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EVIDENCE-BASED MANAGEMENT OF ACUTE MUSCULOSKELETAL PAIN

Australian Acute Musculoskeletal Pain Guidelines Group
EVIDENCE-BASED MANAGEMENT OF ACUTE MUSCULOSKELETAL PAIN

Australian Acute Musculoskeletal Pain Guidelines Group
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).
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Acute Thoracic Spinal Pain

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Scope
Guideline Development Process
Research Agenda for Acute Thoracic Spinal Pain
Summary of Key Messages

Aetiology and Prevalence
Serious Conditions
Mechanical Conditions
Conditions Referring Pain to the Thoracic Spine
Conditions Referring Pain from the Thoracic Spine
Prevalence of Conditions Causing Acute Thoracic Spinal Pain

History
Pain History

Physical Examination
Inspection
Palpation
Movement
Alerting Features of Serious Conditions (see Table 5.7)

Ancillary Investigations
Plain Radiography
Computed Tomography (CT) Scanning
Magnetic Resonance Imaging (MRI)
Other Investigations
Cost Effectiveness of Investigations

Terminology
Recommended Terms

Prognosis
Natural History
Influence of Risk Factors and Diagnostic and Therapeutic Interventions

Interventions
Evidence of Benefit
Other Treatment

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Commonwealth Department of Health and Ageing  
Commonwealth Department of Health and Ageing  
Royal Australian College of General Practitioners  
Australian Osteopathic Association
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.
Summary of Key Messages: Acute Pain Management

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.

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.

Pharmacological Interventions

Specific pharmacological interventions may be required to relieve pain; such agents can be used in conjunction with non-pharmacological interventions.

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.
### Acute Pain Management continued

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<td>Paracetamol or other simple analgesics, administered regularly, are recommended for relief of mild to moderate acute musculoskeletal pain.</td>
<td>CONSENSUS: Steering Committee; NHMRC 1999b</td>
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<td>Where paracetamol is insufficient for pain relief, a non-steroidal anti-inflammatory (NSAID) medication may be used, unless contraindicated.</td>
<td>CONSENSUS: Steering Committee; NHMRC 1999b</td>
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<td>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.</td>
<td>CONSENSUS: Steering Committee; NHMRC 1999b</td>
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<td>Adjuvant agents such as anticonvulsants and antidepressants are not recommended in the management of acute musculoskeletal pain.</td>
<td>CONSENSUS: Steering Committee; NHMRC 1999b</td>
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<td>Any benefits from muscle relaxants may be outweighed by their adverse effects, therefore they cannot be routinely recommended.</td>
<td>CONSENSUS: Steering Committee</td>
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### Summary of Key Messages: Effective Communication

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

### Summary of Key Messages: Acute Low Back Pain

<table>
<thead>
<tr>
<th>Aetiology and Prevalence</th>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>The majority (approximately 95% of cases) of acute low back pain is non-specific; serious conditions are rare causes of acute low back pain.</td>
<td>*LEVEL I, III: Deyo et al. 1992; Suarez-Almazor et al. 1997; Hollingworth et al. 2002</td>
</tr>
<tr>
<td>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.</td>
<td>*LEVEL I, III: van Tulder et al. 1997a; Torgerson and Dotter 1976</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>History</th>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>History enables screening for features of serious conditions; however the reliability and validity of individual features in histories have low diagnostic significance.</td>
<td>*LEVEL III-2: Deyo et al. 1992; van den Hoogen et al. 1995</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Physical Examination</th>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical signs detected during physical and psychosocial assessment must be interpreted cautiously as many tests lack reliability and validity.</td>
<td>*LEVEL III-2: LeBoeuf-Yde et al. 2002; Truchon and Fillion 2000; Knutson 2002; Waddell et al. 1980; Deyo et al. 1992</td>
</tr>
<tr>
<td>A full neurological examination is warranted in the presence of lower limb pain and other neurological symptoms.</td>
<td>*LEVEL IV: Waddell et al. 1982; McCombe et al. 1989</td>
</tr>
</tbody>
</table>
### Ancillary Investigations

<table>
<thead>
<tr>
<th>Evidence-based Management of Acute Musculoskeletal Pain</th>
<th>Evidence Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plain x-rays 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.</td>
<td>*LEVEL III-2: Suarez-Almazor et al. 1997; Hollingworth et al. 2002; Kendrick et al. 2001; Kerry et al. 2002</td>
</tr>
<tr>
<td>Appropriate investigations are indicated in cases of acute low back pain when alerting features (‘red flags’) of serious conditions are present.</td>
<td>*LEVEL III-2: Deyo and Diehl 1986</td>
</tr>
</tbody>
</table>

### Terminology

<table>
<thead>
<tr>
<th>Evidence-based Management of Acute Musculoskeletal Pain</th>
<th>Evidence Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>A specific patho-anatomic diagnosis is not necessary for effective management of acute non-specific low back pain.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
<tr>
<td>Terms to describe acute low back pain with no identifiable pathology include ‘lumbar spinal pain of unknown origin’ or ‘somatic lumbar spinal pain’.</td>
<td>*LEVEL IV: Merskey and Bogduk 1994</td>
</tr>
</tbody>
</table>

### PROGNOSIS

<table>
<thead>
<tr>
<th>Evidence-based Management of Acute Musculoskeletal Pain</th>
<th>Evidence Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>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.</td>
<td>*LEVEL III-2: Croft and Rigby 1994; Schiottz-Christensen et al. 1999</td>
</tr>
<tr>
<td>Recurrences of acute low back pain are not uncommon.</td>
<td>*LEVEL III-3: van den Hoogen et al. 1998; Hurley et al. 2001a</td>
</tr>
<tr>
<td>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.</td>
<td>*LEVEL III-2: Linton 2001; Pincus et al. 2002; Truchon and Fillion 2000</td>
</tr>
</tbody>
</table>

### INTERVENTIONS

#### Evidence of Benefit

<table>
<thead>
<tr>
<th>Evidence-based Management of Acute Musculoskeletal Pain</th>
<th>Evidence Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>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.</td>
<td>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)</td>
</tr>
<tr>
<td>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).</td>
<td>LEVEL II: Based on one study (Nadler et al. 2002)</td>
</tr>
<tr>
<td>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.</td>
<td>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)</td>
</tr>
</tbody>
</table>

#### Conflicting Evidence

<table>
<thead>
<tr>
<th>Evidence-based Management of Acute Musculoskeletal Pain</th>
<th>Evidence Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>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.</td>
<td>LEVEL I: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b) that found numerous RCTs</td>
</tr>
</tbody>
</table>
### Acute Low Back Pain continued

<table>
<thead>
<tr>
<th><strong>Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)</strong> —</th>
<th>There is conflicting evidence that oral and injectable NSAIDs are effective versus placebo or no treatment for acute low back pain.</th>
</tr>
</thead>
<tbody>
<tr>
<td>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.</td>
<td><strong>LEVEL I, II:</strong> Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b; Koes et al. 1997) and numerous RCTs (Amile et al. 1987; Basmajian 1989; Postacchini et al. 1988; Lacey et al. 1984; Nadler et al. 2002)</td>
</tr>
<tr>
<td>There is insufficient evidence that NSAIDs are more effective when compared to muscle relaxants and anti-anxiety agents in acute low back pain.</td>
<td></td>
</tr>
<tr>
<td>NSAIDs are less effective in reducing pain than heat wrap therapy in the first three to four days of acute low back pain.</td>
<td></td>
</tr>
<tr>
<td>Serious adverse effects of NSAIDs include gastrointestinal complications (e.g. bleeding, perforation).</td>
<td><strong>LEVEL I, II:</strong> Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b; Koes et al. 1997) and numerous RCTs (Amile et al. 1987; Basmajian 1989; Postacchini et al. 1988; Lacey et al. 1984; Nadler et al. 2002)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Spinal Manipulation</strong> —</th>
<th>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.</th>
</tr>
</thead>
<tbody>
<tr>
<td>There is insufficient evidence that spinal manipulation is more or less effective than other conservative treatments for acute low back pain.</td>
<td><strong>LEVEL I, II:</strong> Based on systematic reviews (Hsieh et al. 2002)</td>
</tr>
<tr>
<td>Adverse effects of spinal manipulation are rare but potentially serious.</td>
<td><strong>LEVEL IV:</strong> Based on reviews of case studies (Haldeman and Rubinstein 1992; Assendelft et al. 1996; Stevinson and Ernst 2002)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Insufficient Evidence</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acupuncture</strong> —</td>
</tr>
<tr>
<td>Adverse effects of acupuncture are rare but potentially serious.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Analgesics, Compound and Opioid</strong> —</th>
<th>There are no randomised controlled trials investigating the efficacy of opioids and compound analgesics in acute low back pain.</th>
</tr>
</thead>
<tbody>
<tr>
<td>There is evidence that the effect of opioid or compound analgesics is similar to NSAIDs for treatment of acute low back pain.</td>
<td><strong>LEVEL I:</strong> 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)</td>
</tr>
<tr>
<td>In general, opioids and compound analgesics have a substantially increased risk of side effects compared with paracetamol alone.</td>
<td><strong>LEVEL IV:</strong> Based on reviews of case studies (Haldeman and Rubinstein 1992; Assendelft et al. 1996; Stevinson and Ernst 2002)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Analgesics, Simple</strong> —</th>
<th>There are no randomised controlled trials assessing the effectiveness of simple analgesics in acute low back pain.</th>
</tr>
</thead>
<tbody>
<tr>
<td>There is insufficient evidence for the effectiveness of simple analgesics versus NSAIDs in acute low back pain.</td>
<td><strong>LEVEL I, II:</strong> Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b; Bigos et al. 1994) and RCTs (Brown et al. 1986; Videman et al. 1984; Palangio et al. 2002)</td>
</tr>
<tr>
<td>Paracetamol is less effective than heat wrap therapy in acute low back pain.</td>
<td><strong>LEVEL I, II:</strong> Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b; Bigos et al. 1994) and RCTs (Brown et al. 1986; Videman et al. 1984; Palangio et al. 2002)</td>
</tr>
<tr>
<td>There is insufficient evidence for the effect of paracetamol compared to electroacupuncture in mixed populations with low back pain.</td>
<td><strong>LEVEL I, II:</strong> Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b; van Tulder et al. 2002d) of multiple controlled studies</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Back Exercises</strong> —</th>
<th>McKenzie therapy provides similar pain and function outcomes compared to usual care in acute low back pain.</th>
</tr>
</thead>
<tbody>
<tr>
<td>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.</td>
<td><strong>LEVEL I, II:</strong> Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b; van Tulder et al. 2002d) of multiple controlled studies</td>
</tr>
<tr>
<td>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.</td>
<td></td>
</tr>
<tr>
<td>Lateral multifidus muscle exercises reduce recurrences of low back pain compared to usual care in mixed populations with low back pain.</td>
<td></td>
</tr>
</tbody>
</table>
### Evidence-based Management of Acute Musculoskeletal Pain

#### Acute Low Back Pain continued

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Evidence</th>
<th>Level of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Back School</strong></td>
<td>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.</td>
<td>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)</td>
</tr>
<tr>
<td>Bed Rest</td>
<td>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.</td>
<td>LEVEL I, II: Based on systematic reviews (van Tulder et al. 1997b; Hagen et al. 2002) and an RCT (Rozenberg et al. 2002)</td>
</tr>
<tr>
<td><strong>Cognitive Behavioural Therapy</strong></td>
<td>Cognitive behavioural therapy reduces general disability in the long term compared to traditional care in mixed with populations back pain.</td>
<td>LEVEL I: Based on systematic reviews (Turner 1996; van Tulder et al. 2002e)</td>
</tr>
<tr>
<td>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.</td>
<td>LEVEL II: Based on studies by Linton and Andersson (2000) and Linton and Ryberg (2001)</td>
<td></td>
</tr>
<tr>
<td>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.</td>
<td>No Level I or II studies</td>
<td></td>
</tr>
<tr>
<td><strong>Electromyographic Biofeedback</strong></td>
<td>There are no controlled studies testing the effectiveness of electromyographic biofeedback in acute low back pain.</td>
<td>No Level I or II evidence</td>
</tr>
<tr>
<td><strong>Injection Therapy</strong></td>
<td>There is insufficient evidence demonstrating the effectiveness of injection therapy (facet joint, epidural or soft tissue) in the treatment of acute low back pain.</td>
<td>LEVEL II: Based on systematic reviews (Nelemans et al. 2002; Watts and Silagy 1995; Koes et al. 1999) and an RCT (Garvey et al. 1989)</td>
</tr>
<tr>
<td>Adverse effects of injection therapy are rare but serious.</td>
<td>No Level I or II evidence</td>
<td></td>
</tr>
<tr>
<td><strong>Lumbar Supports</strong></td>
<td>There are no controlled studies on the effect of lumbar supports in acute low back pain.</td>
<td>LEVEL I: Based on two systematic reviews (van Tulder et al. 2002c; Bigos et al. 1994)</td>
</tr>
<tr>
<td>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.</td>
<td>LEVEL I: Based on one RCT (Stam et al. 2001)</td>
<td></td>
</tr>
<tr>
<td><strong>Massage</strong></td>
<td>There are no controlled studies for massage therapy in acute low back pain.</td>
<td>LEVEL I, II: Based on systematic reviews (Furlan et al. 2002; Ernst 1999) and RCTs (Cherkin et al. 2001; Preyde 2000)</td>
</tr>
<tr>
<td>Massage is superior to placebo (sham laser) and acupuncture in mixed populations with low back pain.</td>
<td>LEVEL I, II: Based on systematic reviews (Furlan et al. 2002; Ernst 1999) and RCTs (Cherkin et al. 2001; Freyde 2000)</td>
<td></td>
</tr>
<tr>
<td>Massage provides similar effect to back schools (involving exercise and education), corsets and TENS in mixed populations with low back pain.</td>
<td>No Level I or II evidence</td>
<td></td>
</tr>
<tr>
<td>There is conflicting evidence of the effect of massage compared to manipulation and education in mixed populations with low back pain.</td>
<td>No Level I or II evidence</td>
<td></td>
</tr>
<tr>
<td><strong>Multi-Disciplinary Treatment in the Workplace</strong></td>
<td>There are no controlled studies on the effect of multi-disciplinary treatment in the workplace in acute low back pain.</td>
<td>LEVEL I, II: Based on a systematic review (Karjailanen et al. 2002) and RCTs (Loisel et al. 1997; Lindstrom 1992a,b)</td>
</tr>
<tr>
<td>Multi-disciplinary treatment in the workplace improves return to work and subjective disability compared to usual care in mixed populations with low back pain.</td>
<td>No Level I or II evidence</td>
<td></td>
</tr>
<tr>
<td><strong>Topical Treatment</strong></td>
<td>There is insufficient evidence for the effectiveness of spiroflar homeopathic gel or cremol capsici for treatment of acute low back pain.</td>
<td>LEVEL II: Based on one RCT (Stam et al. 2001)</td>
</tr>
</tbody>
</table>
### Acute Low Back Pain continued

| Traction — There are no controlled studies on the effect of traction for acute low back pain. |
| No Level I or II evidence |
| 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: Based on systematic reviews (van der Heijden et al. 1995; van Tulder et al. 1997b) |
| Adverse effects from traction have been reported, including reduced muscle tone, bone demineralisation, thrombophlebitis. |
| LEVEL I, II: Based on a systematic review (van Tulder et al. 1997b) and additional studies (Pengel et al. 2002; Hurley et al. 2001b) |

### Transcutaneous Electrical Nerve Stimulation

| Transcutaneous Electrical Nerve Stimulation — There are no controlled studies on the effect of TENS in acute low back pain. |
| No Level I or II evidence |
| 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 II: Malmivaara et al. 1995; Cherkin et al. 1998; Moffett et al. 1999 |

### Cost Effectiveness

| 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; Moffett et al. 1999 |

### Summary of Key Messages: Acute Thoracic Spinal Pain

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Evidence Level</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aetiology and Prevalence</strong></td>
<td></td>
</tr>
<tr>
<td>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 zygapophysseal joints</td>
<td>*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</td>
</tr>
<tr>
<td>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.</td>
<td>*LEVEL IV: Ross et al. 1994; Patel et al. 1991; Huang et al. 1994</td>
</tr>
<tr>
<td>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.</td>
<td>*LEVEL IV: Deyo and Diehl. 1988</td>
</tr>
</tbody>
</table>

| 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 Murlagh 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 Mackett 1995; Samuels and Kerstein 1993 |
## Acute Thoracic Spinal Pain continued

<table>
<thead>
<tr>
<th>Evidence-based Management of Acute Musculoskeletal Pain</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>In the presence of trauma, x-ray 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.</td>
<td><em>LEVEL IV: Samuels and Kerstein 1993; Durham et al. 1995</em></td>
</tr>
<tr>
<td>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.</td>
<td><em>LEVEL IV: Keene et al. 1982</em></td>
</tr>
<tr>
<td>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.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
</tbody>
</table>

### Terminology

The appropriate labels for non-specific ‘mechanical’ thoracic spinal pain are ‘thoracic spinal pain of unknown origin’ or ‘somatic thoracic spinal pain’.

### PROGNOSIS

There is a lack of published data on the natural history and influence of prognostic risk factors for acute thoracic spinal pain.

### INTERVENTIONS

**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 III-3: Based on cross-sectional and prospective radiological surveys (Heller et al. 1983; Johnson and Lucas 1997)*

*LEVEL III: Based on epidemiological and radiological surveys (van der Donk et al. 1991; Fridenberg and Miller 1963)*

*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)*

*LEVEL III: Based on a prospective epidemiological study (Berglund et al. 2000)*

### Summary of Key Messages: Acute Neck Pain

#### DIAGNOSIS

**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 spondylasis 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. Despite limitations, physical examination is an opportunity to identify features of potentially serious conditions.

Tenderness and restricted cervical range of movement correlate well with the presence of neck pain, confirming a local cause for the pain.

### 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. In symptomatic patients with a history of trauma, radiography is indicated according to the Canadian C-Spine Rule.

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.

Acute neck pain in conjunction with features alerting to the possibility of a serious underlying condition is an indication for MRI.

### Terminology

Except for serious conditions, precise identification of the cause of neck pain is unnecessary. Once serious causes have been recognised or excluded, terms to describe acute neck pain can be either ‘acute idiopathic neck pain’ or ‘acute whiplash-asssociated neck pain’.

### PROGNOSIS

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.

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.

Psychosocial factors are not determinants of chronicity in whiplash-associated neck pain.

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.

### INTERVENTIONS

#### 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.
### Acute Neck Pain

**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 (Bönn 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

**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

**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

**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

**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

<table>
<thead>
<tr>
<th><strong>Multi-Disciplinary Biopsychosocial Rehabilitation</strong> — There are no randomised controlled studies investigating the effect of multi-disciplinary treatment in acute neck pain.</th>
<th>No Level I or II evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>There is insufficient evidence that multi-disciplinary treatment is effective compared to other interventions for reducing neck pain in mixed populations.</td>
<td>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</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Muscle Relaxants</strong> — There are no randomised controlled trials investigating the efficacy of muscle relaxants for the treatment of acute neck pain.</th>
<th>No Level I or II evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle relaxants are no more effective than placebo for neck pain in mixed populations.</td>
<td>LEVEL I, II: Based on a systematic review (Aker et al. 1996) of two studies plus one additional study, all involving mixed populations</td>
</tr>
<tr>
<td>Drowsiness, dizziness and dependency are common adverse effects of muscle relaxants.</td>
<td>LEVEL I: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Neck School</strong> — There are no randomised controlled trials on the effect of neck school for acute neck pain.</th>
<th>No Level I or II evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neck school appears no more effective than no treatment for neck pain in mixed populations.</td>
<td>LEVEL II: Based on one controlled trial (Kamwendo and Linton 1991) involving a mixed population</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)</strong> — There are no randomised controlled trials on the effectiveness of NSAIDs for acute neck pain.</th>
<th>No Level I or II evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>There is evidence that NSAIDs are no more effective than placebo ultrasound for neck pain in mixed populations.</td>
<td>LEVEL I: Based on a systematic review (Aker et al. 1996) that located two studies involving mixed populations</td>
</tr>
<tr>
<td>Serious adverse effects of NSAIDs include gastrointestinal complications. (e.g. bleeding, perforation)</td>
<td>LEVEL I: Based on systematic reviews (Bigos et al. 1994; Henry et al. 1996)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Patient Education</strong> — There are no randomised controlled trials investigating the effect of patient education as a single strategy in the treatment of acute neck pain.</th>
<th>No Level I or II evidence</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th><strong>Spray and Stretch Therapy</strong> — There are no randomised controlled trials investigating the effect of spray and stretch therapy in acute neck pain.</th>
<th>No Level I or II evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spray and stretch therapy appears no more effective than placebo for neck pain in mixed populations.</td>
<td>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)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Traction</strong> — There are no randomised controlled trials investigating the effectiveness of traction for acute neck pain.</th>
<th>No Level I or II evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>In mixed populations, there is evidence that traction is of no benefit compared to a range of other interventions for neck pain.</td>
<td>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</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Transcutaneous Electrical Nerve Stimulation (TENS)</strong> — There is insufficient evidence of benefit from TENS compared to a collar or manual therapy in acute neck pain.</th>
<th>LEVEL I: Based on a systematic review (Gross et al. 2002b) that identified one controlled trial (Nordemar and Thorne 1981) with equivocal results</th>
</tr>
</thead>
</table>
## 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

<table>
<thead>
<tr>
<th>Aetiology and Prevalence</th>
<th>Evidence Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinicians should be alert to the potential for rare, serious conditions (e.g. fracture/dislocation, tumour, infection, inflammatory arthropathies) presenting as acute shoulder pain.</td>
<td><em>LEVEL IV</em>: numerous case studies (Jones et al. 1994; Kaempffe 1995; Barlow and Newman 1994; Welch 1994; Linos et al. 1980)</td>
</tr>
<tr>
<td>Most cases of acute shoulder pain are of 'mechanical' origin and can be managed as acute regional pain.</td>
<td><em>LEVEL III-2, III-3</em>: Torstensen and Hollinshead 1999; Chandnani et al. 1992; Milgrom et al. 1995; Sher et al. 1995</td>
</tr>
<tr>
<td>Biological factors such as age, female gender, past history and response to repetitive physical tasks may contribute to the development of acute shoulder pain.</td>
<td><em>LEVEL III-3</em>: Jones et al. 1994; Cummings et al. 1995; Sambrook 1996; Ekberg et al. 1995; Skov et al. 1996</td>
</tr>
<tr>
<td>Psychosocial factors such as job dissatisfaction and work demands may contribute to the onset of acute shoulder pain.</td>
<td><em>LEVEL III-2</em>: Bergenudd et al. 1994; Ekberg et al. 1995; Marcus et al. 1996; Skov et al. 1996</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>History</th>
<th>Evidence Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Information obtained from the history may alert to the presence of a serious condition as the underlying cause of acute shoulder pain.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
<tr>
<td>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.</td>
<td><em>LEVEL III-2</em>: Narregaard et al. 2002; Litaker et al. 2000</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Physical Examination</th>
<th>Evidence Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>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.</td>
<td><em>LEVEL III-2</em>: Calis et al. 2000; MacDonald et al. 2000; Naredo et al. 2002; Itto et al. 1999; Bennett 1998</td>
</tr>
<tr>
<td>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.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
<tr>
<td>Despite limitations, physical examination is an opportunity to identify features of potentially serious conditions.</td>
<td><em>LEVEL III-2</em>: Bamji et al. 1996; Liesdeck et al. 1997; de Winter et al. 1999; Pai et al. 2000; Narregaard et al. 2002</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ancillary Investigations</th>
<th>Evidence Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>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.</td>
<td><em>LEVEL III</em>: 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)</td>
</tr>
<tr>
<td>There is a need to educate consumers about the limitations of imaging and the risks of radiation exposure.</td>
<td><em>LEVEL IV</em>: Roebuck 1995</td>
</tr>
</tbody>
</table>
### 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

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 of Benefit**

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Evidence Level</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Corticosteroid Injection</strong></td>
<td>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)</td>
</tr>
<tr>
<td><strong>Exercises</strong></td>
<td>LEVEL I: Systematic review of two RCTs (Ginn et al. 1997; Brox et al. 1997)</td>
</tr>
<tr>
<td><strong>Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)</strong></td>
<td>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)</td>
</tr>
<tr>
<td><strong>Serious adverse effects of NSAIDs include gastrointestinal complications (e.g. bleeding, perforation)</strong></td>
<td>LEVEL I: Based on a systematic review (Bigos et al. 1994)</td>
</tr>
<tr>
<td><strong>Ultrasound</strong></td>
<td>LEVEL I: Systematic review of one RCT in acute shoulder pain (Ebenbichler et al. 1999)</td>
</tr>
</tbody>
</table>

**Conflicting Evidence**

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Evidence Level</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acupuncture</strong></td>
<td>LEVEL I: Systematic review (Green et al. 2003) of two RCTs (Kleinhenz et al. 1999; Berry et al. 1980)</td>
</tr>
</tbody>
</table>

**Insufficient Evidence**

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Evidence Level</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Analgesics</strong></td>
<td>No Level I or II evidence</td>
</tr>
<tr>
<td><strong>Extracorporeal Shock Wave Treatment (ESWT)</strong></td>
<td>No Level I or II evidence</td>
</tr>
<tr>
<td>Trials conducted in populations with chronic shoulder pain show conflicting results for ESWT compared with placebo.</td>
<td>LEVEL I: Buchbinder et al. 2003a (systematic review of four RCTs)</td>
</tr>
<tr>
<td><strong>Manual Therapy</strong></td>
<td>LEVEL I: Systematic review located one RCT of 14 patients (Conroy and Hayes 1998)</td>
</tr>
<tr>
<td><strong>Oral Corticosteroids</strong></td>
<td>No Level I or II evidence</td>
</tr>
<tr>
<td>Studies of mixed populations do not report significant benefit from oral corticosteroids compared with placebo or no treatment for adhesive capsulitis.</td>
<td>LEVEL I: Green et al. 1998 (systematic review of two RCTs with methodological limitations)</td>
</tr>
</tbody>
</table>
### Acute Shoulder Pain continued

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

### Summary of Key Messages: Anterior Knee Pain

<table>
<thead>
<tr>
<th>DIAGNOSIS</th>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aetiology and Prevalence</strong></td>
<td></td>
</tr>
<tr>
<td>‘Patellofemoral pain’ is a general term used to describe idiopathic pain arising from the anterior knee/patellofemoral region that is of otherwise unknown origin.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
<tr>
<td>Anterior knee pain is commonly idiopathic; serious causes are rare.</td>
<td>*LEVEL IV: Kaempffle 1995; Ferguson et al. 1997; Kaandorp et al. 1995</td>
</tr>
<tr>
<td>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.</td>
<td>*LEVEL IV: Kujala et al. 2001; Reider et al. 1981a,b; Witvrouw et al. 2000; Tanaka et al. 1989; Cooper et al. 1994</td>
</tr>
<tr>
<td><strong>History</strong></td>
<td></td>
</tr>
<tr>
<td>The history provides information on possible causes of anterior knee pain and assists the identification of serious underlying conditions</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
<tr>
<td><strong>Physical Examination</strong></td>
<td></td>
</tr>
<tr>
<td>Although examination techniques lack specificity for diagnosing knee disorders, physical examination may assist the identification of serious conditions underlying anterior knee pain.</td>
<td>*LEVEL III, IV: Daniel 1991; Cook et al. 2001; Cushnagan et al. 1990; Biedert and Warnke 2001</td>
</tr>
<tr>
<td><strong>Ancillary Investigations</strong></td>
<td></td>
</tr>
<tr>
<td>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.</td>
<td>*LEVEL III, IV: Chapman-J ones et al. 1998; Petit et al. 2001; Stell et al. 1996; Seaberg and Jackson 1994; Bauer et al. 1995</td>
</tr>
<tr>
<td>Suspected fracture in the presence of a normal plain radiograph is an indication for CT scan.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
<tr>
<td>The presence of alerting features of a serious condition is an indication for the use of MRI.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
<tr>
<td>Swelling or potential rupture of anterior knee structures are indications for the use of ultrasound.</td>
<td>*LEVEL IV: Bianchi et al. 1994</td>
</tr>
<tr>
<td><strong>Terminology</strong></td>
<td></td>
</tr>
<tr>
<td>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.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
</tbody>
</table>

### PROGNOSIS

<table>
<thead>
<tr>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>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.</td>
</tr>
</tbody>
</table>
### Anterior Knee Pain continued

<table>
<thead>
<tr>
<th>INTERVENTIONS</th>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Evidence of Benefit</strong></td>
<td></td>
</tr>
<tr>
<td>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.</td>
<td>LEVEL II: Finestone et al. 1993</td>
</tr>
<tr>
<td>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.</td>
<td>LEVEL II: Kannus et al. 1992</td>
</tr>
<tr>
<td>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.</td>
<td>LEVEL I: Based on a systematic review (Crossley et al. 2001) that located one RCT (Eng and Pierrynowski 1993)</td>
</tr>
<tr>
<td>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.</td>
<td>LEVEL II: Based on one RCT (Crossley et al. 2002)</td>
</tr>
<tr>
<td>Eccentric quadriceps exercises produce better functional outcomes compared to standard quadriceps strengthening exercises.</td>
<td>LEVEL I: Based on a systematic review (Crossley et al. 2001) of eight RCTs</td>
</tr>
<tr>
<td><strong>Conflicting Evidence</strong></td>
<td></td>
</tr>
<tr>
<td>Orthoses (Patellofemoral) — There is conflicting evidence that patellofemoral orthoses are effective compared to other interventions and to no treatment for patellofemoral pain.</td>
<td>LEVEL I: Based on two systematic reviews (Crossley et al. 2001; D’hondt et al. 2002)</td>
</tr>
<tr>
<td><strong>Insufficient Evidence</strong></td>
<td></td>
</tr>
<tr>
<td>Acupuncture — There are no randomised controlled studies evaluating the effect of acupuncture for relief of patellofemoral pain.</td>
<td>No Level I or II evidence</td>
</tr>
<tr>
<td>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.</td>
<td>No Level I or II evidence</td>
</tr>
<tr>
<td>Electrical Stimulation — There are no randomised controlled studies of the effectiveness of electrical stimulation of the quadriceps muscle for patellofemoral pain.</td>
<td>No Level I or II evidence</td>
</tr>
<tr>
<td>There is insufficient evidence that one form of electrical stimulation of the quadriceps muscle is superior to another for treating patellofemoral pain.</td>
<td>LEVEL II: Callaghan et al. (2001)</td>
</tr>
<tr>
<td>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.</td>
<td>No Level I or II evidence</td>
</tr>
<tr>
<td>Different types of NSAIDs provide similar relief of patellofemoral pain after five days of use.</td>
<td>LEVEL II: Based on one RCT with limitations (Fulkerson and Folcik 1986)</td>
</tr>
<tr>
<td>Serious adverse effects of NSAIDs include gastrointestinal complications (e.g. bleeding, perforation).</td>
<td>LEVEL I: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 2002)</td>
</tr>
<tr>
<td>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.</td>
<td>LEVEL I, II: Based on two systematic reviews (Crossley et al. 2001; Harrison et al. 2001) and one subsequent RCT (Crossley et al. 2002)</td>
</tr>
<tr>
<td>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).</td>
<td>LEVEL I: Based on a systematic review (Crossley et al. 2001) that located one RCT (Timm 1998)</td>
</tr>
<tr>
<td>Therapeutic Ultrasound — There is insufficient evidence that therapeutic ultrasound is more effective compared to ice massage for the treatment of patellofemoral pain.</td>
<td>LEVEL I: Based on a Cochrane Review (Brosseau et al. 2002b) and two meta-analyses (Gam and J ohannsen 1995; van der Windt et al. 1999)</td>
</tr>
</tbody>
</table>
Anterior Knee Pain continued

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

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


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

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
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 et 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).

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
Chapter 2 • Acute Pain Management

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

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**Figure 2.3**

**Figure 2.4**

**Figure 2.5**


- 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 X-rays 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

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-
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 medication (NHMRC 1999).

The adverse effects of NSAIDs are potentially serious and all people cannot use them. NSAIDs may result in gastrointestinal bleeding, renal dysfunction (particularly in elderly 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)

Review

- Review progress and pain level: confirm favourable outcome; recognise poor progress.
- If symptoms persist, check whether the plan was implemented, reassess for features (‘red flags’) of serious conditions, assess for psychosocial and occupational factors (‘yellow flags’) that may influence presentation, response to treatment and recovery. Explore other potential barriers to recovery and consider alternative interventions.
- Arrange for further follow-up visit as required.

