group 2: Flurbiprofen 200–300mg QID plus physiotherapy	Authors concluded that Flurbiprofen was well tolerated and effective for treatment of acute shoulder pain. There was a statistically significantly greater proportion of participants in the active group with improvement according to investigators' global assessments at all follow-up points (Day 1, 3 or 4, 7 and 14) and at day 7 according to patients' assessments (data not shown for patient assessment of overall improvement). There was a trend in a similar direction for other outcomes reported	Adequate allocation concealment = U Blinding = Y (double) ITT analysis = U (method of analysis not described) LTFU < 15% = Incomplete follow-up (patients who had not responded on day 3 were withdrawn from the study) Bias rating = Moderate

ACUTE SHOULDER PAIN — Table of Included Studies (Interventions) continued

STUDY	METHOD	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY
One NSAID Compared to Another					
Vidal 2001	Double blind randomised controlled trial	*n* = 599 Inclusion criteria: At least 18 years, shoulder pain of acute onset (within 3 days) of non-traumatic origin and > 50 mm on 100mm VAS; diagnosis of one-sided rotator cuff tendonitis (including reactive bursitis and subacromial attrition syndrome) according to stage I or early stage II of impingement syndrome or bicipital tendonitis; painless unrestricted motion of the affected joint immediately before the attack; no symptoms from previous episodes of acute shoulder pain in the last 6 months, symptoms requiring therapy with NSAIDs	Group 1: meloxicam 7.5mg once daily Group 2: meloxicam 15mg once daily Group 3: piroxicam 20mg once daily	Follow-up Day 3, 7 and 14. All treatment groups improved with respect to pain and shoulder score. There were no differences between groups for improvement in pain on active movement on Day 7 (primary endpoint) (Group 1: -47 (SD 25); Group 2: -46 (SD 25); Group 3:-43 (SD 26), p = 0.19)	Adequate allocation concealment = U Blinding = Y (participants and assessors) ITT analysis = Y LTFU < 15% = Y Bias rating = Moderate Groups equivalent at baseline. Sponsored by pharmaceutical company
Gotter 1987	Double blind, randomised controlled trial	*n* = 30 Participants with humeroscapular (acute inflammatory episode of > 4 days with no previous treatment). Duration of symptoms not presented	Group 1: tenoxicam Group 2: piroxicam Both groups received once daily oral doses for 14 days	Both groups improved rapidly with complete remission of symptoms at 14 days: 10/15 tenoxicam versus 6/14 patients treated with piroxicam, p > 0.05. There were no differences in outcome between two treatment groups	Adequate allocation concealment = U Blinding = Y (double) ITT analysis = U (method of analysis not described) LTFU < 15% = Y (One patient lost to follow-up on day 2 in piroxicam group) Bias rating: Moderate to high (likely open label study)
Soave 1982	Double blind randomised controlled trial	*n* = 40 adults with painful shoulder (24 patients) or some other form of soft-tissue rheumatic complaint (epicondylitis *n* = 10, tendonitis *n* = 5, olecranon bursitis *n* = 1), symptoms developing within previous 4 days; localised tenderness over involved area, limitation of motion, pain at rest severe enough to interfere with sleep and pain on motion. Acute shoulder pain in 24/40 patients	Group 1: indomethacin 100mg/day Group 2: indoprofen 800mg/day Both drugs were administered orally × 14 days Participants could seek physiotherapy but no other analgesic therapy	Significant improvement in pain, quality of sleep, range of active motion and patient's assessment found with both drugs, measured on days 48 and 15. No significant difference between the two groups. Results presented graphically One patient in indomethacin group discontinued treatment because of headache and dizziness. Three patients receiving indoprofen developed abdominal pain necessitating dose reduction	Adequate allocation concealment = U Blinding = Y (double blind) ITT analysis = completers only (39/40) LTFU < 15% = Y (0) Bias rating = Low Results for shoulder pain not presented separately
Wielandts 1979	Randomised double blind trial	*n* = 26 Acute tendonitis (25 shoulder, 1 elbow), Duration of symptoms: < 3 months = 23, 3–12 months = 3)	Group 1: 100mg phenylbutazone QID for 7 days Group 2: 100mg fentiazac QID for 7 days	Follow-up 1 week: Both groups improved over time with respect to pain and tenderness. There were no differences between groups	Adequate allocation concealment = U Blinding = Y (double) ITT analysis = completer analysis only LTFU < 15% = N (5/26 LTFU at 1/52, all in phenylbutazone group) Bias rating = Moderate Wyeth supplied the drug

ACUTE SHOULDER PAIN — Table of Included Studies (Interventions) continued

STUDY	METHOD	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY
Corticosteroid Injection					
Adebajo 1990	See NSAID section				
Vecchio 1993	Randomised controlled trial	*n* = 57 Inclusion criteria: clinically defined rotator cuff tendonitis (shoulder pain exacerbated by resistance in at least one of abduction, external or internal rotation, and normal passive motion) Duration of symptoms < 3 months Exclusion criteria included adhesive capsulitis, rotator cuff tears, biceps tendonitis, acromioclavicular arthritis, previous steroid injections into shoulders	Group 1 (28 patients): subacromial injection of 1% lignocaine, 1ml. Group 2 (29 patients): subacromial injection of 40mg methylprednisolone plus 1ml 1% lignocaine NSAIDs were discontinued 1 week prior to study	Authors conclude no significant difference in pain and active and passive ROM between groups. No reported means or standard deviations	Adequate allocation concealment = U Blinding = Y (participants and assessors) ITT analysis = completers analysis only LTFU < 15% = Y Bias rating = Moderate One patient from each group failed to complete 12 week assessment period
Corticosteroid Injection vs NSAID					
Adebajo 1990	See NSAID section				
White 1986	Randomised, controlled trial	*N* = 40 Inclusion criteria: 'Rotator cuff tendonitis' Painful arc between 40–120 degrees abduction, shoulder pain less than 12 weeks duration, no signs of acute calcific tendonitis, no evidence of a systemic inflammatory arthritis or frozen shoulder (defined as external rotation < 30 degrees, abduction < 90 degrees) Exclusion criteria: Active peptic ulcer disease, recent gastrointestinal bleed, contraindication to NSAIDs, evidence of symptomatic acromioclavicular arthritis or bicipitis tendonitis or major rotator cuff tear	Group 1 (20 patients): Subacromial injection (unguided) of 40mg triamcinalone acetonide plus placebo indomethacin tablets 4 × daily Group 2 (20 patients): 25mg indomethacin 4 × daily plus placebo (1cc saline) injection Repeat injection and refill of medication was given after 3 weeks, if necessary All patients were instructed to begin home exercise program of Codman pendulum exercise. 10–15 min twice daily and slow shoulder abduction exerceises using finger-up-the-wall technique	At 6 weeks: no significant difference between groups in pain: mean difference between groups 1.2 (95% CI -3.76, 6.16) At 4 weeks: no significant difference between groups in range of abduction: mean difference between groups -0.18 (95% CI –0.95, 0.58)	Adequate allocation concealment = U Blinding = Y participants and assessors ITT analysis = Y LTFU < 15% = N (25% — 5 patients in each group) Bias rating = Low Appropriate statistical analysis: Yes
Manual Therapy, TENS, Ultrasound, Exercises					
Conroy 1998	Randomised controlled trial	14 participants with primary shoulder impingement syndrome	Group 1: Shoulder joint mobilisation and comprehensive treatment (hot packs, active exercises, stretching, strengthening, soft tissue mobilisation, education) 3 times per week for 3 weeks Group 2: Comprehensive treatment alone	At 3 weeks significant difference between groups in pain: WMD -32.07 mm on VAS (95%CI -58.04, -6.10)-favours addition of mobilisation At 3 weeks no significant difference between groups in range of elevation: WMD –7.28 (-25.74, 11.8)	Adequate allocation concealment = No Blinding = Yes (Assessors only) ITT analysis = No LTFU < 15% = Yes Bias rating = Moderate Between-group comparisons = Yes; Point estimates & variability = Yes