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 NHMRC (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. 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**


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 without any 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 x-rays 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 than 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

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 lifetime 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 sacroccygeal joints
- laterally, by vertical lines tangential to the lateral borders of the lumbar erectors 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
Evidence-based Management of Acute Musculoskeletal Pain

Chapter 4 • Acute Low Back Pain

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

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:

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
<th>Systematic reviews, cross-sectional studies, case series, case reports Adults</th>
</tr>
</thead>
</table>
| 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:

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
<th>Systematic reviews, cohort studies Adults</th>
</tr>
</thead>
</table>
| 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 |
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
- EM BASE — 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 x-ray)
- 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

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<thead>
<tr>
<th>Management Plan</th>
<th>EVIDENCE LEVEL</th>
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<td>It is recommended that the clinician and patient develop a management plan for acute musculoskeletal pain comprising the elements of assessment, management and review:</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
<tr>
<td>• 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.</td>
<td></td>
</tr>
<tr>
<td>• Management — Provide information, assurance and advice to resume normal activity and discuss other options for pain management as needed.</td>
<td></td>
</tr>
<tr>
<td>• Review — Reassess the pain and revise the management plan as required.</td>
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<tr>
<th>Non-Pharmacological Interventions</th>
<th>EVIDENCE LEVEL</th>
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<tbody>
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<th>Pharmacological Interventions</th>
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</thead>
<tbody>
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<td>Specific pharmacological interventions may be required to relieve pain; such agents can be used in conjunction with non-pharmacological interventions.</td>
<td>CONSENSUS: Steering Committee; NHM RC 1999b</td>
</tr>
<tr>
<td>Paracetamol or other simple analgesics, administered regularly, are recommended for relief of mild to moderate acute musculoskeletal pain.</td>
<td>CONSENSUS: Steering Committee; NHM RC 1999b</td>
</tr>
<tr>
<td>Where paracetamol is insufficient for pain relief, a non-steroidal anti-inflammatory (NSAID) medication may be used, unless contraindicated.</td>
<td>CONSENSUS: Steering Committee; NHM RC 1999b</td>
</tr>
<tr>
<td>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.</td>
<td>CONSENSUS: Steering Committee; NHM RC 1999b</td>
</tr>
<tr>
<td>Adjuvant agents such as anticonvulsants and antidepressants are not recommended in the management of acute musculoskeletal pain.</td>
<td>CONSENSUS: Steering Committee; NHM RC 1999b</td>
</tr>
<tr>
<td>Any benefits from muscle relaxants may be outweighed by their adverse effects, therefore they cannot be routinely recommended.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
</tbody>
</table>

### Summary of Key Messages: Effective Communication

| 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                       |
Chapter 4 • Acute Low Back Pain

Summary of Key Messages: Acute Low Back Pain

**DIAGNOSIS**

**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.

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.

**History**

History enables screening for features of serious conditions; however the reliability and validity of individual features in histories have low diagnostic significance.

**Physical Examination**

Clinical signs detected during physical and psychosocial assessment must be interpreted cautiously as many tests lack reliability and validity.

A full neurological examination is warranted in the presence of lower limb pain and other neurological symptoms.

**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.

Appropriate investigations are indicated in cases of acute low back pain when alerting features (‘red flags’) of serious conditions are present.

**Terminology**

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

Terms to describe acute low back pain with no identifiable pathology include ‘lumbar spinal pain of unknown origin’ or ‘somatic lumbar spinal pain’.

**PROGNOSIS**

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.

Recurrences of acute low back pain are not uncommon.

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.

**INTERVENTIONS**

**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.

---

**EVIDENCE LEVEL**


*LEVEL I, III: van Tuylde et al. 1997a; Torgerson and Dotter 1976

*LEVEL III-2: Deyo et al. 1992; van den Hoogen et al. 1995


*LEVEL IV: Waddell et al. 1982; McCombe et al. 1989


*LEVEL III-2: Deyo and Diehl 1986

CONSENSUS: Steering Committee

*LEVEL IV: Merskey and Bogduk 1994

*LEVEL III-2: Croft and Rigby 1994; Schiottz-Christensen et al. 1999

*LEVEL III-3: van den Hoogen et al. 1998; Hurley et al. 2001a

*LEVEL III-2: Linton 2001; Pincus et al. 2002; Truchon and Fillion 2000

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
- **Evidence:** 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).

### Patient Information (Printed)
- **Evidence:** 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.
- **Evidence:** 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.
- **Evidence:** Behavioural therapy interventions are more effective than printed information for preventing long-term disability in mixed populations.

### Conflicting Evidence

#### Muscle Relaxants
- **Evidence:** There is conflicting evidence that muscle relaxants are effective compared to placebo in acute low back pain.
- **Evidence:** There is insufficient evidence to determine whether muscle relaxants are more or less effective compared to NSAIDs for acute low back pain.
- **Evidence:** Drowsiness, dizziness and dependency are common adverse effects of muscle relaxants.

#### Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)
- **Evidence:** There is conflicting evidence that oral and injectable NSAIDs are effective versus placebo or no treatment for acute low back pain.
- **Evidence:** 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.
- **Evidence:** There is insufficient evidence that NSAIDs are more effective when compared to muscle relaxants and anti-anxiety agents in acute low back pain.
- **Evidence:** NSAIDs are less effective in reducing pain than heat wrap therapy in the first three to four days of acute low back pain.
- **Evidence:** Serious adverse effects of NSAIDs include gastrointestinal complications (e.g. bleeding, perforation).

#### Spinal Manipulation
- **Evidence:** 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.
- **Evidence:** There is insufficient evidence that spinal manipulation is more or less effective than other conservative treatments for acute low back pain.
- **Evidence:** Adverse effects of spinal manipulation are rare but potentially serious.

### Insufficient Evidence

#### Acupuncture
- **Evidence:** There is insufficient evidence that acupuncture (dry-needling) is effective compared to injection therapy in acute low back pain.
- **Evidence:** Adverse effects of acupuncture are rare but potentially serious.

#### Analgesics, Compound and Opioid
- **Evidence:** There are no randomised controlled trials investigating the efficacy of opioids and compound analgesics in acute low back pain.
- **Evidence:** There is evidence that the effect of opioid or compound analgesics is similar to NSAIDs for treatment of acute low back pain.
- **Evidence:** In general, opioids and compound analgesics have a substantially increased risk of side effects compared with paracetamol alone.

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**Note:** Levels of evidence are classified as follows:

- **LEVEL I:** Based on systematic reviews and/or RCTs.
- **LEVEL II:** Based on controlled trials.
- **LEVEL III:** Based on cohort studies and case series.
- **LEVEL IV:** Based on expert opinion and case studies.
- **LEVEL V:** Based on case reports.

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**References:**
- Nadler et al. 2002
- Cherkin et al. 1996; Cherkin et al. 1998; Burton et al. 1999; Hazard et al. 2000; Roberts et al. 2002; Linton and Andersson 2000
- Bigos et al. 1994; van Tulder et al. 1997b; van Tulder et al. 2002f; Koes et al. 1997; van Tulder et al. 2002a; Assendelft et al. 1996; Stevinson and Ernst 2002
### Acute Low Back Pain continued

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Evidence/References</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Analgesics, Simple</strong></td>
<td>There are no randomised controlled trials assessing the effectiveness of simple analgesics in acute low back pain. No Level I or II evidence.</td>
</tr>
<tr>
<td></td>
<td>There is insufficient evidence for the effectiveness of simple analgesics versus NSAIDs in acute low back pain. 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.</td>
</tr>
<tr>
<td></td>
<td>Paracetamol is less effective than heat wrap therapy in acute 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.</td>
</tr>
<tr>
<td></td>
<td>There is insufficient evidence for the effect of paracetamol compared to electroacupuncture in mixed populations with low back pain.</td>
</tr>
<tr>
<td><strong>Back Exercises</strong></td>
<td>McKenzie therapy provides similar pain and function outcomes compared to usual care in acute low back pain. LEVEL I, II: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997b) of multiple controlled studies.</td>
</tr>
<tr>
<td></td>
<td>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.</td>
</tr>
<tr>
<td></td>
<td>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, II: Based on systematic reviews (van Tulder et al. 1997b; van Tulder et al. 2002b) and an RCT by Hsieh et al. (2002).</td>
</tr>
<tr>
<td></td>
<td>Lateral multifidus muscle exercises reduce recurrences of low back pain compared to usual care in mixed populations with low back pain.</td>
</tr>
<tr>
<td><strong>Back School</strong></td>
<td>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, II: Based on systematic reviews (van Tulder et al. 1997b; van Tulder et al. 2002b) and an RCT by Hsiet al. (2002).</td>
</tr>
<tr>
<td></td>
<td>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.</td>
</tr>
<tr>
<td><strong>Bed Rest</strong></td>
<td>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: Based on systematic reviews (van Tulder et al. 1997b; Hagen et al. 2002) and an RCT (Rozenberg et al. 2002).</td>
</tr>
<tr>
<td></td>
<td>There is conflicting evidence that bed rest increases disability and rate of recovery compared to staying active in mixed populations with low back pain.</td>
</tr>
<tr>
<td></td>
<td>Bedrest for longer than two days increases the amount of sick leave compared to early resumption of normal activity in acute low back pain.</td>
</tr>
<tr>
<td></td>
<td>There is evidence that prolonged bed rest is harmful.</td>
</tr>
<tr>
<td><strong>Cognitive Behavioural Therapy</strong></td>
<td>Cognitive behavioural therapy reduces general disability in the long term compared to traditional care in mixed with populations back pain. LEVEL I: Based on systematic reviews (Turner 1996; van Tulder et al. 2002e).</td>
</tr>
<tr>
<td></td>
<td>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: Based on studies by Linton and Andersson (2000) and Linton and Ryberg (2001).</td>
</tr>
<tr>
<td></td>
<td>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.</td>
</tr>
<tr>
<td><strong>Electromyographic Biofeedback</strong></td>
<td>There are no controlled studies testing the effectiveness of electromyographic biofeedback in acute low back pain. No Level I or II evidence.</td>
</tr>
<tr>
<td><strong>Injection Therapy</strong></td>
<td>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: Based on systematic reviews (Nelemans et al. 2002; Watts and Silagy 1995; Koes et al. 1999) and an RCT (Garvey et al. 1989).</td>
</tr>
<tr>
<td></td>
<td>Adverse effects of injection therapy are rare but serious.</td>
</tr>
<tr>
<td><strong>Lumbar Supports</strong></td>
<td>There are no controlled studies on the effect of lumbar supports in acute low back pain. LEVEL I, II: Based on two systematic reviews (van Tulder et al. 2002c; Bigos et al. 1994).</td>
</tr>
<tr>
<td></td>
<td>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.</td>
</tr>
</tbody>
</table>
Acute Low Back Pain continued

| Massage — There are no controlled studies for massage therapy in acute low back pain. | No Level I or II evidence |
| 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. |

| 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. | No Level I or II evidence |
| Multi-disciplinary treatment in the workplace improves return to work and subjective disability compared to usual care in mixed populations with low back pain. |

| 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) |
| 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. |

| Transcutaneous Electrical Nerve Stimulation — There are no controlled studies on the effect of TENS in acute low back pain. | No Level I or II evidence |
| 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. |

| 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 x-ray 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.
Evidence-based Management of Acute Musculoskeletal Pain

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 1997; Hollingworth et al. 2002). The majority who prove to have cancer are elderly (Deyo and Diehl 1988).

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).

**Table 4.1**

<table>
<thead>
<tr>
<th>Conditions Associated with the Presence of Acute Low Back Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Serious conditions</strong></td>
</tr>
<tr>
<td>Fracture (traumatic and osteoporotic)</td>
</tr>
<tr>
<td>Tumour: primary (myeloma, tumours of bone, cartilage, neuronal and muscle tissue); secondary (prostate, breast, lung, thyroid, kidney, gastrointestinal, melanoma)</td>
</tr>
<tr>
<td>Infection (osteomyelitis, epidural abscess)</td>
</tr>
<tr>
<td><strong>Neuropathic conditions</strong></td>
</tr>
<tr>
<td>Nerve root entrapment, sciatica, radicular pain</td>
</tr>
<tr>
<td><strong>Mechanical conditions</strong></td>
</tr>
<tr>
<td>Sprains, strains, tears of muscle fascia, ligament, joint or disc</td>
</tr>
<tr>
<td><strong>Visceral conditions</strong></td>
</tr>
<tr>
<td>Arising from abdominal structures, pelvic viscera, renal tract (infection, renal calculi)</td>
</tr>
<tr>
<td>Pancreatitis</td>
</tr>
<tr>
<td>Aortic aneurysm, retroperitoneal disease</td>
</tr>
<tr>
<td><strong>Other conditions</strong></td>
</tr>
<tr>
<td>Metabolic bone disease (Paget’s disease, hyperparathyroidism)</td>
</tr>
<tr>
<td>Osteoarthritis, degenerative joint disease, ankylosing spondylitis, enthesopathy, myositis, disc disruption, discitis</td>
</tr>
</tbody>
</table>

**Table 4.2**

<table>
<thead>
<tr>
<th>Radiological Findings in Patients with Low Back Pain in Primary Care</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lumbar Radiography Findings</td>
</tr>
<tr>
<td>Hollingworth et al. 2002</td>
</tr>
<tr>
<td>Suarez-Almazor et al. 1997</td>
</tr>
<tr>
<td><strong>Normal findings</strong></td>
</tr>
<tr>
<td>N</td>
</tr>
<tr>
<td>------------------------</td>
</tr>
<tr>
<td>Normal findings</td>
</tr>
<tr>
<td>Minor degenerative changes and other findings</td>
</tr>
<tr>
<td>Fracture</td>
</tr>
<tr>
<td>Infection</td>
</tr>
<tr>
<td>Tumour</td>
</tr>
<tr>
<td>Inflammatory disorders</td>
</tr>
<tr>
<td><strong>Total</strong></td>
</tr>
</tbody>
</table>

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

**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)

**>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...
Chapter 4 • Acute Low Back Pain

Evidence-based Management of Acute Musculoskeletal Pain

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.

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.

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).

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.

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.

Table 4.3
Prevalence of Spondylosis in Asymptomatic Individuals and Patients with Lumbar Spinal Pain

<table>
<thead>
<tr>
<th>Age</th>
<th>Asymptomatic</th>
<th>Symptomatic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>n</td>
</tr>
<tr>
<td>All</td>
<td>217</td>
<td>102</td>
</tr>
<tr>
<td>40–49</td>
<td>64</td>
<td>22%</td>
</tr>
<tr>
<td>50–59</td>
<td>69</td>
<td>74%</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Age</th>
<th>Asymptomatic</th>
<th>Symptomatic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>n</td>
</tr>
<tr>
<td>All</td>
<td>217</td>
<td>48</td>
</tr>
<tr>
<td>40–49</td>
<td>64</td>
<td>6%</td>
</tr>
<tr>
<td>60–69</td>
<td>69</td>
<td>48%</td>
</tr>
</tbody>
</table>

Note: N = total number of patients surveyed; n = number affected. The relationship between disc degeneration and symptoms is significant (p < 0.05) on a χ2 test. Based on data from Torgerson and Dotter 1976.
### Table 4.5
Comparison of Somatic Referred and Radicular Pain

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

**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 (Calm 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 x-ray 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 Arthopathies

- 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.
Chapter 4 • Acute Low Back Pain

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)

| Table 4.6
<p>| Alerting Features ('Red Flags') of Serious Conditions Associated with Acute Low Back Pain |</p>
<table>
<thead>
<tr>
<th>Feature or Risk Factor</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms and signs of infection (e.g. fever)</td>
<td>Infection</td>
</tr>
<tr>
<td>Risk factors for infection (e.g. underlying disease process, immunosuppression, penetrating wound)</td>
<td></td>
</tr>
<tr>
<td>History of trauma</td>
<td></td>
</tr>
<tr>
<td>Minor trauma (if &gt; 50 years, history of osteoporosis and taking corticosteroids)</td>
<td>Fracture</td>
</tr>
<tr>
<td>Past history of malignancy</td>
<td>Tumour</td>
</tr>
<tr>
<td>Age &gt; 50 years</td>
<td></td>
</tr>
<tr>
<td>Failure to improve with treatment</td>
<td></td>
</tr>
<tr>
<td>Unexplained weight loss</td>
<td></td>
</tr>
<tr>
<td>Pain at multiple sites</td>
<td></td>
</tr>
<tr>
<td>Pain at rest</td>
<td></td>
</tr>
<tr>
<td>Absence of aggravating features</td>
<td>Aortic aneurysm</td>
</tr>
</tbody>
</table>

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 referred to the low back.

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.
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 (D'eyo 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 (N'ice et al. 1992; N'joo 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).

Loboef-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 (M'ayer 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 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 (M'aher 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 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) (D'oneison 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 (D'oneison 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) (D'efuss et al. 1996). The tests may be positive in some 25% of individuals who have no pain (D'efuss 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

<table>
<thead>
<tr>
<th>Predictors of Chronicity: Waddell's Non-organic Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Superficial tenderness, non-anatomic tenderness</td>
</tr>
<tr>
<td>- Pain reaction to simulation tests for axial loading</td>
</tr>
<tr>
<td>- Pain reaction to simulated rotation</td>
</tr>
<tr>
<td>- Effect of distraction during examination</td>
</tr>
<tr>
<td>- Regional sensory disturbance</td>
</tr>
<tr>
<td>- Regional weakness in non-anatomic distributions</td>
</tr>
<tr>
<td>- Over-reaction during examination</td>
</tr>
<tr>
<td>(overt pain behaviour — grimacing, sighing, guarding, bracing, rubbing)</td>
</tr>
</tbody>
</table>

**Figure 4.1**

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; M Ccombe 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% (H alpin 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; 160 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).
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.

>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.

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

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 erectors 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'.

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.
Recurrences of acute low back pain are not uncommon. (*Level III-3) 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 M M P I — Hysteresis 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;
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).

Mclintoch 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 rehabilitation programs. Having three or more positive Waddell non-organic signs was one of the significant factors in this cohort. Twenty-two studies met their inclusion criteria; all studies had accurate diagnosis of more harmful and chronic conditions, you 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

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 workplace (Work Appgar) 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 communication 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 Lotrie (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-based Management of Acute Musculoskeletal Pain
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 Cochrane 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; Lendroquist et al. 1984; Lindstrom et al. 1992ab; 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 bed rest 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; Almivaara 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
Nadal 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 et al. 1992; 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; Middelton 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.

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 (Pohjoilainen 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; Hösie 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; Hösie 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 felbinafam (Hösie 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.
Clinical Evidence (2002) reports six systematic reviews (Evans and Richards 1996; van Tulder et al. 1997b; Shekelle et al. 1992; Koes et al. 1996; M ohsen-Bandpré 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 greater 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 H unter 1985; M athews 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 mobilization 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 provide 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 Koos 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 \( p < 0.05 \). Anderson 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 acupuncture (dry-needling) is effective compared to injection therapy in acute low back pain. (Level I)
- Adverse effects of acupuncture 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 diurnal 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). These RCTs were located that compared use of a simple analgesic with another treatment.

Two studies (Wiesed et al. 1980; Milgrom et al. 1993) involved male military recruits and were considered low quality in the reviews. Wiesed 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 electroacupuncture 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; Zylkoergold 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 leave in the exercise group at six and 12 weeks and no differences in functional status after one year (n = 103). Stankovic and Johneck (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; Farrel 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 (Faas et al. 1993; Seferlis et al. 1998; Underwood and Morgan 1998) or NSAID use (Waterworth and Hunter 1985). Two (Farrel 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 Johneck 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 Johneck 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 Hooogen 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 Hooogen (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 co interventions 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; M aher 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 (H asembrin 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. H asembrin 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 prolapses; 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 included 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. Vallejones 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. Hohler 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.


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 \((p=0.01)\). 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.
> 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 intervention 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 spongifier 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 spongifier homeopathic gel or cremol capsici for treatment of acute low back pain. (Level II)

### Traction


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. (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 extension; 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).

**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 III)**

**References**


Chapter 4 • Acute Low Back Pain


Brockow T, Dillner A, Franke E, Resch KL (2001). Analgesic effective-


Browning R, Jackson JL, O’Malley PG (2001). Cyclobenzaprine and


Carey TS, Garrett J, Jackman A, McLoughlin C, Fryer J, Smucker DR


Chapter 4 • Acute Low Back Pain


Chapter 4 • Acute Low Back Pain


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


Evidence-based Management of Acute Musculoskeletal Pain


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, inferi- orly 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
- EM BASE — 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), EM BASE, 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:

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
<th>Exclusion criteria</th>
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</thead>
<tbody>
<tr>
<td>Systematic reviews, cross-sectional studies, case series, case reports</td>
<td>Chronic pain</td>
</tr>
<tr>
<td>Adults</td>
<td></td>
</tr>
<tr>
<td>Specific diseases and conditions including those referring pain to the thoracic spine</td>
<td></td>
</tr>
<tr>
<td>Non-specific diseases and conditions of the thoracic spine</td>
<td></td>
</tr>
<tr>
<td>Peer-reviewed journal</td>
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</table>

<table>
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<tr>
<th>Inclusion criteria</th>
<th>Exclusion criteria</th>
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</thead>
<tbody>
<tr>
<td>Systematic reviews, cohort studies</td>
<td>Chronic pain</td>
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<tr>
<td>Adults</td>
<td></td>
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<tr>
<td>Non-specific diseases and conditions of the thoracic spine</td>
<td></td>
</tr>
<tr>
<td>Peer-reviewed journal</td>
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</table>

**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:

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
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<tbody>
<tr>
<td>Systematic reviews, randomised controlled trials, cohort studies, case-control studies</td>
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<tr>
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<tr>
<td>Non-specific diseases and conditions of the thoracic spine</td>
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<tr>
<td>Peer-reviewed journal</td>
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<tr>
<td>Adults</td>
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<tr>
<td>Non-specific diseases and conditions of the thoracic spine</td>
<td></td>
</tr>
<tr>
<td>Peer-reviewed journal</td>
<td></td>
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</tbody>
</table>

**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:

<table>
<thead>
<tr>
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<tbody>
<tr>
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<td>Chronic pain</td>
</tr>
<tr>
<td>Adults</td>
<td></td>
</tr>
<tr>
<td>Non-specific diseases and conditions of the thoracic spine</td>
<td></td>
</tr>
<tr>
<td>Peer-reviewed journal</td>
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</table>

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</tr>
<tr>
<td>Adults</td>
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<tr>
<td>Non-specific diseases and conditions of the thoracic spine</td>
<td></td>
</tr>
<tr>
<td>Peer-reviewed journal</td>
<td></td>
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</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Management Plan</th>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>It is recommended that the clinician and patient develop a management plan for acute musculoskeletal pain comprising the elements of assessment, management and review:</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
<tr>
<td>• 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.</td>
<td></td>
</tr>
<tr>
<td>• Management — Provide information, assurance and advice to resume normal activity and discuss other options for pain management as needed.</td>
<td></td>
</tr>
<tr>
<td>• Review — Reassess the pain and revise the management plan as required.</td>
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</table>

<table>
<thead>
<tr>
<th>Non-Pharmacological Interventions</th>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>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.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pharmacological Interventions</th>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific pharmacological interventions may be required to relieve pain; such agents can be used in conjunction with non-pharmacological interventions.</td>
<td>CONSENSUS: Steering Committee; NHMRC 1999b</td>
</tr>
<tr>
<td>Paracetamol or other simple analgesics, administered regularly, are recommended for relief of mild to moderate acute musculoskeletal pain.</td>
<td>CONSENSUS: Steering Committee; NHMRC 1999b</td>
</tr>
<tr>
<td>Where paracetamol is insufficient for pain relief, a non-steroidal anti-inflammatory (NSAID) medication may be used, unless contraindicated.</td>
<td>CONSENSUS: Steering Committee; NHMRC 1999b</td>
</tr>
<tr>
<td>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.</td>
<td>CONSENSUS: Steering Committee; NHMRC 1999b</td>
</tr>
<tr>
<td>Adjuvant agents such as anticonvulsants and antidepressants are not recommended in the management of acute musculoskeletal pain.</td>
<td>CONSENSUS: Steering Committee; NHMRC 1999b</td>
</tr>
<tr>
<td>Any benefits from muscle relaxants may be outweighed by their adverse effects, therefore they cannot be routinely recommended.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
</tbody>
</table>

Summary of Key Messages: Effective Communication

<table>
<thead>
<tr>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONSENSUS: Steering Committee</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th><strong>DIAGNOSIS</strong></th>
<th><strong>EVIDENCE LEVEL</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aetiology and Prevalence</strong></td>
<td></td>
</tr>
<tr>
<td>Pain may be referred to the upper thoracic spine from visceral structures and cervical spinal structures or arise in the thoracic interspinous ligaments, paraverterbral muscles and zygapophyseal joints</td>
<td><em>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</em></td>
</tr>
<tr>
<td>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.</td>
<td><em>LEVEL IV: Ross et al. 1994; Patel et al. 1991; Huang et al. 1994</em></td>
</tr>
<tr>
<td>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.</td>
<td><em>LEVEL IV: Deyo and Diehl. 1988</em></td>
</tr>
<tr>
<td><strong>History</strong></td>
<td></td>
</tr>
<tr>
<td>History serves to differentiate sources of acute thoracic spinal pain to identify features of potentially serious conditions; however it carries little diagnostic weight.</td>
<td>CONSENSUS: Flynn 1996; Kenna and Murtagh 1989; Corrigan and Maitland 1988</td>
</tr>
<tr>
<td><strong>Physical Examination</strong></td>
<td></td>
</tr>
<tr>
<td>The reliability of palpation for tenderness of the thoracic spine is good but its validity is unknown.</td>
<td><em>LEVEL IV: Christensen et al. 2002</em></td>
</tr>
<tr>
<td>The reliability of motion palpation of the thoracic spine is marginal.</td>
<td><em>LEVEL IV: Love et al. 1987; Christensen et al. 2002</em></td>
</tr>
<tr>
<td>Following blunt trauma, a negative clinical examination in the presence of a clear sensorium makes a thoracic spinal fracture unlikely.</td>
<td><em>LEVEL IV: Durham et al. 1995; Samuels and Kerstein 1993</em></td>
</tr>
<tr>
<td>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.</td>
<td><em>LEVEL IV: Deyo et al. 1988; Malawski et al. 1991; Durham et al. 1995; Samuels and Kerstein 1993</em></td>
</tr>
<tr>
<td><strong>Ancillary Investigations</strong></td>
<td></td>
</tr>
<tr>
<td>In the absence of trauma, plain radiography is of limited use in defining the cause of pain.</td>
<td><em>LEVEL IV: Wood et al. 1995; Nathan 1962; Crawford and Singer 1995</em></td>
</tr>
<tr>
<td>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.</td>
<td><em>LEVEL IV: Frankel et al. 1994; Durham et al. 1995; Meldon and Moeottus. 1995; Samuels and Kerstein 1993</em></td>
</tr>
<tr>
<td>In the presence of trauma, x-ray 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.</td>
<td><em>LEVEL IV: Samuels and Kerstein 1993; Durham et al. 1995</em></td>
</tr>
<tr>
<td>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.</td>
<td><em>LEVEL IV: Keene et al. 1982</em></td>
</tr>
<tr>
<td>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.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
<tr>
<td><strong>Terminology</strong></td>
<td></td>
</tr>
<tr>
<td>The appropriate labels for non-specific ‘mechanical’ thoracic spinal pain are ‘thoracic spinal pain of unknown origin’ or ‘somatic thoracic spinal pain’.</td>
<td>CONSENSUS: Merskey and Bogduk 1994</td>
</tr>
</tbody>
</table>

## PROGNOSIS

<table>
<thead>
<tr>
<th><strong>EVIDENCE LEVEL</strong></th>
</tr>
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<tbody>
<tr>
<td>There is a lack of published data on the natural history and influence of prognostic risk factors for acute thoracic spinal pain.</td>
</tr>
</tbody>
</table>
Acute Thoracic Spinal Pain continued

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.

On the other hand, there is also evidence that...

- 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>
<thead>
<tr>
<th>Table 5.1</th>
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</thead>
<tbody>
<tr>
<td>A Systematic Classification of Causes of Acute Thoracic Pain</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Painful Conditions of the Thoracic Spine</th>
<th>Conditions Referring Pain to the Thoracic Spine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serious conditions</td>
<td>Disorders of cervical zygapophyseal joints, muscles and discs</td>
</tr>
<tr>
<td>Mechanical conditions</td>
<td>Myocardial ischaemia; dissecting thoracic aortic aneurysm; peptic ulcer; acute cholecystitis; pancreatitis; renal colic; acute pyelonephritis</td>
</tr>
<tr>
<td>Infection; fracture; neoplastic disorders; inflammatory disorders; disc protrusion</td>
<td>Discogenic pain; zygapophyseal joint pain; other structures</td>
</tr>
</tbody>
</table>

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).
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 thoracic spine (Weinberg et al. 1972; Bywaters 1974); verte and costovertebral joints as well as the discs in the (Hastings 1994). It has been shown to involve the costotransverse and costovertebral joints as well as the discs 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.

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Inflammation
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 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 (Bhambhan 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

<table>
<thead>
<tr>
<th>Aspect of Presentation</th>
<th>Definition</th>
<th>Proportion of all Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disease Onset</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute</td>
<td>High fever, violent pains and malaise</td>
<td>50%</td>
</tr>
<tr>
<td>Subacute</td>
<td>Fever, moderate pain and slight malaise</td>
<td>33%</td>
</tr>
<tr>
<td>Mild</td>
<td>Afebrile, local pain and otherwise well</td>
<td>17%</td>
</tr>
<tr>
<td>Clinical Features</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infection</td>
<td>fistula, abscess, meningitis</td>
<td>20%</td>
</tr>
<tr>
<td>Neuromotor deficit</td>
<td>paralysis/paresis</td>
<td>5%</td>
</tr>
<tr>
<td>Interval from Onset of Symptoms to Presentation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 3 months</td>
<td></td>
<td>23%</td>
</tr>
<tr>
<td>3–6 months</td>
<td></td>
<td>17%</td>
</tr>
<tr>
<td>6–12 months</td>
<td></td>
<td>13%</td>
</tr>
<tr>
<td>1–8 years</td>
<td></td>
<td>47%</td>
</tr>
</tbody>
</table>

Note: Based on data from Malawski and Lukawski (1992).
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% with more severe back pain and disability. In a cross sectional population-based study, 2992 Caucasian women were x-rayed 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 (Russel 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 (zygapophyseal) 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 |
|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|
| Back pain since previous examination            | Age Adjusted Odds Ratio | 95% CI               | Multiply Adjusted Odds Ratio | 95% CI               |
| Any severity of back pain > none in last 30 days| 5.5                   | 2.5, 12.2            | 4.9                   | 2.1, 11.2            |
| ≥ 3 back disability responses                  | 4.9                   | 2.2, 11.0            | 4.2                   | 1.8, 10.0            |

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 M urtagh 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 (McEnzi 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 x-rays 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.
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 et al. 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 (Magne 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 derived 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).

**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.

\[ \text{Evidence-based Management of Acute Musculoskeletal Pain} \]
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.

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Entities</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rare conditions</td>
<td>Primary and secondary neoplasia</td>
<td>0.63% (Deyo et al. 1988)</td>
</tr>
<tr>
<td></td>
<td>Disc protrusion</td>
<td>0.15% of all surgically treated disc abnormalities (Love and Schorn 1965)</td>
</tr>
<tr>
<td></td>
<td>Rheumatoid arthritis</td>
<td>Unknown</td>
</tr>
<tr>
<td></td>
<td>Spinal infection</td>
<td>&lt;0.01% (Liang and Komaroff 1982)</td>
</tr>
<tr>
<td>Uncommon</td>
<td>Traumatic fractures</td>
<td>Unknown</td>
</tr>
<tr>
<td>Common</td>
<td>Somatic pain</td>
<td>16% of presentations with back pain in primary care (Deyo and Diehl 1988)</td>
</tr>
<tr>
<td></td>
<td>Osteoporotic fractures</td>
<td>6.5% in 50–59 year olds and 77.8% in &gt; 90 year olds (Melton et al. 1989)</td>
</tr>
</tbody>
</table>

**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.
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 Moiré 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 palpative 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.

M inucci (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>
<thead>
<tr>
<th>Level of Agreement</th>
<th>Observed Agreement</th>
<th>Expected Agreement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total agreement (4–0, 0–4)</td>
<td>61</td>
<td>20.75</td>
</tr>
<tr>
<td>Slight agreement (3–1, 1–3)</td>
<td>56</td>
<td>75</td>
</tr>
<tr>
<td>Total disagreement (1–2)</td>
<td>33</td>
<td>54.25</td>
</tr>
</tbody>
</table>

Note: Based on data from Johnston et al. (1983).
Chapter 5 • Acute Thoracic Spinal Pain

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 (McCorm 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; Hwang 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 conditions.
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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 (McCullister 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 5.7

<table>
<thead>
<tr>
<th>Feature or Risk Factor</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minor trauma (if &gt; 50 years, history of osteoporosis and taking corticosteroids)</td>
<td>Fracture</td>
</tr>
<tr>
<td>Major trauma</td>
<td></td>
</tr>
<tr>
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<td></td>
</tr>
<tr>
<td>Night sweats</td>
<td></td>
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<tr>
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</tr>
<tr>
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<td></td>
</tr>
<tr>
<td>Age &gt; 50</td>
<td></td>
</tr>
<tr>
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<td></td>
</tr>
<tr>
<td>Unexplained weight loss</td>
<td></td>
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<tr>
<td>Pain at multiple sites</td>
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<tr>
<td>Pain at rest</td>
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<tr>
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Alerting Features of Serious Conditions Associated with Acute Thoracic Spinal Pain

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Alerting Features of Serious Conditions Associated with Acute Thoracic Spinal Pain

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Evidence-based Management of Acute Musculoskeletal Pain

- 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 x-rayed, 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.

### Table 5.8
Comparison of the Location of Pain in Patients with Scheuermann's Kyphosis with Age and Sex Matched Controls

<table>
<thead>
<tr>
<th>Location of Pain</th>
<th>No. of Patients (n = 67)</th>
<th>No. of Controls (n = 34)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No pain</td>
<td>21 (31%)</td>
<td>21 (62%)</td>
</tr>
<tr>
<td>Back pain</td>
<td>41 (61%)</td>
<td>5 (15%)</td>
</tr>
<tr>
<td>Lower extremity</td>
<td>3 (4%)</td>
<td>5 (15%)</td>
</tr>
<tr>
<td>Back and lower extremity</td>
<td>2 (3%)</td>
<td>3 (9%)</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Age</th>
<th>N</th>
<th>Number with Fractures</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 60 years</td>
<td>82</td>
<td>9 (11%)</td>
</tr>
<tr>
<td>≥ 60 years</td>
<td>17</td>
<td>6 (35%)</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Clinical Status</th>
<th>Size of Study</th>
<th>Prevalence of Fractures</th>
<th>95% Confidence Limits</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td></td>
</tr>
<tr>
<td>Clinical features suggesting injury</td>
<td>24</td>
<td>14</td>
<td>58</td>
</tr>
<tr>
<td></td>
<td>186</td>
<td>32*</td>
<td>17</td>
</tr>
<tr>
<td>No clinical features of injury</td>
<td>55</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>128</td>
<td>10*#</td>
<td>8</td>
</tr>
<tr>
<td>Equivocal features or altered sensorium</td>
<td>20</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>29</td>
<td>5</td>
<td>17</td>
</tr>
</tbody>
</table>

Note: *18 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

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 MR1 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

<table>
<thead>
<tr>
<th>Category</th>
<th>Retrospective Data (n = 1485) Odds Ratio (CI 90%)</th>
<th>Prospective Data Relative Risk (CI 90%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Back pain or tenderness</td>
<td>9 (1.5–13.9)</td>
<td>1 (0.8–2.9)</td>
</tr>
<tr>
<td>Cervical spine fracture</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>Fall ≥ 3m</td>
<td>8 (2.3–16.9)</td>
<td>2 (1.5–4.3)</td>
</tr>
<tr>
<td>Neurologic deficit</td>
<td>10 (3.1–31.6)</td>
<td>N/A</td>
</tr>
<tr>
<td>Ejection from vehicle</td>
<td>6 (2.2–6.3)</td>
<td>2 (1.5–4.9)</td>
</tr>
<tr>
<td>Crash ≥ 80 km/h</td>
<td>2 (1.4–10.1)</td>
<td>2 (1.4–10.1)</td>
</tr>
<tr>
<td>GCS ≤ 8</td>
<td>2 (0.8–2.6)</td>
<td>0</td>
</tr>
</tbody>
</table>

**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).

(Anwand 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 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
Evidence-based Management of Acute Musculoskeletal Pain


Charlton K. Approaches to demonstration of changes of vertebral subluxation in the cervical spine. 1991 M Phil thesis, Griffith University, Nathan, Queensland.


Chapter 5 • Acute Thoracic Spinal 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:

- 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)

**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:

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
<th>Exclusion criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systematic reviews, cross-sectional studies, case series, case reports</td>
<td>Chronic pain</td>
</tr>
<tr>
<td>Adults</td>
<td>Specific diseases and conditions (to identify serious conditions)</td>
</tr>
<tr>
<td>Acute idiopathic neck pain</td>
<td>Acute whiplash-associated neck pain</td>
</tr>
</tbody>
</table>

**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:

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
<th>Exclusion criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systematic reviews, cohort studies</td>
<td>Chronic pain</td>
</tr>
<tr>
<td>Adults</td>
<td>Specific diseases</td>
</tr>
<tr>
<td>Acute idiopathic neck pain</td>
<td>Serious conditions (cervical fracture, infection, neurological conditions, tumour)</td>
</tr>
<tr>
<td>Acute whiplash-associated neck pain</td>
<td>Thoracic spinal pain, throat pain, cervicogenic headache, cervicoradicular pain</td>
</tr>
</tbody>
</table>

**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:

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
<th>Exclusion criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systematic reviews, randomised controlled trials (RCTs)</td>
<td>Chronic pain</td>
</tr>
<tr>
<td>Acute idiopathic neck pain</td>
<td>Specific diseases</td>
</tr>
<tr>
<td>Acute whiplash-associated neck pain</td>
<td>Serious conditions (cervical fracture, infection, neurological conditions, tumour)</td>
</tr>
<tr>
<td>Adults</td>
<td>Thoracic spinal pain, throat pain, cervicogenic headache, cervicoradicular pain</td>
</tr>
</tbody>
</table>
Evidence-based Management of Acute Musculoskeletal Pain

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

<table>
<thead>
<tr>
<th>Management Plan</th>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
</table>
| 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 |

<table>
<thead>
<tr>
<th>Non-Pharmacological Interventions</th>
<th>CONSENSUS: Steering Committee</th>
</tr>
</thead>
<tbody>
<tr>
<td>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.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pharmacological Interventions</th>
<th>CONSENSUS: Steering Committee; NHMRC 1999b</th>
</tr>
</thead>
</table>
| Specific pharmacological interventions may be required to relieve pain; such agents can be used in conjunction with non-pharmacological interventions.  
Paracetamol or other simple analgesics, administered regularly, are recommended for relief of mild to moderate acute musculoskeletal pain.  
Where paracetamol is insufficient for pain relief, a non-steroidal anti-inflammatory (NSAID) medication may be used, unless contraindicated.  
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.  
Adjuvant agents such as anticonvulsants and antidepressants are not recommended in the management of acute musculoskeletal pain.  
Any benefits from muscle relaxants may be outweighed by their adverse effects, therefore they cannot be routinely recommended. | CONSENSUS: Steering Committee; NHMRC 1999b |

CONSENSUS: Steering Committee
### Summary of Key Messages: Effective Communication

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

### Summary of Key Messages: Acute Neck Pain

#### Aetiology and Prevalence

<table>
<thead>
<tr>
<th>Acute neck pain is most commonly idiopathic or attributed to a whiplash accident; serious causes of acute neck pain are rare (&lt;1%).</th>
<th>*LEVEL III-3: Based on cross-sectional and prospective radiological surveys (Heller et al. 1983; Johnson and Lucas 1997)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Degenerative changes, osteoarthrosis or spondylosis of the neck are neither causes nor risk factors for idiopathic neck pain.</td>
<td>*LEVEL III: Based on epidemiological and radiological surveys (van der Donk et al. 1991; Fridenberg and Milner 1963)</td>
</tr>
<tr>
<td>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.</td>
<td>*LEVEL III: Based on multiple epidemiological surveys (Makela et al. 1991; Kamwendo et al. 1991a; Linton and Kamwendo 1989; Vassejien et al. 1995; Fredriksson et al. 2002; Ariens et al. 2001)</td>
</tr>
<tr>
<td>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.</td>
<td>*LEVEL III: Based on a prospective epidemiological study (Berglund et al. 2000)</td>
</tr>
</tbody>
</table>

#### 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; Mor et al. 1985; Youdas et al. 1991; Viikari-Juntura 1987 |
Despite limitations, physical examination is an opportunity to identify features of potentially serious conditions.

Tenderness and restricted cervical range of movement correlate well with the presence of neck pain, confirming a local cause for the pain.

**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.

In symptomatic patients with a history of trauma, radiography is indicated according the Canadian C-Spine Rule.

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.

Acute neck pain in conjunction with features alerting to the possibility of a serious underlying condition is an indication for MRI.

**Terminology**

Except for serious conditions, precise identification of the cause of neck pain is unnecessary.

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'.

**PROGNOSIS**

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.

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.

Psychosocial factors are not determinants of chronicity in whiplash-associated neck pain.

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.

**INTERVENTIONS**

**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.

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.
### 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.

**Evidence:**
- 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.

**Evidence:**
- 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.

**Evidence:**
- No Level I or II evidence

**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.

**Evidence:**
- No Level I or II evidence

**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.

**Evidence:**
- 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.

**Evidence:**
- 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.

**Evidence:**
- No Level I or II evidence

**Electrotherapy**

There is insufficient evidence that electrotherapy is effective compared to no treatment in acute neck pain.

**Evidence:**
- 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.

**Evidence:**
- 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.

**Evidence:**
- 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.

**Evidence:**
- Based on systematic reviews (Karjalainen et al. 2002) that identified two controlled trials and two subsequent trials that all involved mixed populations
### Acute Neck Pain continued

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Evidence of No Benefit</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Muscle Relaxants</strong></td>
<td>There are no randomised controlled trials investigating the efficacy of muscle relaxants for the treatment of acute neck pain.</td>
</tr>
<tr>
<td>Muscle relaxants are no more effective than placebo for neck pain in mixed populations.</td>
<td>Level I, II: Based on a systematic review (Aker et al. 1996) of two studies plus one additional study, all involving mixed populations.</td>
</tr>
<tr>
<td>Drowsiness, dizziness and dependency are common adverse effects of muscle relaxants.</td>
<td>Level I: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 1997)</td>
</tr>
<tr>
<td><strong>Neck School</strong></td>
<td>There are no randomised controlled trials on the effect of neck school for acute neck pain.</td>
</tr>
<tr>
<td>Neck school appears no more effective than no treatment for neck pain in mixed populations.</td>
<td>Level II: Based on one controlled trial (Kamwendo and Linton 1991) involving a mixed population</td>
</tr>
<tr>
<td><strong>Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)</strong></td>
<td>There are no randomised controlled trials on the effectiveness of NSAIDs for acute neck pain.</td>
</tr>
<tr>
<td>There is evidence that NSAIDs are no more effective than placebo ultrasound for neck pain in mixed populations.</td>
<td>Level I: Based on systematic review (Aker et al. 1996) that located two studies involving mixed populations.</td>
</tr>
<tr>
<td>Serious adverse effects of NSAIDs include gastrointestinal complications. (e.g. bleeding, perforation)</td>
<td>Level I: Based on systematic reviews (Bigos et al. 1994; Henry et al. 1996)</td>
</tr>
<tr>
<td><strong>Patient Education</strong></td>
<td>There are no randomised controlled trials investigating the effect of patient education as a single strategy in the treatment of acute neck pain.</td>
</tr>
<tr>
<td></td>
<td>Level I, II: Based on a systematic review (Harms-Ringdahl and Nachemson 2000; Gross et al. 2002b).</td>
</tr>
<tr>
<td><strong>Spray and Stretch Therapy</strong></td>
<td>There are no randomised controlled trials investigating the effect of spray and stretch therapy in acute neck pain.</td>
</tr>
<tr>
<td>Spray and stretch therapy appears no more effective than placebo for neck pain in mixed populations.</td>
<td>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).</td>
</tr>
<tr>
<td><strong>Traction</strong></td>
<td>There are no randomised controlled trials investigating the effectiveness of traction for acute neck pain.</td>
</tr>
<tr>
<td>In mixed populations, there is evidence that traction is of no benefit compared to a range of other interventions for neck pain.</td>
<td>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.</td>
</tr>
<tr>
<td><strong>Transcutaneous Electrical Nerve Stimulation (TENS)</strong></td>
<td>There is insufficient evidence of benefit from TENS compared to a collar or manual therapy in acute neck pain.</td>
</tr>
<tr>
<td></td>
<td>Level I: Based on a systematic review (Gross et al. 2002b) that identified one controlled trial (Nordemar and Thorner 1982) with equivocal results.</td>
</tr>
</tbody>
</table>

### Evidence of No Benefit

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Evidence of No Benefit</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Collars</strong></td>
<td>Soft collars are not effective for acute neck pain compared to advice to resume normal activity and other interventions.</td>
</tr>
<tr>
<td></td>
<td>Level I, II: Based on a systematic review (Harms-Ringdahl and Nachemson 2000) and multiple controlled trials.</td>
</tr>
</tbody>
</table>

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. 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).

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 Musha 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 H art 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 Allegria 1994; Lobitz and Grate 1995). However, motor and sensory deficits usually develop within hours of the onset of pain (Williams and Allegria 1994; Beatty and Winstow 1984; Atsumae 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

<table>
<thead>
<tr>
<th>Prevalence</th>
<th>Threatening</th>
<th>Non-Threatening</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rare (&lt;1%)</td>
<td>Spinal tumours</td>
<td>Retrophyaryngeal tendonitis</td>
</tr>
<tr>
<td></td>
<td>Spinal infection</td>
<td>Rheumatoid arthritis</td>
</tr>
<tr>
<td></td>
<td>Epidural haematoma</td>
<td>Spondylarthropathies</td>
</tr>
<tr>
<td></td>
<td>Aneurysms</td>
<td>Fractures</td>
</tr>
<tr>
<td>Common</td>
<td>Fractures</td>
<td>Torticolis</td>
</tr>
<tr>
<td>Uncommon (&lt;5%)</td>
<td></td>
<td>Idiopathic</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Whiplash</td>
</tr>
</tbody>
</table>

Evidence-based Management of Acute Musculoskeletal Pain
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; Bioussé 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; Hirsh 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 (Harnden and Halia 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 (Elmer 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 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; Macias 1985; Roberge et al. 1988; McNamara 1988; Kreipke et al. 1989; Hoffmann et al. 1992; Gerrels 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 Deyar 1968; Jayakrishnan and Teasdale 2000; Wise et al. 1997; Fielding and Hawkin 1977; van Holseeck and Mackay 1968; Jayakrishnan and Teasdale 2000; Wise et al. 1997). Neurological causes include basal ganglion disorders and phenothiazine toxicity. A putative mechanical cause is extrapment of a meniscoid in a cervical zygapophyseal joint (Merce 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 (Setzé 1958; Signoret et al. 1986), the laminae and articular processes of C2 (Setzé 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 (Schäfner 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).

SPOUSIOUS 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 (Hrinck 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. 1995; 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

<table>
<thead>
<tr>
<th>Medical</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zygapophyseal osteoarthrosis</td>
<td>van der Donk et al. 1991</td>
</tr>
<tr>
<td>Degenerative disc disease</td>
<td>van der Donk et al. 1991</td>
</tr>
<tr>
<td>Previous pain symptoms</td>
<td>Westgard and Jansen 1992</td>
</tr>
<tr>
<td>Social</td>
<td></td>
</tr>
<tr>
<td>Marital status</td>
<td>Westgard and Jansen 1992</td>
</tr>
<tr>
<td>Children</td>
<td>Westgard and Jansen 1992,</td>
</tr>
<tr>
<td>Economic status</td>
<td>Westgard and Jansen 1992; Andersen and Gaardboe 1993b</td>
</tr>
<tr>
<td>Living conditions</td>
<td>Westgard and Jansen 1992</td>
</tr>
<tr>
<td>Exercise</td>
<td>Westgard and Jansen 1992</td>
</tr>
<tr>
<td>Workload at home</td>
<td>Westgard and Jansen 1992</td>
</tr>
<tr>
<td>Activities outside work</td>
<td>Westgard and Jansen 1992</td>
</tr>
<tr>
<td>Smoking</td>
<td>Makela et al. 1991</td>
</tr>
<tr>
<td>Occupational</td>
<td></td>
</tr>
<tr>
<td>Prolonged sitting at a work station</td>
<td>Kamwendo et al. 1991a (Part I)</td>
</tr>
<tr>
<td>Ergonomic variables</td>
<td>Kamwendo et al. 1991b (Part II)</td>
</tr>
</tbody>
</table>

Table 6.3
Medical, Social and Occupational Risk Factors Weakly Associated with Neck Pain

<table>
<thead>
<tr>
<th>Medical</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous injury</td>
<td>Makela et al. 1991</td>
</tr>
<tr>
<td>Social</td>
<td></td>
</tr>
<tr>
<td>Female gender</td>
<td>van der Donk et al. 1991</td>
</tr>
<tr>
<td>Education level &lt; 8 years</td>
<td>Makela et al. 1991</td>
</tr>
<tr>
<td>Education level 8-12 years</td>
<td>Makela et al. 1991</td>
</tr>
<tr>
<td>Occupation</td>
<td></td>
</tr>
<tr>
<td>Clerical</td>
<td>Makela et al. 1991</td>
</tr>
<tr>
<td>Industry</td>
<td>Makela et al. 1991</td>
</tr>
<tr>
<td>Agriculture</td>
<td>Makela et al. 1991</td>
</tr>
<tr>
<td>Occupational</td>
<td></td>
</tr>
<tr>
<td>Physical stress at work</td>
<td>Makela et al. 1991</td>
</tr>
<tr>
<td>Mental stress at work</td>
<td>Makela et al. 1991</td>
</tr>
<tr>
<td>Working with machines</td>
<td>Kamwendo et al. 1991a (Part I)</td>
</tr>
</tbody>
</table>

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

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 Middelx Ltd Hospital Questionnaire and the Maudsley Personality Inventory (Piddi-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 cooperation between workers (Linton and Kamwendo 1989; Ariens et al. 2001)
- lack of camaraderie (Linton and Kamwendo 1989)
- lack of possibility to influence or vary one’s 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. M any 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 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.

**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; Barsness et al. 1995; Lord et al. 1996; Grubb and Kelly 2000; Schellhas et al. 1996) have shown that pain stemming from the zygopophyseal 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.

**Time 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:

Table 6.5
Alerting Features of Serious Conditions Associated with Acute Neck Pain

<table>
<thead>
<tr>
<th>Feature or Risk Factor</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms and signs of infection (e.g. fever, night sweats)</td>
<td>Infection</td>
</tr>
<tr>
<td>Risk factors for infection (e.g. underlying disease process, immunosuppression, penetrating wound, exposure to infectious diseases)</td>
<td></td>
</tr>
<tr>
<td>History of trauma</td>
<td>Fracture</td>
</tr>
<tr>
<td>Use of corticosteroids</td>
<td></td>
</tr>
<tr>
<td>Past history of malignancy</td>
<td>Tumour</td>
</tr>
<tr>
<td>Age &gt; 50 years</td>
<td></td>
</tr>
<tr>
<td>Failure to improve with treatment</td>
<td></td>
</tr>
<tr>
<td>Unexplained weight loss</td>
<td></td>
</tr>
<tr>
<td>Dysphagia, headache, vomiting</td>
<td></td>
</tr>
<tr>
<td>Neurological symptoms in the limbs</td>
<td>Neurological condition</td>
</tr>
<tr>
<td>Cerebrovascular symptoms or signs, anticoagulant use</td>
<td>Cerebral or spinal hemorrhage</td>
</tr>
<tr>
<td>Cardiovascular risk factors, transient ischaemic attack</td>
<td>Vertebral or carotid aneurysm</td>
</tr>
</tbody>
</table>
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 (Drouiche 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 asymptomatic; the other had a fracture of the lamina of C6.

Occult fractures in asymptomatic patients are rare and are reported in case reports (Hambrayrah 1972; Mault and Sachatello 1977; Bresler and Rich 1982; Walter et al. 1984; Haines 1986; Ogden and Dunn 1986; McKee et al. 1990; Magee 1991; Magee 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; Miviris et al. 1989; Roberge 1993). Accordingly, provided the operational criteria are strictly followed, occult fracture is unlikely in the absence of symptoms.

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 (M.C. Amara 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.

If 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; McDonald 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 interval ranging from 98% to 100%.

**Key Message**
In symptomatic patients with a history of trauma, radiography is indicated according to the Canadian C-Spine Rule. (*Level III*)

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**Figure 6.1**
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; Wilibeger and M aron 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.

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 H andal 1995; Barnsley 1998).

Descriptive studies (Mirvis et al. 1989; Gerrets 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 (Schlehauf 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 (Blacksn 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; Yekin 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 anteroposterior 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; T eresi 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 myofacial 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, 29% 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 (Kash 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. Any 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>
<thead>
<tr>
<th>Demographic</th>
<th>Older Age</th>
<th>Female gender</th>
<th>Having dependents</th>
<th>Not employed full-time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical</td>
<td>Neck pain on palpation</td>
<td>Muscle pain</td>
<td>Headache</td>
<td>Pain or numbness radiating to the upper limb</td>
</tr>
</tbody>
</table>

Table 6.6

Factors Associated with Chronic Neck Pain After Whiplash: Insurance Data

Note: Based on insurance claims data from Suissa et al. 2001 and Harder et al. 1998.
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; Norris and Watt 1983). Competent follow-up studies have shown chronicity to be independent of litigation (Norris and Watt 1983; Maaimaris et al. 1988; Pennie and Agambarn 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 (Spitzer 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,

### Table 6.7

Demographic and Clinical Factors Associated with Chronic Neck Pain After Whiplash

<table>
<thead>
<tr>
<th>Demographic</th>
<th>Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Past History</td>
<td>Of headache</td>
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<tr>
<td></td>
<td>Of head injury</td>
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<tr>
<td>Clinical</td>
<td>Impaired neck movements</td>
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<tr>
<td></td>
<td>Initial pain intensity</td>
</tr>
<tr>
<td></td>
<td>Initial headache intensity</td>
</tr>
<tr>
<td>Psychometric</td>
<td>Nervousness score</td>
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<tr>
<td></td>
<td>Neuroticism score</td>
</tr>
<tr>
<td></td>
<td>Test score on focused attention</td>
</tr>
</tbody>
</table>

Note: Based on data from Radanov et al. 1991.
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 Harrms-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-Kiukaanniemi 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-Kiukaanniemi 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 and calcic iontophoresis. At one and six months after treatment, pain scores were significantly less in the multi-modal group.
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 interaction.
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 (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)

**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).

**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 Azelman 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.

**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 (opoid 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).

**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 one only 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 Organ 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. Hendricks and Organ (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 are no randomised controlled studies investigating the effect of multi-disciplinary treatment in acute neck pain. (No Level I or II studies)

**Multi-Disciplinary Treatment**

Multi-disciplinary treatment comprises a combination of treatment modalities, including physical treatments for musculoskeletal pain and psychological, behavioural and educational interventions.


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 muscle relaxants on neck pain.

> Muscle relaxants 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, tiredness 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 Messages**

> 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) located 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 effect 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 Nachelson (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; Rosenfeld et al. 2000) and no more effective than TENS (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; Nordemar and Thorner 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


Bernstein SA (1975). Acute cervical pain associated with soft-tissue calcium deposition anterior to the interspace of the first and


Chapter 6 • Acute Neck Pain


Chapter 6 • Acute Neck 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.
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**

<table>
<thead>
<tr>
<th><strong>DIAGNOSIS</strong></th>
<th><strong>EXCLUSION CRITERIA</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Inclusion criteria</strong></td>
<td>Systematic reviews, cross-sectional studies</td>
</tr>
<tr>
<td><strong>Exclusion criteria</strong></td>
<td>Chronic pain</td>
</tr>
<tr>
<td></td>
<td>Specific diseases and conditions, fracture/disclocation, neoplasm, infection, inflammatory arthropathies, pain referred from/to the shoulder, intrinsic neurological conditions, hemiplegic shoulder pain</td>
</tr>
</tbody>
</table>

**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 |
| **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) |
| **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
• 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

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.

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.

Pharmacological Interventions
Specific pharmacological interventions may be required to relieve pain; such agents can be used in conjunction with non-pharmacological interventions.

Paracetamol or other simple analgesics, administered regularly, are recommended for relief of mild to moderate acute musculoskeletal pain.

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

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.

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

Any benefits from muscle relaxants may be outweighed by their adverse effects, therefore they cannot be routinely recommended.
## Summary of Key Messages: Acute Shoulder Pain

<table>
<thead>
<tr>
<th>DIAGNOSIS</th>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aetiology and Prevalence</td>
<td></td>
</tr>
<tr>
<td>Clinicians should be alert to the potential for rare, serious conditions (e.g. fracture/dislocation, tumour, infection, inflammatory arthropathies) presenting as acute shoulder pain.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
<tr>
<td>Most cases of acute shoulder pain are of ‘mechanical’ origin and can be managed as acute regional pain.</td>
<td><em>LEVEL III-2, III-3: Torstensen and Hollinshead 1999; Chandnani et al. 1992; Milgrom et al. 1995; Sher et al. 1995</em></td>
</tr>
<tr>
<td>Biological factors such as age, female gender, past history and response to repetitive physical tasks may contribute to the development of acute shoulder pain.</td>
<td><em>LEVEL III-3: J ones et al. 1994; Cummings et al. 1995; Sambrook 1996; Ekberg et al. 1995; Skov et al. 1996</em></td>
</tr>
<tr>
<td>Psychosocial factors such as job dissatisfaction and work demands may contribute to the onset of acute shoulder pain.</td>
<td><em>LEVEL III-2: Bergenudd et al. 1994; Ekberg et al. 1995; Marcus et al. 1996; Skov et al. 1996</em></td>
</tr>
<tr>
<td>History</td>
<td></td>
</tr>
<tr>
<td>Information obtained from the history may alert to the presence of a serious condition as the underlying cause of acute shoulder pain.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
<tr>
<td>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.</td>
<td><em>LEVEL III-2: Nørregaard et al. 2002; Litaker et al. 2000</em></td>
</tr>
<tr>
<td>Physical Examination</td>
<td></td>
</tr>
<tr>
<td>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.</td>
<td><em>LEVEL III-2: Calis et al. 2000; MacDonald et al. 2000; Naredo et al. 2002; Ito et al. 1999; Bennett 1998</em></td>
</tr>
<tr>
<td>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.</td>
<td><em>LEVEL III-2: Bamji et al. 1996; Liesdeck et al. 1997; de Winter et al. 1999; Pal et al. 2000; Nørregaard et al. 2002</em></td>
</tr>
<tr>
<td>Despite limitations, physical examination is an opportunity to identify features of potentially serious conditions.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
</tbody>
</table>
**Ancillary Investigations**

<table>
<thead>
<tr>
<th>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.</th>
<th><em>LEVEL III: Numerous studies (Torstensen and Hollinshead 1999; Teefey et al. 2000a,b; Tempelhof et al. 1999; Migrom et al. 1995; Chandnani et al. 1992; Sher et al. 1995; Sher et al. 1998; Blanchard et al. 1999a)</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>There is a need to educate consumers about the limitations of imaging and the risks of radiation exposure.</td>
<td><em>LEVEL IV: Roebuck 1995</em></td>
</tr>
</tbody>
</table>

**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**

| 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 Winden 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 of Benefit**

<table>
<thead>
<tr>
<th>Corticosteroid Injection — 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.</th>
<th>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)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exercises — Exercises may improve shoulder pain compared to placebo in people with rotator cuff disease in both the short and longer term.</td>
<td>LEVEL I: Systematic review of two RCTs (Ginn et al. 1997; Brox et al. 1997)</td>
</tr>
<tr>
<td>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.</td>
<td>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)</td>
</tr>
<tr>
<td>Serious adverse effects of NSAIDs include gastrointestinal complications (e.g. bleeding, perforation)</td>
<td>LEVEL I: Based on a systematic review (Bigos et al. 1994)</td>
</tr>
<tr>
<td>Ultrasound — Therapeutic ultrasound may provide short-term pain relief in calcific tendonitis compared to placebo.</td>
<td>LEVEL I: Systematic review of one RCT in acute shoulder pain (Ebenbichler et al. 1999)</td>
</tr>
</tbody>
</table>

**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) |
Acute Shoulder Pain continued

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
- Serious conditions

**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, osteopenia 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
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)
- polychondritis
- 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 A 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 myotendinous 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 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; O’zaki 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 Zarin 1981; Mabian 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 downwards 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; Ohlsson 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.
Chapter 7 • Acute Shoulder Pain

The problem was described by Duplay (1872) as ‘périarthrite scapulo-humérale’ 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 (Nevaiser and Nevaiser 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 cuff tears to be common in both symptomatic (Torstensen and Hollinshed 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 occurring 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 etiology; 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 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

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Threatening Conditions</th>
<th>Non-threatening Conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rare causes (&lt;1%)</td>
<td>Neoplasia (&lt;1%)</td>
<td>Impingement syndrome (males 4%)</td>
</tr>
<tr>
<td></td>
<td>Septic arthritis (&lt;0.01%)</td>
<td>Frozen shoulder (2%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Osteoporotic fractures (3-6% over age 60)</td>
</tr>
<tr>
<td>Uncommon causes (&lt;5%)</td>
<td>Inflammatory arthropathies (&lt;5% by conditions and age)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rotator cuff tears (&gt;50% over age 50)</td>
</tr>
<tr>
<td>Common causes</td>
<td></td>
<td>Impingement syndrome (females 9%)</td>
</tr>
<tr>
<td>Unknown</td>
<td></td>
<td>Minor sprains</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fractures of healthy bones</td>
</tr>
</tbody>
</table>
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)

Chapter 7 • Acute Shoulder Pain

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 joint may be 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

Periodic 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

A 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.
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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.

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 hypoesthesia, suggesting neurological deficit, and hyperaesthesia, suggesting allodynia,
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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.

---

### Table 7.3

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Kappa</th>
<th>Standard Error</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain deep in the shoulder</td>
<td>0.15</td>
<td>0.08</td>
</tr>
<tr>
<td>Pain in the upper shoulder</td>
<td>0.09</td>
<td>0.07</td>
</tr>
<tr>
<td>Pain in the front of the shoulder</td>
<td>0.15</td>
<td>0.08</td>
</tr>
<tr>
<td>Pain in the back of the shoulder</td>
<td>0.49</td>
<td>0.10</td>
</tr>
<tr>
<td>Pain on lifting or throwing</td>
<td>0.26</td>
<td>0.16</td>
</tr>
<tr>
<td>Pain at rest</td>
<td>0.54</td>
<td>0.08</td>
</tr>
</tbody>
</table>

Note: Based on data from Nørregaard et al. (2002).

### Table 7.4

<table>
<thead>
<tr>
<th>Features in History</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Likelihood Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of trauma</td>
<td>0.36</td>
<td>0.73</td>
<td>1.33</td>
</tr>
<tr>
<td>Pain on shoulder movement</td>
<td>0.98</td>
<td>0.10</td>
<td>1.10</td>
</tr>
<tr>
<td>Night pain</td>
<td>0.88</td>
<td>0.20</td>
<td>1.10</td>
</tr>
</tbody>
</table>

Note: Based on data from Litaker et al. (2000).
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.

Inclinometry (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**

<table>
<thead>
<tr>
<th>Ranges</th>
<th>ICCs (6 physiotherapists)</th>
<th>ICCs (6 rheumatologists)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total shoulder flexion</td>
<td>0.82</td>
<td>0.73</td>
</tr>
<tr>
<td>Total shoulder abduction</td>
<td>0.88</td>
<td>0.56</td>
</tr>
<tr>
<td>External rotation in neutral</td>
<td>0.95</td>
<td>0.30</td>
</tr>
<tr>
<td>External rotation in abduction</td>
<td>0.73</td>
<td>0.19</td>
</tr>
<tr>
<td>Internal rotation in abduction</td>
<td>0.48</td>
<td>0.02</td>
</tr>
<tr>
<td>Hand behind back</td>
<td>0.71</td>
<td>0.80</td>
</tr>
</tbody>
</table>

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**

<table>
<thead>
<tr>
<th>Physical Signs</th>
<th>Kappa</th>
<th>Standard Error</th>
</tr>
</thead>
<tbody>
<tr>
<td>Painful arc</td>
<td>0.93</td>
<td>0.11</td>
</tr>
<tr>
<td>Painful resisted external rotation</td>
<td>0.90</td>
<td>0.11</td>
</tr>
<tr>
<td>Painful resisted internal rotation</td>
<td>0.54</td>
<td>0.11</td>
</tr>
<tr>
<td>Painful resisted abduction</td>
<td>0.81</td>
<td>0.11</td>
</tr>
<tr>
<td>Acromioclavicular joint ‘stress’</td>
<td>0.80</td>
<td>0.11</td>
</tr>
</tbody>
</table>

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 Myynes 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

<table>
<thead>
<tr>
<th>Physical Sign(s)</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Likelihood Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain on passive forward elevation (Neer test)</td>
<td>89%</td>
<td>31%</td>
<td>1.29</td>
</tr>
<tr>
<td>Pain on passive internal rotation at 90° flexion (Hawkins test)</td>
<td>92%</td>
<td>25%</td>
<td>1.23</td>
</tr>
<tr>
<td>Pain on passive horizontal adduction with elbow flexed</td>
<td>82%</td>
<td>28%</td>
<td>1.14</td>
</tr>
<tr>
<td>Painful arc between 60° and 120° of active shoulder abduction</td>
<td>33%</td>
<td>81%</td>
<td>1.74</td>
</tr>
<tr>
<td>Sudden drop on active adduction from horizontal (drop arm test)</td>
<td>8%</td>
<td>97%</td>
<td>2.60</td>
</tr>
<tr>
<td>Shoulder pain on resisted forearm supination (Yergason test)</td>
<td>37%</td>
<td>86%</td>
<td>2.64</td>
</tr>
<tr>
<td>Pain on resisted shoulder flexion (Speed test)</td>
<td>69%</td>
<td>56%</td>
<td>1.57</td>
</tr>
<tr>
<td>All 7 of the above ‘impingement’ tests positive</td>
<td>4%</td>
<td>97%</td>
<td>1.57</td>
</tr>
</tbody>
</table>

Note: Based on data from Calis et al. (2000).

Table 7.8

<table>
<thead>
<tr>
<th>Physical Sign(s)</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Likelihood Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain on passive forward elevation (Neer test) (M)</td>
<td>75%</td>
<td>48%</td>
<td>1.44</td>
</tr>
<tr>
<td>Pain on internal rotation at 90° flexion (Hawkins test) (M)</td>
<td>92%</td>
<td>44%</td>
<td>1.64</td>
</tr>
<tr>
<td>Both Neer and Hawkins tests positive (M)</td>
<td>71%</td>
<td>51%</td>
<td>1.45</td>
</tr>
<tr>
<td>Neer, Hawkins, Yocum and other tests all positive (N)</td>
<td>43%</td>
<td>88%</td>
<td>3.6</td>
</tr>
</tbody>
</table>

Note: Based on data from Macdonald et al. 2000 (M) and Naredo et al. 2002 (N).