ACUTE SHOULDER PAIN — Table of Included Studies (Interventions) continued

STUDY	METHOD	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY
Ebenbichler 1999	RCT design with random allocation to groups	54 participants (61 shoulders) Radiographically verified calcific tendonitis. Mild-mod pain for > 4 weeks or restricted ROM	Compared ultrasound therapy (15 mins, .89MHz, 2.5w cm^2, pulsed 1:4, transducer size 5 cm?) versus sham ultrasound with 24 treatment sessions (first 15 were daily then last 9 were 5 times weekly)	Assessed following treatment course and at 9 months Assessment of change from baseline in calcium deposits on radiography 100 point Constant score (pain, AROM, strength, ADLs), Pain (pain score and VAS and on abduction (4 point scale) 4. QOL 10cm VAS Following treatment significant difference between groups in perceived recovery: RR 1.81 (95%CI 1.26, 2.60) favours ultrasound Significant difference between groups in radiological appearance of calcific tendonitis in the short term (end of treatment) (RR 4.53 (1.46, 14.07)) and long term (9 month follow-up) (RR 3.74 (1.62, 8.66)) At 9 months no significant difference between groups for perceived recovery: RR 1.26 (95%CI 0.9, 1.77)	Adequate allocation concealment = Y Blinding = Y (assessors, subjects, therapists) ITT analysis = N LTFU < 15% = Y Bias rating = Low Baseline comparability: Yes; Between-group comparisons: Yes; Point estimates & variability: Yes
Shehab 2000	RCT design with random allocation to groups	50 female participants with painful shoulder movement of at least 1 month's duration. Diagnosis confirmed with provocative testing	Group 1:Transcutaneous Nerve Stimulation (TNS), 30 mins 50Hz_ to anterior and posterior shoulder 3–5 times a week for 13 sessions Group 2:US (0.5W for 10 mins, increased by 0.1W for each session) 3–5 times a week for 13 sessions Both groups had ice and stretching	Pain post intervention: Median (Range) TENS 0(0–.65) US 0.5(0–2.75). Significantly better in US group Flexion score post intervention: Median (Range) TENS 140 (120–160) US 175 (115–180). Significantly better in US group Abduction score post intervention: Median (Range) TENS 130 (116.7–156.5) US 180 (101.2–180). Significantly better in US group	Adequate allocation concealment = Y Blinding = Y (assessors only) ITT analysis = Y LTFU < 15% = N Bias rating = Low Baseline comparability: Yes; Between-group comparisons: Yes; Point estimates & variability: Yes
Acupuncture					
Kleinhenz 1999	RCT design with random allocation to groups	52 Athletes with rotator cuff disease, excluding rotator cuff tear on ultrasound. Inclusion criteria: rotator cuff disease due to sport; 18 — 50 years old; shoulder pain for > 4 weeks; no acupuncture therapy in past 6 months. Exclusion criteria: cervical or thoracic pain; previous surgery; rotator cuff tear; calcific tendonitis; arthritis	Group 1: Eight acupuncture sessions in 4 weeks Group 2: Identical regimen of placebo ultrasound	At 4 weeks significant difference between groups in Constant-Murley score (a composite score of pain, function and range of motion WMD 10.83 (2.46, 19.20) out of a possible 100 favours acupuncture At 4 months difference between groups WMD 3.53 (0.74, 6.32) — statistically significant but clinically unimportant benefit in favour of acupuncture	Adequate allocation concealment = Y Blinding = Y (assessors, therapists and subjects) ITT analysis = Y LTFU < 15% = Y Bias rating = Low Baseline comparability: Yes; Between-group comparisons: Yes; Point estimates and variability: Yes

ACUTE SHOULDER PAIN — Table of Excluded Studies (Interventions)