Table 7.9

<table>
<thead>
<tr>
<th>Physical Sign(s)</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Likelihood Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain on passive forward elevation (‘Neer test’) (M)</td>
<td>83%</td>
<td>51%</td>
<td>1.69</td>
</tr>
<tr>
<td>Pain on internal rotation at 90° flexion (‘Hawkins test’) (M)</td>
<td>88%</td>
<td>43%</td>
<td>1.54</td>
</tr>
<tr>
<td>Both ‘Neer’ and ‘Hawkins’ tests positive (M)</td>
<td>83%</td>
<td>56%</td>
<td>1.89</td>
</tr>
<tr>
<td>‘Neer’, ‘Hawkins’, ‘Yocum’ and other tests all positive (N)</td>
<td>79%</td>
<td>50%</td>
<td>1.58</td>
</tr>
<tr>
<td>Pain on external rotation in elevation (‘the full can test’) (I)</td>
<td>66%</td>
<td>64%</td>
<td>1.83</td>
</tr>
<tr>
<td>Pain on internal rotation in elevation (‘the empty can test’) (I)</td>
<td>63%</td>
<td>55%</td>
<td>1.40</td>
</tr>
</tbody>
</table>

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.

Evidence-based Management of Acute Musculoskeletal Pain

Table 7.10
Validity of Physical Signs of Biceps Tendon Lesions

<table>
<thead>
<tr>
<th>Physical Sign(s)</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Likelihood Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neer, Hawkins, Yocum and other tests all positive (N)</td>
<td>74%</td>
<td>58%</td>
<td>1.76</td>
</tr>
<tr>
<td>Pain on resisted shoulder flexion (Speed test) (B)</td>
<td>90%</td>
<td>14%</td>
<td>1.05</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Feature or Risk Factor</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms and signs of infection (e.g. fever)</td>
<td>Infection</td>
</tr>
<tr>
<td>Risk factors for infection (e.g. underlying disease process, immunosuppression, penetrating wound)</td>
<td>Fracture/dislocation</td>
</tr>
<tr>
<td>History of trauma</td>
<td></td>
</tr>
<tr>
<td>Sudden onset of pain</td>
<td></td>
</tr>
<tr>
<td>Past history of malignancy</td>
<td>Tumour</td>
</tr>
<tr>
<td>Age &gt; 50 years</td>
<td></td>
</tr>
<tr>
<td>Failure to improve with treatment</td>
<td></td>
</tr>
<tr>
<td>Unexplained weight loss</td>
<td></td>
</tr>
<tr>
<td>Pain at multiple sites</td>
<td></td>
</tr>
<tr>
<td>Pain at rest</td>
<td></td>
</tr>
</tbody>
</table>

> 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 x-ray beam is impeded by tissue in its path to produce an image on a radiosensitive plate. Radiographic images depend on the relative radiolucenties 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...
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 Oettle 1993; Tyson 1995). No studies have quantified those abilities. One paper reports sensitivity of 78% and specificity of 98% for plain x-ray 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 investigation 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 unreported 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
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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.

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 (M Ilgrom et al. 1995; Tempelhof et al. 1999).

### Table 7.12

<table>
<thead>
<tr>
<th>Authors</th>
<th>N</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Likelihood Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>D’Erme et al. (1993)</td>
<td>15</td>
<td>83%</td>
<td>0%*</td>
<td>0.83*</td>
</tr>
<tr>
<td>Brandt et al. (1989)</td>
<td>58</td>
<td>75%</td>
<td>43%</td>
<td>1.30</td>
</tr>
<tr>
<td>Miller et al. (1989)</td>
<td>56</td>
<td>58%</td>
<td>93%</td>
<td>8.30</td>
</tr>
<tr>
<td>Middleton et al. (1986)</td>
<td>100</td>
<td>91%</td>
<td>91%</td>
<td>10.00</td>
</tr>
<tr>
<td>Farin et al. (1996)</td>
<td>86</td>
<td>89%</td>
<td>95%</td>
<td>18.00</td>
</tr>
<tr>
<td>Mack et al. (1988)</td>
<td>99</td>
<td>88%</td>
<td>96%</td>
<td>22.00</td>
</tr>
<tr>
<td>Mack et al. (1985)</td>
<td>72</td>
<td>93%</td>
<td>97%</td>
<td>31.00</td>
</tr>
</tbody>
</table>

Note: * The specificity of 0% and low likelihood ratio were due to a lack of true negative scores in the results.

### Table 7.13

<table>
<thead>
<tr>
<th>Authors</th>
<th>N</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Likelihood Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>D’Erme et al. (1993)</td>
<td>9</td>
<td>86%</td>
<td>0%*</td>
<td>0.86*</td>
</tr>
<tr>
<td>Brandt et al. (1989)</td>
<td>38</td>
<td>71%</td>
<td>29%</td>
<td>1.00</td>
</tr>
<tr>
<td>Kurol et al. (1991)</td>
<td>58</td>
<td>42%</td>
<td>88%</td>
<td>3.50</td>
</tr>
<tr>
<td>Brenneke and M organ (1992)</td>
<td>120</td>
<td>78%</td>
<td>82%</td>
<td>4.30</td>
</tr>
<tr>
<td>Teeffey et al. (2000b)</td>
<td>120</td>
<td>94%</td>
<td>85%</td>
<td>6.30</td>
</tr>
<tr>
<td>Crass et al. (1988)</td>
<td>108</td>
<td>90%</td>
<td>92%</td>
<td>11.00</td>
</tr>
<tr>
<td>Mack et al. (1985)</td>
<td>47</td>
<td>100%</td>
<td>91%</td>
<td>11.00</td>
</tr>
<tr>
<td>Wiener and Seitz (1993)</td>
<td>225</td>
<td>95%</td>
<td>94%</td>
<td>16.00</td>
</tr>
<tr>
<td>Farin et al. (1996)</td>
<td>86</td>
<td>87%</td>
<td>98%</td>
<td>44.00</td>
</tr>
<tr>
<td>Mack et al. (1988)</td>
<td>90</td>
<td>91%</td>
<td>98%</td>
<td>46.00</td>
</tr>
</tbody>
</table>

Note: * The specificity of 0% and low likelihood ratio were due to a lack of true negative scores in the results.

### Table 7.14

<table>
<thead>
<tr>
<th>Age Groups</th>
<th>All Tears (M Ilgrom et al. 1995) N = 90</th>
<th>Complete Tears (Tempelhof et al. 1999) N = 411</th>
</tr>
</thead>
<tbody>
<tr>
<td>30–39</td>
<td>6%</td>
<td>–</td>
</tr>
<tr>
<td>40–49</td>
<td>10%</td>
<td>–</td>
</tr>
<tr>
<td>50–59</td>
<td>33%</td>
<td>13%</td>
</tr>
<tr>
<td>60–69</td>
<td>53%</td>
<td>20%</td>
</tr>
<tr>
<td>70–79</td>
<td>70%</td>
<td>31%</td>
</tr>
<tr>
<td>&gt;80</td>
<td>80%</td>
<td>51%</td>
</tr>
</tbody>
</table>

Note: Based on data from M Ilgrom et al. (1995) and 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 neurorrhomas (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: \( \kappa \) Score Ranges Between Five Experienced Radiologists

<table>
<thead>
<tr>
<th></th>
<th>Partial Tears</th>
<th>Complete Tears</th>
<th>All Tears</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \kappa )</td>
<td>0.17–0.44</td>
<td>0.73–0.88</td>
<td>0.63–0.80</td>
</tr>
</tbody>
</table>

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 *

<table>
<thead>
<tr>
<th>Authors</th>
<th>N</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Likelihood Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Torstensen and Hollinshead (1999)</td>
<td>15</td>
<td>73%</td>
<td>58%</td>
<td>1.7</td>
</tr>
<tr>
<td>Gross et al. (1990)</td>
<td>22</td>
<td>91%</td>
<td>69%</td>
<td>2.9</td>
</tr>
<tr>
<td>Iannotti et al. (1991)</td>
<td>39</td>
<td>88%</td>
<td>93%</td>
<td>13.0</td>
</tr>
<tr>
<td>Green and Christensen (1994)</td>
<td>33</td>
<td>75%</td>
<td>100%</td>
<td>*</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Authors</th>
<th>N</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Likelihood Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Torstensen and Hollinshead (1999)</td>
<td>24</td>
<td>96%</td>
<td>49%</td>
<td>1.9</td>
</tr>
<tr>
<td>Tuite et al. (1998)</td>
<td>110</td>
<td>67%</td>
<td>77%</td>
<td>2.9</td>
</tr>
<tr>
<td>Blanchard et al. (1999b)</td>
<td>54</td>
<td>81%</td>
<td>78%</td>
<td>3.7</td>
</tr>
<tr>
<td>Maurer et al. (1997)</td>
<td>14</td>
<td>79%</td>
<td>88%</td>
<td>6.6</td>
</tr>
<tr>
<td>Zlatkin et al. (1989)</td>
<td>32</td>
<td>91%</td>
<td>88%</td>
<td>7.6</td>
</tr>
<tr>
<td>Evancho et al. (1988)</td>
<td>31</td>
<td>69%</td>
<td>94%</td>
<td>12.0</td>
</tr>
<tr>
<td>Balich et al. (1997)</td>
<td>222</td>
<td>84%</td>
<td>94%</td>
<td>14.0</td>
</tr>
<tr>
<td>Ianotti et al. (1991)</td>
<td>88</td>
<td>100%</td>
<td>95%</td>
<td>20.0</td>
</tr>
</tbody>
</table>

Table 7.18
MRI Findings of Rotator Cuff Tears in 96 People Without Symptoms

<table>
<thead>
<tr>
<th>Age Groups</th>
<th>Partial Tears</th>
<th>Complete Tears</th>
<th>All Tears</th>
</tr>
</thead>
<tbody>
<tr>
<td>19–39</td>
<td>4%</td>
<td>0%</td>
<td>4%</td>
</tr>
<tr>
<td>40–60</td>
<td>24%</td>
<td>4%</td>
<td>28%</td>
</tr>
<tr>
<td>&gt;60</td>
<td>26%</td>
<td>28%</td>
<td>54%</td>
</tr>
<tr>
<td>ALL</td>
<td>20%</td>
<td>15%</td>
<td>34%</td>
</tr>
</tbody>
</table>

Note: Based on data from Sher et al. (1995).

Table 7.19
MRI Findings of Rotator Cuff Tears in 100 People Without Symptoms

<table>
<thead>
<tr>
<th>Age Groups</th>
<th>Partial Tears</th>
<th>Complete Tears</th>
<th>All Tears</th>
</tr>
</thead>
<tbody>
<tr>
<td>19–39</td>
<td>8%</td>
<td>0%</td>
<td>8%</td>
</tr>
<tr>
<td>40–60</td>
<td>27%</td>
<td>4%</td>
<td>31%</td>
</tr>
<tr>
<td>&gt;60</td>
<td>27%</td>
<td>27%</td>
<td>54%</td>
</tr>
<tr>
<td>ALL</td>
<td>22%</td>
<td>14%</td>
<td>36%</td>
</tr>
</tbody>
</table>

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
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 Niederer 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

<table>
<thead>
<tr>
<th>MRI Findings</th>
<th>Symptomatic People</th>
<th>Asymptomatic People</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acromioclavicular osteophytes</td>
<td>11</td>
<td>7</td>
</tr>
<tr>
<td>Anterior instability</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Posterior instability</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Abnormal labral morphology</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Abnormal labral signal</td>
<td>11</td>
<td>10</td>
</tr>
<tr>
<td>Bony glenoid defect</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Joint fluid</td>
<td>7</td>
<td>10</td>
</tr>
<tr>
<td>Absent subacromial fat</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Absent subdeltoid fat</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Supraspinatus depression</td>
<td>13</td>
<td>6</td>
</tr>
<tr>
<td>Abnormal tendon morphology</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>Abnormal tendon signal</td>
<td>13</td>
<td>6</td>
</tr>
<tr>
<td>Impingement</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Tendonitis</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Partial rotator cuff tear</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Tendon discontinuity</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>Complete rotator cuff tear</td>
<td>6</td>
<td>0</td>
</tr>
</tbody>
</table>

Note: Based on data from Chandnani et al. (1992).
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 (Merckel et al. 1978)
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 esti-
mations 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.

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 clini-
cians, 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 accu-
ratey 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 scientifi-
cally valid. Inaccurate description or use of inappropriate terms
obscures the diagnosis, hinders communication and under-
standing 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

<table>
<thead>
<tr>
<th>Authors</th>
<th>Clinicians</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bamji et al. (1996)</td>
<td>Rheumatologists</td>
<td>'only 46%'</td>
</tr>
<tr>
<td>Liesdeck et al. (1997)</td>
<td>GPs and physiotherapists</td>
<td>'low' (κ = 0.31)</td>
</tr>
<tr>
<td>de Winter et al. (1999)</td>
<td>Physiotherapists</td>
<td>'moderate' (κ = 0.45)</td>
</tr>
<tr>
<td>Pal et al. (2000)</td>
<td>Emergency room doctors</td>
<td>'low'</td>
</tr>
<tr>
<td>Narregaard et al. (2002)</td>
<td>Orthopaedic surgeon and rheumatologist</td>
<td>'poor' (κ &lt; 0.4)</td>
</tr>
</tbody>
</table>
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 tendinitis’, ‘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

Table 7.22
Short Term Recovery of Acute Shoulder Pain

<table>
<thead>
<tr>
<th></th>
<th>2 weeks</th>
<th>6 weeks</th>
<th>12 weeks</th>
<th>25 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>9%</td>
<td>48%</td>
<td>76%</td>
<td>91%</td>
<td></td>
</tr>
</tbody>
</table>

Note: Based on data from Winters et al. (1997b).

Table 7.23
Longer Term Recovery of Acute Shoulder Pain

<table>
<thead>
<tr>
<th></th>
<th>1 month</th>
<th>3 months</th>
<th>6 months</th>
<th>12 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>23%</td>
<td>44%</td>
<td>51%</td>
<td>59%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>49%</td>
<td>59%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Based on data from van der Windt et al. (1996) (upper figures) and Winters et al. (1997b) (lower figures).

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 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
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.

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 tendinitis (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>
<thead>
<tr>
<th>Table 7.24</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recovery of Disability Associated with Acute Shoulder Pain</td>
</tr>
<tr>
<td>6 months</td>
</tr>
<tr>
<td>21%</td>
</tr>
</tbody>
</table>

Note: Based on data from Croft et al. (1996).
trials of corticosteroid injections for shoulder pain, there was little overall evidence to guide treatment 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 injection 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.

Table 7.25
Biological Risk Factors for Shoulder Pain as Shown in Various Reports

<table>
<thead>
<tr>
<th>Factors</th>
<th>Subjects</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Work above shoulder height</td>
<td>Industrial workers</td>
<td>Bjelle et al. (1979)</td>
</tr>
<tr>
<td></td>
<td>Car assembly workers</td>
<td>Punnett et al. (2000)</td>
</tr>
<tr>
<td></td>
<td>Forestry workers</td>
<td>Miranda et al. (2001)</td>
</tr>
<tr>
<td></td>
<td>Urban manual workers</td>
<td>Pope (2001)</td>
</tr>
<tr>
<td>Low frequency vibration</td>
<td>Industrial workers</td>
<td>Futatsaka et al. (1985)</td>
</tr>
<tr>
<td>Repetitive work tasks</td>
<td>Semi-rural community</td>
<td>Ekberg et al. (1995)</td>
</tr>
<tr>
<td></td>
<td>Shoulder patients</td>
<td>English et al. (1995)</td>
</tr>
<tr>
<td></td>
<td>Urban manual workers</td>
<td>Pope (2001)</td>
</tr>
<tr>
<td>Heavy workload</td>
<td>Forestry workers</td>
<td>Miranda et al. (2001)</td>
</tr>
<tr>
<td>Work pace</td>
<td>Semi-rural community</td>
<td>Ekberg et al. (1995)</td>
</tr>
<tr>
<td>Driving for long periods</td>
<td>Commercial travellers</td>
<td>Skov et al. (1996)</td>
</tr>
<tr>
<td>Shift work</td>
<td>Male workers</td>
<td>Fredriksson et al. (1999)</td>
</tr>
<tr>
<td>Sleep disturbance</td>
<td>Male and female workers</td>
<td>Bergenudd and Nilsson (1994)</td>
</tr>
<tr>
<td>Smoking</td>
<td>Male workers</td>
<td>Bergenudd and Nilsson (1994)</td>
</tr>
<tr>
<td></td>
<td>Commercial travellers</td>
<td>Skov et al. (1996)</td>
</tr>
<tr>
<td></td>
<td>Video display unit users</td>
<td>Marcus and Gerr (1996)</td>
</tr>
<tr>
<td></td>
<td>Female sewing machinists</td>
<td>Kaergaard and Andersen (2000)</td>
</tr>
<tr>
<td>Caffeine consumption</td>
<td>Video display unit users</td>
<td>Marcus and Gerr (1996)</td>
</tr>
<tr>
<td>Female gender</td>
<td>Semi-rural community</td>
<td>Ekberg et al. (1995)</td>
</tr>
<tr>
<td></td>
<td>Commercial travellers</td>
<td>Skov et al. (1996)</td>
</tr>
<tr>
<td></td>
<td>Female sewing machinists</td>
<td>Kaergaard and Andersen (2000)</td>
</tr>
</tbody>
</table>

Table 7.26
Psychosocial Risk Factors for Shoulder Pain as Shown in Various Reports

<table>
<thead>
<tr>
<th>Factors (‘yellow flags’)</th>
<th>Subjects</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Job dissatisfaction</td>
<td>Male and female workers</td>
<td>Bergenudd and Nilsson (1994)</td>
</tr>
<tr>
<td></td>
<td>Video display unit users</td>
<td>Marcus and Gerr (1996)</td>
</tr>
<tr>
<td>Uncertain work demands</td>
<td>Semi-rural community</td>
<td>Ekberg et al. (1995)</td>
</tr>
<tr>
<td></td>
<td>Commercial travellers</td>
<td>Skov et al. (1996)</td>
</tr>
<tr>
<td></td>
<td>Newly employed workers</td>
<td>Nahit et al. (2001)</td>
</tr>
<tr>
<td>Poor support at work</td>
<td>Video display unit users</td>
<td>Marcus and Gerr (1996)</td>
</tr>
<tr>
<td></td>
<td>Female sewing machinists</td>
<td>Kaergaard and Andersen (2000)</td>
</tr>
<tr>
<td>High mental workload</td>
<td>Male workers</td>
<td>Fredriksson et al. (1999)</td>
</tr>
<tr>
<td></td>
<td>Urban manual workers</td>
<td>Pope (2001)</td>
</tr>
<tr>
<td>Psychological distress</td>
<td>Urban workers</td>
<td>Macfarlane et al. (1998)</td>
</tr>
<tr>
<td></td>
<td>Forestry workers</td>
<td>Miranda et al. (2001)</td>
</tr>
<tr>
<td></td>
<td>Semi-rural community</td>
<td>van der Windt et al. (2002)</td>
</tr>
<tr>
<td></td>
<td>Semi-rural community</td>
<td>Badcock et al. (2002)</td>
</tr>
<tr>
<td>Immigrant status</td>
<td>Semi-rural community</td>
<td>Ekberg et al. (1995)</td>
</tr>
</tbody>
</table>
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-inflammatory 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 triamcinolone acetonide to indoethacin (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 (Patri 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 (Wilcoxon Matched Pairs Test for related samples: sum of difference scores 22, p < 0.001) (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 = 2.6; 95% CI 1.18, 68.74) (Buchbinder et al. 2002, van der Windt et al. 1998).

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 indoethacin 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 x-rays 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 fenitropraz (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 (Kleinheinz 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) (WM D = 10.83; 95% CI 2.46, 19.20) but no difference at four months (WM D = 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 analgesics for acute shoulder pain. There are no randomised controlled trials of analgesics (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 (WM D = -32.07mm on VAS; 95% CI –58.04, –6.10). There was however no significant difference between groups in range of elevation (WM D = -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**


Evidence-based Management of Acute Musculoskeletal Pain
Evidence-based Management of Acute Musculoskeletal Pain


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Chapter 7 • Acute Shoulder Pain


Chapter 7 • Acute Shoulder Pain


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; 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; Matheron 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
Study Selection Criteria

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:

**DIAGNOSIS**

| 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**

<table>
<thead>
<tr>
<th>Management Plan</th>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
</table>
| 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 |

<table>
<thead>
<tr>
<th>Non-Pharmacologic Interventions</th>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>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.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pharmacologic Interventions</th>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
</table>
| Specific pharmacologic interventions may be required to relieve pain; such agents can be used in conjunction with non-pharmacologic interventions.  
  Paracetamol or other simple analgesics, administered regularly, are recommended for relief of mild to moderate acute musculoskeletal pain.  
  Where paracetamol is insufficient for pain relief, a non-steroidal anti-inflammatory (NSAID) medication may be used, unless contraindicated.  
  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.  
  Adjuvant agents such as anticonvulsants and antidepressants are not recommended in the management of acute musculoskeletal pain.  
  Any benefits from muscle relaxants may be outweighed by their adverse effects, therefore they cannot be routinely recommended. | CONSENSUS: Steering Committee; NHM RC 1999b |

**Summary of Key Messages: Effective Communication**

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

<table>
<thead>
<tr>
<th>DIAGNOSIS</th>
<th>EVIDENCE LEVEL</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aetiology and Prevalence</strong></td>
<td></td>
</tr>
<tr>
<td>‘Patellofemoral pain’ is a general term used to describe idiopathic pain arising from the anterior knee/patellofemoral region that is of otherwise unknown origin.</td>
<td>CONSENSUS: Steering Committee</td>
</tr>
<tr>
<td>Anterior knee pain is commonly idiopathic; serious causes are rare.</td>
<td>*LEVEL IV: Kaempfte 1995; Ferguson et al. 1997; Kaandorp et al. 1995</td>
</tr>
<tr>
<td>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.</td>
<td>*LEVEL IV: Kujala et al. 2001; Reider et al. 1981a,b; Witvrouw et al. 2000; Tanaka et al. 1989; Cooper et al. 1994</td>
</tr>
</tbody>
</table>

| 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-J ones 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 |
| --- | --- |

<table>
<thead>
<tr>
<th>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.</th>
<th>No Level I or II evidence</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Electrical Stimulation — There are no randomised controlled studies of the effectiveness of electrical stimulation of the quadriceps muscle for patellofemoral pain.</th>
<th>No Level I or II evidence</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>There is insufficient evidence that one form of electrical stimulation of the quadriceps muscle is superior to another for treating patellofemoral pain.</th>
<th>LEVEL II: Callaghan et al. (2001)</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>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.</th>
<th>No Level I or II evidence</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Different types of NSAIDs provide similar relief of patellofemoral pain after five days of use.</th>
<th>LEVEL II: Based on one RCT with limitations (Fulkerson and Folcik 1986)</th>
</tr>
</thead>
</table>

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<tr>
<th>Serious adverse effects of NSAIDs include gastrointestinal complications (e.g. bleeding, perforation).</th>
<th>LEVEL I: Based on systematic reviews (Bigos et al. 1994; van Tulder et al. 2002)</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>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.</th>
<th>LEVEL I, II: Based on two systematic reviews (Crossley et al. 2001; Harrison et al. 2003) and one subsequent RCT (Crossley et al. 2002)</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>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).</th>
<th>LEVEL I: Based on a systematic review (Crossley et al. 2001) that located one RCT (Timm 1998)</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Therapeutic Ultrasound — There is insufficient evidence that therapeutic ultrasound is more effective compared to ice massage for the treatment of patellofemoral pain.</th>
<th>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)</th>
</tr>
</thead>
</table>

| 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.

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; Orini et al. 1998) and retinaculum (Kasim and Fullkerson 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 Fullkerson 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 (Crossey et al. 2002). Malalignment leading to elevated patellofemoral joint stress has been promoted as a factor by some and refuted by others (Fullkerson 1989; Grelsamer and Klein 1998; Grelsamer 2000; Osterbridge 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 (Crossey 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) (Buchalter and Powers 2002)
- Gender, muscle strength and motion (Cintalan 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 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; Fullkerson and Huergerford 1990; Insall 1979; Kivimaki et al. 1994). However, several studies have shown poor correlation between arthritic 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 x-ray 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; Maciachin and Emery 1974). Softening and fribillation of the patella can be a natural consequence of aging (Bennett et al. 1942; Collins and McEachin 1961; Owrre 1936). The relationship between patellofemoral pain and chondromalacia patellae remains unclear (Kannus et al. 1999).

Table 8.1

Potential Causes of Anterior Knee Pain

<table>
<thead>
<tr>
<th>Serious conditions</th>
<th>Fracture; Tumour; Infection; Inflammatory arthropathies; Osteonecrosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intrinsic mechanical conditions</td>
<td>Patella (patellar instability and dislocation; infrapatellar contracture syndrome; medial patellar subluxation); Patellar tendon (patellar tendinopathy; Osgood-Schlatter Disease); Sinding-Larsen-Johansson Syndrome; Quadriceps tendonopathy; Fat Pad Syndrome; Prepatellar and infrapatellar bursitis; Plicae; Osteochondritis dissecans; Sprains and strains</td>
</tr>
<tr>
<td>Refferred pain</td>
<td>Somatic conditions (e.g. diseases of the hip joint)</td>
</tr>
<tr>
<td>Neurological conditions</td>
<td>Infrapatellar neuralgia</td>
</tr>
</tbody>
</table>
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 osteoepenia 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 (Kämpfeffe 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 857 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 (Kandorp et al. 1995), have diabetes (Kandorp et al. 1995), have rheumatoid arthritis (Kandorp et al. 1995), have a skin infection (Kandorp 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 (Dutenbeck 1972; Huse and Ahmed 1977; Senegor 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, hypeaesthesia, hypoaes- thesia, 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 impinge- ment and inflammation of the infrapatellar fat pad (Kebs and Parker 1994).

The clinical features include tenderness on palpation and pain in the retro- and infrapatellar regions aggravated by move- ment 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 chordoma (Krebs and Parker 1994).

Osteonecrosis
Osteonecrosis is generally an idiopathic condition, usually found in the medial femoral condyle (Ahlbäck 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 Hungarford 1990), shallow patellar depth (Malghe and Madalou 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).

Infraapatellar 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 infraapatellar 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 tendinitis’. 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 (M Atava 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 (Outterbridge 1964) and illiotibial band tightness in ballet dancers (Winslow and Yoder 1995) have been identified as risk factors for patellofemoral pain.

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 oblique 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 anteverision (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 N essan 1997); antever- sion, 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 (Miligrom 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; Granfil Leave and Moretz 1978; Jackson et al. 1978; Kallen 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 (Fel son 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 (M Allindon 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 Fel son 1988; Kivimaki et al. 1992; Lindberg and Montgomery 1987; Tanaka et al. 1989). Burstsits and other anterior soft tissue changes occur more often in workers who kneel frequently (Tanaka et al. 1989; T hun et al. 1987; Watkins et al. 1958). The most common lesions in kneeling workers are meniscal lesions (Holikov et al. 1985) osteoarthritis (Kasch and Enderlin 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).

Table 8.2

Prevalence of Conditions Presenting as Anterior Knee Pain

<table>
<thead>
<tr>
<th>Prevalence</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rare causes</td>
<td>Tumour</td>
</tr>
<tr>
<td></td>
<td>Infection</td>
</tr>
<tr>
<td></td>
<td>Neurological conditions</td>
</tr>
<tr>
<td>Uncommon causes</td>
<td>Fracture</td>
</tr>
<tr>
<td></td>
<td>Osteonecrosis</td>
</tr>
<tr>
<td></td>
<td>Inflammatory arthropathies</td>
</tr>
<tr>
<td></td>
<td>Pain referred to the knee</td>
</tr>
<tr>
<td>Common causes</td>
<td>Mechanical conditions</td>
</tr>
</tbody>
</table>

Note: As there is limited evidence available, Table 8.2 was developed through consensus.
between patellofemoral pain and work is less convincing (M baruk 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 (H unter 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, 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**

While 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 (T homee 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 (M ckenzie 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 spondyloarthritides. 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 seous 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 to 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 semitendinosus tendon passing over the medial tibial condyle (Bae and Kwon 1997).

### Alerting Features of Serious Conditions

Features alerting to the possibility of a serious condition may be identified during clinical assessment. While the predictive value 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

<table>
<thead>
<tr>
<th>Feature or Risk Factor</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major trauma</td>
<td>Fracture or tendon and ligament rupture, osteonecrosis</td>
</tr>
<tr>
<td>Sudden onset of pain</td>
<td>Fracture or tendon and ligament rupture, osteonecrosis</td>
</tr>
<tr>
<td>Minor trauma (if &gt; 50 years, history of osteoporosis and taking corticosteroids)</td>
<td></td>
</tr>
<tr>
<td>Fever, night sweats, signs of inflammation (large, warm effusion)</td>
<td>Infection (e.g. septic arthritis), crystal arthritis</td>
</tr>
<tr>
<td>Risk factors for infection (e.g. underlying disease process, immunosuppression, penetrating wound)</td>
<td>Tumour</td>
</tr>
<tr>
<td>Past history of malignancy</td>
<td></td>
</tr>
<tr>
<td>Age &gt; 50</td>
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<tr>
<td>Failure to improve with treatment</td>
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<tr>
<td>Unexplained weight loss</td>
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<tr>
<td>Pain at multiple sites</td>
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<td>Pain at rest</td>
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<tr>
<td>Night pain</td>
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</tbody>
</table>
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; Haracker 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 (Haracker et al. 1990).

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 tendinopathy, 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 tendinopathy 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 tendinopathy 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 (Berquist et al. 1993; Cusnashan et al. 1990; Ike and O’Rourke 1995; Jones et al. 1992). Although crepitus is generally considered unreliable and invalid (Cusnashan 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 0.39–0.50) 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 x-ray 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 x-rays in general practice found that 50% of knee x-rays were ordered according to the Royal College of Radiologists guidelines (1995), in which the indications for x-ray are locking or restricted movement. Of the 1153 x-rays 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 were 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 x-ray 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 x-rays (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; M eyer 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 x-ray 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 x-rays taken in an emergency department revealed that about 92% were negative for fracture (Stiël et al. 1995). As a consequence, the 'Ottawa Knee Rule' was developed following a multi-centre trial (Stiël et al. 1996). This rule has been validated and found to be reliable (Stiël et al. 1997; Wason and Sox 1996).

The rule states that a conventional x-ray 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 x-rays. 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, x-rays 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 x-rayed.
- Of those remaining, only those who cannot walk four weight-bearing steps in the emergency department are x-rayed. 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 x-rays 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)

Radionuclide 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 Holsbeek and Introcasa 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 Holsbeek 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. (1998) found ultrasound to be more accurate than CT scan for chronic patellar tendinitis 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 tendinopathy, 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 anatomic-ical 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 (Miligrom 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...
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 intra-articular 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 (lidocaine) 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, Crosley 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, Crosley 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; Kowal et al. 1996; McVilien 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 = −0.10; 95% CI −1.17, 0.92). Harrison et al. (1999) compared a McConnel-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 McConnel program group compared to the supervised exercises but no difference between the McConnel program and the home exercise program (effect size for pain = −0.45; 95% CI −1.20, 0.27). Overall, Crosley 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 pain is unknown, it is thought that weakened quadriceps muscles or an imbalance in the strength of the vastus lateralis and vastus medialis obliquis 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 muscles 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 muscles 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 diflunisal 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 treatment 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 Error = 0.44).

**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 databases (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 x-ray diagnosis of a fracture were subject to a cost-effectiveness evaluation (Nichol et al. 1999). The evaluation was performed in Canada and concluded that x-ray 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 x-ray 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


Evidence-based Management of Acute Musculoskeletal Pain


Chapter 8 • Anterior Knee Pain


Anterior Knee Pain

Chapter 8

Evidence-based Management of Acute Musculoskeletal Pain


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 specialising in musculoskeletal medicine. A unidisciplinary 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.
Evidence-based Management of Acute Musculoskeletal Pain

(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.

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**Table 9.1**

<table>
<thead>
<tr>
<th>Question</th>
<th>Study Type</th>
<th>Level of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intervention</td>
<td>Systematic review</td>
<td>I</td>
</tr>
<tr>
<td></td>
<td>Randomised controlled trial</td>
<td>II</td>
</tr>
<tr>
<td></td>
<td>Cohort study</td>
<td>III-2</td>
</tr>
<tr>
<td></td>
<td>Case-control study</td>
<td>III-2</td>
</tr>
<tr>
<td>Diagnostic test/performance</td>
<td>Systematic review</td>
<td>*I</td>
</tr>
<tr>
<td></td>
<td>Cross-sectional study</td>
<td>*III-3</td>
</tr>
<tr>
<td></td>
<td>Case series</td>
<td>*IV</td>
</tr>
<tr>
<td>Prediction and prognosis</td>
<td>Systematic review</td>
<td>*I</td>
</tr>
<tr>
<td></td>
<td>Cohort/survival study</td>
<td>*III-2</td>
</tr>
</tbody>
</table>

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**

<table>
<thead>
<tr>
<th>Level of Evidence</th>
<th>Study Design</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Evidence obtained from a systematic review of all relevant randomised controlled trials.</td>
</tr>
<tr>
<td>II</td>
<td>Evidence obtained from one or more properly designed randomised controlled trials.</td>
</tr>
<tr>
<td>III-1</td>
<td>Evidence obtained from well-designed pseudo randomised controlled trials (alternate allocation or some other method).</td>
</tr>
<tr>
<td>III-2</td>
<td>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.</td>
</tr>
<tr>
<td>III-3</td>
<td>Evidence obtained from comparative studies with historical control, two or more single arm studies, or interrupted time series without parallel control group.</td>
</tr>
<tr>
<td>IV</td>
<td>Evidence obtained from case series, either post-test or pre-test/post-test.</td>
</tr>
<tr>
<td>Consensus</td>
<td>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.</td>
</tr>
</tbody>
</table>

Chapter 9 • Process Report

• 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.