STUDY	REASON
Aaras 2001	Not shoulder-specific — shoulder data not presented separately; not acute; not randomised
Arslan 2001	Mean duration of symptoms greater than 3 months
Bain 2001	Trial of intra-operative pain relief. Pain > 6 months. Not relevant
Bang 2000	Mean duration of symptoms > 5 months
Barber 2001	Does not fit inclusion criteria
Berry 1980	Duration of symptoms greater than 3 months; used for mixed populations
Binder 1984b	Duration of symptoms greater than 3 months; used for mixed data
Binder 1986	Chronic pain
Blockey 1954	Frozen shoulder, periarthritis not defined; not acute (some acute patients but not reported separately)
Bottoni 2002	Fracture dislocation rather than acute (non-specific) shoulder pain
Brosseau 2002a	Exclude as relates to knee tendonitis only
Brox 1993/7	Duration of symptoms greater than 3 months
Bulgen 1984	Duration of symptoms greater than 3 months
Carter 2001	Case series
Ceccherelli 2001	Chronic pain
Chiou 2001	All chronic subjects
Dacre 1989	Duration of symptoms at least 4 weeks and periarthritis. Duration of symptoms not reported
Dahan 2000	Mean duration of pain is 1 year; inclusion criteria is at least 1 month of surgery
Dal Conte 1990	Duration of symptoms greater than 3 months
Downing 1986	Duration of symptoms greater than 3 months
England 1989	Duration of symptoms greater than 3 months
Fankhauser 2002	Not an RCT
Ferrante 1998	Results not separate for shoulder; subjects had pain duration > 6 months
Gam 1995	Exclude as includes many musculoskeletal conditions; not specific to shoulder
Ginn 1997	Duration of symptoms greater than 3 months (used for mixed data)
Green 2002	Mixture of acute and chronic pain duration (used to obtain data on mixed populations)
Haake 2002	Exclude due to chronic pain > 6 months

ACUTE SHOULDER PAIN — Table of Excluded Studies (Interventions) continued

STUDY	REASON
Horneij 2001	Heterogeneous group — prospective prevention trial
Invargarsson 1996	Surgery trial. Duration of symptoms unlikely to be less than 3 months
Johansson 2002	Not acute shoulder pain (based on references included). Not stated in paper but studies included in SR are on chronic patients
Jones 1999	Likely not to be acute but no duration given (2nd or 3rd stage capsulitis)
Karatas 2002	Duration of symptoms not defined except for > 4 weeks; likely to be > 12 weeks; duration of study f/u is 60 minutes
Karjalainen 2002	Greater than 3 weeks; chronic
Ketola 2002	Neck/shoulder pain. Topic not relevant
Kivimaki 2001	Pain duration > 12 weeks; no control (placebo); poor inclusion definition; inadequate manipulation
Klein 2002	Polio survivors therefore not representative of the general population presenting to primary practice; maximum duration of symptoms unclear
Leclaire 1999	Duration of symptoms greater than 3 months
Lee 1973	Duration of symptoms greater than 3 months
Lesprit 2001	Case series
Lindh 1993	Surgery trial. Duration of symptoms unlikely to be less than 3 months
Moore 1976	Mean duration of symptoms not reported
Nicholson 1985	Duration of symptoms greater than 3 months
Nykanen 1995	Duration of symptoms reported to be greater than 2 months; mean duration not reported but unlikely to be less than 3 months
Oldervoll 2001	Not a randomised trial; not acute duration; not just shoulder pain
Perron 1997	Duration of symptoms greater than 3 months
Price 2002	Post stroke
Reid 1996	Unlikely to be acute
Ritchie 1995	Not a RCT
Rizk 1991	Chronic pain (mean duration = 13.2 weeks)
Rompe 2001	Pain duration greater than 3 months (> 12 months)
Saunders 1995	Duration of symptoms greater than 3 months
Schmitt 2001	Chronic pain (> 6 months)
Shibata 2001	Chronic pain
Sileghem 1991	Chronic pain — definition of diagnosis unclear

ACUTE SHOULDER PAIN — Table of Excluded Studies (Interventions) continued

STUDY	REASON
Snels 2000a	Hemiplegic shoulder pain therefore exclude
Snels 2000b	Survey
Spangehl 2002	Exclude: chronic pain; mean duration > 12 months
Speed 2002	Not acute — useful for chronic
Sperber 2001	For post-traumatic instability
Sun 2001	Duration of pain > 3/12 based on mean duration of symptoms
Taverna 1990	Duration of symptoms greater than 3 months
van der Heijden 1996	3/16 RCTs included are acute; included studies poor quality; unable to pool results. The three studies were reviewed separately
van der Heijden 1997	Physiotherapy for soft tissue disorders
van der Windt 1998	Half the participants had pain for greater than three months (used for mixed data)
van der Windt 1995	Efficacy of NSAIDs for shoulder pain
van der Windt 1999	Not shoulder-specific
Waling 2002	Not acute — includes cervical spine
Walsh 2001	Case report
Wang 2001	No comparison group (case series). All had pain longer than 6 months
Weldon 2001	Not a systematic review or a RCT
Winters 1997b	Heterogeneous, variable treatments. Different diagnostic groups
Winters 1999a	Not acute
Withrington 1985	RCT lignocaine and steroid vs saline; outcomes pain and paracetamol count; no significant difference; exclude due to chronic pain
Zuinen 1993	Met inclusion criteria but not relevant question (i.e. comparing diclofenac and misoprostol to diclofenac alone. Concluded equal benefit with regard to shoulder symptoms, increased gastro-intestinal adverse events with additional misoprostol