Aetiology and Prevalence

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.

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 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.

Appendix E: Tables of Included and Excluded Studies

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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.

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 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

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.

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.
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 the 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).

### Table 9.3

**Criteria for Categorising Interventions**

<table>
<thead>
<tr>
<th>Category</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evidence of Benefit</td>
<td>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.</td>
</tr>
<tr>
<td>Conflicting Evidence</td>
<td>Interventions for which there have been a number of similar controlled trials that have achieved conflicting results.</td>
</tr>
<tr>
<td>Insufficient Evidence</td>
<td>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.</td>
</tr>
<tr>
<td>Evidence of No Benefit</td>
<td>Interventions that have demonstrated no effect vs. placebo or natural history and have confidence intervals that exclude a clinically important benefit.</td>
</tr>
</tbody>
</table>

### 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.
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 be 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


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 (2000c). How to Use the Evidence: Assessment and Application of Scientific Evidence. Canberra: NHMRC


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)

Manipulation (spinal) is a mechanical form of therapy 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)

Odds ratio 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).

<table>
<thead>
<tr>
<th>Kappa Score</th>
<th>Agreement</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.8–1.0</td>
<td>Very Good</td>
</tr>
<tr>
<td>0.6–0.8</td>
<td>Good</td>
</tr>
<tr>
<td>0.4–0.6</td>
<td>Moderate</td>
</tr>
<tr>
<td>0.2–0.4</td>
<td>Fair</td>
</tr>
<tr>
<td>0.0–0.2</td>
<td>Poor</td>
</tr>
</tbody>
</table>


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.
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.

## Table of Unit Costs

(November 2002)

<table>
<thead>
<tr>
<th>Service*</th>
<th>MBS ITEM NO.</th>
<th>FEE ($)</th>
<th>85% REBATE</th>
</tr>
</thead>
<tbody>
<tr>
<td>General practitioner</td>
<td>36 (Level A, B, C)</td>
<td>55.95</td>
<td>47.60</td>
</tr>
<tr>
<td></td>
<td>23 (Level A, B, C)</td>
<td>29.45</td>
<td>25.05</td>
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<tr>
<td>Surgeon</td>
<td>104</td>
<td>69.35</td>
<td>58.95</td>
</tr>
<tr>
<td></td>
<td>105</td>
<td>34.80</td>
<td>29.60</td>
</tr>
<tr>
<td>Consultant physician</td>
<td>110</td>
<td>122.35</td>
<td>104.00</td>
</tr>
<tr>
<td></td>
<td>116</td>
<td>61.25</td>
<td>52.10</td>
</tr>
<tr>
<td>Physiotherapist</td>
<td></td>
<td>Mean 50.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mean 40.00</td>
<td></td>
</tr>
<tr>
<td>Chiropractor</td>
<td></td>
<td>60.00-100.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>40.00</td>
<td></td>
</tr>
<tr>
<td>Osteopath</td>
<td></td>
<td>100.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>60.00-80.00</td>
<td></td>
</tr>
<tr>
<td>Acupuncture</td>
<td>173</td>
<td>21.65</td>
<td>18.45</td>
</tr>
</tbody>
</table>

### Interventions

<table>
<thead>
<tr>
<th>Intervention</th>
<th>FEE ($)</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Massage therapy (1 hour)</td>
<td>(average from Cost Study)</td>
<td>49.00 (range 40-60)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TENS</th>
<th>Machine</th>
<th>buy</th>
<th>hire</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Replacement gel pads</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Battery (9v)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>120-220</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>60-68/month</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>16.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>5.60</td>
<td></td>
</tr>
</tbody>
</table>
## Unit Costs continued

<table>
<thead>
<tr>
<th>MBS ITEM NO.</th>
<th>FEE ($)</th>
<th>85% REBATE</th>
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<tbody>
<tr>
<td>50124</td>
<td>24.00</td>
<td>20.40</td>
</tr>
<tr>
<td>39013</td>
<td>88.55</td>
<td>75.30</td>
</tr>
<tr>
<td>From RNSH pharmacy</td>
<td>10.05/ea</td>
<td>3.10/ea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.25/ea</td>
</tr>
<tr>
<td>PBS 8440F</td>
<td>32.09</td>
<td></td>
</tr>
<tr>
<td>PBS 1300K</td>
<td>13.65</td>
<td></td>
</tr>
<tr>
<td>RRP</td>
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<td></td>
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<td>PBS 1746X</td>
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<td>PBS 1215Y</td>
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</tr>
<tr>
<td>56625</td>
<td>317.90</td>
<td>270.25</td>
</tr>
<tr>
<td>59751</td>
<td>475.00</td>
<td>419.40</td>
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<tr>
<td>59751</td>
<td>131.15</td>
<td>111.50</td>
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<tr>
<td>55828</td>
<td>99.90</td>
<td>84.95</td>
</tr>
<tr>
<td>55808</td>
<td>99.90</td>
<td>84.95</td>
</tr>
</tbody>
</table>

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.
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

<table>
<thead>
<tr>
<th>SUSPECTED CONDITION</th>
<th>REGION OF PAIN</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fracture</strong></td>
<td>Lumbar Spinal</td>
</tr>
<tr>
<td>History of significant trauma</td>
<td>Cervical Spinal</td>
</tr>
<tr>
<td>History of minor trauma in association with corticosteroid use, age over 50, history of osteoporosis</td>
<td>Thoracic Spinal</td>
</tr>
<tr>
<td>History or previous fracture or metabolic disease</td>
<td>Shoulder</td>
</tr>
<tr>
<td>Positive for Canadian C-spine rule</td>
<td>Knee</td>
</tr>
<tr>
<td>Positive for Ottawa Knee Rule</td>
<td></td>
</tr>
<tr>
<td><strong>Infection</strong></td>
<td>Lumbar Spinal</td>
</tr>
<tr>
<td>Fever</td>
<td>Cervical Spinal</td>
</tr>
<tr>
<td>Sweating</td>
<td>Thoracic Spinal</td>
</tr>
<tr>
<td>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)</td>
<td>Shoulder</td>
</tr>
<tr>
<td><strong>Tumour</strong></td>
<td>Lumbar Spinal</td>
</tr>
<tr>
<td>Palpable mass</td>
<td>Cervical Spinal</td>
</tr>
<tr>
<td>Past history of malignancy</td>
<td>Thoracic Spinal</td>
</tr>
<tr>
<td>Age &gt; 50 years</td>
<td>Shoulder</td>
</tr>
<tr>
<td>Failure to improve with treatment</td>
<td>Knee</td>
</tr>
<tr>
<td>Unexplained weight loss</td>
<td></td>
</tr>
<tr>
<td>Pain not relieved by rest</td>
<td></td>
</tr>
<tr>
<td><strong>Crystal arthritis</strong></td>
<td>Lumbar Spinal</td>
</tr>
<tr>
<td>Joint effusion</td>
<td>Cervical Spinal</td>
</tr>
<tr>
<td><strong>Aneurysm</strong></td>
<td>Lumbar Spinal</td>
</tr>
<tr>
<td>Cardiovascular risk factors</td>
<td>Cervical Spinal</td>
</tr>
<tr>
<td>Anticoagulants</td>
<td>Thoracic Spinal</td>
</tr>
<tr>
<td>Transient ischaemic attacks</td>
<td>Shoulder</td>
</tr>
<tr>
<td>Bruits</td>
<td>Knee</td>
</tr>
<tr>
<td>Recent history of torsion to neck</td>
<td></td>
</tr>
<tr>
<td>Absence of musculoskeletal signs</td>
<td></td>
</tr>
<tr>
<td><strong>Osteonecrosis</strong></td>
<td>Lumbar Spinal</td>
</tr>
<tr>
<td>Immunosuppression</td>
<td>Cervical Spinal</td>
</tr>
<tr>
<td>Renal dialysis</td>
<td>Thoracic Spinal</td>
</tr>
<tr>
<td>Use of corticosteroids</td>
<td>Shoulder</td>
</tr>
<tr>
<td>Diabetes, alcoholism</td>
<td>Knee</td>
</tr>
</tbody>
</table>

**Note:** ESR: erythrocyte sedimentation rate; FBC: full blood count; CRP: C-reactive protein; MRI: magnetic resonance imaging; IEPG: immunoelectrophoretogram; MRA: magnetic resonance angiography.
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. R.L. 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 Murtough (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 (Australasian 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)
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
- Acute Low Back Pain — Table of Included Studies (Diagnosis)
- Acute Low Back Pain — Table of Excluded Studies (Diagnosis)
- Acute Low Back Pain — Table of Included Studies (Prognosis)
- Acute Low Back Pain — Table of Excluded Studies (Prognosis)
- Acute Low Back Pain — Table of Included Studies (Interventions)
- Acute Low Back Pain — Table of Excluded Studies (Interventions)
- Acute Low Back Pain — Table of Included Studies (Cost Effectiveness)
- Acute Low Back Pain — Table of Excluded Studies (Cost Effectiveness)
- Acute Thoracic Pain — Table of Included Studies (Diagnosis)
- Acute Thoracic Pain — Table of Excluded Studies (Diagnosis)
- Acute Thoracic Pain — Table of Included Studies (Prognosis)
- Acute Thoracic Pain — Table of Excluded Studies (Prognosis)
- Acute Thoracic Pain — Table of Included Studies (Interventions)
- Acute Thoracic Pain — Table of Excluded Studies (Interventions)
- Acute Neck Pain — Table of Included Studies (Diagnosis)
- Acute Neck Pain — Table of Excluded Studies (Diagnosis)
- Acute Neck Pain — Table of Included Studies (Prognosis)
- Acute Neck Pain — Table of Excluded Studies (Prognosis)
- Acute Neck Pain — Table of Included Studies (Interventions)
- Acute Neck Pain — Table of Excluded Studies (Interventions)
- Acute Shoulder Pain — Table of Included Studies (Diagnosis)
- Acute Shoulder Pain — Table of Excluded Studies (Diagnosis)
- Acute Shoulder Pain — Table of Included Studies (Prognosis)
- Acute Shoulder Pain — Table of Excluded Studies (Prognosis)
- Acute Shoulder Pain — Table of Included Studies (Interventions)
- Acute Shoulder Pain — Table of Excluded Studies (Interventions)
- Acute Shoulder Pain — Table of Excluded Studies (Interventions)
- Acute Shoulder Pain — Table of Excluded Studies (Cost Effectiveness)
- Anterior Knee Pain — Table of Included Studies (Diagnosis)
- Anterior Knee Pain — Table of Excluded Studies (Diagnosis)
- Anterior Knee Pain — Table of Included Studies (Prognosis)
- Anterior Knee Pain — Table of Excluded Studies (Prognosis)
- Anterior Knee Pain — Table of Included Studies (Interventions)
- Anterior Knee Pain — Table of Excluded Studies (Interventions)
## EFFECTIVE COMMUNICATION — Table of Included Studies

<table>
<thead>
<tr>
<th>STUDY</th>
<th>METHODS</th>
<th>PARTICIPANTS</th>
<th>INTERVENTIONS</th>
<th>RESULTS</th>
<th>QUALITY SUMMARY</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abenheim 1995</td>
<td>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.</td>
<td>n = 2500, 2347 met criteria of more than 1 day off but no compensation in previous 2 years for back pain.</td>
<td>N/A</td>
<td>13.3% had no diagnosis within 7 days (48% of those went on to become chronic); 43.8% had no diagnosis in first week; only 8–9% were given a specific diagnosis. Older workers were 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 = E Data extractors were blinded; the outcome measure was independent (computer database) Drop out rate = U Overall bias rating = Moderate</td>
<td>Adequate allocation concealment = Y Blinding = double blinded plus outcome assessor blindedITT analysis = U LTFU = 15% (N; 88% followed) Bias = Moderate</td>
<td>Specific diagnoses (i.e. neck, thoracic or low back pain) are not described in the 8.9% with a specific diagnosis.</td>
</tr>
<tr>
<td>Burton 1999</td>
<td>Randomised controlled trial in primary care setting in 5 general practices and 1 osteopathic practice in England. 88% of eligible patients agreed to participate.</td>
<td>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 = 81 (41 F; 42 M); mean age 42.6 ± 12.2 years. Control group: n = 79 (48 F; 31 M); mean age 44.7 ± 12.2 years.</td>
<td>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 discusion of patient’s involvement and responsibilities. Traditional booklet advised against activity if in pain, described spinal injury and damage, encouraged passivity.</td>
<td>Significant reduction in mean belief scores at 3/12 and 6/12 p = 0.002, and at 12/12 p = 0.05. For clinically important improvement, at 25/22 RR = 2.52; 1.77–3.40 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 blindedITT analysis = U LTFU = 15% (N; 79% followed) Bias = Moderate</td>
<td>Adequate allocation concealment = U Blinding = double blinded plus outcome assessor blindedITT analysis = U LTFU = 15% (N; 88% followed) Bias = Moderate</td>
<td>Doesn’t describe method of randomisation.</td>
</tr>
<tr>
<td>Croft 1998</td>
<td>Consecutive cohort of new presentations, South Manchester LBP cohort.</td>
<td>n = 800 presentations to GP for back pain over 1 year. Mean duration of pain 3 weeks. M = 203; F = 297 Excluded if presented in past 3/12</td>
<td>59% of subjects did not consult again for their back pain in the next 3/12, 30% had repeat consultation but this occurred within the first 3/12. Patients &gt; 30 years were 3 times more likely to have repeat consultation than younger low back pain patients, 25% stated fully recovered at 12/12.</td>
<td>Selection bias minimised = N Adjusted for confounders = Y Measurement bias minimised = U Drop out rate = 20% Overall bias rating = Moderate</td>
<td>Adequate allocation concealment = U Blinding = N ITT analysis = Y LTFU = 15% (N; 17% followed) Bias = Moderate</td>
<td>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 38% compared with 23% in the study sample. Therefore the estimates of poor outcome stated by the authors may be overestimates.</td>
</tr>
<tr>
<td>Dayo 1997</td>
<td>RCT. Stratified by number of prior episodes and employment status.</td>
<td>Low risk subjects from consecutive patients presenting to walk in clinic of public hospital. 82% were Hispanic. Study comprised immediate x-ray (n = 43; F24, M19); mean age 34.3 years, mean duration of pain 12.6 days) vs education (n = 48; F24, M25); mean age 32.5 years, mean duration of pain 16.6 days</td>
<td>Immediate lumbar spine x-ray vs education (5 minutes. brief advice) vs x-ray at 3/12 if unimproved</td>
<td>50% of eligible enrolled. Follow up at 3/12 was 92% and at 12/12 was 83%. No significant difference in any of the self reported measures of pain, satisfaction with care, functional ability, days off work. Overall the 46% of x-ray group had an x-ray vs. 20.3% of the education group over 3/12. Adequate allocation concealment = U Blinding = N ITT analysis = Y LTFU = 15% (N; 17% followed) Bias = Moderate</td>
<td>Adequate allocation concealment = U Blinding = N ITT analysis = Y LTFU = 15% (N; 17% followed) Bias = Moderate</td>
<td>Patients also randomised to receive 2 days vs. 7 days of bed rest but these groups were combined for this report and analysis.</td>
</tr>
</tbody>
</table>
### EFFECTIVE COMMUNICATION — Table of Included Studies continued

<table>
<thead>
<tr>
<th>STUDY</th>
<th>METHODS</th>
<th>PARTICIPANTS</th>
<th>INTERVENTIONS</th>
<th>RESULTS</th>
<th>QUALITY SUMMARY</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indahl 1995</td>
<td>Quasi-randomised study</td>
<td>Patients with &gt;8 weeks sick leave with a sick leave certificate</td>
<td>Conventional care versus mini-back school (advice to be active, education about pain, reassurance). All subjects were fully examined including x-ray and CT scan. The interventions 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</td>
<td>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</td>
<td>Adequate allocation concealment: = N (intervention group given even number and control group given odd numbers) Blinding = N ITT analysis = Y LTFU &lt; 15% = Y Bias = moderate</td>
<td>Difficult to determine method of randomisation. Patients with subacute pain 8–12 weeks. No documention of conventional care</td>
</tr>
<tr>
<td>Indahl 1998</td>
<td>Five year follow-up of 500 subjects from the original cohort (Indahl 1995 study)</td>
<td>Interventions group = 245 (125 M; 89.5% mean age 68.6 years + 15); Control group = 244 (125 M; 94 F; mean age 68.9 + 13.3); Norway National Insurance Office provided the follow-up data</td>
<td>As above (mini-back school vs. conventional care)</td>
<td>RTW at 5 years; Interventions group = 83% and Control group = 38%. Permanent disability (i.e., no RTW); Interventions group = 10% and Control group = 36%. 38% 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 (68%) compared with the intervention group (48%), p = 0.03</td>
<td>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 &lt; 15% = N Bias = moderate</td>
<td>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 documention of conventional care</td>
</tr>
<tr>
<td>McGuirk 2003</td>
<td>Open-label, non-randomised trial of 13 special GPs (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</td>
<td>Treatment X 3 months according to evidence-based guidelines for acute low back pain versus usual care</td>
<td>1.4% found to have features alerting to the presence of serious conditions associated with their LBP</td>
<td>Adequate allocation concealment: = N (N/A) Blinding = multi-blinded ITT analysis = Y LTFU &lt; 15% = Y Bias = high (non-randomised)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Evidence-based Management of Acute Musculoskeletal Pain

#### ACUTE LOW BACK PAIN — Table of Included Studies (Diagnosis)

<table>
<thead>
<tr>
<th>STUDY</th>
<th>METHODS</th>
<th>PARTICIPANTS</th>
<th>TEST</th>
<th>RESULTS</th>
<th>QUALITY SUMMARY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hollingworth 2002</td>
<td>An observational/descriptive study of the impact of guidelines for lumbar spine radiography on GP practice provides useful background information</td>
<td>Median duration of low back pain was 30 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</td>
<td>Primary outcome, Roland Morris scale; secondary VAS, EuroQol, satisfaction with care, use of health services. Randomised to x-ray (n = 250) and non-x-ray (n = 211)</td>
<td>At 3 months, more pain and disability and medical attention in x-ray group (RR 1.26, 95% C.I. 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 x-ray but not at 3/12. Overall, 80% of patients would want x-ray if given the choice. An abnormal finding on x-ray made no difference to the outcome by the Roland Score.</td>
<td>Adequate allocation concealment = Y ITT analysis = Y LTFU &lt; 15% = N (93.6% at completion; follow-up at 3/12 and 9/12) Blinded = N Bias rating = Moderate Groups similar at baseline</td>
</tr>
<tr>
<td>Kendrick 2001</td>
<td>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</td>
<td>No x-ray (n = 76; mean age 44, past hx back pain 57%, bad pain 20%).</td>
<td>Randomised to either x-ray or no x-ray (observational). Questionnaires provided at baseline, 6 weeks and 1 year</td>
<td>x-ray group had no effect on physical functioning, pain or disability but had small improvement in psychosocial well-being compared to non-x-ray group (n = 211) RR: 89%, 73%, 69%</td>
<td>Adequate allocation concealment = Y ITT analysis = Y LTFU &lt; 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</td>
</tr>
<tr>
<td>Knutson 2002</td>
<td>Randomised unblinded controlled trial, prospective with an observational arm to enable comparisons</td>
<td>24 volunteers of the community at various social gatherings including exercise classes; 50% M; mean age: 35 years (11–65); 82% had had back pain in the past; Community based</td>
<td></td>
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</tr>
<tr>
<td>Kerry 2002</td>
<td>Randomised unblinded controlled trial, prospective with an observational arm to enable comparisons</td>
<td>No x-ray (n = 76; mean age 44, past hx back pain 57%, bad pain 20%). X-ray (n = 65; mean age 41, past history of back pain 54%, bad pain 22%). Patients from UK general practices. First presentation for low back pain</td>
<td>Randomised to either x-ray or no x-ray (observational). Questionnaires provided at baseline, 6 weeks and 1 year</td>
<td>x-ray group had no effect on physical functioning, pain or disability but had small improvement in psychosocial well-being compared to non-x-ray group (n = 211) RR: 89%, 73%, 69%</td>
<td>Adequate allocation concealment = Y ITT analysis = Y LTFU &lt; 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</td>
</tr>
<tr>
<td>Leboeuf-Yde 2002</td>
<td>Cross-sectional survey of leg length asymmetry, foot rotation and pelvic crest unleveling examiner was blinded</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Leboeuf-Yde 2002</td>
<td>Population based sample of volunteers from a twin register</td>
<td>2572 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</td>
<td></td>
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</tbody>
</table>

**Notes:**

- N/A: Not applicable
- Useful for additional information and for summary of radiography findings

**Appendix E:** Tables of Included and Excluded Studies
### ACUTE LOW BACK PAIN — Table of Excluded Studies (Diagnosis)

<table>
<thead>
<tr>
<th>STUDY</th>
<th>REASON</th>
</tr>
</thead>
<tbody>
<tr>
<td>Borenstein 2001</td>
<td>Descriptive view — opinion of single author. Mostly discussing chronic low back pain. Qualitative review focused on herniated disc related low back pain</td>
</tr>
<tr>
<td>Haijiao 2001</td>
<td>Not a diagnostic test study. No information on duration of pain</td>
</tr>
<tr>
<td>Hollenberg 2002</td>
<td>MRI study, did not specify duration of back pain. Primarily adolescent population all with clinical diagnosis of sport-related stress injury to spine</td>
</tr>
<tr>
<td>Hunter 2001</td>
<td>Concerned with chronic/persistent pain. Review includes neck and carpal tunnel and non-specific low back pain</td>
</tr>
<tr>
<td>Kilpikoski 2002</td>
<td>Chronic pain subjects (average duration of pain = 14 years)</td>
</tr>
<tr>
<td>McNally 2001</td>
<td>Patient group not well defined, other back pain sites; pain &gt; 6 weeks but cannot isolate the less than 12 week group; select group unresponsive to therapy; descriptive study of MRI findings only</td>
</tr>
<tr>
<td>Patel 2000</td>
<td>Qualitative review. No original data</td>
</tr>
<tr>
<td>Yelland 2001</td>
<td>Not concerned with low back pain only. Doesn't meet inclusion criteria. Does not specifically deal with acute pain — no duration given</td>
</tr>
<tr>
<td>Zuberbier 2001</td>
<td>No specific information on patient spectrum. Review is at least partly based on studies of chronic pain subjects (based on the reference list)</td>
</tr>
</tbody>
</table>
## ACUTE LOW BACK PAIN — Table of Included Studies (Prognosis)

<table>
<thead>
<tr>
<th>STUDY</th>
<th>METHOD</th>
<th>PARTICIPANTS</th>
<th>RESULTS</th>
<th>QUALITY SUMMARY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abenheim 1995</td>
<td>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</td>
<td>Medical records were retrieved for the diagnosis; f/u × 24/12 and recorded duration of compensated absence from work</td>
<td>Selection bias minimized; measurement bias minimized; adjusted for confounders; dropout rate U; overall bias rating = moderate</td>
<td><em>Evidence-based Management of Acute Musculoskeletal Pain</em></td>
</tr>
<tr>
<td>Croft 1998</td>
<td>Consecutive cohort of new presentations. The South Manchester LBP cohort</td>
<td>n = 2500; 2147 met criteria of more than 1 day off but no compensation in previous 2 years for back pain</td>
<td>Selection bias not accounted for; measurement bias minimized; adjusted for confounders; dropout rate U; overall bias rating = moderate</td>
<td></td>
</tr>
<tr>
<td>Fransen 2002</td>
<td>Prospective cohort study</td>
<td>1464 hemets (compensation cohort) with 884 (51.5%) providing data, 3/68 (5.5%) of these not meeting inclusion criteria and 2/68 (2.9%) of these meeting criteria for outpatient treatment</td>
<td>Selection bias not accounted for; measurement bias minimized; adjusted for confounders; dropout rate U; overall bias rating = Low</td>
<td></td>
</tr>
<tr>
<td>Fritz 2001</td>
<td>Sequential selection of non-contact patients from a random sample of acute low back pain patients selected for a telephone interview (n = 5000)</td>
<td>The mean age of the study sample was 37.4 years; 50% prior history low back pain; baseline Oswestry 42.9 to 26.4 at 4 weeks</td>
<td>Selection bias not accounted for; measurement bias minimized; adjusted for confounders; dropout rate U; overall bias rating = Low</td>
<td></td>
</tr>
</tbody>
</table>

### Notes
- Specific diagnoses (i.e. neck, thoracic or low back pain) are not described in the study.
- A subsample of the non-interviewed group (n = 44) were followed (not stated how chosen) and found to have better outcomes than those who participated. Therefore the estimates of poor outcome stated by the authors may be overestimates.
- Possibly has to be excluded by reference to radiating pain however it is not clear if this is a true radiating pain or a somatic referral.
- Setting may not be typical of GP practice. Setting may not be typical of GP practice.
- Inpatient if acute low back pain, no available beliefs and pain work status.
- Inpatient if acute low back pain, no available beliefs and pain work status.
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<tr>
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<tbody>
<tr>
<td>Fritz 2000</td>
<td>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</td>
<td>69 same cohort as Fritz et al. 2001. Rear avoidance, non-organic signs (0-5)/non-organic symptoms (05). Setting = referral to physiotherapist.</td>
<td>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 &lt;3.00 for all and generally &lt;1.0 for selection 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: 0.20; 2: 0.11; 3: 0.5; 4: 3.0. None of the non-organic tests served as effective screening tool.</td>
<td>Measurement bias minimised = U Bias rating = None Selection bias minimised = Y Adjusted for confounders = Y</td>
<td>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. Does involve ALBP. This is not necessarily representative of ps presenting to GP practice.</td>
</tr>
<tr>
<td>Hurley 2001a</td>
<td>Prospective cohort study</td>
<td>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 &lt;3 months.</td>
<td>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 ALBP5Q (a biopsychosocial risk profile score) to predict chronicity. Although the score was correlated with pain and disability at 12/12, sensitivity (53%) and specificity (69%) for predicting recurrences were low. A high score had a high sensitivity (59%) for predicting work loss at 12 months but a lower specificity (81%).</td>
<td>Measurement bias minimised = N Drop out rate = 38% Bias rating = moderate Selection bias minimised = U Adjusted for confounders = Y</td>
<td>Include because within 12 week period and results presented separately by duration of pain. Claimant cohort 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.</td>
</tr>
<tr>
<td>Krause 2001</td>
<td>Retrospective cohort study to determine the association of psychosocial factors with duration of work disability</td>
<td>Claimant cohort with acute LBP n = 433 (70% male; mean age 37.3 years). 33% had previous back LTI.</td>
<td>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 &gt; 1 year: p = 0.001.</td>
<td>Measurement bias minimised = U Drop out rate = 0 Retrospective Bias rating = Low Selection bias minimised = U Adjusted for confounders = U</td>
<td></td>
</tr>
<tr>
<td>Linton 1998</td>
<td>Prospective cohort study. Looked at accumulated leave at 6/12 and whether a screening tool could predict this</td>
<td>Recruited from 15 health clinics in Sweden. Included acute/subacute back and neck pain, n = 142 eligible; 137 participated. Mean age 46.6 years (22-63). F = 45% 59% Swedish; 59% back, 53% should, 53% neck pain.</td>
<td>At 6/126/12% had no sick leave; 18% required short term leave (1-30 days) and 20% had long term leave. Factors associated with 7% of chronic pain sufferers: belief that one should not work with current pain levels (less avoidance; perceived chance of working within 6/12 light work: problems with work function); stress; previous number of sick leave days.</td>
<td>LTU &lt;15% = Y Bias = high (non-randomised) Adequate allocation concealment = N (N/A) Blinding = unblinded ITT analysis = Y</td>
<td></td>
</tr>
<tr>
<td>Linton 2001</td>
<td>A systematic review of the literature on the role of psychosocial workplace factors on back pain</td>
<td>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.</td>
<td>Number of studies = 21 Quality assessment = Y Data pooled = N Heterogeneity = Y</td>
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</table>
### ACUTE LOW BACK PAIN — Table of Included Studies (Prognosis) continued

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<tr>
<td>McGuirk 2001</td>
<td>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</td>
<td>Participants had LBP for less than 12 weeks with pain present for at least several days. Workers’ compensation recipients excluded. 437/545 participated out of those eligible. Median duration of pain approximately 2 weeks</td>
<td>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 = 66%; at 6/12 = 70%; at 12/12 = 78%; Full recovery at 3/12 for usual care: arM = 48%; 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%.</td>
<td>Adequate allocation concealment = N</td>
<td>ITT analysis = Y</td>
</tr>
<tr>
<td>Pincus 2002</td>
<td>Systematic review of prospective cohort studies in acute or sub-chronic low back pain, minimum 12 month follow-up</td>
<td>Identified 25 studies, 18 independent cohorts</td>
<td>Only two got three star rating. Six met acceptability criteria: methodology, psychosocial 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.</td>
<td>Number of studies = 25</td>
<td>Quality assessment = Y</td>
</tr>
<tr>
<td>Pulliam 2001</td>
<td>Designed as a prospective study and only presented as a cross sectional analysis with low response rate and modelling</td>
<td>574/524 screened; divided into 28 low and 29 high risk of chronic back pain disability based on Gatchel.</td>
<td>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 I disorders (affective, anxiety, somatoform, substance abuse) did not appear to be independent predictors of unfavourable outcome.</td>
<td>Measurement bias minimised = N</td>
<td>Drop out rate = not a follow-up study</td>
</tr>
<tr>
<td>Schiottz-Christensen 1999</td>
<td>Prospective cohort study in general practice in Denmark. 330 GPs invited to participate; 132 agreed, 75 enrolled. Range recruited (1-41; median 6)</td>
<td>524/123 patients under 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.</td>
<td>Adequately randomised = N</td>
<td>Blinding = N</td>
<td>ITT analysis = Y</td>
</tr>
<tr>
<td>Shaw 2001</td>
<td>Systematic review to synthesise findings from available studies of prognostic factors for acute OLBP disability</td>
<td>22 studies met criteria for inclusion</td>
<td>Prognostic factors: low workplace support; personal stress; shorter job tenure; prior episodes; heavier work load; majority with no modified duties; delayed reporting; severity of pain; functional impact; radiologic 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.</td>
<td>Number of studies = 22</td>
<td>Quality assessment = N</td>
</tr>
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</table>

*OLBP = acute or sub-chronic low back pain; LTFU = loss to follow-up; ITT = intention to treat; arM = anchor for meaningfulness; SNAP = Somatic Symptom and Pain Questionnaire; MMPI = Minnesota Multiphasic Personality Inventory; LTFU: <15% = U; Bias rating = High; Measurement bias minimised = N; Drop out rate = not a follow-up study; Bias rating = High; Selection bias minimised = N; Adjusted for confounders = Y; 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.
### ACUTE LOW BACK PAIN — Table of Included Studies (Prognosis) continued

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<tr>
<td>Truchon 2000</td>
<td>Systematic review of three electronic databases (Medline, PsycInfo, Sociofile) plus review of reference lists 1987–1999. Looked at prospective studies of LBP &lt; 12 weeks</td>
<td>Reviewed 38 articles that met criteria: looking at occupational factors that influence LBP</td>
<td>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.</td>
<td>Number of studies = 18</td>
<td>Not clear whether articles double-read</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Measurement bias minimised = N</td>
<td>Data pooled = U</td>
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<td>Measurement bias minimised = N</td>
<td>Heterogeneity = Y</td>
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<td></td>
<td>Measurement bias minimised = N</td>
<td>Bias rating = Low</td>
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<td></td>
<td></td>
<td></td>
<td>Measurement bias minimised = N</td>
<td>Adjusted for confounders = Y</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Measurement bias minimised = N</td>
<td>Not much detail on patient population but gives reference to baseline study, Spine 1999*unsure how referred initially</td>
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<p>| Werneke 2000     | 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. | 223 pts with LBP (original cohort described elsewhere); mean age 37.8 (99.52%) males; 38.2% multiple sites; 30.7% leg pain at intake; 79.9% high pain at intake and mean 13.3 + 4.4 days pain; 10% workers comp. Lot of psychosocial factors (depressive (4%), somatisation (4%), fear-avoidance beliefs (4%), 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 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) <em>Dependent (outcomes at 12 months) see Table: pain pattern classification (non-centralisation) and leg pain at intake were the strongest predictive variables of chronicity | Bias rating = Low                     |                                                         |
|                  |                                                                        |                                                                              | Measurement bias minimised = N                                                        | Adjusted for confounders = Y          |                                                         |
|                  |                                                                        |                                                                              | Measurement bias minimised = N                                                        | Not much detail on patient population but gives reference to baseline study, Spine 1999</em>unsure how referred initially |                                                         |</p>
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<tbody>
<tr>
<td>BenDebba 2002</td>
<td>Chronic LBP mean 2.52 years for current episode. Non-randomised — describing interventions. Chronic study population (average pain duration = 10 years)</td>
</tr>
<tr>
<td>Dionne 2001</td>
<td>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</td>
</tr>
<tr>
<td>Hemmel 2002</td>
<td>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</td>
</tr>
<tr>
<td>Hudak 1996</td>
<td>A critical appraisal of the research; not primary research</td>
</tr>
<tr>
<td>Kaiser 2001</td>
<td>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</td>
</tr>
<tr>
<td>Kjellman 2001</td>
<td>Inadequate description/definition of duration of pain at baseline (50% had sick leave of &gt;28 days or more); primary focus is comparison of pain episodes/impact on ADLs between pain sites (back pain vs neck/shoulder); backgroup included sciatica disc problems</td>
</tr>
<tr>
<td>Kuch 2001</td>
<td>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</td>
</tr>
<tr>
<td>Kummel 2001</td>
<td>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</td>
</tr>
<tr>
<td>Linton 2001</td>
<td>Broad review of back and neck pain and which interventions prevent neck and back pain. Does not meet inclusion criteria</td>
</tr>
<tr>
<td>Linton 2002</td>
<td>Looks at effect of abuse on pain; does not meet inclusion criteria for acute pain</td>
</tr>
<tr>
<td>Loisel 2002</td>
<td>Includes thoracic spine pain, subacute rather than acute pain, radiating pain, neurological signs and surgery — contaminated sample</td>
</tr>
<tr>
<td>Lundin 2001</td>
<td>Study deals with onset of LBP at follow-up in a cohort who had back x-rays at baseline approx. 10 years earlier. Not acute LBP. Seems high rate of self-reported pain. Different definition of pain — could be whole spine</td>
</tr>
<tr>
<td>McIntosh 2000b</td>
<td>No description of duration of LBP; no description of consumer population and no prognosis; no results given</td>
</tr>
<tr>
<td>Musgrave 2001</td>
<td>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</td>
</tr>
<tr>
<td>OSullivan 2000</td>
<td>Commentary — not original</td>
</tr>
<tr>
<td>Shannon 2001</td>
<td>Duration of LBP uncertain and discusses risk factors for onset of LBP; low numbers</td>
</tr>
<tr>
<td>Sleg 2001</td>
<td>Uncontrolled intervention. Most subjects chronic. Results not shown separately for acute/chronic</td>
</tr>
<tr>
<td>Torp 2001</td>
<td>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</td>
</tr>
<tr>
<td>Trainor 2002</td>
<td>Qualitative review. No original data. Discusses risk factors for development of LBP in athletes</td>
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**ACUTE LOW BACK PAIN — Table of Included Studies (Interventions)**