ACUTE SHOULDER PAIN — Table of Excluded Studies (Cost Effectiveness)

STUDY	REASON
Bongers 2001	Exclude because not a cost effectiveness study; a narrative review

SUMMARY TABLES

ANTERIOR KNEE PAIN — Table of Included Studies (Diagnosis)

STUDY	TESTS	PARTICIPANTS	RESULTS	QUALITY SUMMARY	NOTES
Chapman-Jones 1998	Retrospective study (chart review) to investigate utility of MRI vs plain radiography for non-specific knee pain. All had plain film and then MRI and the corresponding results were compared	*n* = 103 patients with non-specific knee pain	Demonstrated that patients with non-specific knee pain having plain xray will have a high probability of a negative finding irrespective of any anatomical derangement present	N/A	Not acute pain only; only those who went on for MRI included. Those who did not have MRI (regardless of whether plain film was normal or abnormal) were excluded. The paper highlights the role of MRI but use of MRI is not commonly required in non-specific knee pain

ANTERIOR KNEE PAIN — Table of Excluded Studies (Diagnosis)

STUDY	REASON
Abrahams 2001	Personal opinion only
Baker 2000	Opinion only
Cesarelli 2000	Requires EMG; pain duration unknown
Davies 2000	Orthopaedic based sample; little detail regarding subjects; not relevant to primary care setting
Ferrari 1998	Not a systematic review. Clinical examination, diagnosis recommendation largely unreferenced. Useful MRI references
Fouts 1999	General overview — not a systematic review or primary research; < 7% of all knee injuries involve a clinically significant fracture
Fulkerson 2000	Not a systematic review. No recommendation of one diagnostic strategy over another
Fulkerson 2002	Orthopaedic review — not relevant in primary care. Not specifically acute pain. Not systematic or cross sectional. Opinion only
Grelsamer 2000	Individual opinion
Hayes 2001	Same as Petersen & Hayes 2000; chronic knee pain
Irrgang 2001	Validation of tool; no data on duration
Lee 2001	Mechanistic study on cadavers
Livingston 1999	Aetiologic; pain duration not reported

ANTERIOR KNEE PAIN — Table of Included Studies (Prognosis)

STUDY	METHOD	PARTICIPANTS	RESULTS	QUALITY SUMMARY	NOTES
Nimon 1998	Case series	Adolescent girls, mean age 15.5 yrs presenting to orthopaedic surgeons. Duration of acute knee pain unknown but likely to be longer term	Average follow-up 16 years (14–20): 73% improved; 27% same or worse. Suggests that pain will persist for many years but eventually the majority improve without any significant long-term problems	Not applicable (small series; no statement about length of symptoms at diagnosis)	Provides information on natural history

ANTERIOR KNEE PAIN — Table of Excluded Studies (Prognosis)

STUDY	REASON
Callaghan 2000a	No long-term follow-up; no data on prognosis
Kaeding 2001	Equated acute knee pain/patellofemoral pain with chondromalacia patella. General opinion based overview
Lam 2001	Mechanistic study; no follow-up therefore no prognostic data; chronic pain > 6/12
Petersen 2000	Chronic knee pain — duration, mean 33.9 months (*SD* > 52.1) median 7.7 months (range 0.4–234)
Sanchis-Alfonso 1998	Histological study of knee tissue; not relevant
Selfe 2001	Not relevant to these guidelines
Wolfe 2002	Not relevant in primary care — specific diagnosis of osteoarthritis. Symptom duration is > 2 weeks at first visit