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<tr>
<td>Browning 2001</td>
<td>Meta-analysis of cyclobenzaprine and back pain</td>
<td>Mixture of acute and chronic back and neck pain; 12 of the 14 studies only included ALBP (sensitivity analysis showed no difference between studies for acute and chronic LBP)</td>
<td>Compares cyclobenzaprine to placebo, not other medications. Used variable doses of cyclobenzaprine from 10-60mg daily between trials.</td>
<td>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 (oral pain, muscle spasm, tenderness to palpation, range of motion, ADL). Local pain improvement days 1-4 0.41 (0.23, 0.58) and day 5-9 0.38 (0.21, 0.56) and days &gt;9 0.41 (0.24, 0.58). Efficacy was greatest in the first few days declining after the first week. Cyclo group experienced more adverse effects. Drowsiness p &lt;0.001, dry mouth p = 0.02, dizziness = 0.04, nausea p = 0.70 any 53% compared with 28% in placebo p = 0.002. 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 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 &lt; 15% = Y (0 at 1 year; &lt; 15% at 3 years) Bias = Moderate Recall bias (patients not blinded; main outcome measure patient self-report)</td>
<td>Number of RCTs = 14 Quality assessment = Y Data pooled = Y Heterogeneity = Y Applicability of results = not available in Australia</td>
<td></td>
</tr>
<tr>
<td>Furian 2002</td>
<td>Massage for LBP: a systematic review of RCT, quasi-RCT or CCT for massage for NSLBP. Cochrane Review</td>
<td>Patients with acute, subacute and chronic non-specific LBP</td>
<td>Any type of massage (M) using hands or mechanical device used to treat low back pain.</td>
<td>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 = exercises; M &gt; 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.</td>
<td>Number of RCTs = 8 (2 included mixed subacute/chronic; 1/8 included only ALBP patients) Quality assessment = Y, blinded Data pooled = N Heterogeneity = Y</td>
<td>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</td>
</tr>
<tr>
<td>Hagen 2002</td>
<td>Bed rest and advice to stay active: a systematic review included quasi-randomised studies</td>
<td>9 (n = 1435 subjects in total). Trials included patients with acute low back pain with or without radiation of pain below the knee</td>
<td>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</td>
<td>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 &lt; 15% = Y (0 at 1 year; &lt; 15% at 3 years) Bias = Moderate Recall bias (patients not blinded; main outcome measure patient self-report)</td>
<td>Number of RCTs = 9 Quality assessment = Y (9 included low risk of bias) Data pooled = Y Some outcomes only Heterogeneity = Y</td>
<td></td>
</tr>
<tr>
<td>Hides 2001</td>
<td>RCT; randomisation method not described</td>
<td>First episode of unilateral mechanical low back pain of less than 3 weeks presenting to emergency department</td>
<td>Intervention group (n = 24) 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.</td>
<td>There was reduced multifidus muscle size on the affected side in the short term (results reported in earlier study). In the control group, 94% 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 &lt; 15% = Y (0 at 1 year; &lt; 15% at 3 years) Bias = Moderate Recall bias (patients not blinded; main outcome measure patient self-report)</td>
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**EVIDENCE-BASED MANAGEMENT OF ACUTE MUSCULOSKELETAL PAIN**
### Evidence-Based Management of Acute Musculoskeletal Pain

**ACUTE LOW BACK PAIN — Table of Included Studies (Interventions)** continued

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</thead>
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<tr>
<td>Hilde 2002</td>
<td>Advice to stay active: systematic review</td>
<td>43 (n = 495 subjects in total). Included acute, subacute &amp; chronic LBP with &amp; without radiation below the knee; excluded red flag conditions</td>
<td>Advice to stay active as a single treatment for acute low back pain with and without sciatica.</td>
<td>Number of RCTs = 4 Quality assessment = Y</td>
<td>Data pooled = N Heterogeneity = Y</td>
<td>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.</td>
</tr>
<tr>
<td>Hsieh 2002</td>
<td>RCT tools to assess the effect of four conservative treatments for subacute low back pain</td>
<td>200 pts in an outpatient physiotherapy clinic in the USA. Assessed at baseline, after 6/12 after therapy completed. Subjects had LBP ≥3 weeks and ≤6 months from current episode or pain-free ≥3 1/2</td>
<td>Back school vs. myofascial therapy, joint manipulation and combined MPT and JM. Back school comprised education, and body mechanics for daily activities and supervised home program.</td>
<td>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.</td>
<td>Adequate allocation concealment = U Blinding = Y (examiners) ITT analysis = Y LTFU &lt; 15% = Y (8% at 3/52, 11% at 6/12) Bias = Low</td>
<td>Includes some pts with pain ≥3 months, mean duration of pain 10.7–11.5 (+ 6–7 weeks). No control group.</td>
</tr>
<tr>
<td>Hurley 2003b</td>
<td>RCT investigating interventional electrode placement in acute low back pain compared with control treatment. Single blind, randomised controlled trial with 3 1/2 follow-up</td>
<td>N = 58.48 remaining at 3 1/2 GP and self-referrals to the study in GP physiotherapy departments, hospital and university settings</td>
<td>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</td>
<td>1) MPQ mean values 5.0, 2.0 and 3.0, p = 0.052 2) RMDQ mean values 2.0, 1.0 and 1.0, p = 0.049 3) EQ — SD mean 0.0, 0.8 and 1.0, p = 0.049. There is a trend toward improvement in pain and disability scores with IFT over the spinal nerve area compared with painful area or control.</td>
<td>Adequate allocation concealment = Y Blinding = N, U ITT analysis = Y LTFU &lt; 15% = N (20% LTFU at 7/12) Bias = High</td>
<td>69% had LBP &lt; 3 months duration. Sample size eligible consumer only 60. p values borderline.</td>
</tr>
<tr>
<td>Karjalainen 2002</td>
<td>Cochrane Review (last updated 1999)</td>
<td>2 RCTs (n = 238 subjects in total)</td>
<td>Effectiveness of multi-disciplinary rehabilitation for subacute (≥4 week) and &lt;12 week low back pain among working age adults. Intervention was a physician consultation plus either a psychological, vocational or social intervention, or combination of these.</td>
<td>Moderate evidence of positive effects of multi-disciplinary rehabilitation for subacute low back pain: workplace visit increases the effectiveness</td>
<td>Number of RCTs = 2 Quality assessment = Y (Both studies low quality, but data missing on several quality assessment criteria) Data pooled = N Heterogeneity = Y</td>
<td>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.</td>
</tr>
<tr>
<td>Little 2001</td>
<td>RCT factorial trial to assess effectiveness of a booklet of physician advice to take regular exercise</td>
<td>N = 311. R 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 231 (77%) of pts</td>
<td>1. Receive a detailed self-mgt booklet 2. Advice to take regular exercise 3. Back school 4. Neither</td>
<td>Follow-up at 1 week: authors concluded that pain and functional outcomes can be improved with the use of either advice to exercise (p = 0.098) or provision of a booklet (p = 0.0049), but the effect was reduced if both of these interventions were used (p = 0.058). Reviewers conclude that result for exercise was not significant; follow-up at 3 weeks — no significant difference in pain/function between all groups.</td>
<td>Adequate allocation concealment = N Blinding = N (data collectors) ITT analysis = U LTFU &lt; 15% = N (23% LTFU) Bias = Moderate</td>
<td></td>
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</tbody>
</table>
## ACUTE LOW BACK PAIN — Table of Included Studies (Interventions) continued

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<tr>
<th>STUDY</th>
<th>METHODS</th>
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<th>RESULTS</th>
<th>QUALITY SUMMARY</th>
<th>NOTES</th>
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</thead>
<tbody>
<tr>
<td>Maher 1999</td>
<td>Systematic review evaluating 'activity prescription' for non-specific low back pain (NSLBP)</td>
<td>Acute (&lt;6 weeks) and subacute (6 weeks to 3 months) NSLBP. 9 RCTs for exercise therapy for acute NSLBP</td>
<td>'Activity prescription', i.e. bed rest, advice to alter activity levels and exercise</td>
<td>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.</td>
<td>Number of RCTs = 9</td>
<td>Search of Medline &amp; CINAHL (up to 1999)</td>
</tr>
<tr>
<td>Nadler 2002</td>
<td>Prospective, randomised single blind comparative efficacy trial</td>
<td>N = 371; F = 216; M = 155, with acute low back pain, 11 settings/sites</td>
<td>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 follow-up</td>
<td>Outcomes discussed: pain, muscle stiffness, lateral trunk flexibility. At day 2, significantly better for group 1 than 2 (p = 0.0008) and 3 (p = 0.0007) but only looked at results for 2 days.</td>
<td>Adequate allocation concealment = Y</td>
<td>FAU 2 days only. Patients equivalent at baseline</td>
</tr>
<tr>
<td>Nelemans 2002</td>
<td>Cohrane review of steroid injection therapy</td>
<td>25 RCTs: 'Benign' LBP of at least 4wks duration; included studies with chronic pain subjects</td>
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<tr>
<td>Palangio 2002</td>
<td>RCT — combination hydrocodone and ibuprofen vs acetaminophen, Computer-randomised, double-blind, Intention to treat analysis and &gt;95% follow-up</td>
<td>N = 197; mean age 49y. Adults with moderate to severe acute LBP requiring opioid or opioid-narcotic combination therapy, enrolled within 48 hours of onset pain, self-rated pain moderate-severe</td>
<td>Hydrocodone 7.5mg and ibuprofen 200mg (HC/IB) vs. combination oxycodone 5mg and acetaminophen 325mg (OW/A)</td>
<td>No difference between groups in daily pain relief and SF-36 scores at 8days.</td>
<td>Adequate allocation concealment = Y</td>
<td>Follow-up = 8days. No difference between groups at baseline</td>
</tr>
<tr>
<td>Pengel 2002</td>
<td>Systematic review and meta-analysis of RCTs</td>
<td>Subacute (6weeks to 3 months) non-specific LBP with or without referral. Also used alternate definition of subacute (7 days to 6 months)</td>
<td>Manipulation, back schools, exercise, advice, TENS, massage, corset, cognitive behaviour treatment, coordination of primary care</td>
<td>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.</td>
<td>Number of RCTs = 13</td>
<td>Lack of a uniform definition for subacute LBP is a problem</td>
</tr>
<tr>
<td>Pohjalainen 2000</td>
<td>RCT — randomised, blinded to category, well designed</td>
<td>104 (5 withdrew); F = 52; M = 52. Median age 42 (29-63); Finnish outpatient clinic. Patients had &gt;4 weeks duration LBP with no sciatica or radicular pain</td>
<td>Comparisons nimesulide (COX-2 selective anti-inflammatory) with ibuprofen</td>
<td>Significant improvement in Oswestry score at 10 days for COX-2 group (p &lt; 0.002). There was no significant difference in pain relief between groups.</td>
<td>Adequate allocation concealment = Y</td>
<td>Excluded LBP &gt;4 weeks duration, sciatica or radiating pain. Reviewer got different values than author on t-test analysis</td>
</tr>
<tr>
<td>STUDY</td>
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<tr>
<td>Rozenberg 2002</td>
<td>RCT — randomised to normal activity or 4 days bed rest</td>
<td>278 multi-centre ambulatory patients. Bedrest = 137; activity = 140</td>
<td>4 days bed rest vs. normal activity (as much activity as pain would allow). All received analgesics and muscle relaxants</td>
<td>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</td>
<td>Adequate allocation concealment = Y</td>
<td>Blinding = N ITT analysis = Y LTFU = 13% = Y (2.5%) Bias = Moderate</td>
</tr>
<tr>
<td>Stam 2001</td>
<td>RCT</td>
<td>683 patients in 19 GP practices in UK. SRL = 83; CCC = 78 M = 87; F = 74</td>
<td>Spironflor (SRL) homeopathic gel vs. cremeol capisci (CCC)</td>
<td>No significant difference in VAS pain after 1 week</td>
<td>Adequate allocation concealment = U ITT analysis = Y LTFU = 15% = Y Bias = Low</td>
<td>Participants not blinded. No placebo or control. Equivalent at baseline.</td>
</tr>
<tr>
<td>van Tulder 2002</td>
<td>Cochrane Review</td>
<td>Mixed settings, hospital, outpatient and general practice</td>
<td>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</td>
<td>There is no difference in pain intensity between NSAIDs and placebo (9/11 trials but significant heterogeneity p &lt; 0.01) but increased global improvement (RR = 1.1495% CI 1.10, 1.19; 6/9 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.</td>
<td>Number of RCTs = 51 (total); 9ALBP specific trials of NSAID vs placebo analysed separately Quality assessment = Y Data pooled = Y Heterogeneity = N</td>
<td>Includes trials where non-specific back pain may be associated with sciatica and chronic/acute LBP.</td>
</tr>
<tr>
<td>van Tulder 2000</td>
<td>Exercise therapy for LBP. Systematic review</td>
<td>10 trials on acute LBP included</td>
<td>Eight different exercise therapies included compared with numerous controls (other conservative treatment, usual care, manual therapy, back school and NSAIDs)</td>
<td>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</td>
<td>Number of RCTs = 10 Quality assessment = Y Data pooled = Y Heterogeneity = Y Due to heterogeneity, difficult to interpret and apply results</td>
<td>Included acute and chronic back pain. Problems with pooling different types of exercise therapies; care needs to be taken with interpretation. Heterogeneity likely to be significant.</td>
</tr>
<tr>
<td>van Tulder 2002a</td>
<td>Cochrane Review of acupuncture</td>
<td></td>
<td></td>
<td></td>
<td>Number of RCTs = 11 (only one met the criteria for this update) Quality assessment = Y Data pooled = N Heterogeneity = Y</td>
<td>Mix of acute and subacute (12 weeks or less) and chronic with results pooled; poor quality studies to review</td>
</tr>
<tr>
<td>van Tulder 2002d</td>
<td>Cochrane Review of exercise therapy for low back pain</td>
<td></td>
<td></td>
<td>The evidence summarised in this systematic review does not indicate that specific exercises are effective for the treatment of acute low back pain</td>
<td>Number of RCTs = 19 (12 studies on acute low back pain; only 1 RCT met the criteria for this update Quality assessment = Y Data pooled = N Heterogeneity = Y</td>
<td></td>
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</table>
### ACUTE LOW BACK PAIN — Table of Included Studies (Interventions) continued

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<tr>
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<tr>
<td>van Tulder 2002c</td>
<td>Cochrane Review of lumbar supports</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Number of RCTs = 6 (studies on use of lumbar support for treatment) Quality assessment = Y Data pooled = N Heterogeneity = Y</td>
</tr>
<tr>
<td>van Tulder 2002b</td>
<td>Back schools for non-specific LBP, Cochrane Review</td>
<td>Two trials (n = 217, n = 100)</td>
<td>Back school = education and skills program including supervised exercises</td>
<td>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</td>
<td>Number of RCTs = 2/15 were LBP specific Quality assessment = Y (blinded) Data pooled = N Heterogeneity = N</td>
<td>Mixed populations with radiating pain, or unspecified. Acute LBP analysed separately. Question feasibility, cost</td>
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## ACUTE LOW BACK PAIN — Table of Excluded Studies (Interventions)

<table>
<thead>
<tr>
<th>STUDY</th>
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<tbody>
<tr>
<td>Anonymous 2001a</td>
<td>This is an editorial only. Not a systematic review</td>
</tr>
<tr>
<td>Anonymous 2001b</td>
<td>Is for chronic low back pain and is only an editorial. Editorial comment only</td>
</tr>
<tr>
<td>Blomberg 1994</td>
<td>Did not meet inclusion criteria due to inclusion of patients with leg pain and herniated discs</td>
</tr>
<tr>
<td>Brockow 2001</td>
<td>Majority of patients had pain &gt;3 months (i.e. 137/140). Only 34/140 had LBP. Others had neck, or neck plus back pain</td>
</tr>
<tr>
<td>Cardon 2002</td>
<td>Involved children and preventive interventions. Primary prevention. Not an RCT</td>
</tr>
<tr>
<td>Chubaskik 2001</td>
<td>59% of patients had LBP &gt;3 months, and results not presented separately for those &lt;3 months. Lack of definition re: 6 months susceptibility</td>
</tr>
<tr>
<td>Cooperstein 2001</td>
<td>Not LBP intervention. Description of chiropractic techniques. Describes manipulative techniques</td>
</tr>
<tr>
<td>Del Mar 2001</td>
<td>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</td>
</tr>
<tr>
<td>Dreman 2001</td>
<td>Narrative review. Not a systematic review. Not specific to Acute LBP</td>
</tr>
<tr>
<td>Ernst 2000</td>
<td>Not a systematic review. A literature review on the effects of manipulation</td>
</tr>
<tr>
<td>Ernst 2001</td>
<td>None of included RCT were ALBP</td>
</tr>
<tr>
<td>Fisk-Ritson 2003</td>
<td>Narrative review</td>
</tr>
<tr>
<td>Frank 2001</td>
<td>Narrative review</td>
</tr>
<tr>
<td>Garfin 2002</td>
<td>Not a systematic review. Lit review of exercise therapy</td>
</tr>
<tr>
<td>Glouu yod 2001</td>
<td>Preventive strategy</td>
</tr>
<tr>
<td>Heigh 2001</td>
<td>Not a systematic review. Not reviewing acute non-specific LBP</td>
</tr>
<tr>
<td>Hawk 1999</td>
<td>Not acute LBP. Not a RCT</td>
</tr>
<tr>
<td>Hammilla 2002</td>
<td>Chronic LBP (&gt;7 weeks); patients with pain &lt;3 months not reported separately. Chronic lower back pain</td>
</tr>
<tr>
<td>Homey 2001</td>
<td>Not acute LBP. Prevention study</td>
</tr>
<tr>
<td>Hurwitz 2002</td>
<td>Mostly chronic LBP. Duration of pain greater than 3 months in most participants. Majority (60%) had sciatica</td>
</tr>
<tr>
<td>Jacobson 2002</td>
<td>Not an RCT (before and after study). Chronic LBP</td>
</tr>
<tr>
<td>Jordanning 2000</td>
<td>No methods, inclusion criteria included — narrative review. Not a systematic review</td>
</tr>
<tr>
<td>Kowac 2002</td>
<td>Subjects have sub-acute or chronic LBP and data is not presented separately for pain &lt;3 months (our definition of acute LBP). Included patients with chronic back pain &gt;12 weeks and those with referred pain</td>
</tr>
<tr>
<td>Larsen 2002</td>
<td>Preventive strategy; orthoses</td>
</tr>
<tr>
<td>Linton 2001</td>
<td>Preventive strategy involves both back and neck pain and does not meet criteria for inclusion</td>
</tr>
<tr>
<td>Linton 2001</td>
<td>Addressing primary prevention</td>
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</table>
### ACUTE LOW BACK PAIN — Table of Excluded Studies (Interventions) continued

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<thead>
<tr>
<th>STUDY</th>
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</thead>
<tbody>
<tr>
<td>Loisel 2001</td>
<td>Study is for subacute LBP (&gt;6 weeks) and data not presented separately for acute LBP (&lt;3 months by our definition). Not an RCT</td>
</tr>
<tr>
<td>Lorig 2002</td>
<td>Chronic LBP</td>
</tr>
<tr>
<td>Maier-Riehle 2001</td>
<td>Chronic LBP (mean 7.0 years)</td>
</tr>
<tr>
<td>Newsomenger 2000</td>
<td>Letter to the editor; not primary research</td>
</tr>
<tr>
<td>Newton-John 2000</td>
<td>Not an RCT. Describes a process</td>
</tr>
<tr>
<td>Onorato 2001</td>
<td>Subacute/chronic LBP</td>
</tr>
<tr>
<td>Pantilimon 2002</td>
<td>Chronic LBP</td>
</tr>
<tr>
<td>Quittan 2002</td>
<td>Narrative review. Not a systematic review or lit review</td>
</tr>
<tr>
<td>Ramley 2001</td>
<td>Review not systematic — not only LBP — looks at acupuncture for many conditions</td>
</tr>
<tr>
<td>Rosinol 2000</td>
<td>Likely to have substantial bias. More of an implementation study</td>
</tr>
<tr>
<td>Schreiber 2001</td>
<td>Participants only included if pain &gt;3 months duration; included neck and LBP</td>
</tr>
<tr>
<td>Sheppard 2001</td>
<td>Review of RCT only (&gt;chronic pain). Critical appraisal and is CLBP</td>
</tr>
<tr>
<td>Smith 2002</td>
<td>Narrative Review. Not a systematic review. Not specific to acute LBP</td>
</tr>
<tr>
<td>Strand 2001</td>
<td>Mean duration of back pain in participants is 10 years (+10 years)</td>
</tr>
<tr>
<td>Susman 2001</td>
<td>This is an editorial only. Not an RCT</td>
</tr>
<tr>
<td>van Deem 2001</td>
<td>Lit review — not RCT — narrative — healthy subjects</td>
</tr>
<tr>
<td>van Tulder 2002e</td>
<td>Addresses behavioural treatment for chronic LBP only</td>
</tr>
<tr>
<td>Veneema 2000</td>
<td>Does not distinguish acute LBP from chronic</td>
</tr>
<tr>
<td>Van Keef 2001</td>
<td>Not an RCT. Describes a process</td>
</tr>
<tr>
<td>Yesser 2001</td>
<td>Addressing primary prevention and is not a study of acute LBP. Back pain neck pain (summary of a review therefore exclude)</td>
</tr>
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</table>
### ACUTE LOW BACK PAIN — Table of Included Studies (Cost Effectiveness)

<table>
<thead>
<tr>
<th>AUTHOR</th>
<th>TITLE</th>
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<tbody>
<tr>
<td>Malmivaara 1995</td>
<td>The treatment of low back pain: bed rest, exercises, or ordinary activity?</td>
<td>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 = 69). FU at 3/52, 12/52. LTFU 11% no ITT analysis. No blindness. 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. 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 economic. Cochrane reviewer conclusion: small sample with high LTFU and self-reported outcomes.</td>
</tr>
<tr>
<td>Cherkin 1998</td>
<td>A comparison of physical therapy, chiropractic manipulation, and provision of an educational booklet for the treatment of patients with lower back pain.</td>
<td>RCT Group 1: educational booklet (n = 66); Group 2: chiropractic (n = 122); Group 3: McKenzie method (n = 133). 2 year follow-up. Interviewer blinded to subject assignment up to 4 weeks. ITT analysis: Group 2 had a 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. Conclusions: small sample with high LTFU and self-reported outcomes.</td>
</tr>
<tr>
<td>Moffett 1999</td>
<td>Randomised controlled trial of exercise for low back pain: clinical outcomes, costs, and preferences.</td>
<td>RCT; n = 187. Group 1: received community-based exercise program (n = 93) vs Group 2: usual primary care management which may/may not involve referral to physiotherapist (n = 94). Measured costs of direct medical cost to the NHS and included indirect costs (lost work days) at 12/52. Analysis of clinical improvement at 12/52: 64% (54–74) had clinically important improvement (no raw data shown). Group 2: had 39% (25–54) clinically important improvement. RCT; NNT 3.4 — only 5 people needed to do the exercise program for one more person to have clinically important improvement. Exercise group more clinically effective and more cost effective: cost of care Group 1: 300.15 (SD 582.77) UK pounds (1996) vs Group 2: 508.43 (SD 110.79) UK pounds (1996). Cost of days off work: Group 1: 378 days off work in 12/12 (cost = 17,010) vs group 2: 607 days off work in 12/12 (cost = 27,315).</td>
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### ACUTE LOW BACK PAIN — Table of Excluded Studies (Cost Effectiveness)

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<tr>
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<tbody>
<tr>
<td>Carey 1995</td>
<td>The outcomes and costs of care for acute low back pain among patients seen by primary care clinicians, chiropractors, and orthopaedic surgeons.</td>
<td>Observational study only — not a CEA — not an RCT — patients had significantly different demographic and prognostic characteristics — age, ethnic, income.</td>
</tr>
<tr>
<td>Mitchell 1994</td>
<td>Effectiveness and cost-effectiveness of employer-issued back belts in areas of high risk for back injury.</td>
<td>Prevention study — not an RCT — lots of confounding not continued for heavyweights and previous injury had to wear belts.</td>
</tr>
<tr>
<td>Sinclair 1997</td>
<td>The effectiveness of an early active intervention programme for workers with soft-tissue injuries: the early claimant cohort study.</td>
<td>Don't know duration of back pain; cohort study not RCT.</td>
</tr>
<tr>
<td>Skargren 1998</td>
<td>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.</td>
<td>Neck and back included together — don't know if they adjusted for these as subgroups.</td>
</tr>
<tr>
<td>Skargren 1997</td>
<td>Cost and effectiveness analysis of chiropractic and physiotherapy treatment for low back and neck pain: 6 month follow-up.</td>
<td>Have neck and back included; cannot tell if they have done subgroups and cannot determine duration of pain.</td>
</tr>
<tr>
<td>STUDY</td>
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<tr>
<td>Bryner 1989</td>
<td>Dolorimeter applied to region of transverse processes of each thoracic vertebra from T1 to T12 greater than 50 newtons regarded as normal if no flinching or pain reported from the subject</td>
<td>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</td>
</tr>
<tr>
<td>Christensen 2002</td>
<td>3 palpation procedures for the detection of spinal biomechanical 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</td>
<td>N = 107 subjects; 22 dropouts; 43 subjects enrolled in the interobserver reliability protocol and 54 and 25 respectively participated in the intrarater protocol. Setting: Dept of Nuclear Medicine, Denmark</td>
</tr>
<tr>
<td>Delamarter 1990</td>
<td>Discusses primary osseous neoplasms of the thoracic and lumbar spine. Case series of 29 adults and children with these rare tumors</td>
<td>N = 29 adults and children with primary osseous lesions between 1965 and 1985</td>
</tr>
<tr>
<td>Fukui 1997</td>
<td>Investigated the patterns of pain induced by distorting the cervical and lumbar zygapophyseal joints</td>
<td>N = 15 patients with back pain suspected to be of zygapophyseal origin</td>
</tr>
<tr>
<td>Kleineman 1978</td>
<td>Describes the clinical course of metastatic cancer of the spinal column in a 2 year retrospective study</td>
<td>N = 77 patients with documented metastatic cancer to the spinal column. Ages: 30–85 (mean 62); M = 48; F = 57; 51% had thoracic spine involvement</td>
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<tr>
<td>STUDY</td>
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<tr>
<td>Ozaki Toshifumi 2002</td>
<td>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</td>
<td>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 166 months in 1980s and 86 months in 1990s. Patient age range between 8 and 89 years. Average age 168 years with osteoid osteoma and 188 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</td>
</tr>
<tr>
<td>Patel 1991</td>
<td>Describes the events leading to 4 features of acute pain, previous medical history, VAS in patients with osteoporotic vertebral fractures</td>
<td>n = 30 patients with acute painful vertebral compression fracture within 14 days of the event, F = 26; M = 4. Women had early menopause (mean age 44, 30-54 yrs)</td>
</tr>
</tbody>
</table>
## ACUTE THORACIC PAIN — Table of Excluded Studies (Diagnosis)

<table>
<thead>
<tr>
<th>STUDY</th>
<th>REASON</th>
</tr>
</thead>
<tbody>
<tr>
<td>Comeaux 2001</td>
<td>Examination of normal subjects without pain. Not relevant in primary care. Based on osteopathic palpation skills. Study on normal subjects without pain — not been reviewed</td>
</tr>
<tr>
<td>Haneline 2000</td>
<td>Referred pain from thoracic spine. Does not meet inclusion criteria</td>
</tr>
<tr>
<td>Kandaberow 1997</td>
<td>Expert opinion, not primary research</td>
</tr>
<tr>
<td>Keating 2001</td>
<td>Investigates non-symptomatic subjects</td>
</tr>
<tr>
<td>LaBlanc 1994</td>
<td>Does not meet inclusion criteria. Deals with chest wall pain arising from fractures of the ribs and sternum</td>
</tr>
<tr>
<td>Mitra 1996</td>
<td>Chronic thoracic spinal pain</td>
</tr>
<tr>
<td>O’Neill 1989</td>
<td>Does not meet selection criteria</td>
</tr>
<tr>
<td>Quest 1987</td>
<td>Not primary research and not a systematic review; unsuitable references, too old for possible red flag value. Narrative review and 2 case studies</td>
</tr>
<tr>
<td>Raine 1994</td>
<td>Subject had dorsal spine pain. Pain excluded in entry criteria</td>
</tr>
<tr>
<td>Sward 1991</td>
<td>Dissection of pain not specified. Convenience samples for both cases and controls lead to a high chance of selection bias</td>
</tr>
<tr>
<td>Triano 1999</td>
<td>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</td>
</tr>
<tr>
<td>Wise 1992</td>
<td>Does not meet inclusion criteria</td>
</tr>
<tr>
<td>Wood 1999</td>
<td>Chronic pain</td>
</tr>
<tr>
<td>Yelland 2002</td>
<td>Does not meet criteria</td>
</tr>
</tbody>
</table>

## ACUTE THORACIC PAIN — Table of Excluded Studies (Prognosis)

<table>
<thead>
<tr>
<th>STUDY</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Cockerill 2000</td>
<td>Cross-sectional study with high risk or bias. Based on survey but pain distinction not specified</td>
</tr>
<tr>
<td>Evans 2000</td>
<td>Can’t be used for prognosis — it’s a cross-sectional survey. Pain &gt; 12 weeks — representative of primary care. Not relevant</td>
</tr>
<tr>
<td>Harreby 1995</td>
<td>Study examines risk factors for low back pain. This study is about the progression of risk factors for LBP. Based on children not adults</td>
</tr>
<tr>
<td>Hudak 1996</td>
<td>Review of criteria for assessing studies of prognosis of soft tissue musculoskeletal disorders in general — not specific to thoracic spine</td>
</tr>
<tr>
<td>Musgrave 2001</td>
<td>Study of risk factors. Doesn’t distinguish thoracic pain from other back pain</td>
</tr>
<tr>
<td>Norlander 1997</td>
<td>Neck and shoulder pain not thoracic spinal pain. Not relevant in primary care</td>
</tr>
<tr>
<td>Norlander 1996</td>
<td>Neck and shoulder pain not thoracic spinal pain. Not relevant in primary care</td>
</tr>
<tr>
<td>Vanichkachorn 2000</td>
<td>This is a narrative review article by two authors. May provide a useful perspective</td>
</tr>
<tr>
<td>Vedderkropp 2001</td>
<td>Not an adult population. Study of back pain in children and adolescents only</td>
</tr>
</tbody>
</table>

Acute Thoracic Pain
### ACUTE THORACIC PAIN — Table of Included Studies (Interventions)

<table>
<thead>
<tr>
<th>STUDY</th>
<th>METHODS</th>
<th>PARTICIPANTS</th>
<th>INTERVENTIONS</th>
<th>RESULTS</th>
<th>QUALITY SUMMARY</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schiller 2001</td>
<td>RCT</td>
<td>N = 30; 15 to control group and 15 to intervention</td>
<td>Spinal manipulation versus non-functional ultrasound</td>
<td>Outcomes measured on Oswestry, NRS, McGill Pain Questionnaire; SM has greater benefits than ultrasound: p &lt; .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</td>
<td>Adequate allocation concealment = U Blinding =</td>
<td>Small sample size. Type II error. Pre-intervention pain duration not described. Groups equivalent at baseline. Follow-up for 4/52</td>
</tr>
</tbody>
</table>

### ACUTE THORACIC PAIN — Table of Excluded Studies (Interventions)

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<tr>
<th>STUDY</th>
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<tbody>
<tr>
<td>Giles 1999</td>
<td>Chronic pain. Part of the data relates to thoracic spine (pain duration &gt;12 weeks) and compares three therapies</td>
</tr>
<tr>
<td>Haldeman S 1993</td>
<td>Does not meet criteria. Not T spine, not RCT, not pain duration &lt;12 weeks</td>
</tr>
</tbody>
</table>
## ACUTE NECK PAIN — Table of Included Studies (Diagnosis)

<table>
<thead>
<tr>
<th>STUDY</th>
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</thead>
<tbody>
<tr>
<td>Gordon 2002</td>
<td>Randomised population based study to determine frequency, duration and prevalence of waking C-spine pain and stiffness by gender and age</td>
<td>Incidence of 38% cervical spine pain on awakening — more frequent among females and decline in pain with age; males = peak prevalence in 40–59 year group</td>
<td>Selection bias minimised = Y Measurement bias minimised = Y Verification bias minimised = Y Compared to reference standard = N/A Confounding avoided = Y Bias rating = Moderate</td>
<td>Authors noted that there is little validated advice re the mgt and prevention of people waking with such symptoms *Chronicity is not defined Telephone survey</td>
<td></td>
</tr>
<tr>
<td>Johnson 1997</td>
<td>Value of C-spine radiographs in pts presenting with vague, non-localising and non-specific neck pain</td>
<td>Not a cost effective method of diagnosing cause of cervical pain</td>
<td>Selection bias minimised = U Measurement bias minimised = U Verification bias minimised = U Compared to reference standard = U Confounding avoided = U Bias rating = Moderate</td>
<td>Include for information on the fact that millions of dollars are spent unnecessarily on c x-rays *does not mention duration of pain</td>
<td></td>
</tr>
<tr>
<td>Mihmanli 2001</td>
<td>Case report of inflammation of vertebral bone associated with acute calcific tendonitis of the longus colli muscle</td>
<td>N/A</td>
<td>N/A</td>
<td>Case report not research study but useful for differential diagnosis</td>
<td></td>
</tr>
<tr>
<td>Muffoletto 2001</td>
<td>Case studies: hematogenous pyogenic facet joint infection of the subaxial cervical spine</td>
<td>Two cases reported</td>
<td>N/A</td>
<td>Case report not research study but useful for differential diagnosis; very rare condition. Use for additional information only</td>
<td></td>
</tr>
<tr>
<td>Nolla 2002</td>
<td>Case series of pyogenic vertebral osteomyelitis (spontaneous) neck or neck pain reported in all cases with mean duration of 48 days +40 days pain was mechanical in 25% of patients</td>
<td>64 patients</td>
<td>N/A</td>
<td>Include for useful descriptive data on incidence of QM in neck</td>
<td></td>
</tr>
<tr>
<td>Ring 1984</td>
<td>Case series (&lt; 5) of acute calcific retropharyngeal tendonitis</td>
<td></td>
<td>Selection bias minimised = U Measurement bias minimised = Y Verification bias minimised = Y Compared to reference standard = Y Confounding avoided = N/A Bias rating = Low</td>
<td>Include as it provides descriptive data and reassuring data on an unusual differential diagnosis</td>
<td></td>
</tr>
<tr>
<td>Sandmark 1995</td>
<td>To assess sensitivity and specificity of 5 manual tests for pain provocation in the neck for use in epidemiological studies</td>
<td>Sample of 75 men randomly selected — asymptomatic and symptomatic for neck pain</td>
<td>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</td>
<td>Selection bias minimised = Y Measurement bias minimised = Y Verification bias minimised = Y Compared to reference standard = Y Self report of neck pain was the criterion standard Confounding avoided = N/A Bias rating = Low</td>
<td>Could be used in the revised section on physical examination</td>
</tr>
<tr>
<td>Wang 1999</td>
<td>Flexion-extension x-rays in acutely injured may not be useful due to the decreased CROM and there are reports of serious neurological injuries occurring with the use of such x-rays</td>
<td></td>
<td>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</td>
<td>Algorithm on page 115–6 may be useful; suitable to base guidelines on why not to take flexion-extension x-rays</td>
<td></td>
</tr>
<tr>
<td>STUDY</td>
<td>REASON</td>
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<td></td>
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<tr>
<td>Gordin 2001</td>
<td>Descriptive review of procedures for chronic neck pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bono 2000</td>
<td>Opinion piece, No raw data, Mostly chronic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Demure 2000</td>
<td>Does not present the results for neck separately from ‘neck/shoulder’</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hudak 1998</td>
<td>Opinion piece, Not a study or a review of data. **Could be used, if wanted, for its scoring system for articles on prognosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jull 1998</td>
<td>Chronic neck pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jull 1997</td>
<td>Current pain — but chronicity history (chronic) not known</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kligman 2000</td>
<td>Case report, No evidence of causative link to neck pain. Could be incidental finding</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stolk-Horreveld 1999</td>
<td>Abstract — headache — insufficient data provided</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sutner 2002</td>
<td>Chronic, Single case-report not a study</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Walsh 2000</td>
<td>Descriptive study only. Chronicity not defined</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wemecke 1999</td>
<td>Not relevant in primary care</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### ACUTE NECK PAIN — Table of Included Studies (Prognosis)

<table>
<thead>
<tr>
<th>Study</th>
<th>Method</th>
<th>Participants</th>
<th>Results</th>
<th>Quality Summary</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ariens 2001</td>
<td>3-year prospective cohort study to see whether work related job demands are risk factors for neck pain</td>
<td>Males and females from 34 companies in the NL, 1384 workers met the criteria but 26.8% loss to follow-up</td>
<td>Neck pain recorded at 1 and 3 years; work factors assessed at baseline. Low co-working support was found to be predictive of neck pain RR = 2.1 (1.3–3.3) and also high job demands RR = 2.1 (1.3–3.3)</td>
<td>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</td>
<td>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.</td>
</tr>
<tr>
<td>Borchgrevink 1997</td>
<td>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 MRI. Patients completed the CMC-I at intake and then 6 months later. Assessed neck pain, neck stiffness and headache, taking pre-symptoms into account</td>
<td>88 (43M/45F) pts &gt;40A recruited from ER of MRI done in 4 days of injury *nearly all covered by insurance *Norway, university hospital</td>
<td>Follow-up of 1 year in total. No clinical consultations during first 6 months and second 12 months following MRI. Results do not support the view that pre-morbid personality traits predict outcome for neck sprain pts</td>
<td>Selection bias minimised = Y, Adjusted for confounders = Y, Measurement bias minimised = Y, Drop out rate = 11%</td>
<td>Selection bias minimised = Y, Adjusted for confounders = Y, Measurement bias minimised = Y, Drop out rate = 6 months, 5%; 12 months, 30%; 24 months, 64%</td>
</tr>
<tr>
<td>Borghouts 1998</td>
<td>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</td>
<td>n = 43 consecutive patients; 38 participated in study *38/39 had been in rear end collisions *neck pain present in all patients</td>
<td>A higher severity of pain and a previous history of neck pain seems 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 any 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 &gt;6 months</td>
<td>Number of RCTs = 23, Quality assessment = Y, Heterogeneity = Y</td>
<td></td>
</tr>
<tr>
<td>Brison 2000</td>
<td>Prospective study of factors associated with development of WAD &gt; rear end collisions. Aim to describe the natural course of symptoms among patients from acute stages of injury — 2 years post injury</td>
<td>n = 352 mostly female of people presenting to ER post rear end collision *minimal data *noise due to low rate of litigation</td>
<td>62% experienced WAD D post-collision with decline to 37% at 3/12</td>
<td>Selection bias minimised = Y, Adjusted for confounders = N, Measurement bias minimised = Y, Drop out rate = 6 months, 5%; 12 months, 30%; 24 months, 64%</td>
<td>Bias rating = Low</td>
</tr>
<tr>
<td>Fredriksen 2002</td>
<td>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</td>
<td>Age range 22–59 years *included if acute whiplash injury *excluded if acute whiplash injury within past 14 days *n = 43 consecutive patients *39 participated in study</td>
<td>Associations between several exposures in the work environment and seeking care for neck or shoulder pain were found *reduction period for neck or shoulder pain may be short</td>
<td>Selection bias minimised = Y, Adjusted for confounders = Y, Measurement bias minimised = Y, Drop out rate = N, Bias rating = Low</td>
<td>Worth considering if we are looking at etiology of neck pain even though pain was of greater duration than 3 months</td>
</tr>
<tr>
<td>Karlsborg 1997</td>
<td>Prospective study of 39 patients with acute whiplash injury &gt; 12 days, 1 month and 7 months post injury; MRI of brain and cervical spine done at 12 post-injury and 6 if abnormalities were found</td>
<td>Age range 22–59 years *included if acute whiplash injury within past 14 days *n = 43 consecutive patients; 39 participated in study</td>
<td>Total recovery rate (asymptomatic) was 29% at 12 months, 52% at 12 months *correlation between MRI and clinical findings was poor *outcome was related more to stressful life events than clinical findings *85% resumed work within 1/12</td>
<td>Selection bias minimised = Y, Adjusted for confounders = Y, Measurement bias minimised = Y, Drop out rate = U, Bias rating = Low</td>
<td>Selection bias minimised = Y, Adjusted for confounders = Y, Measurement bias minimised = Y, Drop out rate = 0, Bias rating = Low</td>
</tr>
<tr>
<td>Kasch 2001</td>
<td>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</td>
<td>n = 146 recruited over 1 year in Canada *involved in rear end collision and seen within 2–4 days of injury in ER *excluded those with chronic neck pain</td>
<td>Initial pain intensity did not predict long term handicap after injury; poor prognosis related to reduced CROM and High initial VAS score (sens. and spec. were 73% and 92%)</td>
<td>Selection bias minimised = Y, Adjusted for confounders = U, Measurement bias minimised = Y, Drop out rate = 6% at 12/12, Bias rating = Low</td>
<td>Discussions 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</td>
</tr>
</tbody>
</table>

Appendix E • Tables of Included and Excluded Studies
### ACUTE NECK PAIN — Table of Excluded Studies (Prognosis)

<table>
<thead>
<tr>
<th>STUDY</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Hudak 1998</td>
<td>A perspective; not primary research or a review</td>
</tr>
<tr>
<td>Palmer 2001</td>
<td>Other neurological/pain presentations</td>
</tr>
<tr>
<td>Schrader 1996</td>
<td>Interpretation may exceed the comparative data; methodological limitations</td>
</tr>
<tr>
<td>Tilnoff 2002</td>
<td>Descriptive study of incidence in nurses; Not study of acute, non-specific cervical pain — chronically some &gt;6 months</td>
</tr>
<tr>
<td>Virani 2001</td>
<td>Survey of opinion only, not study of non-specific acute pain — chronic sufferers &gt;6 months</td>
</tr>
</tbody>
</table>
## ACUTE NECK PAIN — Table of Included Studies (Interventions)

<table>
<thead>
<tr>
<th>STUDY</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Bonk 2000</td>
<td>Prospective RCT of activity vs collar and the natural history of whiplash in Germany</td>
<td>Recruited from hospital ER within 3 days of accident. Active therapy = 47, collar n = 50, control = approx 50</td>
<td>Group 1, active therapy (AT) 3 wks vs Group 2, collar (CT) 3 wks vs asymptomatic control group. All allowed to use amphetamine.</td>
<td>AT resulted in significantly faster recovery rate from acute whiplash injury at 3/52 compared to CT. At 4/52, there was no difference between AT and healthy controls and at 12/52, CT did not differ from healthy controls. AT, postural advice and exercises had better outcomes than passive modalities. COST: AT 1/48, took time off work; CT 2/52, took time off work.</td>
<td>Adequate allocation concealment = U Blinded = N ITT analysis = N</td>
<td>Randomisation method not discussed. Unblinded treatment and unblinded f/u at 12/52. Matched controls</td>
</tr>
<tr>
<td>Gross 2002a</td>
<td>Cochrane systematic review on patient education for mechanical neck disorders</td>
<td>RCTs involving patients n = 271 with mechanical neck pain. Inception cohort was from primary care in all 3 studies</td>
<td>Systematic review of patient education using individualized or group instructional strategies. Has not been shown to be beneficial in reducing pain for mechanical neck disorders.</td>
<td>Conclusion was that patient education using individualized or group instructional strategies has not been shown to be beneficial in reducing pain for mechanical neck disorders.</td>
<td>Systematic review = Y Number of RCTs = 3 Quality assessment = Y Data pooled = Y Heterogeneity = Y Applicability of results = Y</td>
<td>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</td>
</tr>
<tr>
<td>Gross 2002b</td>
<td>Cochrane review to assess CCT and RCT of physical medicine modalities for mechanical neck disorders</td>
<td>13 trials included. Excluded patients with neurological deficit</td>
<td>Electromagnetic therapy, laser therapy, exercise, traction, acupuncture, heat/cold, electrotherapies, cervical orthoses and cognitive behavioural rehab strategies.</td>
<td>Some support for EMT in reducing pain. Some data against the use of laser therapy: 3 articles using laser therapy did not differ from placebo p = 0.026 for 6-10 sessions of treatment. Little information available from trials to support the use of these modalities for mechanical neck pain.</td>
<td>Systematic review = Y Number of studies = 13 Quality assessment = Y Data pooled = Y Heterogeneity = Y Applicability of results = Y</td>
<td>Unable to find separate analysis of acute neck pain and concerned about 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</td>
</tr>
<tr>
<td>Gross 2002c</td>
<td>Systematic review of manual therapy for mechanical neck disorders</td>
<td>20 RCTs</td>
<td>Systematic review = Y Number of RCTs = 20 Quality assessment = Y Data pooled = Y Heterogeneity = Y Applicability of results = Y</td>
<td>Multimodal care consisting of mobilisation massage or manipulation and exercise alone or in combination with physical modalities and infrequent collar use appears more effective than application of these therapies alone, specific physical medicine modalities and continuous collar use with rest for pain reduction. Treatment advantages for pain ranged from 6% to 41%.</td>
<td>Search date: December 1999</td>
<td></td>
</tr>
<tr>
<td>Hoving 2002</td>
<td>RCT with blinded outcome assessments</td>
<td>Patients with non-specific neck pain referred by GPs, age 18-70 years, pain or stiffness in the neck x 2 weeks or more, no treatment for neck pain in previous 6 months. 74% had neck pain of &lt;3 months</td>
<td>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.</td>
<td>Data collected at 3 weeks and 7 weeks. At 7 weeks success rate was high for MT as for UC and higher for PT than for UC, but not statistically significant. MT 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.</td>
<td>Adequate allocation concealment = U Blinded = N ITT analysis = Y</td>
<td>Mixed sample (acute and chronic). No separate analysis of acute and chronic neck pain. Contentious principal outcome measure</td>
</tr>
<tr>
<td>Karjalainen 2002</td>
<td>Multi-disciplinary biopsychosocial rehab for neck and shoulder pain among working age adults; a Cochrane Review</td>
<td></td>
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</table>
### ACUTE NECK PAIN — Table of Included Studies (Interventions) continued

<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>McKinney 1989</td>
<td>Single blind prospective randomized trial to determine whether manipulative physiotherapy or patients ability to perform home exercise program influence reduction in pain and ROM</td>
<td>Receiving physio n = 66; receiving analgesia and a c-collar n = 33. Recruited from ER in Belfast</td>
<td>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</td>
<td>Patients receiving out patient physiotherapy and those receiving advice on home exercise Group 2 and 3 had similar pain outcomes to each other at 1 and 2 months and had significantly less neck pain (p &lt; 0.001) and greater cervical movement (p &lt; 0.001) at 1 and 2 months vs patients receiving rest (Group 1)</td>
<td>Adequate allocation concealment = Y Blinded = Y single ITT analysis = N LTFU &lt; 15% = N (&gt; 20%) Bias = Moderate</td>
<td></td>
</tr>
<tr>
<td>McLean 2001</td>
<td>RCT to determine the effect of ‘microbreaks’ on muscle activation behaviour, perceived discomfort and on worker productivity</td>
<td>n = 15 pain free at inception</td>
<td>Random assignment to three experimental groups (microbreaks at 20 minute intervals, 40 minute intervals, and at own discretion)</td>
<td>Regularly scheduled microbreaks reduced subjective discomfort (p &lt; 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</td>
<td>Adequate allocation concealment = Y Blinded = N ITT analysis = N LTFU &lt; 15% = Y Bias = Moderate</td>
<td></td>
</tr>
<tr>
<td>Musly 1996</td>
<td>RCT looking at early cervical passive mobilisation of acute whiplash injuries</td>
<td>n = 63 (2 patients lost to follow up); Recruited from ER, Dublin</td>
<td>Rest and initial cervical passive mobilisation with a soft collar vs early active cervical passive mobilisation</td>
<td>At 6 weeks a significant improvement in actively treated group in terms of CROM (p &lt; 0.05) and pain intensity (p &lt; 0.025) resemble each other</td>
<td>Adequate allocation concealment = Y Blinded = Y/NU Blinding of data collectors not specifically stated ITT analysis = Y LTFU &lt; 15% = N (10%) Bias = Moderate</td>
<td>Follow-up at 8/52</td>
</tr>
<tr>
<td>Peeters 2001</td>
<td>Systematic review (Cochrane-style) to assess efficacy of conservative treatment for whiplash (same as Verhagen 2002)</td>
<td>Systematic review (Cochrane-style) to assess efficacy of conservative treatment for whiplash (same as Verhagen 2002)</td>
<td>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)</td>
<td>Systematic review = Y Number of RCTs = 11 Quality assessment = Y Data pooled = N Heterogeneity = Y Applicability of results = Y</td>
<td>No discussion of duration of pain. Pre-1988 publications</td>
<td></td>
</tr>
<tr>
<td>Provinciali 1996</td>
<td>RCT — single blind prospective trial looking at multi-modal treatment to prevent late whiplash syndrome</td>
<td>60 patients enrolled following rear end collision within past 48 days or less. Subjects were diagnosed with ‘cervico-encephalics syndrome’. Subjects well-matched</td>
<td>Multi-modal treatment (MMT) consisting of postural training, manual technique, psychological support, and control treatment using physical agents such as electrical and sonic modalities (TENS and U/S)</td>
<td>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 3 and 6 months (p &lt; 0.001)</td>
<td>Adequate allocation concealment = U Blinded = Y single (data collectors) ITT analysis = Y LTFU &lt; 15% = Y Bias = Low</td>
<td>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</td>
</tr>
<tr>
<td>Verhagen 2002</td>
<td>Cochrane Review assessing efficacy of conservative treatment for whiplash</td>
<td></td>
<td>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)</td>
<td>Systematic review = Y Number of RCTs = 11 Quality assessment = Y Data pooled = N Heterogeneity = Y Applicability of results = Y</td>
<td></td>
<td>10 of the RCTs related to acute pain</td>
</tr>
<tr>
<td>STUDY</td>
<td>REASON</td>
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<tr>
<td>Brockow 2001</td>
<td>Majority had pain &gt;3 months — fails inclusion criteria. Chronic neck pain and back pain</td>
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<tr>
<td>Dabbs 1995</td>
<td>Narrative view, not experimental or systematic review. Not a scientific paper. A narrative of reviewed papers including back pain</td>
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<tr>
<td>Ernst 2002</td>
<td>Does not satisfy any inclusion criteria. SR-observational report citing case control studies. Prospective studies and a systematic review; a commentary only</td>
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<tr>
<td>Hoving 2001</td>
<td>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</td>
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<tr>
<td>Horneij 2001</td>
<td>Not acute neck pain; Outside scope of guidelines</td>
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<tr>
<td>Ketola 2002</td>
<td>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</td>
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<tr>
<td>Linton 2001</td>
<td>Not acute pain. Included back, neck or upper back pain. Also out of the scope of this project</td>
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<tr>
<td>Munchau 2001</td>
<td>Not neck pain — not an experimental study; not non-specific neck pain; inclusion criteria not fulfilled</td>
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<tr>
<td>Schneiker 2001</td>
<td>Chronic subjects in study</td>
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<tr>
<td>Skargren 1997</td>
<td>No separate analysis of acute (&lt;3 months) neck pain. Fails inclusion criteria. Neck and back pain not differentiated</td>
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<tr>
<td>Sterling 2001</td>
<td>Not acute pain. Neck pain &gt; 3 months</td>
<td></td>
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<tr>
<td>Sterner 2001</td>
<td>A retrospective cohort study. Fails inclusion criteria. Chronic symptoms &gt;3 months</td>
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<tr>
<td>Wheeler 1998</td>
<td>Chronic neck pain</td>
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<tr>
<td>Wood 2001</td>
<td>Not acute neck pain; No control group — test two versions of application of cervical manipulation — predominantly chronic patients</td>
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</table>
## ACUTE SHOULDER PAIN — Table of Included Studies (Diagnosis)

<table>
<thead>
<tr>
<th>STUDY</th>
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</thead>
<tbody>
<tr>
<td>Bennett 1998</td>
<td>Speeds test evaluated in a prospective study to determine sensitivity, specificity, PPV and NPV</td>
<td>n = 45; 31 male, 14 female *variety of diagnoses including acute and chronic</td>
<td>PPV = 29%; NPV = 88%; sensitivity = 60%; specificity = 133%</td>
<td>Speeds test versus arthroscopy</td>
<td>Selection bias minimised = Y</td>
<td>Duration of pain is unstated however this is not relevant in the case of diagnostic tests</td>
</tr>
<tr>
<td>Blanchard 1999a</td>
<td>RCT comparing MRI and arthrography for diagnostic impact in shoulder patients *f/u 10/12</td>
<td>Patients referred from a rheumatology clinic *29 shoulders randomised to MRI; 24 to arthrography</td>
<td>MRI and arthrography had similar therapeutic impact although MRI was associated with a significant shift towards surgical intervention</td>
<td>Selection bias minimised = U</td>
<td>Duration of pain is unstated however this is not relevant in the case of diagnostic tests</td>
<td></td>
</tr>
<tr>
<td>Fraenkel 2000</td>
<td>Improving the selective use of plain radiographs *descriptive study, not analytic *prospective f/u of patients for 3/12</td>
<td>Patients presenting to ER with shoulder pain, excluding 'red flag' conditions *206 had shoulder x-ray of which 88% were TU</td>
<td>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</td>
<td>Plain x-ray</td>
<td>Selection bias minimised = U</td>
<td>Useful for better utilisation of x-rays; pain duration unknown and half patients had no x-ray, however 88% TU and 66% TU had pain &lt; 1 day; some patients had specific conditions and unknown pain duration so not a pure sample; low risk of bias</td>
</tr>
<tr>
<td>Green 1998b</td>
<td>Standardised protocol for measurement of ROM shoulder using plurimeter-V inclinometer and assessment of inter/intrarater reliability</td>
<td>Mean duration of symptoms = 165 months (9–24 months). 3 of the 6 patients had history of shoulder trauma; 4 had previous shoulder surgery</td>
<td>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</td>
<td>Selection bias minimised = Y</td>
<td>Subjects not acute; useful reference re the reliability of physical examination</td>
<td></td>
</tr>
<tr>
<td>Llaker 2000</td>
<td>Retrospective chart review looking at how well bedside history and physical exam predict arthrography results in older patients suspected of having rotator cuff tears</td>
<td>n = 486; on retrospective patients with suspected RCTs</td>
<td>Weakness with external rotation; age &gt; 65 years; and night pain were predictors of rotator cuff tears</td>
<td>Selection bias minimised = Y</td>
<td>Mixed data; useful as a reference to the validity of medical history taking</td>
<td></td>
</tr>
<tr>
<td>MacDonald 2000</td>
<td>Looked at the diagnostic accuracy of Hawkins and Neer subacromial impingement signs</td>
<td>n = 446; on retrospective patients with suspected rotator cuff tears</td>
<td>Selection bias minimised = Y</td>
<td>Included as a reference to the validity of physical examination</td>
<td></td>
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</tbody>
</table>
### ACUTE SHOULDER PAIN — Table of Included Studies (Diagnosis) continued

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<tr>
<td>Naredo 2002</td>
<td>Compared clinical diagnosis established by physical examination with high frequency ultrasonographic findings in patients with shoulder pain. All underwent exam and US was carried out 1 week later by a third examiner with no knowledge of the clinical findings.</td>
<td>n = 38, with first flare of shoulder pain. All had clinical diagnosis of periarticular disorders. Excluded patients with chronic pain or previous trauma.</td>
<td>Clinical assessment showed low accuracy in diagnosis of periarticular shoulder lesions through physical examination. US should be used when possible to aid diagnosis and treatment.</td>
<td>Selection bias minimised = Y Measurement bias minimised = Y Veriﬁcation bias minimised = U Compared to reference standard = Y Confounding avoided = U Bias rating = Moderate</td>
<td>Reference to validity of physical examination</td>
<td></td>
</tr>
<tr>
<td>Norgaard 2002</td>
<td>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. US exam was performed followed by clinical exam performed randomly by orthopedic surgeon or rheumatologist who had no knowledge of pt history.</td>
<td>86 consecutive patients with long-standing shoulder pain of at least 2, 3 and 6 month duration (mean symptom duration = 25 months). They either had steroid injection without lasting effect or this was not indicated.</td>
<td>Accuracy of clinical tests and diagnoses in comparison with arthroscopic ﬁndings was low and only slightly better when the results of US became available in addition to clinical examination.</td>
<td>Selection bias minimised = Y Measurement bias minimised = Y Veriﬁcation bias minimised = Y Compared to reference standard = Y Confounding avoided = U Bias rating = Low</td>
<td>Although duration of pain is mixed study included as a reference</td>
<td></td>
</tr>
<tr>
<td>Pal 2000</td>
<td>Clinical audit of practice in emergency department (QA exercise to determine how people with shoulder pain were managed).</td>
<td>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.</td>
<td>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.</td>
<td>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</td>
<td>Reference to bursitis mechanism</td>
<td></td>
</tr>
<tr>
<td>Reins 1998</td>
<td>Case series of patients with subtle greater tuberosity fractures who were sent for MRI because of possible rotator cuff tear.</td>
<td>n = 17; Sydney Australia</td>
<td>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.</td>
<td>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</td>
<td>Reference to bursitis mechanism</td>
<td></td>
</tr>
<tr>
<td>Sommer 2001</td>
<td>Differential expression of cytokines and nitric oxide synthase isoforms in rotator cuff bursa. Subacromial bursal samples were collected at open surgery.</td>
<td>n = 28, consecutive patients examined following arthroscopy 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.</td>
<td>A mid-substance location and presence of fluid was more commonly associated with acute tears; non-visualised cuff and absence of any fluid more commonly associated with chronic tears.</td>
<td>Selection bias minimised = Y Measurement bias minimised = Y Veriﬁcation bias minimised = Y Retrospectively &amp; independently Compared to reference standard = Y Confounding avoided = Y Bias rating = Low</td>
<td>Reference to bursitis mechanism</td>
<td></td>
</tr>
<tr>
<td>Tickle 2000a</td>
<td>Study to identify differences in the sonographic appearance of acute and chronic full-thickness rotator cuff tears.</td>
<td>n = 28, consecutive patients examined following arthroscopy 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.</td>
<td>A mid-substance location and presence of fluid was more commonly associated with acute tears; non-visualised cuff and absence of any fluid more commonly associated with chronic tears.</td>
<td>Selection bias minimised = Y Measurement bias minimised = Y Veriﬁcation bias minimised = Y Retrospectively &amp; independently Compared to reference standard = Y Confounding avoided = Y Bias rating = Low</td>
<td>Reference to bursitis mechanism</td>
<td></td>
</tr>
<tr>
<td>Toftsen 1989</td>
<td>Looked at utility of MRI as a diagnostic tool in comparison with arthroscopy.</td>
<td>n = 31 with first flare of shoulder pain. All had clinical diagnosis of periarticular disorders. Excluded patients with chronic pain or previous trauma.</td>
<td>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.</td>
<td>Selection bias minimised = N Measurement bias minimised = N Veriﬁcation bias minimised = N Compared to reference standard = N Confounding avoided = N Bias rating = Moderate to High</td>
<td>Pain duration not mentioned — a range of conditions were involved; useful for reference only</td>
<td></td>
</tr>
</tbody>
</table>
## ACUTE SHOULDER PAIN — Table of Excluded Studies (Diagnosis)

<table>
<thead>
<tr>
<th>STUDY</th>
<th>REASON</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bencardino 2000</td>
<td>Chronic shoulder pain. Not relevant in primary care</td>
</tr>
<tr>
<td>Binkert 2001</td>
<td>Exclude — not relevant to these guidelines; doesn't meet selection criteria</td>
</tr>
<tr>
<td>Blanchard 1999a</td>
<td>No pain data/small numbers</td>
</tr>
<tr>
<td>Brackida 1999</td>
<td>Patient group not relevant</td>
</tr>
<tr>
<td>Bunker 2000</td>
<td>Maybe useful as a pathological reference only</td>
</tr>
<tr>
<td>Cameron 1999</td>
<td>High chronic pain — specific diagnosis</td>
</tr>
<tr>
<td>Clanie 1998</td>
<td>Chronic pain &gt;4/12</td>
</tr>
<tr>
<td>Coen 1999</td>
<td>Doesn't meet any inclusion criteria</td>
</tr>
<tr>
<td>Cuic 2003</td>
<td>Myocardial infarction. Article does not deal with acute shoulder pain</td>
</tr>
<tr>
<td>Fernand 2000</td>
<td>Chronic medication pain</td>
</tr>
<tr>
<td>Gertsman 1998</td>
<td>Not relevant; looks at patient perceptions of shoulder conditions on general health status</td>
</tr>
<tr>
<td>Johnson 2001</td>
<td>Needs review</td>
</tr>
<tr>
<td>Kim 2001</td>
<td>Apparent selection bias; therefore exclude</td>
</tr>
<tr>
<td>Law 1991</td>
<td>Case report of chronic pain (5 year duration)</td>
</tr>
<tr>
<td>Lee 2002</td>
<td>Chronic only</td>
</tr>
<tr>
<td>Murrell 2001</td>
<td>Does not meet criteria; data appears incomplete</td>
</tr>
<tr>
<td>Saetan 2000</td>
<td>Chronic — no shoulder specific data — review paper — no data</td>
</tr>
<tr>
<td>Scuttellani 1998</td>
<td>Review paper — not acute</td>
</tr>
<tr>
<td>Sitthi 2002</td>
<td>Chronic except one subject with 2/12 duration of symptoms. No differentiation between anterior and posterior impingment</td>
</tr>
<tr>
<td>Taufrey 2000a</td>
<td>Chronic</td>
</tr>
<tr>
<td>Vilkari-Juntura 2000</td>
<td>Irrelevant to these guidelines; mixed data on neck and shoulder</td>
</tr>
<tr>
<td>Wodland 2000</td>
<td>Doesn't meet any inclusion criteria</td>
</tr>
<tr>
<td>Yanagisawa 2001</td>
<td>Chronic pain. Study related to the mechanics of pain (subacromial)</td>
</tr>
<tr>
<td>STUDY/REFERENCE</td>
<td>METHOD</td>
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</tr>
<tr>
<td>MacFarlane 1998</td>
<td>Prospective cohort study over 3 years to determine the natural history of shoulder symptoms in the general population and whether long-term outcome can be predicted on the basis of clinical (pain related) and individual (host related) factors</td>
</tr>
<tr>
<td>Miranda 2008</td>
<td>Prospective study of work-related risk factors and physical exercise as predictors of shoulder pain among forestry workers; questionnaire administered to 3000 employees with 75% response rate</td>
</tr>
<tr>
<td>Nahit 2001</td>
<td>Questionnaire given to newly employed workers to determine presence of shoulder pain present in past month lasting for &gt;1 day</td>
</tr>
<tr>
<td>Punnett 2000</td>
<td>Cohort study conducted in auto-assembly plant to evaluate risk of shoulder disorders associated with non-neutral postures; questionnaire administered to 7000 employees with 75% response rate</td>
</tr>
<tr>
<td>Solomon 2000</td>
<td>Examined medical records to determine whether treating physicians documented key history and examination findings to determine whether documentation associated with patient satisfaction and clinical outcome</td>
</tr>
<tr>
<td>Solomon 2003</td>
<td>To examine the factors that influence referral of patients with musculoskeletal pain and whether referral influences outcomes. Follow-up 12/12</td>
</tr>
<tr>
<td>Tempelhof 1999</td>
<td>Looked at the prevalence of rotator cuff tears in asymptomatic shoulders using US</td>
</tr>
<tr>
<td>Tuite 1998</td>
<td>To determine the relative distribution of the locations of rotator cuff tears and sensitiviy of of anterior versus posterior tears on MR images</td>
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### ACUTE SHOULDER PAIN — Table of Included Studies (Prognosis) continued