ANTERIOR KNEE PAIN — Table of Included Studies (Interventions)

STUDY	METHODS	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY	NOTES
Arroll 1997	A critical review of clinical trials of non-operative therapy	5 studies included: *n* = 219. Age range 14.8–32.3; 38% female	Non-operative therapy was evaluated (shoe orthoses, knee sleeves, NSAIDs, injection therapy)	Five RCTs were located (two comparing injection therapies, one comparing wedged vs flat shoe insoles, one comparing knee sleeves and one comparing two NSAID agents)	Number of studies = 5 Quality assessment = Y Data pooled = N Heterogeneity = Y	An old review so relevant recent papers not included. Problem with lack of standardised outcome measures Search 1966–1995 of broader pain conditions than this guideline addresses
Brosseau 2002	Cochrane Review on therapeutic ultrasound for treating patellofemoral pain syndrome	*N* = 29	1. ice massage vs 2. ultrasoundplus ice massage	In group 2 there was 46% (6/13) with improved pain relief compared with 31%(4/13); the difference was not clinically or statistically significant. One study only; insufficient evidence (quality and quantity) for inclusion of ultrasound in evidence-based recommendations	Number of RCTs = 1 Quality assessment = Y Data pooled = N/A Heterogeneity = N/A Well-designed review but only had one study which scored poorly for methodological quality	Assessed side effects and effectiveness of ultrasound for patellofemoral pain *may be Type II error due to small sample size and cannot draw conclusions. One study only so insufficient evidence (quality and quantity) for inclusion of ultrasound in evidence based recommendations
Callaghan 2001b	Pilot study to compare two types of electrical stimulation of the quadriceps in the treatment of patellofemoral pain	*N* = 14 (M = 2; F = 12) patients with patellofemoral pain, quadriceps atrophy and normal gait parameters	Participants randomised to receive either sequential mixed frequency stimulation or simultaneous mixed frequency electrical stimulation of quadriceps	Main outcomes were related to muscle function not pain. Both groups improved; no difference between the two. Concluded that electrical stimulation of quadriceps may help	Adequate allocation concealment = U Blinding = U ITT analysis = N LTFU < 15% = no follow-up Bias = High Groups comparable at baseline	No mention of duration of pain so unclear how generalisable. Tertiary referral setting so likely to be more severe than general practice setting
Crossley 2001	Systematic review of physical interventions for patellofemoral pain	Studies: *N* = 456; Taping: *N* = 240; Foot orthoses: *N* = 20; patellofemoral orthoses/braces: *N* = 218 (*N* represents total number of participants, including control and treatment groups)		No strong evidence for one superior physical intervention. There is no data for physical interventions in treatment of patellofemoral pain however there was a trend of some treatment effect from education, stretching and quadriceps strengthening (including concentric exercises). 2 studies for taping found insufficient evidence for taping in treatment of patellofemoral pain but cannot discount effectiveness of this measure. 2 studies on patellofemoral orthoses. Reviewed studies on use of laser, acupuncture, sacroiliac joint manipulation and chiropractic patellofemoral joint mobilisation — more research is needed. Progressive resistance brace or corrective foot orthoses also looked at and more studies needed — poor quality of existing studies	RCTs = 16 Quality assessed = Y Data pooled = N Heterogeneity = Y (narrative summary of various interventions)	Well designed review. All studies in the review except one involved > 12 week duration of pain; other interventions involved mixed acute/chronic populations, but were predominantly chronic