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<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>van der Windt 1996</td>
<td>Shoulder disorders in general practice — what are the prognostic indicators of outcome <em>prospective f/u study (observational)</em> by 11 Dutch GPs</td>
<td>n = 349 pts with new episodes of shoulder pain with f/u at 1, 3, 6, 12 months</td>
<td>At 1/12, 23% showed complete recovery; at 3/12, 44% fully recovered, at 12/12, 59% fully recovered; 43% had persistent symptoms after 12/12</td>
<td>Selection bias minimised = Y Adjusted for confounders = Y Measurement bias minimised = Y Drop out rate = 4% Bias rating = Low</td>
<td>Included for information on long term course of shoulder pain; although has mixed data; useful as a reference only</td>
</tr>
<tr>
<td>Winters 1999a</td>
<td>Prospective study in general practice of the long term course of shoulder complaints <em>assessed diagnostic category and fluctuations in pain</em> all were given the same treatment with NSAID during first 2 weeks of inclusion *after this, the GP prescribed therapy tailored to the patients</td>
<td>n = 349 pts with new episodes of shoulder pain with f/u at 1, 3, 6, 12 months</td>
<td>The character of the symptoms changed considerably in the first week; 51% had recurrent complaints after 26/52 (assessed at visit) and 43% after 12–18 months (assessed via survey)</td>
<td>Selection bias minimised = Y Adjusted for confounders = U Measurement bias minimised = N Drop out rate = 7% Bias rating = Moderate</td>
<td>Included for information on long term course of shoulder pain; although has mixed data; useful as a reference only</td>
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## ACUTE SHOULDER PAIN — Table of Excluded Studies (Prognosis)

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<tr>
<th>STUDY</th>
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<tbody>
<tr>
<td>Albright 2001</td>
<td>Chronic pain</td>
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<tr>
<td>Brulin 2002</td>
<td>Follow-up study — issue of interest — pain — 5 years previously</td>
</tr>
<tr>
<td>Chiou 2002</td>
<td>As per all exclusion criteria. Not specifically acute pain in patients</td>
</tr>
<tr>
<td>Feldman 2002</td>
<td>Can’t separate local causes of shoulder pain from those referred</td>
</tr>
<tr>
<td>Fredriksson 1999</td>
<td>No baseline shoulder data. No specificity of data, diagnosis</td>
</tr>
<tr>
<td>Goh 1997</td>
<td>Retrospective. Duration of pain not specified</td>
</tr>
<tr>
<td>Griggs 2000</td>
<td>As per all exclusion criteria. Majority of patients chronic</td>
</tr>
<tr>
<td>Hirschhorn 2000</td>
<td>Not relevant patient group. Case report</td>
</tr>
<tr>
<td>Hudel 1996</td>
<td>No data. Review article</td>
</tr>
<tr>
<td>Johansson 1994</td>
<td>Diagnosis by questionnaire. Not specified for acute or chronic. 12-month period — no indication of site preparation</td>
</tr>
<tr>
<td>Kaergaard 2000</td>
<td>Data mixed — neck and shoulder</td>
</tr>
<tr>
<td>Leppola 1998</td>
<td>Chronic capsulitis &gt;312. Chronic disease</td>
</tr>
<tr>
<td>Lowe 2001</td>
<td>Eight subjects recruited, 4/8 had musculoskeletal pain in the past 12/12; results do not differentiate for shoulder pain</td>
</tr>
<tr>
<td>Rahme 1998</td>
<td>Chronic patients &gt;1-year duration</td>
</tr>
<tr>
<td>Smith 2001</td>
<td>Chronic pain — association between two chronic conditions</td>
</tr>
<tr>
<td>Winters 1999b</td>
<td>50% of sample acute — 28% chronic — 25%</td>
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</tbody>
</table>
### ACUTE SHOULDER PAIN — Table of Included Studies (Interventions)

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<tbody>
<tr>
<td>Adebajo 1990</td>
<td>Randomised, controlled trial</td>
<td>60 patients. Inclusion criteria: symptoms &lt;3 months and rotator cuff tendinitis defined by pain exacerbated by resisted movement, an abduction with a painful arc or external rotation; active range of movement frequently limited by pain and passive range always &gt;active range of movement; normal glenohumeral range of passive movement. Exclusion criteria: Systemic inflammatory arthritis, recent peptic ulceration or gastrointestinal bleeding; or sensitivity to NSAID or triamcinolone; shoulder injection within previous 3 months; glenohumeral arthritis, acromioclavicular arthritis, bicipital tendinitis or a suspected rotator cuff tear (weak arm elevation, positive ‘diplopia’ sign) or a high riding humerus seen radiologically; local infection. NSAIDs stopped at least 1 week before study entry.</td>
<td>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 3ml 0.5% lignocaine.</td>
<td>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)).</td>
<td>Adequate allocation concealment = Y Blinding = Y (participants and outcome assessors blinded) ITT analysis = Y LTFU &lt; 12% = Y (No loss to follow-up) Bias rating = Low</td>
</tr>
<tr>
<td>Ginsberg 1991</td>
<td>Randomised double blind crossover trial</td>
<td>n = 30 (2 with epicondylitis; 28 with peri-artitis of the shoulder)*</td>
<td>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.</td>
<td>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.</td>
<td>Adequate allocation concealment = U Blinding = Y ITT analysis = U LTFU &lt;12% = No mention of any patients lost to follow-up Bias rating = Low</td>
</tr>
<tr>
<td>Mena 1986</td>
<td>Randomised double blind placebo-controlled trial</td>
<td>n = 48. Inclusion criteria: Ages 14–70 years with acute bursitis or tendinitis of the shoulder and minimum duration of symptoms =4 days.</td>
<td>Group 1: Placebo NSAID plus physiotherapy. Group 2: Flurbiprofen 200–300mg QID plus physiotherapy.</td>
<td>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 3, 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.</td>
<td>Adequate allocation concealment = U Blinding = Y (double) ITT analysis = U (method of analysis not described) LTFU &lt;12% = Incomplete follow-up (patients who had not responded on day 3 were withdrawn from the study) Bias rating = Moderate</td>
</tr>
<tr>
<td>STUDY</td>
<td>METHOD</td>
<td>PARTICIPANTS</td>
<td>INTERVENTIONS</td>
<td>RESULTS</td>
<td>QUALITY SUMMARY</td>
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</tbody>
</table>
| Vidal 2001 | Double blind randomised controlled trial | n = 599 | 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 = 0.69) | 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 | Group 1: tenoxicam 
Group 2: piroxicam | 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 (21 patients) or some other form of soft-tissue rheumatic complaint (epicondylitis n=10, tendinitis n=5, olecranon bursitis n=3), 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 100mg/day | Significant improvement in pain, quality of sleep, range of active motion and patients 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 (1) 
Bias rating: Low 
Results for shoulder pain not presented separately |
| Wiellandts 1979 | Randomised double blind trial | n = 36 | Group 1: 100mg phenylbutazone QID for 7 days 
Group 2: 100mg fenetylic acid 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 = completers analysis only 
LTFU < 15% = N (5/26 LTFU at 1/2, all in phenylbutazone group) 
Bias rating: Moderate 
Wyeth supplied the drug |
<table>
<thead>
<tr>
<th>STUDY</th>
<th>METHOD</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Adebajo 1990</td>
<td>See NSAID section</td>
<td>n = 57 Inclusion criteria: clinically defined rotator cuff tendinitis (shoulder pain exacerbated by resistance in at least one of abduction, external or internal rotation, and normal passive motion) Duration of symptoms &lt; 3 months Exclusion criteria included adhesive capsulitis, rotator cuff tears, biceps tendinitis, acromioclavicular arthritis, previous steroid injections into shoulders</td>
<td>Group 1 (28 patients): subacromial injection of 1% lidocaine, 1ml. Group 2 (29 patients): subacromial injection of 40mg methylprednisolone plus 1ml 1% lidocaine NSAI Ds were discontinued 1 week prior to study.</td>
<td>Authors conclude no significant difference in pain and active and passive ROM between groups. No reported means or standard deviations.</td>
<td>Adequate allocation concealment = U Blinding = Y (participants and assessors) ITT analysis = completers analysis only LTFU &lt; 15% = Y Bias rating = Moderate One patient from each group failed to complete 12 week assessment period.</td>
</tr>
<tr>
<td>Vecchio 1993</td>
<td>Randomised controlled trial</td>
<td>n = 50 Inclusion criteria: ‘Rotator cuff tendinitis’ Painful arc between 40–120 degrees abduction, shoulder pain less than 3 weeks duration, no signs of calcific tendinitis, no evidence of a systemic inflammatory arthritis or frozen shoulder (defined as external rotation &lt; 30 degrees, abduction &lt; 30 degrees) Exclusion criteria: Active peptic ulcer disease, recent gastrointestinal bleed, contraindication to NSAIDs, evidence of symptomatic acromioclavicular arthritis or bicipital tendinitis or major rotator cuff tear</td>
<td>Group 1 (20 patients): Subacromial injection (unguided) of 40mg triamcinolone acetonide plus placebo indomethacin tablets 4 mg daily. Group 2 (20 patients): 25mg indomethacin 4 mg daily plus placebo (1cc saline) injection in the subacromial bursa. Repeat injection and refill of medication was given after 3 weeks, if necessary. All patients were instructed to begin home exercise program of Codyman pendulum exercises 10–15 min twice daily and slow shoulder abduction exercises using finger-up-the-wall technique.</td>
<td>At 6 weeks: no significant difference between groups in pain: mean difference between groups 1.2 (95% CI -0.36, 0.6) At 4 weeks: no significant difference between groups in range of abduction: mean difference between groups 0.18 (95% CI 0.03, 0.33)</td>
<td>Adequate allocation concealment = U Blinding = Y (participants and assessors) ITT analysis = Y LTFU &lt; 15% = N (25% — 5 patients in each group) Bias rating = Low Appropriate statistical analysis: Yes</td>
</tr>
<tr>
<td>Conroy 1998</td>
<td>Randomised controlled trial</td>
<td>n = 50 Inclusion criteria: primary shoulder impingement syndrome 3 times per week for 3 weeks</td>
<td>Group 1: Shoulder joint mobilisation and comprehensive treatment (hot packs, active exercises, stretching, strengthening, soft tissue mobilisation, education) Group 2: Comprehensive treatment alone</td>
<td>At 3 weeks: significant difference between groups in pain: WMD = -2.07 mm on VAS (95% CI -3.07, -1.07) — favours addition of mobilisation At 3 weeks: no significant difference between groups in range of elevation: WMD = 7.28 (95% CI -2.54, 11.18)</td>
<td>Adequate allocation concealment = No Blinding = Y (Assessors only) ITT analysis = No LTFU &lt; 15% = Yes Bias rating = Moderate Between group comparisons = Yes; Point estimates &amp; variability = Yes</td>
</tr>
<tr>
<td>White 1995</td>
<td>Randomised controlled trial</td>
<td>n = 50 Inclusion criteria: ‘Rotator cuff tendinitis’ Painful arc between 40–120 degrees abduction, shoulder pain less than 3 weeks duration, no signs of calcific tendinitis, no evidence of a systemic inflammatory arthritis or frozen shoulder (defined as external rotation &lt; 30 degrees, abduction &lt; 30 degrees) Exclusion criteria: Active peptic ulcer disease, recent gastrointestinal bleed, contraindication to NSAIDs, evidence of symptomatic acromioclavicular arthritis or bicipital tendinitis or major rotator cuff tear</td>
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<td>Adequate allocation concealment = U Blinding = Y (participants and assessors) ITT analysis = Y LTFU &lt; 15% = N (25% — 5 patients in each group) Bias rating = Low Appropriate statistical analysis: Yes</td>
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<tr>
<td>Ebenbichler</td>
<td>RCT design with random allocation</td>
<td>54 participants (61 shoulders)</td>
<td>Compared ultrasound therapy (15 mins, 88MHz, 2.5w/cm², pulsed 1:4, transducer size 5cm²) vs sham ultrasound with 24 treatment sessions (final 15 were daily then last 9 were 5 times weekly)</td>
<td>Assessed following treatment course and at 9 months</td>
<td>Adequate allocation concealment = Y</td>
</tr>
<tr>
<td>1999</td>
<td></td>
<td>Radiographically verified calcific tendinitis. Mild/mod pain for &gt;4 weeks or restricted ROM</td>
<td>Assessment of change from baseline in calcium deposits on radiography and pain (AROM, strength, ADLs).</td>
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<td>Adequate allocation concealment = Y</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Pain (pain score and VAS and reduction in pain (4-point scale)) 4.</td>
<td>QOL, 10cm VAS</td>
<td>Adequate allocation concealment = Y</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Follow-up: significant difference treatment (RR 1.80 (95%CI 1.26, 2.60) favours ultrasound)</td>
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<td>Significant difference radiological appearance calcific tendinitis in the short term (end of treatment) RR 1.43 (1.14, 1.77); and long term (9-month follow-up) RR 1.37 (1.02, 1.67).</td>
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<td>Adequate allocation concealment = Y</td>
</tr>
<tr>
<td>Shehab 2000</td>
<td>RCT design with random allocation</td>
<td>50 female participants with painful shoulder movement of at least 1 month's duration. Diagnosis confirmed with provocative testing</td>
<td>Group 1: Transcutaneous Nerve Stimulation (TNS), 30 mins 50Hz to anterior and posterior shoulder 3–5 times a week for 13 sessions</td>
<td>Pain post intervention: Median (Range) TENS 100 (60–150) US 80–160 (95%CI 1.26, 2.60)</td>
<td>Adequate allocation concealment = Y</td>
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<td>Group 2: Ultrasonic therapy (US), 15 mins, increased by 0.1W for each session; 3–5 times a week for 13 sessions</td>
<td>Pain post intervention: Median (Range) TENS 100 (60–150) US 80–160 (95%CI 1.26, 2.60)</td>
<td>Adequate allocation concealment = Y</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Both groups had ice and stretching</td>
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<td>Group 2: Ultrasonic therapy (US), 15 mins, increased by 0.1W for each session; 3–5 times a week for 13 sessions</td>
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<td>Adequate allocation concealment = Y</td>
</tr>
<tr>
<td>Kleinhenz 1999</td>
<td>RCT design with random allocation</td>
<td>52 Athletes with rotator cuff disease, excluding rotator cuff tear or ultrasound. Inclusion criteria: rotator cuff disease due to sport; 18–50 years old; shoulder pain for &gt;4 weeks; no acupuncture therapy in past 6 months.</td>
<td>Group 1: Eight acupuncture sessions in 4 weeks. Group 2: Identical regimen of placebo ultrasound</td>
<td>Assessment of change from baseline 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</td>
<td>Adequate allocation concealment = Y</td>
</tr>
<tr>
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<td>Group 1: Eight acupuncture sessions in 4 weeks. Group 2: Identical regimen of placebo ultrasound</td>
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<td>Adequate allocation concealment = Y</td>
</tr>
</tbody>
</table>

**Evidence-based Management of Acute Musculoskeletal Pain**
### ACUTE SHOULDER PAIN — Table of Excluded Studies (Interventions)

<table>
<thead>
<tr>
<th>STUDY</th>
<th>REASON</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aaras 2001</td>
<td>Not shoulder-specific — shoulder data not presented separately; not acute; not randomised</td>
</tr>
<tr>
<td>Arslan 2001</td>
<td>Mean duration of symptoms: greater than 3 months</td>
</tr>
<tr>
<td>Bain 2001</td>
<td>Trial of intra-operative pain relief. Pain &gt;6 months. Not relevant</td>
</tr>
<tr>
<td>Bang 2000</td>
<td>Mean duration of symptoms: &gt;5 months</td>
</tr>
<tr>
<td>Barber 2001</td>
<td>Does not fit inclusion criteria</td>
</tr>
<tr>
<td>Berry 1990</td>
<td>Duration of symptoms: greater than 3 months; used for mixed populations</td>
</tr>
<tr>
<td>Binder 1994b</td>
<td>Duration of symptoms: greater than 3 months; used for mixed data</td>
</tr>
<tr>
<td>Binder 1996</td>
<td>Chronic pain</td>
</tr>
<tr>
<td>Blockey 1994</td>
<td>Frozen shoulder, periartitis not defined; not acute (some acute patients but not reported separately)</td>
</tr>
<tr>
<td>Bottomi 2002</td>
<td>Fracture dislocation rather than acute (non-specific) shoulder pain</td>
</tr>
<tr>
<td>Brosseau 2002a</td>
<td>Exclude as relates to knee tendinitis only</td>
</tr>
<tr>
<td>Brox 1993/7</td>
<td>Duration of symptoms: greater than 3 months</td>
</tr>
<tr>
<td>Bulgen 1984</td>
<td>Duration of symptoms: greater than 3 months</td>
</tr>
<tr>
<td>Carter 2001</td>
<td>Case series</td>
</tr>
<tr>
<td>Caccavelli 2001</td>
<td>Chronic pain</td>
</tr>
<tr>
<td>Chou 2001</td>
<td>All chronic subjects</td>
</tr>
<tr>
<td>Dacre 1989</td>
<td>Duration of symptoms: at least 4 weeks and periartitis. Duration of symptoms: not reported</td>
</tr>
<tr>
<td>Daham 2000</td>
<td>Mean duration of pain is 1 year; inclusion criteria is at least 1 month of surgery</td>
</tr>
<tr>
<td>Del Conte 1990</td>
<td>Duration of symptoms: greater than 3 months</td>
</tr>
<tr>
<td>Downing 1996</td>
<td>Duration of symptoms: greater than 3 months</td>
</tr>
<tr>
<td>England 1989</td>
<td>Duration of symptoms: greater than 3 months</td>
</tr>
<tr>
<td>Fankhauser 2002</td>
<td>Not an RCT</td>
</tr>
<tr>
<td>Fers expenses</td>
<td>Results not separate for shoulder; subjects had pain duration &gt;6 months</td>
</tr>
<tr>
<td>Gann 1995</td>
<td>Exclude as includes many musculoskeletal conditions not specific to shoulder</td>
</tr>
<tr>
<td>Glen 1997</td>
<td>Duration of symptoms: greater than 3 months (used for mixed data)</td>
</tr>
<tr>
<td>Green 2002</td>
<td>Mixture of acute and chronic pain duration (used to obtain data on mixed populations)</td>
</tr>
<tr>
<td>Haiske 2002</td>
<td>Exclude due to chronic pain: &gt;6 months</td>
</tr>
<tr>
<td>STUDY</td>
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<tr>
<td>Horneij 2001</td>
<td>Heterogeneous group — prospective prevention trial</td>
</tr>
<tr>
<td>Invargarsson 1996</td>
<td>Surgery trial. Duration of symptoms unlikely to be less than 3 months</td>
</tr>
<tr>
<td>Johansson 2002</td>
<td>Not acute shoulder pain (based on references included). Not stated in paper but studies included in SR are on chronic patients</td>
</tr>
<tr>
<td>Jones 1999</td>
<td>Likely not to be acute but no duration given (2nd or 3rd stage capsulitis)</td>
</tr>
<tr>
<td>Karakes 2002</td>
<td>Duration of symptoms not defined except for &gt;4 weeks; likely to be &gt;12 weeks; duration of study is 60 minutes</td>
</tr>
<tr>
<td>Karjalainen 2002</td>
<td>Greater than 3 weeks; chronic</td>
</tr>
<tr>
<td>Ketola 2002</td>
<td>Neck/shoulder pain. Topic not relevant</td>
</tr>
<tr>
<td>Kivirikko 2001</td>
<td>Pain duration &gt;12 weeks; no control (placebo); poor inclusion definition; inadequate manipulation</td>
</tr>
<tr>
<td>Klein 2002</td>
<td>Polio survivors therefore not representative of the general population presenting to primary practice; maximum duration of symptoms unclear</td>
</tr>
<tr>
<td>Leclaire 1999</td>
<td>Duration of symptoms greater than 3 months</td>
</tr>
<tr>
<td>Lee 1993</td>
<td>Duration of symptoms greater than 3 months</td>
</tr>
<tr>
<td>Lеспри 2001</td>
<td>Case series</td>
</tr>
<tr>
<td>Lindh 1988</td>
<td>Surgery trial. Duration of symptoms unlikely to be less than 3 months</td>
</tr>
<tr>
<td>Moore 1976</td>
<td>Mean duration of symptoms not reported</td>
</tr>
<tr>
<td>Nicholson 1985</td>
<td>Duration of symptoms greater than 3 months</td>
</tr>
<tr>
<td>Nykanen 1995</td>
<td>Duration of symptoms reported to be greater than 2 months; mean duration not reported but unlikely to be less than 3 months</td>
</tr>
<tr>
<td>Oldervoll 2001</td>
<td>Not a randomised trial; not acute duration; not just shoulder pain</td>
</tr>
<tr>
<td>Peron 1997</td>
<td>Duration of symptoms greater than 3 months</td>
</tr>
<tr>
<td>Price 2002</td>
<td>Post stroke</td>
</tr>
<tr>
<td>Reid 1986</td>
<td>Unlikely to be acute</td>
</tr>
<tr>
<td>Ritchie 1995</td>
<td>Not a RCT</td>
</tr>
<tr>
<td>Risk 1991</td>
<td>Chronic pain (mean duration = 13.2 weeks)</td>
</tr>
<tr>
<td>Rompe 2001</td>
<td>Pain duration greater than 3 months (&gt;12 months)</td>
</tr>
<tr>
<td>Saunders 1995</td>
<td>Duration of symptoms greater than 3 months</td>
</tr>
<tr>
<td>Schmitt 2001</td>
<td>Chronic pain (&gt;6 months)</td>
</tr>
<tr>
<td>Shibata 2001</td>
<td>Chronic pain</td>
</tr>
<tr>
<td>Sileghem 1995</td>
<td>Chronic pain — definition of diagnosis unclear</td>
</tr>
</tbody>
</table>
### ACUTE SHOULDER PAIN — Table of Excluded Studies (Interventions) continued

<table>
<thead>
<tr>
<th>STUDY</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Snels 2000a</td>
<td>Hemiplegic shoulder pain therefore exclude</td>
</tr>
<tr>
<td>Snels 2000b</td>
<td>Survey</td>
</tr>
<tr>
<td>Spangehl 2002</td>
<td>Exclude chronic pain; mean duration &gt;12 months</td>
</tr>
<tr>
<td>Speed 2002</td>
<td>Not acute — useful for chronic</td>
</tr>
<tr>
<td>Spender 2001</td>
<td>For post-traumatic instability</td>
</tr>
<tr>
<td>Sun 2001</td>
<td>Duration of pain &gt;3/12 based on mean duration of symptoms</td>
</tr>
<tr>
<td>Taverna 1990</td>
<td>Duration of symptoms greater than 3 months</td>
</tr>
<tr>
<td>van der Heijden 1996</td>
<td>3/16 RCTs included are acute; included studies poor quality; unable to pool results. The three studies were reviewed separately</td>
</tr>
<tr>
<td>van der Heijden 1997</td>
<td>Physiotherapy for soft tissue disorders</td>
</tr>
<tr>
<td>van der Windt 1998</td>
<td>Half the participants had pain for greater than three months (used for mixed data)</td>
</tr>
<tr>
<td>van der Windt 1995</td>
<td>Efficacy of NSAIDs for shoulder pain</td>
</tr>
<tr>
<td>van der Windt 1999</td>
<td>Not shoulder-specific</td>
</tr>
<tr>
<td>Walsingham 2002</td>
<td>Not acute — includes cervical spine</td>
</tr>
<tr>
<td>Walsh 2001</td>
<td>Case report</td>
</tr>
<tr>
<td>Wang 2001</td>
<td>No comparison group (case series); All had pain longer than 6 months</td>
</tr>
<tr>
<td>Weldon 2001</td>
<td>Not a systematic review or a RCT</td>
</tr>
<tr>
<td>Winters 1997b</td>
<td>Heterogeneous, variable treatments; Different diagnostic groups</td>
</tr>
<tr>
<td>Winters 1999a</td>
<td>Not acute</td>
</tr>
<tr>
<td>Withjington 1985</td>
<td>RCT lignocaine and steroid vs saline; outcomes pain and paracetamol count; no significant differences; exclude due to chronic pain</td>
</tr>
<tr>
<td>Zuinem 1993</td>
<td>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 gastrointestinal adverse events with additional misoprostol</td>
</tr>
</tbody>
</table>

### ACUTE SHOULDER PAIN — Table of Excluded Studies (Cost Effectiveness)

<table>
<thead>
<tr>
<th>STUDY</th>
<th>REASON</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bongers 2001</td>
<td>Exclude because not a cost effectiveness study; a narrative review</td>
</tr>
</tbody>
</table>
### SUMMARY TABLES

#### ANTERIOR KNEE PAIN — Table of Included Studies (Diagnosis)

<table>
<thead>
<tr>
<th>STUDY</th>
<th>TESTS</th>
<th>PARTICIPANTS</th>
<th>RESULTS</th>
<th>QUALITY SUMMARY</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chapman-Jones 1998</td>
<td>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</td>
<td>n = 128 patients with non-specific knee pain</td>
<td>Demonstrated that patients with non-specific knee pain having plain x ray will have a high probability of a negative finding irrespective of any anatomic derangement present.</td>
<td>N/A</td>
<td>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.</td>
</tr>
</tbody>
</table>

#### ANTERIOR KNEE PAIN — Table of Excluded Studies (Diagnosis)

<table>
<thead>
<tr>
<th>STUDY</th>
<th>REASON</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abrahams 2001</td>
<td>Personal opinion only</td>
</tr>
<tr>
<td>Baker 2000</td>
<td>Opinion only</td>
</tr>
<tr>
<td>Cesarelli 2000</td>
<td>Requires EMG, pain duration unknown</td>
</tr>
<tr>
<td>Davies 2000</td>
<td>Orthopedic based sample; little detail regarding subjects; not relevant to primary care setting</td>
</tr>
<tr>
<td>Ferrar 1996</td>
<td>Not a systematic review. Clinical examination, diagnosis recommendation largely unreferenced. Useful MRI references</td>
</tr>
<tr>
<td>Fouts 1999</td>
<td>General overview — not a systematic review or primary research; &lt;7% of all knee injuries involve a clinically significant fracture</td>
</tr>
<tr>
<td>Fulkerson 2000</td>
<td>Not a systematic review. No recommendation of one diagnostic strategy over another</td>
</tr>
<tr>
<td>Fulkerson 2002</td>
<td>Orthopedic review — not relevant in primary care. Not specifically acute pain. Not systematic or cross sectional. Opinion only</td>
</tr>
<tr>
<td>Grelsamer 2000</td>
<td>Individual opinion</td>
</tr>
<tr>
<td>Hayes 2001</td>
<td>Same as Petersen &amp; Hayes 2000; chronic knee pain</td>
</tr>
<tr>
<td>Irrgang 2001</td>
<td>Validation of tool; no data on duration</td>
</tr>
<tr>
<td>Lee 2001</td>
<td>Mechanistic study on cadavers</td>
</tr>
<tr>
<td>Livingston 1999</td>
<td>Aetiologic; pain duration not reported</td>
</tr>
</tbody>
</table>
## Appendix E • Tables of Included and Excluded Studies

### Evidence-based Management of Acute Musculoskeletal Pain

#### Anterior Knee Pain — Table of Included Studies (Prognosis)

<table>
<thead>
<tr>
<th>Study</th>
<th>Method</th>
<th>Participants</th>
<th>Results</th>
<th>Quality Summary</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nimon 1998</td>
<td>Case series</td>
<td>Adolescent girls, mean age 16.5 yrs presenting to orthopaedic surgeons. Duration of acute knee pain unknown but likely to be longer term.</td>
<td>Average follow-up 16 years (14-20): 79% improved, 27% same or worse. Suggests that pain will persist for many years but eventually the majority improve without any significant long-term problems.</td>
<td>Not applicable (small series; no statement about length of symptoms at diagnosis)</td>
<td>Provides information on natural history</td>
</tr>
</tbody>
</table>

#### Anterior Knee Pain — Table of Excluded Studies (Prognosis)

<table>
<thead>
<tr>
<th>Study</th>
<th>Reason</th>
</tr>
</thead>
<tbody>
<tr>
<td>Callaghan 2000a</td>
<td>No long-term follow-up, no data on prognosis</td>
</tr>
<tr>
<td>Lam 2001</td>
<td>Mechanistic study; no follow-up therefore no prognostic data; chronic pain &gt;6/12</td>
</tr>
<tr>
<td>Petersen 2000</td>
<td>Chronic knee pain — duration, mean 38.9 months (SD &gt;52.1) median 7.7 months (range 0.4–234)</td>
</tr>
<tr>
<td>Sanchis-Alfonso 1998</td>
<td>Histological study of knee tissue; not relevant</td>
</tr>
<tr>
<td>Selfe 2001</td>
<td>Not relevant to these guidelines</td>
</tr>
<tr>
<td>Wolfe 2002</td>
<td>Not relevant in primary care — specific diagnosis of osteoarthritis. Symptom duration is &gt;2 weeks at first visit</td>
</tr>
<tr>
<td>STUDY</td>
<td>METHODS</td>
</tr>
<tr>
<td>-------</td>
<td>---------</td>
</tr>
<tr>
<td>Arol 1997</td>
<td>A critical review of clinical trials of non-operative therapy</td>
</tr>
<tr>
<td>Broseau 2002</td>
<td>Cochrane Review on therapeutic ultrasound for treating patellofemoral pain syndrome</td>
</tr>
<tr>
<td>Callaghan 2003b</td>
<td>Pilot study to compare two types of electrical stimulation of the quadriceps in the treatment of patellofemoral pain</td>
</tr>
<tr>
<td>Crossley 2003</td>
<td>Systematic review of physical interventions for patellofemoral pain</td>
</tr>
</tbody>
</table>
### ANTERIOR KNEE PAIN — Table of Included Studies (Interventions) continued

<table>
<thead>
<tr>
<th>STUDY</th>
<th>METHODS</th>
<th>PARTICIPANTS</th>
<th>INTERVENTIONS</th>
<th>RESULTS</th>
<th>QUALITY SUMMARY</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crossley 2002</td>
<td>Randomised controlled trial</td>
<td>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.</td>
<td>Group 1: n = 33 received physical therapy, quadriceps muscle retraining, patellar joint mobilisation, patellar taping and daily home exercises. Group 2: n = 34 received placebo (sham ultrasound, light application of nontherapeutic gel, placebo taping).</td>
<td>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.</td>
<td>Adequate allocation concealment = Y, ITT analysis = Y, LTFU = 15% = Y (6%). Bias = Low. Groups similar at baseline.</td>
<td>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.</td>
</tr>
<tr>
<td>D’hondt 2002</td>
<td>Cochrane Review on orthotic devices for treating patellofemoral pain syndrome</td>
<td>Trials involving n = 362.</td>
<td>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 Gomori’s bandage, the Protonics orthosis and the Palumbo dynamic patellar brace and the Cho-pat strap.</td>
<td>Inconclusive; no special recommendation for practice are made. Only 1 trial was considered high quality which compared McConnell to Gomori’s and there was a trend to favour McConnell regimen but the study only involved 10 subjects. Palumbo vs Cho-pat showed no difference. Protonics vs no trt showed all outcomes significantly favours 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.</td>
<td>Number of RCTs = 5, Quality assessment = Y, Data pooled = N, Heterogeneity = Y.</td>
<td>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.</td>
</tr>
<tr>
<td>Harrison 2001</td>
<td>Review article on patellofemoral pain syndrome — looks at aetiology, diagnosis and non-surgical management</td>
<td>12 articles reviewed.</td>
<td>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 form or other factors. Biofeedback, manual therapy, NSAIDs and sleeves have not been found to be more effective than simple exercises.</td>
<td>Adequate allocation concealment = Y, ITT analysis = Y, LTFU = 15% = Y (6%). Bias = Low. Groups similar at baseline.</td>
<td>Number of RCTs = 12, Quality assessment = N, Data pooled = N, Heterogeneity = Y.</td>
<td>Useful for epidemiological data.</td>
</tr>
</tbody>
</table>
### ANTerior Knee Pain — Table of Excluded Studies (Interventions)

<table>
<thead>
<tr>
<th>STUDY</th>
<th>REASON</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brosseau 2002</td>
<td>Not relevant to anterior knee pain. Discusses ilio-tibial band syndrome</td>
</tr>
<tr>
<td>Cohen 2001</td>
<td>Exclude due to a theoretical study on cadavers</td>
</tr>
<tr>
<td>Dursun 2001</td>
<td>Pain not &lt;12 weeks</td>
</tr>
<tr>
<td>Gotlin 2000</td>
<td>Not a systematic overview</td>
</tr>
<tr>
<td>Herrington 2000</td>
<td>Narrative review</td>
</tr>
<tr>
<td>Meiss 2001</td>
<td>For selected patellofemoral conditions; non-specific patellofemoral pain; chronic conditions; narrative review</td>
</tr>
<tr>
<td>Neptune 2000</td>
<td>Not relevant; mechanistic study not involving patients in pain</td>
</tr>
<tr>
<td>Powers 1998</td>
<td>Narrative review</td>
</tr>
<tr>
<td>Rillman 2000</td>
<td>Exclude due to surgical treatment for patellofemoral instability</td>
</tr>
<tr>
<td>Roush 2000</td>
<td>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 tendinopathy, plica syndrome etc.</td>
</tr>
<tr>
<td>Salisich 2002</td>
<td>Exclude due to not an RCT; small sample n =10</td>
</tr>
<tr>
<td>Selfe 2001</td>
<td>Not relevant to these guidelines; a validation of outcome measurement rather than clinical assessment</td>
</tr>
<tr>
<td>Suter 1999</td>
<td>Underlying assumptions are unproven; chronic pain</td>
</tr>
<tr>
<td>Suter 2000</td>
<td>Basic science (construct validity); chronic pain</td>
</tr>
<tr>
<td>Tang 2001</td>
<td>Basic science — not relevant</td>
</tr>
<tr>
<td>Tolkin 2000</td>
<td>Mechanistic study in asymptomatic study; not relevant for this guideline although a good study</td>
</tr>
<tr>
<td>Young 2001</td>
<td>Review of studies of primary prevention; not relevant</td>
</tr>
</tbody>
</table>