ANTERIOR KNEE PAIN — Table of Included Studies (Interventions) continued

STUDY	METHODS	PARTICIPANTS	INTERVENTIONS	RESULTS	QUALITY SUMMARY	NOTES
Crossley 2002	Randomised controlled trial	*N* = 71 with non-specific patellofemoral pain of 1 month or longer and no other specific pathologic condition. Participants were aged 40 or younger to reduce chance of degenerative joint disease as cause of pain	Group 1 *n* = 33 received physical therapy (quadriceps muscle retraining, patellofemoral joint mobilisation, patella taping and daily home exercises); Group 2 *n* = 34 received placebo (sham ultrasound, light application of nontherapeutic gel, placebo taping)	Physical therapy group demonstrated significantly greater reduction in scores for average, worst pain and disability than placebo group after 6 weeks of physical therapy; better outcomes seen in younger participants. Physical therapy appears effective in reducing short-term pain and disability in patellofemoral pain	Adequate allocation concealment = Y Blinding = double blind ITT analysis = Y LTFU < 15% = Y (6%) Bias = Low Groups similar at baseline	Long-term follow-up of both groups was not possible as placebo group offered physical therapy intervention at completion of 6 week trial Those receiving physical therapy were followed up at 3 months and their pain had continued to abate
D'hondt 2002	Cochrane Review on orthotic devices for treating patellofemoral pain syndrome	5 trials involving *n* = 362	Evidence from RCTs too limited to draw definitive conclusions about the use of knee and foot orthotics for the treatment of patellofemoral pain. Compared McConnell's taping and modified Couman's bandage, the Protonics orthosis and the Palumbo dynamic patellar brace and the Cho-pat strap	Inconclusive; no special recommendation for practice are made. Only 1 trial was considered high quality which compared McConnell to Couman's and there was a trend to favour McConnell regimen but the study only involved 18 subjects. Palumbo vs Cho-pat showed no difference *n* = 59. Protonics vs no trt *n* = 100 showed all outcomes significantly favouring the Protonics orthosis. 1. home exercise vs 2. same with monitored therapy vs 3. same with McConnell and biofeedback showed that biofeedback with McConnell and home exercise produced better outcomes than home exercise alone or home exercise with monitored therapy. Concentric vs eccentric exercises in 76 participants; 60 completed the study with non significant difference between the groups on favour of concentric	Number of RCTs = 5 Quality assessment = Y Data pooled = N Heterogeneity = Y	Lack of standard outcome measures makes it difficult to compare between studies; no global rating of change. A number of important references have yet to be included. Reviewers graded the strength of the evidence as limited therefore unable to make firm recommendations re foot and knee orthotics
Harrison 2001	Review article on patellofemoral pain syndrome — looks at aetiology, diagnosis and non-surgical management	12 articles reviewed		Advice to consider quadriceps strengthening especially in the short term. For most patients other interventions may be considered when a basic home program has been ineffective over the short term. Taping appears to reduce pain in some patients; because of its low cost and simplicity it should be considered particularly for patients with changes in patellar orientation and mobility. There is a lack of valid clinical measures for patellar mobility and orientation that make it hard to tell whether the effect of taping results from mechanical improvement of from other factors. Biofeedback, manual therapy, NSAIDs and sleeves have not been found to be more effective than simple exercises	Number of RCTs = 12 Quality assessment = N Data pooled = N Heterogeneity = Y English language search between 1995–2000	Useful for epidemiological data

ANTERIOR KNEE PAIN — Table of Excluded Studies (Interventions)

STUDY	REASON
Brosseau 2002	Not relevant to anterior knee pain. Discusses ilio-tibial band syndrome
Cohen 2001	Exclude due to a theoretical study on cadavers
Dursun 2001	Pain not < 12 weeks.
Gotlin 2000	Not a systematic overview
Herrington 2000	Narrative review
Mears 2001	For selected patellofemoral conditions not non-specific patellofemoral pain; chronic conditions; narrative review
Neptune 2000	Not relevant; mechanistic study not involving patients in pain
Powers 1998	Narrative review
Rillman 2000	Exclude due to surgical treatment for patellofemoral instability
Roush 2000	Study does not specifically address patellofemoral pain — see inclusion criteria. This study is not limited to the specific diagnosis — it includes individuals with a number of diagnostic entities including patellar tendonopathy, plica syndrome etc.
Salsich 2002	Exclude due to not an RCT; small sample *n* = 10
Selfe 2001	Not relevant to these guidelines; a validation of outcome measurement rather than clinical assessment
Suter 1999	Underlying assumptions are unproven; chronic pain
Suter 2000	Basic science (construct validity); chronic pain
Tang 2001	Basic science — not relevant
Tobin 2000	Mechanistic study in asymptomatic study; not relevant for this guideline although a good study
Yeung 2001	Review of studies of primary prevention. Not relevant